DIVISION
OF
SAFETY AND HYGIENE

DESK REFERENCE GUIDE:

CHEMICAL OR NOISE RELATED OCCUPATIONAL DISEASE CLAIMS

April 2004

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CANCER FACTS
National Cancer Institute * National Institutes of Health

Questions and Answers About Asbestos Exposure

1. What is asbestos?

"Asbestos" is the name given to a group of minerals that occur naturally as masses of strong, flexible fibers that can be separated into thin threads and woven. These fibers are not affected by heat or chemicals and do not conduct electricity. For these reasons, asbestos has been widely used in many industries. Four types of asbestos have been commonly used:

- Chrysotile, or white asbestos (curly, flexible white fibers), which accounts for about 90 percent of the asbestos currently used in industry;
- Amosite (straight, brittle fibers that are light gray to pale brown in color);
- Crocidolite, or blue asbestos (straight blue fibers); and
- Anthophyllite (brittle white fibers).

Chrysotile asbestos, with its curly fibers, is in the serpentine family of minerals. The other types of asbestos, which all have needle-like fibers, are known as amphiboles.

Asbestos fiber masses tend to break easily into a dust composed of tiny particles that can float in the air and stick to clothes. The fibers may be easily inhaled or swallowed and can cause serious health problems.

2. How is asbestos used?

Asbestos has been mined and used commercially in North America since the late 1800s, but its use increased greatly during World War II. Since then, it has been used in many industries. For example, the building and construction industry uses it for strengthening cement and plastics as well as for insulation, fireproofing, and sound absorption. The shipbuilding industry has used asbestos to insulate boilers, steampipes, hot water pipes, and nuclear reactors in ships. The automotive industry uses asbestos in vehicle brakeshoes and clutch pads. More than 5,000 products contain or have contained asbestos, some of which are listed below:

- Asbestos cement sheet and pipe products used for water supply and sewage piping, roofing and siding, casings for electrical wires, fire protection material, chemical tanks, electrical switchboards and components, and residential and industrial building materials;

- Friction products, such as clutch facings; brake linings for automobiles, railroad cars, and airplanes; and industrial friction materials;

- Products containing asbestos paper, such as table pads and heat-protective mats, heat and electrical wire insulation, industrial filters for beverages, small appliance components, and underlying material for sheet flooring;

- Asbestos textile products, such as packing components, roofing materials, heat- and fire-resistant clothing, and fireproof draperies; and

- Other products, including ceiling and floor tile; gaskets and packings; paints, rings, and sealants; caulking and patching tape; and plastics.

In the late 1970s, the U.S. Consumer Product Safety Commission banned the use of asbestos in wallboard patching compounds and gas fireplace because these products released excessive amounts of asbestos fibers into the environment. In addition, asbestos was voluntarily withdrawn by manufacturers of electric hair dryers. These and other regulatory actions, coupled with widespread public concern about the hazards of asbestos, have resulted in a significant annual decline in U.S. use of asbestos: Domestic use of asbestos amounted to about 560,000 metric tons in 1979, but it had dropped to about 55,000 metric tons by 1989.

3. What are the health hazards of exposure to asbestos?

Exposure to asbestos may increase the risk of several serious diseases:

- Asbestosis-a chronic lung ailment that can produce shortness of breath and permanent lung damage and increase the risk of dangerous lung infections;

- Lung cancer;

- Mesothelioma-a relatively rare cancer of the thin membranes that line the chest and abdomen; and

- Other cancers, such as those of the larynx and of the gastrointestinal tract.

4. Who is at risk?

Since the early 1940s, millions of American workers have been exposed to asbestos dust, including many of the 4.5 million men and women who worked in shipyards during the peak shipbuilding years of World War II. Health hazards from asbestos dust have been recognized in workers exposed in shipbuilding trades, asbestos mining and milling, manufacturing of asbestos textiles and other asbestos products, insulation work in the construction and building trades, brake repair, and a variety of other trades. Demolition workers, drywall removers, and firefighters also may be exposed to asbestos dust. As a result of Government regulations and improved work practices, today's workers (those without previous exposure) are likely to face smaller risks than did those exposed in the past.

Although it is known that the risk to workers increases with heavier exposure and longer exposure time, investigators have found asbestos-related diseases in some shipyard workers exposed to high levels of asbestos fibers for only brief periods (as little as 1 or 2 months). Even workers who may not have worked directly with asbestos but whose jobs were located near contaminated areas have developed asbestosis, mesothelioma, and other cancers associated with asbestos exposure.

Generally, workers who develop asbestos-related diseases show no signs of illness until many years after first exposure. For example, the time between first exposure to asbestos and the appearance of lung cancer is generally 15 years or more; a lag of 30 to 35 years is not unusual. The lag period for development of mesothelioma and asbestosis is even greater, often as long as 40 to 45 years.

There is also some evidence that family members of workers heavily exposed to asbestos face an increased risk of developing mesothelioma and perhaps other asbestos-related diseases. This risk is thought to result from exposure to asbestos dust brought into the home on the shoes, clothing, skin, and hair of workers.

5. How great is the risk?

Not all workers exposed to asbestos will develop diseases related to their exposure. In fact, many will experience no ill effects.

Asbestos that is bonded into finished products such as walls, tiles, and pipes poses no risk to health as long as it is not damaged or disturbed (for example, by sawing or drilling) in such a way as to release fibers into the air. When asbestos particles are set free and inhaled, however, exposed individuals are at risk of developing an asbestos-related disease. Once these nearly indestructible fibers work their way into body tissues, they tend to stay there indefinitely.

The risk of developing asbestos-related diseases varies with the type of industry in which the exposure occurred and with the extent of the exposure. In addition, different

types of asbestos fibers may be associated with different health risks. For example, results of several studies suggest that crocidolite and amosite are more likely than chrysotile to cause lung cancer, asbestosis, and, in particular, mesothelioma. Even so, no fiber type can be considered harmless, and proper safety precautions should always be taken by people working with asbestos.

6. How does smoking affect risk?

Many studies have shown that the combination of smoking and asbestos exposure is particularly hazardous. Cigarette smokers, on the average, are 10 times as likely to develop lung cancer as are nonsmokers. For nonsmokers who work with asbestos, the risk is about five times greater than for those in the general population. By contrast, smokers who also are heavily exposed to asbestos are as much as 90 times more likely to develop lung cancer than are non-exposed individuals who do not smoke. Smoking does not appear to increase the risk of mesothelioma, however.

There is evidence that quitting smoking will reduce the risk of lung cancer among asbestos-exposed workers, perhaps by as much as half or more after at least 5 years without smoking. People who were exposed to asbestos on the job at any time during their life or who suspect they may have been exposed *should not smoke.* If they smoke, they should stop.

7. Who needs to be examined?

Individuals who have been exposed (or suspect they have been exposed) to asbestos dust on the job or at home via a family contact should inform their physician of their exposure history and any symptoms. A thorough physical examination, including a chest x-ray and lung function tests, may be recommended. Interpretation of the chest x-ray may require the help of a specialist who is experienced in reading X-rays for asbestos-related diseases. Other tests also may be necessary.

As noted earlier, the symptoms of asbestos-related diseases may not become apparent for many decades after exposure. If any of the following symptoms develop, a physical examination should be scheduled without delay:

- Shortness of breath;
- A cough or a change in cough pattern;
- Blood in the sputum (fluid) coughed up from the lungs;
- Pain in the chest or abdomen;

• Difficulty in swallowing or prolonged hoarseness; and/or

• Significant weight loss.

8. What are the treatments for asbestos-related diseases?

The key to successful treatment of asbestos-related diseases lies in early detection. The health problems caused by asbestosis are due mainly to lung infections, like pneumonia, that attack weakened lungs. Early medical attention and prompt, aggressive treatment offer the best chance of success in controlling such infections. Depending on the situation, doctors may give a vaccine against influenza or pneumococcal pneumonia as a protective measure.

Treatment of cancer is tailored to the individual patient and may include surgery, anticancer drugs, radiation, or combinations of these therapies. Information about cancer treatment is available from the National Cancer Institute-supported Cancer Information Service, whose toll-free telephone number is 1-800-4-CANCER.

9. How can workers protect themselves?

Employers are required to follow regulations dealing with asbestos exposure on the job that have been issued by the Occupational Safety and Health Administration (OSHA), the Federal agency responsible for health and safety regulations in the workplace. Regulations related to mine safety are enforced by the Mine Safety and Health Administration (MSHA). Workers should use all protective equipment provided by their employers and follow recommended work practices and safety procedures. Workers who are or who have been exposed to asbestos should not smoke cigarettes.

Workers who are concerned about asbestos exposure in the workplace should discuss the situation with other employees, their union, and their employers. If necessary, OSHA can provide more information or make an inspection. Area offices of OSHA are listed in the "United States Government' section of telephone directories' blue pages (under “Department of Labor”), or can be found on web at www.osha.gov. Mine workers may contact MSHA's Office of Standards, Variances, and Regulation at Room 627, 4015 Wilson Boulevard, Arlington, VA 22203.

The National Institute for Occupational Safety and Health (NIOSH) is another Federal agency that is concerned with asbestos exposure in the workplace. The Institute conducts asbestos-related research, evaluates work sites for possible health hazards, and makes safety recommendations. In addition, NIOSH distributes publications on the health effects of asbestos exposure and can suggest additional sources of information. The address is Office of Information, National Institute of Occupational Safety and Health, 4676 Columbia Parkway/ Mailstop C-19, Cincinnati, OH 45226. The toll-free telephone number is 1-800-35-NIOSH (1-800-356-4674).

10. What should people who have been exposed to asbestos do?

It is important for exposed individuals to:

- Stop smoking;
- Get regular health checkups;
- Get prompt medical attention for any respiratory illness; and
- Use all protective equipment, work practices, and safety procedures designed for working around asbestos.

11. Will the Government provide examinations and treatment or pay for such services? What about insurance coverage?

Medical services related to asbestos exposure are available through the Government only for certain groups of eligible individuals. In general, exposed individuals must pay for their own medical services unless they are covered by private or Government health insurance. Medicare may reimburse people with symptoms of asbestos-related diseases for the costs of diagnosis and treatment (following review of medical procedures for appropriateness). General and specific information about benefits is available from the Medicare office serving each state; for the telephone number of the nearest office, call 1-800-772-1213.

People with asbestos-related diseases also may qualify for financial help, including medical payments, under state workers' compensation laws. Because eligibility requirements vary from state to state, workers should contact the workers' compensation program in the state where the last exposure occurred.

If exposure occurred during employment with a Federal agency (military or civilian), medical expenses and other compensation may be covered by the Federal Employees' Compensation Act. Workers who are or were employed in a shipyard by a private employer may be covered under the Longshoremen and Harbor Workers' Compensation Act. Information about eligibility or how to file a claim is available from the U.S. Department of Labor, Office of Workers' Compensation Programs, Room S-3229, 200 Constitution Avenue NW, Washington, DC 20210.

Retired military personnel and their eligible dependents may receive health care at any Department of Defense medical facility, Department of Veterans Affairs (VA) hospital, or Public Health Service hospital. Where no Federal facility is available, civilian facilities may be used under the Civilian Health and Medical Program for the Uniformed Services. Those over age 65 may be

covered by Medicare. Former members of the military who believe they may have a service-related medical problem may inquire about care at a VA facility or telephone the local VA office.

Workers also may wish to contact their international union for information on other sources of medical help and insurance matters. One organization, the Asbestos Victims Special Fund Trust, provides financial assistance to asbestos victims who have not received workers' compensation or compensation through legal avenues. Information is available from the Trust at Suite M- 11, 1500 Walnut Street, Philadelphia, PA 19102.

12. Is there a danger of nonoccupational exposure from products contaminated with asbestos particles?

Asbestos is so widely used that the entire population has been exposed to some degree. Air, beverages, drinking water, food, drug and dental preparations, and a variety of consumer products all may contain small amounts of asbestos. In addition, asbestos fibers are released into the environment from natural deposits in the earth and as a result of wear and deterioration of asbestos products.

The U.S. Environmental Protection Agency (EPA) regulates the general public's exposure to asbestos in buildings, drinking water, and the environment. The EPA's Toxic Substances Control Act (TSCA) Assistance Office can answer questions about toxic substances, including asbestos. Printed material is available on a number of topics, particularly on controlling asbestos exposure in schools and other buildings. The TSCA office can provide information about accredited laboratories for asbestos testing and can refer inquirers to other resources on asbestos. Questions may be directed to the TSCA Assistance Office, U.S. Environmental Protection Agency, 7408 M Street SW, Washington, DC 20024; the telephone number is 202-554-1404.

The Consumer Product Safety Commission (CPSC) is responsible for the regulation of asbestos in consumer products. The CPSC maintains a toll-free information line on the potential hazards of commercial products; the telephone number is 1-800-638-2772. In addition, CPSC provides information about laboratories for asbestos testing, guidelines for repairing and removing asbestos, and general information about asbestos in the home. Publications are available from the Office of Public Affairs, Consumer Product Safety Commission, 4330 East-West Highway, Bethesda, MD 20816.

The U.S. Food and Drug Administration is concerned with asbestos contamination of foods, drugs, and cosmetics and will answer questions on these topics. The address is Office of Consumer Affairs, Food and Drug Administration, HFE-88, 5600 Fishers Lane, Rockville, MD 20857.

13. What other organizations offer information related to asbestos exposure?

The American Lung Association and the American Cancer Society can provide information about lung disease, cancer, and smoking. Local chapters of these organizations are listed in telephone directories. Material about cancer and how to quit smoking is available by calling the National Cancer Institute - supported Cancer Information Service (CIS). The CIS, a program of the National Cancer Institute, provides a nationwide telephone service for cancer patients and their families, the pubic, and health care professionals. CIS information specialists have extensive training in providing up-to-date and understandable information about cancer and cancer research. They can answer questions in English and Spanish and can send free printed material. In addition, CIS offices serve specific geographic area and have information about cancer-related services and resources in their region. The toll-free number of the CIS is 1-800-4-CANCER (1-800-422-6237).

ASBESTOS-RELATED DISEASE CLAIMS

EMPLOYMENT RECORDS

Check to see if injured worker worked for numerous employers out of a union hall. Some unions will have a little information, but it is rarely site-specific. For example, Business Agent Gerald Hall at the Asbestos Workers Union, Local # 45, in Toledo, OH has reported they have records of who the employer was, but not locations where they worked. He said 75%[1] of their work was at powerhouses or refineries since that was where most asbestos was found.

When available, Social Security records should give dates of employment for the injured worker's employers, but these records will not show where the injured worker was assigned to work. Contractors and unions rarely keep this information. Many injured workers provide lists of coworkers who they remember working with at specific sites where asbestos was being applied or removed. This is often done in conjunction with civil suits against the asbestos manufacturers. If a lawsuit is in progress, the injured worker's attorney might be able to provide you with additional information on work sites.

The presence of asbestos on the equipment, however, **DOES NOT** necessarily mean workers were exposed to asbestos while working around the equipment. Asbestos exposure is not synonymous with asbestos disease.

Was the injured worker employed in construction prior to the regulation of asbestos? Most employers and employees considered asbestos an inert material and were not concerned about the generation of dust prior to the mid-1970s. Unfortunately, we cannot determine where or when the first or last, injurious exposure to asbestos may have occurred. Conditions began to improve slowly after regulations to limit dust exposure went into effect in the mid-1970s, although compliance was not immediate. Workers with 20-40 years of experience were likely to have been exposed more intensely during the earlier years of their employment.

AIR SAMPLING INFORMATION

You may need to contact an industrial hygienist in one of the Safety and Hygiene offices for interpretation of any industrial hygiene data included in the file.

The following gives some insight into how heavy dust exposures were in the past: Nicholson, Perkel and Selikoff summarized average fiber concentrations per cubic centimeter (f/cc) in insulation work prior to 1970.[1,2] The average fiber concentrations reported were 2.7 f/cc to 6.3 f/cc for light and heavy construction and 2.9 f/cc to 6.6 f/cc for marine work. The authors noted during certain operations (cement mixing, hand-saw or band saw cutting, removal) extremely high concentrations were observed (100 f/cc).

In 1986 OSHA cited studies by Berry and Finkelstein as justification for lowering the permissible exposure limit for asbestos from 2 f/cc to 0. 1 f/cc. These studies showed workers exposed to concentrations of asbestos close to or below former exposure limits (100 f/cc-years) were at substantial excess risk of asbestosis. (100 f/cc-years is the cumulative lifetime exposure (50 years) permitted by OSHA's former 2 f/cc exposure limit).

1. Nicholson, W.J.; Perkel, G.; Selikoff, L, "Occupational Exposure to Asbestos: Populations at Risk and Projected Mortality - 1980 to 2030," American Journal of Industrial Medicine 3: 1982, pp. 259-311.

2. "Revised Recommended Asbestos Standard, NIOSH. US Department of Health, Education, and Welfare, (DHEW/NIOSH Publication No. 77-169),1977.

3. EPA 560-5-85, -Guidance for Controlling Asbestos-Containing Materials in Buildings," June. 1985. 4. "29 CFR Parts 1910 and 1926 Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite Final Rules," Federal Register, June 20, 1986, pp. 22612 - 22774.

WORKERS AT RISK

The following list is by no means complete. As noted in the introduction to this manual these are some of the **MORE COMMON OCCUPATIONS** for which asbestos-related OD claims were recently investigated. The absence of a particular occupation in the following discussion **DOES NOT MEAN** asbestos exposure could not have occurred.

The NIOSH publications, Occupational Respiratory Diseases (DHHS/NIOSH Publication No. 86-102) and Occupational Diseases, A Guide to Their Recognition (DHEW/NIOSH Publication No. 77-181) are good references to check for additional information on asbestos exposure potential.

Asbestos workers and insulators

There's no doubt workers in these trades were exposed to asbestos. **Their job duties often included cutting, sawing, handling, and applying insulation material. These workers would have some of the highest risk of developing asbestos-related diseases.**
The frequency, intensity and duration of exposure are not known, but, in general, asbestos workers were exposed frequently and intensely to asbestos. In cases which involve union hall employees, we cannot determine where the injured worker was working when the first or last exposure occurred, nor can we determine which exposure initiated the disease process.

Until the early 1970s when OSHA (Occupational Safety and Health Administration) and EPA (Environmental Protection Agency) regulations went into effect, most industrial insulation contained asbestos and was generally handled without any measures to prevent exposure to high concentrations of dust. Conditions began to improve slowly after regulations to limit dust exposure went into effect in the mid- 1970s, although compliance was not immediate.

Carpenters

Carpenters may have been exposed to asbestos at sites while fireproofing materials were sprayed on structural steel or other asbestos products were used.[1] While cutting asbestos materials without local exhaust ventilation, wetting techniques, or respiratory protection would create high exposures, carpenters would not typically do this work on a daily basis. In general, carpenters were not exposed as frequently and as intensely to asbestos as were other trades, such as pipefitters, plumbers, and insulators. Asbestosis is more frequently reported among these other workers than among carpenters.

1 Bodeur, Paul, The Asbestos Hazard 1980.

Construction workers, general

It has been estimated that **70 percent of workers who have been exposed to asbestos are or were employed in the building construction trades.** These include pipefitters, welders, electricians, plumbers, carpenters, and others who may have been at sites while fireproofing materials were sprayed on structural steel or other asbestos products were used.[1]

1 Bodeur, Paul, The Asbestos Hazard 1980.

Electricians

Electricians frequently work in confined spaces with old asbestos insulation and, in the past, stripped old asbestos from pipes, beams, and wires in order to install or repair equipment. A study of a small group of electricians involved in a renovation project where asbestos exposure was documented showed radiological evidence of asbestosis in about 16%; for those employed more than 20 years, the percentage was 25%. Electricians in shipyards have shown a prevalence of about 60%.[1]

1 Hodgson, MJ; Parkinson, DK; Sabo, S; Owens, GR, and Feist, JK "Asbestosis Among Electricians," Journal of Occupational Medicine Vol. 30, No. 8, August, 1988, pp. 638-640.

Maintenance workers in schools and universities

Potential sources for exposure include: Installation of pipes and various parts on sinks, toilets, kitchen machinery and boilers Disassembling, cleaning, and reassembling boilers Pipes were covered with asbestos-containing insulation, boiler gaskets, other gaskets

Disassembling and reassembling boilers could create some dust, depending on the location and condition of the asbestos insulation on the boilers and associated parts. Changing gaskets would result in minimal exposure.

A school system may have a written policy which prohibits maintenance men and other personnel from disturbing, repairing or removing asbestos. These policies may have been implemented about the time EPA and OSHA requirements became effective for schools, which would have been about 1983. Find out if such a policy was in effect for the entire length of the injured worker's employment.

Also find out which buildings the injured worker worked in. The school system should have detailed records about the presence of asbestos for each building.

Millwrights

Millwrights set up and dismantle equipment. On some sites, it is possible equipment could have been erected and dismantled in areas where asbestos was used for insulation. Millwrights or others may have knocked asbestos off surfaces while working in these areas. At other times, millwrights probably erected and dismantled equipment in asbestos-free areas. Exposures would have been intermittent and generally would not be comparable to the high exposures experienced by those who routinely tore off and replaced asbestos insulation (asbestos workers, pipefitters, plumbers, boilermakers). Thus, the risk for asbestosis would be less than those employed in these other trades.

By the early 1980s, strict asbestos regulations were in effect, but compliance was not universal. Union employees, such as mil1wrights, were generally aware of the regulations and required personal protective equipment before working around asbestos or required the work to be done by asbestos abatement workers. In general, millwrights would not be expected to have experienced as much exposure as pipefitters and insulators who worked more directly with asbestos-containing materials.

It would be very difficult to determine when or at which site the claimant received his "last injurious" exposure to asbestos.

Plumbers/ Pipefitters

Asbestos was commonly used in the past for pipe and pipe fitting insulation, boiler and tank insulation, and HVAC duct insulation. There is little doubt most of the piping in powerhouses, coke ovens, blast furnaces, and chemical plants was covered with asbestos insulation during this time period. These materials are generally described as friable and as such pose a greater risk of releasing fibers into the air than non-friable materials. [1,2]

A plumber/pipefitter who worked prior to asbestos regulation would be at risk for developing asbestos-related disease. It would be very difficult, however, to determine when or at which site the "last injurious" exposure to asbestos occurred.

More intense exposure would be expected during the earlier years of employment, before regulations limiting employee exposure to asbestos were in effect. Before such regulations, pipefitters commonly mixed, sawed, and removed asbestos insulation without any precautions to minimize dust; as a result exposures to asbestos dust were high. By the early 1980s, strict asbestos regulations were in effect, but compliance was not universal. Union employees were generally aware of the regulations and required personal protective equipment before working around asbestos or required the work to be done by asbestos abatement workers.

It is likely these workers were exposed to asbestos during many years of employment in construction. The frequency, intensity and duration of exposure are not known, but, in general, pipefitters were exposed frequently and intensely to asbestos.

1. Patty's Industrial Hygiene and Toxicology, General Principles Vol. IB, 4th Edition, edited by George D. Clayton & Florence E. Clayton, John Wiley & Sons, New York, 1991.

2. "Guidance for Controlling Asbestos-Containing Materials in Buildings," EPA 560-5-85, June, 1985.

Roofers

Roofers can be exposed to asbestos during tear-off of deteriorated asbestos shingles and other asbestos-containing materials.

Steel -Makers

Asbestos was commonly used to insulate furnaces and other hot process equipment in steel and other industries in the decades prior to the early 1970s. Asbestos insulation may have been used on various pipes and equipment in the mill. It was also commonly used in the caulking between firebricks lining industrial furnaces. Employees who installed or tore out and replaced bricks in these situations could be exposed to asbestos from such caulking. While asbestos was a good insulator for steam-associated equipment, its use around molten steel was limited since it would degrade at such high temperatures.

Employees who worked in melting departments in the steel industry have claimed they covered ladles and furnaces with brick and asbestos millboard. They have also described dust exposures from using compressed air to blow off the work area and from cutting asbestos millboard. Company industrial hygienists have reported the casting floor mold platforms of continuous casting equipment were covered with asbestos fiber board coverings. If a claimant cut asbestos millboard and/or used compressed air to blow off asbestos insulated equipment, he probably was exposed to some asbestos, especially if protective measures were not in place. Steel workers have reported throwing raw asbestos onto the molten metal in ladles to protect against heat loss.

COMMON ASBESTOS-RELATED DISEASES

Asbestos is known to cause asbestosis, pleural changes, and lung cancer, as well as other cancers.

ASBESTOSIS

Asbestosis is a type of pulmonary fibrosis due to asbestos exposure. It tends to produce diffuse interstitial fibrosis and usually presents a restrictive pattern of breathing impairment. Asbestosis might be expected to develop within 15 to 20 years among workers regularly exposed to asbestos, though this varies with the dust levels. Cases have been reported as progressing from first exposure to death within 10 years and in some occupations, such as spraying asbestos, an exposure of as little as three years may result in the development of asbestosis.[1]

The following quote by Murphy demonstrates the difficulty of diagnosing of asbestos related disease, and the importance of high quality X-rays and their interpretation: [2]

" …chest roentgenogram is the best means to exclude many other diseases as well as to quantitate their importance when present. The major problem is with the nonspecificity of the findings, particularly in detecting slight or early disease Bilateral, small, irregular, and linear opacification (more predominant in the lower lung fields) and associated, pleural disease are the most commonly accepted features of asbestosis. When found in an advanced degree with known exposure, specificity for asbestosis is high. However, when irregular, linear, basal opacities are present in slight profusion, they are easily confused with the normal vascular markings, with shadowing caused by poor inspiration, with soft tissue densities due to breast or other fatty tissue, and by the effect of low kilovoltage - making this interpretation one of the most subjective in radiology."

1. Morgan. W.K.C., M.D. and A. Seaton, M.D., Occupational Lung Diseases, W.B. Saunders Co. 1984, pp. 296-297.

2 Murphy, R_ "Asbestos Related Disease: Difficulties in Diagnosing Occupationally Related Illness," Frontiers in Medicine, 2/10/91.

LUNG CANCER

Cigarette smoke is a well established cause of lung cancer, (Check page 66 of this manual). Studies have indicated smoking and asbestos exposure together increase the risk for lung cancer dramatically. NIOSH reported the following information regarding the lung cancers associated with asbestos.[1]

- **The majority of asbestos-associated bronchial carcinomas arise in lungs that also show asbestosis,**

- **The lung cancers occur at a slightly earlier age than in non-exposed individuals,**
- **They arise in relation to the fibrotic lesions and thus are more common in the periphery of the lower lobes,**
- **All histological types of cancer occur; most studies show a preponderance of adenocarcinomas.**

1. Occupational Respiratory Diseases US Department of Health and Human Services, DHHS(N10SH) Publication No. 86-102, Sept., 1986.

3. Bohlig, H. and Calavrezos, A., "Development, radiological zone patterns, and importance of diffuse pleural thickening in relation to occupational exposure to asbestos," British Journal of Industrial Medicine 1987; 44: 673-681.

MESOTHELIOMA Mesothelioma is **a rare tumor of the lining of the cavity of the chest, abdomen, or, pericardium, presenting with pain and effusion of fluid into the cavity.**[1] The tumor grows mainly locally and leads to death within months rather than years. In a publication by the National Institute for Occupational Safety and Health (NIOSH), Dr. Ruth Lillis states "...asbestos is widely accepted as the causative agent in the vast majority of mesothelioma cases. Asbestos is not the only cause of mesothelioma; mesothelioma was known long before any substantial quantity of asbestos was ever mined or used. However, asbestos is considered the most important cause of mesothelioma in modem times. [1]

The development of mesothelioma is associated with low levels of exposure and a short duration of exposure (several weeks to several months). Although a dose response relationship may exist, it has not been quantified. Pulmonary interstitial fibrosis (asbestosis) is not necessarily a precursor to mesothelioma.[3,4] Dr. Raymond Murphy states, "When mesothelioma occurs in an asbestos worker with a prolonged period from onset of initial exposure to asbestos, it is almost certain that a cause-and-effect relationship exists. In cases where exposure history is known, and mesothelioma has been found at autopsy, the average duration elapsed since initial exposure is over thirty years. When the exposure duration is much shorter, for example, less than ten years, the cause and effect- relationship becomes more difficult to assess. An extremely important question arises as to the cause-and-effect relationship between mesothelioma and slight or indirect exposure to asbestos." [5]

According to NIOSH, the population at risk for developing mesothelioma includes the following:[2]

- **All occupations with direct contact and handling of asbestos,**
- **Employees with other occupations (electricians, welders, painters, carpenters, etc.) who work or have worked, even for short periods, in areas where asbestos has been handled by others,**

- **Family members (household contacts) of asbestos workers who have been exposed to asbestos fibers brought into the household by the worker, and**
- **Individuals who have resided in the vicinity (one mile) of an asbestos plant, shipyard, or other source of asbestos contamination.**

The latency period for malignancies due to asbestos exposure, including mesothelioma, is generally more than 20 years. An average latency period between initial exposure and subsequent development of the tumor has been reported as 40 years.[3]

1 Encyclopedia of Occupational Health and Safety Vol. 1, edited by Dr. Luigi Parmeggiani . International Labour Office, Geneva, 1989.

2 Occupational Respiratory Diseases, US Dept. of Health and Human Services, DHHS(NIOSH) Publication No. 86-102, September, 1986.

3 Zenz, Carl, M. D., ScD., Occupational Medicine Principles and Practical Applications Second Edition, Year Book Medical Publishers, Inc., Chicago, 1988.

4 Occupational Lung Diseases, W.K.C. Morgan, M.D., and Anthony Seaton, M.D., W. B. Saunders Co., 1984.

5 Murphy, Raymond MD, "Asbestos Related Disease: Difficulties in Diagnosing Occupationally Related Illness," Frontiers in Medicine Vol. 9, 1987.

PLEURAL THICKENING AND PLEURAL PLAQUES / INDICATORS OF EXPOSURE

Pleural thickening and pleural plaques are common indicators of asbestos exposure, not disease according to the National Institute for Occupational Safety and Health (NIOSH).[1] NIOSH reports the plaques are more common than the pulmonary parenchymal lesions of asbestosis, thus their presence does not necessarily imply coexistent asbestosis. In 1987 Bohlig and Calavrezos reported their studies of 1204 asbestos-exposed workers and 622 non-exposed controls. Bilateral diffuse pleural thickening was the most frequent and probably the earliest detectable sequela of asbestos exposure. In the group they studied, there was not a single case of parenchymal involvement that did not also have concomitant, usually advanced, diffuse pleural thickening. These authors also noted pleural plaques were extremely rare in the unexposed group.[2]

Additional information on asbestos related pleural disease is available in the referenced text.[1] The Bureau of Workers' Compensation library can provide much more information on this topic if it is required. You can call (614) 466-7388 or 1-800-282-3045 extension 7388 for assistance.

1. Occupational Respiratory Diseases, US Department of Health and Human Services, DHHS (NIOSH) Publication No. 86-102, Sept., 1986.

2. Bohlig, H. and Calavrezos. A.. "Development, radiological zone patterns, and importance of diffuse pleural thickening in relation to occupational exposure to asbestos," British Journal of Industrial Medicine, 1987 44: 673-681.

RELATED FACTORS

While some asbestos-related diseases, such as mesothelioma, have been associated with relatively low "bystander" exposures, pulmonary fibrosis from asbestos exposure (asbestosis) has a more definite dose-response and is associated with high exposures, such as those experienced by asbestos workers, insulators and pipefitters in the past.

The **latency period between exposure to asbestos and detection of asbestos-related malignancies is generally more than 20 years**, but shorter periods have been reported. Pleural calcification is not seen commonly until 20 years after onset of exposure. Symptomatic asbestosis is uncommon before at least 20 - 30 years of exposure. Death rates for lung cancer in asbestos workers who smoke are much higher than in nonsmoking workers.[1]

OTHER EXAMPLES OF OCCUPATIONAL DISEASES

ASTHMA

Individuals with asthma complain of wheeziness and shortness of breath. Initially these symptoms may occur only while the individual is at work, but later may persist at home and on the weekends. Workers who are atopic (genetically predisposed) are more prone to develop occupational asthma and may do so on relatively short exposure. Non-atopic individuals may also be affected although their symptoms often do not appear for several years; that is, until they have become sensitized. The sensitizing agent is most often a gas, vapor or dust. Usually, the individual who develops occupational asthma has worked directly with the sensitizing agent.

In cases of asthma, air sampling results must be interpreted cautiously. Low levels of air contaminants might aggravate an existing asthma condition, but cause no problem for a healthy worker.

There are many agents known to cause asthma. The following lists are not complete:[1]

Natural products such as: vegetable gums, flax seed, castor bean, soybean, natural glues, animal danders and other animal antigens, coffee bean, insect debris, detergent enzymes, grain dust and grain products, orris root, flour, papain, mushroom dust and moldy compost, wood dusts, natural resins, animal fat, oil, fish meal and emulsions, tobacco dust, pancreatic extracts;

Synthetic, inorganic products such as: complex platinum salts, nickel salts, chromium salts, sodium and potassium persulphates; and

Synthetic, organic products such as:
diisocyanates (toluene diisocyanate, diphenylmethane diisocyanate, hexamethylene diisocyanate);

anhydrides (phthallic anhydride, tetrachlorophthallic anhydride, trimellitic anhydride);

amines (aminoethyl ethanolamine, dimethyl ethanolamine, ethylene diamine, paraphenylenediamine, diethylene triamine, diethylene tetramine);

pharmaceuticals (penicillin, ampicillin, spiramycin, phenylglycine acid chloride, sulphathiasole, bromelin, amprofium hydrochloride, suphone chloramides);

miscellaneous (formaldehyde, piperazine, organophosphorous insecticides, pyrolysis products of polyvinyl chloride, alkylaryl polyether alcohol, tartrazine, products of heated adhesives)

1. Occupational Respiratory Diseases DHHS (NIOSH) Publication No. 86-102, September, 1986.

Information developed by Division of Safety and Hygiene

BERYLLIOSIS AND CHRONIC BERYLLIUM DISEASE (CBD)

Beryllium and its compounds are highly toxic. The route of entry into the body is almost always by inhalation. The exposure limits established for beryllium are very low, even lower than the exposure limits for lead.

Berylliosis can be chronic or acute. Chronic pulmonary berylliosis is a systemic disease which produces granulomata throughout the body, particularly affecting the lungs. The disease may represent a hypersensitivity reaction and an immunological basis for the disease is generally accepted. [1,2] The onset of symptoms may occur while the worker is still exposed to beryllium or long after exposure has ceased. There may be a previous history of acute beryllium pneumonitis.[2] Some authors report the lack of a well established dose-response relationship in chronic beryllium disease cases. Dr. Otto Preuss, retired from his former position as Corporate Medical Director for Brush Wellman, reported the development of CBD requires no prolonged exposure period but a predisposition for sensitization by beryllium ions and a minimum exposure. [1]

CBD usually is of long duration with exacerbations and remissions. The disease can vary from asymptomatic and non-disabling to severely disabling disease which may result in right heart failure. In its mildly disabling form, the disease results in some nonproductive cough and dyspnea following unusual levels of exertion. Joint pain and weakness are common complaints. [3,4]

According to Proctor and Hughes' Chemical Hazards of the Workplace the presence of beryllium in tissue is an indicator of exposure, but the concentration is often not in direct proportion to the severity of the disease. These authors report an individual's reactivity to beryllium can be assessed with a "lymphocyte blast transformation test of lymphocytes from peripheral blood or bronchoalveolar lavage", with the latter providing a better index of reactivity. [5] An article published in 1988 by Dr. Markham and others describes the usefulness of these tests (bronchoalveolar cell proliferation response to beryllium salts) as a diagnostic tool for chronic beryllium disease. The authors reported their findings support the hypothesis that chronic beryllium disease is a cell-mediated type of
hypersensitivity pneumonitis. Copies of this article are available from the BWC library.

1. Zenz, Carl, Occupational Medicine, Principles and Practical Applications Second Edition, Year Book Medical Publishers, Chicago, 1988.

2. Morgan and Seaton, Occupational Lung Diseases, 2nd. Edition, W. B. Saunders Co., Philadelphia, 1994.

3. Occupational Diseases, A Guide to Their Recognition Revised Edition, DHEW (NIOSH) Publication No., 77-18 1, June 1977,

4. Occupational Respiratory Diseases DHHS (NIOSH) Publication No. 86-102, September, 1986.

5. Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by G. J. Hathaway, N. H. Proctor, J. P. Hughes, and M. L. Fischman.

CARBON MONOXIDE POISONING

Carbon monoxide (CO) is a chemical asphyxiant. It interferes with the blood's ability to deliver oxygen to the tissues. **If present in certain concentrations, carbon monoxide might produce headache, light headedness, and nausea. Higher concentrations can result in unconsciousness or coma.**

Common sources of carbon monoxide in industry are gasoline or propane-powered fork trucks, gas heaters, and cigarette smoke. Replacing gasoline or propane-powered trucks with electric trucks, fitting gas heaters with automatic controls to bring in outside combustion air, and increasing general ventilation are common recommendations. Non-occupational sources of carbon monoxide include car exhaust, faulty home heating systems, and cigarette smoke. Methylene chloride, a chemical used to strip furniture finish, is metabolized into carbon monoxide in the body.

Carboxyhemoglobin, a complex of CO and hemoglobin can be detected in the blood. The American Conference of Governmental Industrial Hygienists (ACGIH) states blood samples must be collected within 15 minutes after the end of the shift for the Biological Exposure Index to be applied. At the end of an 8-hour exposure, carboxyhemoglobin approaches steady state. The time the blood sample was collected is important because of the half-life of carboxyhemoglobin and because emergency response personnel frequently administer oxygen to suspected carbon monoxide poisoning victims. Therefore, reported carboxyhemoglobin levels could be higher at the time of the injured worker's injury than reported later in the hospital.

Detailed information about CO is available in the Documentation of Threshold Limit Values and Biological Exposure Indices, published by the ACGIH.[1] The current l995 Biological Exposure Index (BEI) is 3.5% carboxyhemoglobin. The ACGIH notes carboxyhemoglobin is usually present in a significant amount in biological specimens collected from subjects who have not been occupationally exposed and states such background levels are included in the BEI value. Definitions of these terms, TLV and BEI are included in the glossary.

The following BEI documentation gives the **typical saturation levels of carboxyhemoglobin in populations without occupational exposure to carbon monoxide:**

Endogenous production	**0.4%-0.7%**
during pregnancy	**up to 2.6%**
patients with hemolytic anemia	**4%-6%**
Urban population	**1%-2%**
Commuters on urban highways	**5% or more**
Tobacco smokers	
cigarettes, one pack per day	**5% - 6% average**
cigarettes, 2 - 3 pack per day	**7% - 9% average**
cigars	**up to 20%**
Methylene chloride exposure	
at 50 ppm for 8 hours per day	**1.5%-2.5%**

These results correlate well with those reported in a study of traffic workers:[2]

4. 1% for light smokers
5.4 % for those smoking at least 20 cigarettes (one pack) per day

The graph included in the ACGIH documentation shows the relationship between carboxyhemoglobin in blood and duration of exposure to various concentrations of carbon monoxide. A carboxyhemoglobin (COHb) level of 8%-9% corresponds to an 8-hour exposure of approximately 0.005% or 50 ppm, which is the permissible exposure limit currently enforced by OSHA. A carboxyhemoglobin level of more than 20% is considered toxic, according to Hamilton and Hardys Industrial Toxicology.[2]

According to Patty's Industrial Hygiene and Toxicology, a small amount of COHb is produced in the body and accounts for a COHb of about 0.4% to 0.7%. Some diseases such as hemolytic anemia cause higher percentages.[1] This reference states tobacco smoking is a major source of carbon monoxide exposure in many adults. Cigarette smoke contains over 2% carbon monoxide (CO); the average concentration of carbon monoxide in the smoke that reaches the lungs is about 400 ppm.

1 Documentation of Threshold Limit Values and Biological Exposure Indices American Conference of Governmental Industrial Hygienists, Cincinnati, OK 1994.

2 Hamilton and Hardy's industrial Toxicology Fourth Edition, edited by Asher J. Finkel, published by John Wright - PSG Inc., Boston. 1983.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

The following table is from Occupational Respiratory Diseases, DHHS (NIOSH) Publication 86-102, pp. 504. Please check this reference for a thorough discussion of chronic bronchitis, emphysema and COPD. Please also refer to page 66 of this manual (smoking).

LIST OF CAUSATIVE AGENTS

	Definite	Probable	Possible
Aldehydes (acrolein, formaldehyde)	+		
Ammonia	+		
Brick Dust		+	
Cadmium (emphysema)			+
Chlorine	+		
Chloromethyl Methyl Ether	+		
Chromium	+		
Coal Mine Dust (bronchitis, emphysema)	+		
Cobalt		+	
Coke Oven	+		
Cotton Dust	+		
Diesel Exhaust	+		
Endotoxin	+		
Grain Dust (wheat, barley)	+		
Osmium Tetroxide	+		
Oxides of Nitrogen		+	
Paraquat			+
Phosgene	+		
Polychlorinated Biphenyls		+	
Pottery Dust	+		
Sodium Hydroxide	+		
Toluene Diisocyanate	+		
Tungsten Carbide		+	
Vanadium		+	
Vinyl Chloride Monomer		+	
Western Red Cedar	+		
Wood Dust	+		

HEARING LOSS CLAIMS

Information to obtain from the company includes:

- Hearing test results, if any, when hired and any subsequent audiograms
- Noise measurements in the injured worker's work area, if any
- Job description

According to authors of Noise & Hearing Conservation Manual, noise-induced hearing loss is generally characterized by poorer hearing in the high tones and relatively good hearing in the lower test frequencies. [1] Even if an audiogram, shows a similar pattern with one reflecting a noise-induced hearing loss, the similarity alone is not enough to determine if hearing loss was due to occupational noise. Other causes of hearing loss are: aging, nonoccupational noise sources (power tools, lawnmowers, amplified music, military noise exposure), medical abnormalities, and use of certain medications.

Since aging is also a cause of hearing loss, the Occupational Safety and Health Administration (OSHA) allows employers to adjust the results of the most recent audiogram to allow for the contribution of aging to the change in hearing level.[2] However, such baseline audiograms; were often not conducted.

1 Noise and Hearing Conservation Manual 4th Edition, edited by Elliott Berger, Jeffrey Morrill, W. Dixon Ward, and Larry Royster, American Industrial Hygiene Association, Akron, Ohio, 1988.

2 CFR 1910.95, Occupational Noise Exposure, US Department of Labor, Occupational Safety and Health Administration.

HISTOPLASMOSIS

Histoplasmosis is caused by *Histoplasma capsulatum*, a fungus which grows particularly well in soil contaminated with fecal material of birds or bats. **(Histoplasmosis is not common as an occupational disease, but endemic to the Ohio area). Populations at risk include farmers and persons who live and work in endemic areas of the infection,** such as the Ohio and Mississippi River Valleys. **The disease is transmitted by the inhalation of aerosols produced by the fungus during the disturbance of soil containing the fungus.** Such disturbances would include cleaning or razing old chicken coops, demolishing buildings occupied by pigeons, disturbing ground under pigeon roosting sites, etc.

Health effects on humans are often subclinical and vary depending on dose and immune response of the individual. Histoplasmosis usually results in a febrile illness with influenza-like symptoms, cough and, pneumonitis. Recovery usually occurs within 2 to 3 weeks. Chronic forms may be severe and difficult to treat.[1,2]

According to the National Institute for Occupational Safety and Health (NIOSH) almost all residents in some areas of the central United States are infected regardless of occupation and in most cases they are infected in childhood. In Ohio, more than half of the individuals in farm areas who were tested had positive skin reactions to histoplasmin.[3] Some additional information about the disease, including a map showing endemic areas, can be obtained from the Ohio Health Department.

1. Zenz, Carl, M.D., ScD., Occupational Medicine Principles and Practical Applications Second Edition, Year Book Medical Publishers, Inc., Chicago, 1988.

2. Occupational Diseases: A Guide to their Recognition Revised Edition, U. S. Dept. of Health, Education, and Welfare, DHEW (NIOSH) Publication No. 77-181, Jane, 1977.

3. Occupational Respiratory Diseases U. S. Dept. of Health and Human Services, DHHS (NIOSH) Publication No. 86-102, 1987.

LEAD POISONING

Unfortunately, we are still investigating claims of lead poisoning due to use of lead-based paint, the melting of lead alloys, and other processes.

These files usually contain blood lead test results. For example:

Sample collected	Blood lead	Protoporphorin Free (FEP)	Zinc protoporphorin (ZPP)
3/25/94	**64 mcg/dl**	**70 mcg/dl**	**77 mcg/dl**
5/3/94	**54 mcg/dl**	**64 mcg/dl**	**70 mcg/dl**
7/1/94	**43 mcg/dl**	**29 mcg/dl**	**31 mcg/dl**
10/10/94	**37 mcg/dl**		

mcg/dl = micrograms per deciliter (This is also sometimes abbreviated ug/l)

The paint in this example contained 30% lead chromate, 30% lead molybdate, and 5% lead sulfate. Even if a company has switched to a lead free paint, or paint with a lower lead content, employees would still be exposed to lead when they sand on the old paint.

There are strict federal (OSHA) requirements for lead, including air monitoring when there is potential lead exposure. A company should have air monitoring results, and if respirators are required, documentation showing the proper respirator was selected and fit.

Exposure to lead and other agents in paint can occur from inhalation of lead containing paint mist due to an inadequately fitted respirator. Another route of exposure can be accidental ingestion of lead due to inadequate personal hygiene (leading to contamination of food and tobacco products).

In the United States, lead in blood of adults without occupational exposure is usually between 10 mcg/dl and 35 mcg/dl; 98% of the population has concentrations below 50 mcg/dl. For industrially exposed adults, the normal blood lead level is less than 40 mcg/dl; 80 mcg/dl is considered toxic. The Occupational Safety and Health Administration (OSHA) requires employers to take specific actions to reduce exposures when blood lead levels reach 40 mcg/dl, and requires employees whose blood lead levels reach 50 mcg/dl to be removed from exposure.[1] According to Occupational Medicine Principles and Practical Applications, elevated ZPP/FEP values are significant because they show functional change and they persist out of proportion to current blood lead levels which drop after exposure ceases.[2] The normal ZPP range is below 40 - 50 mcg/dl, and for adults who are exposed to lead, the limit for ZPP after one month of exposure to lead is 100 mcg/dl.

According to Proctor and Hughes' Chemical Hazards of the Workplace , signs and symptoms of lead exposure include: weakness, lassitude, and insomnia; facial pallor and pallor of the eye grounds; anorexia, weight loss, and malnutrition; constipation, abdominal discomfort and tenderness, and colic; anemia; lead line on gingival tissues; signs of motor

weakness, including paralysis of the extensor muscles of the wrist and less often of the ankles; encephalopathy; and nephropathy.[3] Additional information about the significance of zinc protoporphorin tests is available in Reference #3 from the BWC Library.

I Documentation of Threshold Limit Values and Biological Exposure Indices American Conference of Governmental Industrial Hygienists, Cincinnati, OH, 1992.

2 Zenz. Carl. M. D., Sc. D., Occupational Medicine Principles and Practical Applications. Second Edition, Year Book Medical Publishers, Chicago, 1988.

3 Proctor and Hughes' Chemical Hazards of the Workplace, Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James P. Hughes, and Michael L. Fischman, Van Nostrand Reinhold, New York, 1991.

METAL FUME FEVER

Metal fume fever results from the inhalation of minute particles of oxides of various metals. **Zinc, copper, and magnesium are the chief causes,** but cadmium, iron, manganese, nickel, selenium, tin, and antimony are sometimes also responsible.[1] **Welding galvanized steel is the most common cause for metal fume fever.[2]**

The disease has an acute onset and there is usually a four to eight hour lag before symptoms of sudden thirst and metallic taste in the mouth occur. Later, symptoms resemble the flu, and a leukocytosis is often present. While many of the metallic oxides cause only transient symptoms with no harmful side effects, cadmium fume is more hazardous and can cause permanent lung damage.[1]

1 Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James P. Hughes, Michael L. Fischman, Van Nostrand Reinhold, New York, 1991.

2 Zenz, Carl, Occupational Medicine Principles and Practical Applications Second Edition. Year Book Medical Publishers, 1988.

3 Hawley's Condensed Chemical Dictionary Eleventh Edition, edited by N. Irving Sax and Richard J. Lewis, Van Nostrand Reinhold Co., New York, 1987.

PULMONARY EDEMA

Pulmonary edema can result from massive exposures to irritant gases and fumes. The National Health Institute for Occupational Safety and Health (NIOSH) lists ammonia, phosgene, toluene diisocyanate, cadmium oxide, and nitrogen dioxide as occupational causes of pulmonary edema.[1] Such exposures are also associated with other symptoms, such as upper respiratory tract irritation from high exposures to ammonia or phosgene, toluene diisocyanate, and metal fume fever from exposure to cadmium oxide. NIOSH reports such massive exposures can damage the alveolar-capillary membrane or parenchyma of the lung. Pulmonary edema has been occasionally reported among workers with high exposures to freshly generated finely divided particulate metallic oxides, combustion products of fluorocarbon polymers, organic dusts containing bacterial endotoxins, and possibly mycotoxins.[2]

1. Occupational Respiratory Diseases U. S. Department of Health and Human Services, DHHS (NIOSH) Publication 86-102, April, 1977, p. 306.

2. Dorland's Illustrated Medical Dictionary 27th Edition, W. B. Saunders Co., Philadelphia, PA. 1988.

SARCOIDOSIS According to Dorland's Illustrated Medical Dictionary, 27th Edition, sarcoidosis is a chronic, progressive, systemic granulomatous reticulosis of *unknown occupational* etiology, involving almost any organ or tissue, including the skin, lungs, lymph nodes, liver, spleen, eyes, and small bones of the hands and feet. This reference indicates the acute form has an abrupt onset and spontaneous remission rate, while the chronic form is insidious in onset and progresses.[1]

Another reference indicates sarcoidosis is more common among young blacks and describes the disease as one in which multiple, tiny patches of inflammation suddenly appear in one or several parts of the body, frequently in the lungs.[2] This article states no one knows why sarcoidosis occurs or why it clears up, as it usually does on its own.

A third article also states the cause of sarcoidosis is unknown, but fists the following as possible causes that have been considered:[3]

a) Hypersensitivity response to such agents as atypical mycobacteria, fungi, and pine pollen;
b) Genetic predisposition (suggested by a slightly higher incidence of sarcoidosis within the same family;) and
c) An extrinsic cause suggested by similar illnesses caused by beryllium and zirconium.

1. Dorland's Illustrated Medical Dictionary 27th edition, W. B. Saunders Co., Philadelphia, 1988.

2. The American Medical Association Family Medical Guide, 3rd edition, edited by Dr. Charles B. Clayman, Random House, N.Y., 1994.

3. Professional Guide to Diseases, 4th edition, edited by Genell J. Subck-Sharpe, Springhouse Corp., Springhouse, PA, 1991.

SPECIAL AGENTS AND PROCESSES

ALUMINUM OXIDE

Aluminum oxides are considered to be nuisance dusts, but can cause a minimal pulmonary nodular response. A 1988 study of workers in an aluminum production facility showed 7%-8% of the workers had small irregular opacities as determined by chest x-ray. The prevalence of opacities was increased among smokers and among nonsmokers with high cumulative dust exposures. A slight, but significant, decrement in ventilatory function among nonsmoking workers was also observed. These findings were consistent with a minor degree on nonspecific chronic industrial bronchitis. The bronchitis was associated with excessive protracted nuisance dust exposure (i.e., 100 milligram-years for more than 20 years).[1]

1. Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James. P. Hughes, Michael L. Fischman, Van Nostrand Reinhold, New York, 1991.

BRICK MAKING According to the International Labour Office's Encyclopedia of Occupational Health and Safety the materials used for making bricks and tiles do not usually produce highly dangerous dust unless the content of free silica in the clay is relatively high. The silica content in clay deposits can vary between 3% to as high as 35%, depending on the quality of the deposit.[1] In many brickyards, the clay pit was adjacent to the factory where the finished articles were made. Dust hazards increased with mechanization and low moisture levels.

1. Encyclopedia of Health and Safety, Third (Revised) Edition, edited by Dr. Luigi Parineggiani. International Labour Office, Geneva, 1989.

Information developed by Division of Safety and Hygiene

CEDAR WOOD DUST

Dr. Anthony Seaton reports workers may develop hypersensitivity to wood dust as well as to fungal spores and substances used in the treatment of wood. He lists cedar, oak, mahogany, western red cedar, iroko, cedar of Lebanon, and zebrawood as causes of asthma.[1] According to Hamilton and Hardy's Industrial Toxicology occupational asthma and rhinitis have been particularly noticeable among employees who work with western red cedar *(Thuja plicala).* The authors report the ingredient in red cedar extract that appears to cause bronchoconstriction appears to be plicatic acid, and an inhalation provocation test can be used to confirm the diagnosis.[2] The National Institute for Occupational Safety and Health (NIOSH) lists western red cedar as a definite causative agent for chronic bronchitis.[3]

The American Conference of Governmental Industrial Hygienists reports there are numerous reports of occupational dermatitis and asthma resulting from exposure to allergenic dusts (dogwood, mulberry, myrtle, red cedar). The recommended exposure limits for wood dust exclude the issue of occupational asthma and related allergic respiratory response associated with exposure to red cedar dust and similar woods.[4]

1. Collingwood, G. R and Warren D. Brush, Knowing Your Trees revised by Devereux Butcher. American Forestry Association, Washington, D. C., 1974.

2. Morgan, W. Keith C. and Anthony Seaton, Occupational Lung Diseases, 2nd. Edition, W. B. Saunders Co., Philadelphia, 1984.

3. Hamilton and Hardy's Industrial Toxicology Fourth Edition, revised by Asher J. Finkel, John Wright, PSG, Inc., Boston, 1933.

4. Occupational Respiratory Diseases, US Dept. of Health and Human Services, DHHS(NIOSH) Publication No. 86-102, April, 1987.

5. " Wood Dusts," Documentation of the Threshold Limit Values American Conference of Governmental Industrial Hygienists, Cincinnati, OH, 1994.

CEMENT DUST *Concrete* contains gravel, pebbles, sand, broken stone, blast-furnace slag or cinders. This material is embedded in mortar or *cement* (usually Portland cement in the US).[1] Portland cement is a finely divided, gray powder. It contains lime, alumina, silica, and iron oxide (as tetracalcium., aluminoferrate, tricalcium, aluminate, tricalcium silicate, and dicalcium silicate). Small amounts of magnesia, sodium, potassium, and sulfur are also present. Free silica content is usually less than one percent.[1]

The National Institute for Occupational Safety and Health (NIOSH) states cement dust can cause non-specific bronchitis and obstructive respiratory impairment. NIOSH also reports workers in several industries, including ceramics and cement, have more chronic bronchitis than can be accounted for by such factors as age, smoking, and air pollution.[4]

There have been reports of pneumoconioses among cement workers. However, these relate to exposures that occurred primarily in the mining, quarrying, or crushing of silica containing raw materials.[3,4] Trace amounts of hexavalent chromium in Portland cement can cause allergic contact dermatitis.[5] Additional information is available from our library.

At a plant where cement trucks are loaded, an operator may use a control panel to regulate the delivery of cement through various gates and hoppers into trucks. The panel may be inside a control room, but operators would typically go outside to check trucks, etc. and would be exposed to some dust.

I Hawley's Condensed Chemical Dictionary Eleventh Edition, revised by N. I. Sax and R_ J. Lewis, Sr., Van Nostrand Reinhold Company, New York, 1997.

2 Pattv's Industrial Hygiene and Toxicology Volume 2B , Third Revised Edition. edited by George D. Clayton & Florence E. Clayton, John Wiley & Sons, New York. 1991.

3 Patty's Industrial Hygiene and Toxicology Volume IB, 4th Edition, edited by George D. Clayton & Florence E. Clayton, John Wiley & Sons, New York, 1991.

4 Occupational Respiratory Diseases US Department of Health and Human Services, DHHS (NIOSH) Publication No. 86-102, September, 1986.

5 Zenz, Carl, M.D., Sc.D., Occupational Medicine Principles and Practical Applications Second Edition, Year Book Medical Publishers, Inc., Chicago, 1988.

CHEMICAL BLOWING AGENTS IN PLASTICS MANUFACTURING

The following information is from a chapter entitled "Plastic Processing Operations" in the referenced text.1 Chemical blowing agents (CBAs) are added to the polymer (resin) during processing to form tiny gas cells throughout the plastic product. The gas is liberated from a chemical reaction or decomposition upon heating. The CBA powder can be dry tumbled with the polymer, mixed in as a concentrate in a suitable polymer carrier, or metered directly into the system as a dispersion in a compatible carrier. Organic CBAs can be compounded into the resin by the formulator or mixed with pellets by the processor. Compounds such as alcohols, glycols, antioxidants, metal stabilizers, and various metal salts are added routinely to control CBA decomposition temperature. The authors of this chapter recommend avoiding unnecessary dust exposures and skin contact with organic CBAs since many are potent skin, eye and upper respiratory irritants. They recommend local exhaust ventilation whenever possible at dumping and mixing stations and measures to eliminate manual handling operations.

Azodicarbonamide (also known as 1,1'-azobisformamide) is the most commonly used organic CBA- The manufacturer, Uniroyal Chemical Company, Inc., classifies the azodicarbonamide as a sensitizer. The MSDS states, "Repeated, minimal inhalation exposure can cause respiratory sensitization and occupational asthma. Exposure to decomposition gases can cause irritation to eyes, lungs, and mucous membranes. Individuals with respiratory problems should avoid inhalation toxicity. " The manufacturer strongly recommends use of local exhaust ventilation for mixing operations and where ventilation is inadequate, use of respiratory protection.

1 Industrial Hygiene Aspects of Plant Operations, Vol. 1, Unit Operations and Product Fabrication edited by Lewis J. Cralley and Lester V. Cralley, Macmillan Publishing Co., New York, 1984, pp. 270-273.

COBALT

Many high speed tools are manufactured from cemented tungsten carbide. Cases of asthma and obstructive lung syndrome have been reported among workers involved in the manufacture of cemented tungsten carbide and also among workers grinding with such tools. Cobalt has been implicated as the causative agent. [1,2]

1. Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James. P. Hughes, Michael L. Fischman, Van Nostrand Reinhold, New York, 199 1.

2. Morgan, W. Keith C. and Anthony Seaton, Occupational Lung Diseases, 2nd Edition, W. B. Saunders Co., Philadelphia. 1984.

COKE OVEN EMISSIONS

Coke oven emissions contain confirmed carcinogens with the lung, skin, and kidneys as targets.[1,2] The Occupational Safety and Health Administration (OSHA) has specifically regulated coke oven emissions since 1977.

An archived industrial hygiene report (1942) described the duties of a coke oven operator as follows:

- running the weigh car under hoppers,
- filling the car with coal,
- running the weigh car directly over the ovens,
- charging the ovens through charge holes,
- backing the weigh car away from the ovens,
- sweeping the spillage into the charging holes, and
- sealing the charging holes.

The investigator noted the oven operator was exposed to coal dust, to dust when the coke was pushed out of the oven, to gases, coal tar vapors and other contaminants. He reported the dust blackened the faces of the operators and the clothes of anyone who happed to be around. The carcinogenic properties of the emissions were not recognized at that time.

In a 1979 report concerning an investigation of a claim filed by coke oven maintenance man, a Division of Safety and Hygiene engineer reported the greatest risk for cancer among workers in this industry was to employees who worked on top of the ovens. This risk is due to exposure to coke oven emissions until 1977. This was about the time the federal government began to regulate coke oven emissions as carcinogenic.

1. Occupational Diseases - A Guide to Their Recognition DHEW (NIOSH) Publication No. 77-123.

2. Proctor and Hughes' Chemical Hazards of the Workplace, Third Edition, edited by G. Hathaway, N. Proctor, J. Hughes, and M. Fischman, Van Nostrand Reinhold, New York, 1991.

COOLANTS

Machining coolants may contain amines, but general statements about exposure to airborne amines from coolant use is difficult because of the variety of products. Amine content can vary from 2% to 15%. The way the product is used also varies widely and affects exposure, for example, some shops enclose the equipment which contains the coolant, but at other shops, the operator can get covered with coolant mist. Machining equipment with enclosures which activate before the equipment can operate keep the coolant from splashing out of the machine.

A bacterial or mold growth in water soluble coolants is possible and occurs sometimes when tramp oil from parts to be machined, hydraulic lines, or other sources gets into the coolant reservoir. Airborne coolant mist can be a source of exposure to these microorganisms and their byproducts. Biocides may be used to control this growth.

CRYSTALLINE FREE SILICA

Crystalline free silica (quartz) causes silicosis, a form of disabling, progressive, and sometimes fatal pulmonary fibrosis. Silicosis is characterized by the presence of typical nodulation in the lungs. The clinical signs and symptoms of this disease tend to be progressive with continued exposure to dust containing free silica, with advancing age, and with continued smoking habits. The symptoms can continue to progress after dust exposure ceases.[1]

While using a torch to cut castings in a foundry, a claimant would be exposed to the oxides of iron and other metals. While metallic oxides would not cause silicosis, they may cause changes on x-rays and interfere with their interpretation. Iron oxide fume and dust can cause a benign pneumoconiosis called siderosis. The retained dust produces x-ray shadows that may be indistinguishable from fibrotic pneumoconiosis.[1]

1. Proctor and Hughes' Chemical Hazards of the Workplace, Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James. P. Hughes, Michael L. Fischman Van Nostrand Reinhold, New York, 1991.

DIESEL EMISSIONS

Diesel exhaust can be irritating to the eyes and respiratory tract; however, studies of workers operating diesel-powered equipment generally have not shown significant adverse health effects. Diesel emissions contain a number of particulates and gases, including carbon monoxide, carbon dioxide, sulfur dioxide, formaldehyde, and nitrogen dioxide. Typically, diesel engines do not produce as much carbon monoxide as gasoline engines, but diesel emissions can be irritating to the eyes and respiratory tract.

Few conclusions about the health effects of chronic exposure can be drawn from available studies. While one study of members of a heavy equipment operators union showed a significantly higher mortality rate from emphysema, other studies of workers with long-term, direct exposure to diesel emissions (coal workers, iron ore workers, railway workers) have not shown significant adverse health effects. [1,2]

1 Occupational Lung Diseases W.K-C. Morgan., M.D., A- Seaton, M.D., W.B. Saunders Company, 1984.

2 Zenz, Carl. M.D., Sc.D., Occupational Medicine Principles and Practical Applications Second Edition, Year *Book* Medical Publishers, Inc., Chicago, 1998.

EPOXIES

Many components of epoxy systems are severe skin irritants and/or skin sensitizers and are well known to cause allergic contact dermatitis. Some components can also cause respiratory irritation, sensitization, and asthmatic symptoms. According to Dr. Toby Mathias, who wrote the chapter on dermatitis in Occupational Medicine Principles and Practical Applications, careful investigation of contact dermatitis due to epoxy resin almost always demonstrates sensitization to the epoxy resin, reactive diluent, or hardener. Glycidal ethers which are used as reactive diluents in epoxy resins are primary skin and eye irritants and potential skin sensitizing agents.

1. Zenz, Carl, M.D., ScD., Occupational Medicine Principles and Practical Applications Second Edition, Year Book Medical Publishers, Inc., Chicago, 1988.

FELDSPAR

Feldspar is composed of a mixture of the silicates of sodium, potassium, and aluminum. One reference states the crystalline silica content of feldspar ranges from 12 to 25%, but elsewhere states feldspar contains no free silicon dioxide.[1] According to this author, feldspar, even with no free silica content, can cause fibrosis of the lung.

1 Levy, Stuart A., MD, "An Overview of Occupational Pulmonary Disorders," in Occupational Medicine: Principle and Practical Applications Second Edition, edited by Carl Zenz, MD, Year Book medical Publishers, Chicago, 1988.

FIBERGLASS

Fiberglass itself is an undisputed mechanical irritant and can cause temporary eye, skin, and upper respiratory tract irritation. Symptoms of upper respiratory tract irritation increase with the dose and duration of exposure and may include burning and itching of the nose and throat, cough, nasal congestion, and rhinitis. Symptoms resolve once exposure ceases.[1]

In 1986 The National Institute for Occupational Safety and Health (NIOSH) reported there were no specific clinical, x-ray, or pulmonary function test changes that are characteristic of fibrous glass exposure.[2] This conclusion was repeated in a joint report by Dr. Philip Harber, Director, UCLA Occupational Environmental Medicine Program and Dr. William B. Bunn M, Mobile Oil Corporation in September, 1993.[3]

Several references were reviewed for information on fiberglass's ability to cause allergic reactions. According to an American Industrial Hygiene Association (AIHA) publication, exposure to fiberglass does not sensitize the lung and cause allergic reactions. There have been cases of asthma reported in a fiberglass manufacturing plant, but agents other than fiberglass were reported to have caused the asthma.[1] A Michigan Department of Public Health publication describes the upper respiratory irritation caused by fiberglass as "not an allergy, but a mechanical irritation characterized by coughing and wheezing. The effects should subside upon removal from the work area and should impose no further burden upon the worker's health."[4]

During manufacturing, glass fibers are coated with an epoxy resin and heat cured. Workers applying the epoxies can be sensitized to the resins, resulting in skin rashes and upper respiratory irritations. However, once resins are cured, sensitized workers exhibit no adverse skin rashes with prolonged handling of the fibrous glass materials.[5]

1 Bender, JR, Konzen, JL, and Devirt, GE, "Occupational Exposure, Toxic Properties, and Work Practice Guidelines for Fiber Glass," American Industrial Hygiene Association. Fairfax, VA, 199 1.

2 Occupational Respiratory Diseases DHHS (NIOSH) Publication No. 86-102., 1986, pp. 444-45 1.

3 Harber, Philip, NID, MPH and William B. Bunn M, MD, JD, MPK "Does Fiberglas Exposure Produce Significant Effects on Pulmonary Function?", The OEM Report, Vol. 7, No. 9, September, 1993, pp. 78 -80.

4 Grubb, Greg, Industrial Hygienist, Michigan Department of Public Health, "Fiberglass: Tomorrow's Asbestos Today?, MOH Vol. 25, No. 1, Summer, 1989, pp. 1-3.

5 Castillo, Jeff, ASP, "Fiberglass: Information You Should Know," Professional -Safety- November, 1992, pp. 29- 32.

6 Konzen, Jon, M. D. "Observations on Fiberglass in Relation to Health" in Occupational Medicine, Principles and Practical Applications Second Edition, edited by Carl Zenz, M. D. Sc.D., Year Book Medical publishers, Chicago, 1988, pp. 1067 - 1071.

FORMALDEHYDE

Formaldehyde can be very irritating to the eyes, mucous membranes, and respiratory tract, depending upon the concentration. Formaldehyde has been reported to cause asthmatic reactions. These have been suggested to be sometimes due to hypersensitivity and sometimes to a direct irritant effect. In the cases described, the formaldehyde exposures were 1.5 ppm, 3.2 ppm, and 20.7 ppm. A summary of irritation effects of formaldehyde is available from the American Conference of Governmental Industrial Hygienist (ACGIH) shows eye irritation as the only complaint at the lowest levels studied (0.02 ppm).

1. Burge, P. Sherwood, L. M. G. Harries, W. K- Lam, L M O'Brien, P. A- Patchett, "Occupational Asthma due to Formaldehyde," Thorax 1985, pp. 255-260.

2. Documentation of the Threshold Limit Values and Biological Exposure Indices, American Conference of Governmental Industrial Hygienists, Cincinnati, OH 1993.

FOUNDRIES

Chippers and grinders who worked in foundries in the past were commonly exposed to excessive amounts of free crystalline silica. The exposures resulted from sand which adhered to the casting after shake-out and to the partial solubilization of silica from the casting molds into the outer portions of the metal. The airborne dust released by grinding such castings has been reported to contain 10% silica.[1]

Molders were commonly overexposed to crystalline free silica in the past in foundries using sand molding agents. While using a torch to cut castings at the foundry, the claimant may have been exposed to the oxides of iron, zinc, and other metals.

While metallic oxides would not cause silicosis, they may cause changes on x-rays and interfere with their interpretation. The authors of Proctor and Hughes' Chemical Hazards of the Workplace state that iron oxide fume and dust can cause a benign pneumoconiosis called siderosis. The retained dust produces x-ray shadows that may be indistinguishable from fibrotic pneumoconiosis.[2]

Magnesium oxide fume is a mild irritant of the eyes and nose and may cause metal fume fever, an illness similar to influenza. Exposure to zinc oxide fume causes metal fume fever, but zinc oxide dust is considered a nuisance dust that has little adverse effect on the lung if exposures are kept under reasonable control. Aluminum oxides are also considered to be nuisance dusts, but can cause a minimal pulmonary nodular response.[2]

Employees who work in a foundry and who are involved in pouring molten brass or other alloys containing lead are at an increased risk of lead poisoning. The lead fume can be controlled to some degree by stringent engineering controls including ventilation. However, these controls often must be supplemented with personal protective equipment such as respirators. The respirators must have the right kind of filters to remove airborne lead dust and fume. Additionally, the respirator must be fitted, cleaned, stored, and maintained according to specific standards.

1. Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James. P. Hughes, Michael L. Fischman, Van Nostrand Reinhold, New York, 199 1.

2. Industrial Hygiene Aspects of Plant Operations: Unit Operations and Product Fabrication Vol. 2, edited by LJ Cralley, LV Cralley, MacMillan Publishing Co,, New York, 1984.

INDOOR AIR QUALITY

Ask the employer if OSHA or another agency such as OSHA On-site Consultation or the National Institute for Occupational Safety and Health (NIOSH) has recently visited the facility to investigate air quality problems. Have any of the other employees in the area or building where the claimant worked complained of the air quality or related breathing problems to their work environment?

Sometimes, private consulting firms will conduct extensive surveys in buildings where there are complaints. Air monitoring might including samples for carbon monoxide, carbon dioxide, formaldehyde, and airborne microorganisms, temperature and humidity levels. These results will usually be compared against OSHA or ASHRAE standards. ASHRAE (American Society of Heating, Refrigerating, and Air-Conditioning Engineers) recommends temperature and humidity values stay within specific ranges for comfort.

If the levels of fungi in the budding are considerably lower than those outdoors, this would indicate the filtration system was effectively cleaning the fresh intake air. Often, the industrial hygienist or engineer will visually examine the drip pans in the HVAC units and ductwork downstream from the cooling coils. If these are dirty or show signs of microbial growth, this could lead to problems during the cooling season. Regular inspection, cleaning, and disinfection of the cooling coils and drain pans is recommended.

IONIZING RADIATION

Exposure to radiation and radioactive materials in laboratories and in industry has become a more important problem with increase use. Types of radiation include alpha rays, beta rays, and gamma rays. Other forms of emitted radioactivity includes x-rays and neutrons.

Occupational exposure can and does occur. Work locations that have potential radiation exposures are well known and dosimeters or badges are used to monitor work exposure. These records should be available.

Any worker who has worked around radiation for several years may develop cancer. Cancers include:
Bone, leukemia, lung cancer, multiple myeloma (malignant tumor – cells normally found in bone marrow), non-hodgkins lymphoma, cancers of thyroid, breast, esophagus, stomach, pharynx, small intestine, pancreas, bile ducts, gall bladder, salivary glands, urinary bladder, brain, colon, ovary, and liver.

The latency period for exposure to radiation is from 2 to 40 years. Hamilton and Hardy state, "Leukemia was noted in Hiroshima and Nagasaki survivors with a peak incidence of the chronic granulocytic form at five to eight years after the acute exposure in 1945…… Excess myelomas has been noted by Cuzick with a preponderance of cases occuring 15 to 25 years after exposure."

LATEX

Individuals who are allergic to latex rubber can react to starch powders used inside some gloves because of its ability to adsorb proteins from latex and act as an allergen carrier. Abstracts of the papers describing this situation can be obtained from the BWC library. Another abstract reviewed described a case of a cardiac nurse who had an anaphylactic reaction to latex. The gloves she used were labeled as specially formulated for hands allergic to latex, but they were just latex gloves with cornstarch powder.

MAGNISIUM OXIDE FUME

Magnesium oxide fume is a mild irritant of the eyes and nose and may cause metal fume fever, an illness similar to influenza. Exposure to zinc oxide fume causes metal fume fever, but zinc oxide dust is considered a nuisance dust that has little adverse effect on the lungs if exposures are kept under reasonable control.

1. Encyclopedia of Health and Safety Third (Revised) Edition, edited by Dr. Luigi Parmeggiani. International Labour Office, Geneva, 1989.

PAINTING

There have been claims for squamous cell carcinoma of the lung as a result of exposure to various agents in paints filed by lifelong painters. Sometimes the injured worker will mention specific agents such as solvents (e.g., naphtha, methyl ethyl ketone, petroleum distillates, toluene, butyl acetate), crystalline silica, polyurethane, carbon black, and asbestos.

To investigate the carcinogenic potential of such agents, you can obtain information from the publication, "Guide to Occupational Exposure Values - 1999", published by the American Conference of Governmental Industrial Hygienists (ACGIH).[1] Using the various classification systems in this reference, you would find, for the above list, only asbestos is defined as a confirmed human carcinogen. Crystalline silica (quartz) is "probably" and "reasonably anticipated to be" a human carcinogen.

It is not clear how a painter would have been exposed to asbestos and silica. It is not likely painters would be working in construction areas during the application or removal of asbestos. Sanding surfaces covered with paints or plaster containing asbestos is a possibility, but whether such activity actually occurred and the frequency of such occurrence is not known. Exposure to respirable silica dust from sand blasting operations is another possibility, but no information is available on this activity either.

There are several examples of cancer among workers in other trades, including chimney sweeps, mule spinners, and steel mill workers, but it is not clear how a painter would be exposed to agents such as carbon black and polycyclic aromatic hydrocarbons (PAHs) . The International Agency for Research on Cancer (L&RC) has not classified carbon black regarding its carcinogenicity to humans. The National Institute for Occupational Safety and Health (NIOSH) considers carbon black a potential carcinogen only when PAHs are present.[2] In paint, carbon black is added as a pigment. The potential for a painter's exposure to PAHs from carbon black, when used in paint as a pigment, is questionable.

It should be kept in mind the list of agents provided by the injured worker may not be complete. In the above example, the painter could also have been exposed to lead chromate and/or zinc chromate, both of which were used extensively (and are still used) in paints as pigments and corrosion control agents. The injured worker in this example may not have realized the paint he used contained chromates. Lead chromate and zinc chromate are considered carcinogenic to humans by a number of agencies.

In Occupational Medicine Principles and Practical Applications, contributing authors Axelson and Hogstedt stated studies have shown excess respiratory system and stomach cancer mortality among painters. These authors explained it is not entirely clear if the indicated cancer hazard among the painters studied resulted from solvent exposure or to other agents such as pigments. They pointed out some asbestos exposure may have occurred from grinding on plaster.[3]

1 "Guide to Occupational Exposure Values - 1999 , American Conference of Governmental Industrial Hygienists, Cincinnati, OH 1999.

2 Documentation of Threshold Limit Values and Biological Exposure Indices, American Conference *of* Governmental Industrial Hygienists, Cincinnati, OH 1999.

3 Axelson, Olav, MD, and Christer Hogstedt, MD, "On the Health Effects of Solvents," in Occupational Medicine Principles and Practical Applications by Carl Zenz, Second Edition, year Book Medical Publishers, *Inc., Chicago*, 1988.

PARAFFIN WAX FUME

The American Conference of Governmental Industrial Hygienists (ACGIH) has established a threshold limit value of 2 milligrams per cubic meter (mg/m3), as an 8-hour time-weighted average (TWA) for paraffin wax fume. In the documentation for this value, the ACGIH reported paraffin wax fume has a low intrinsic toxicity. They cited a 1938 publication which described working around molten parafin as "uncomfortable and nauseating". The ACGIH recommended the TLV-TWA to "minimize the potential for irritation of respiratory passages and other unpleasant effects that can be associated with occupational exposure to the fume of paraffin wax." The ACGIH did not discuss any specific occupational diseases or adverse effects from exposure to paraffin wax fume other than respiratory irritation.[1] The material safety data sheet for the paraffin wax states overexposure to fumes may cause respiratory tract irritation.

1 American Conference of Governmental Industrial Hygienists, Documentation of Threshold Limit Values and Biological Exposure Indices, Sixth Edition, Cincinnati, OH 1993.

PLATING

Plating processes are often highly automated and involve numerous tanks in the following general order: cleaner, caustic, rinse, acid pickle, rinses, heated caustic, rinses, nickel semi-bright, nickel bright, rinses, chrome, rinses. The parts then enter the dryer and are unracked. The parts on the conveyor automatically enter the tanks; there is no manual placement of parts in the tank. The nickel tanks are a brightening dip rather than electroplating tanks, are only slightly heated (800^{o}F), and generate no visible emissions.

Rackers hang the parts to be plated on a conveyor. In the more automated systems the rackers and unrackers do not usually monitor the activity in the tanks themselves. Thus, they are not exposed to the same extent as a plater in a manually operated plating operation would be exposed. The general ventilation may not adequately protect a worker from irritating vapors and mists if the worker would be assigned to work directly at the tanks. This situation could aggravate an existing respiratory condition.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends the use of local exhaust ventilation (collector hoods, ductwork, fans) with specific plating baths and processes.[1] The purpose of such ventilation is to control the generation of acid and alkaline mists and other air contaminants.

Employees may work at a specific plating line, for example, an electroless nickel line for many years, or they may be a floater working on many different lines. Respiratory protection, while sometimes available, is not routinely used by platers. Platers working on manual lines, rather than automated lines, lift containers of parts in and out of the cleaning and plating solutions using hoists. They may rack and unrack parts and apply or remove a masking material. The masking material can be a viscous liquid into which parts are dipped and may contain solvents such as perchloroethylene and VM&P naphtha.

If chemicals are added to the plating baths through an automated piping system, there would be less exposure than if chemicals are manually added. Sometimes, additions are made to tanks from 5-gallon jugs. This may occur several times per day. The jug may have a valve which allows this chemical to drip or flow into the tank so the employee does not have to pour the liquid into the tank. Electroless nickel tanks may be cleaned about once a day and platers may take turns doing this job. This involves pumping the contents of the electroless nickel tank into another tank and pumping diluted nitric acid into the emptied tank.

The ventilation systems provided for plating tanks should be in good condition and visibly removing mists from the tops of the tanks. The exhaust systems, including the components located on the roof, should be inspected daily.

Trace amounts of acid mists, alkaline mists, and ammonia vapor may, on occasion, escape even if extensive ventilation systems are provided. These air contaminants could be irritating to the upper respiratory tract.

Information to obtain for plating claims

- Obtain a copy of the injured worker's job description.
- Which plating lines did the injured worker work on?
- Was he or she always assigned to this position?
- What other kinds of plating lines did the injured worker work at if any?
- List the baths and contents of each bath on these line.
- If available, include a diagram of the shop showing the location of these baths.
- Was local exhaust ventilation provided for the specific tanks where the injured worker worked? Does the company have dates of installation and maintenance records for these systems? Was it functioning well and were preventative maintenance records maintained?
- If general (dilution) ventilation is provided, please describe.
- Did the injured worker relate his/her illness to any specific incident (such as a spill or fan malfunction), work process change, or chemical exposure?
- Did other workers in the area complain of similar symptoms?
- Was the injured worker required to wear respiratory protection as part of his job?
- If so, what kind was provided?
- Were fit tests provided?
- If so, what kind?
- Are these records available?
- During the term of the injured worker's employment did the company sponsor any physical examinations such as chest x-rays, pulmonary function tests, nasal exams, etc.?
- If so, please provide copies of the results or direction as to how these results can be obtained. Was any air sampling conducted to characterize the injured worker's exposure to air contaminants, including organic solvents, silica, acids, alkali , etc.
- If so, please provide copies of the results. (This request includes representative sampling results, even if the injured worker did not personally wear sampling equipment)

1 Industrial Ventilation: A Manual for Recommended Practice 20th Edition, American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio 1988.

2 Industrial Hygiene Aspects of Plant Operations; Vol. 2, Unit Operations and Product Fabrication, edited by Lewis J. Cralley and Lester V. Crailey, Macmillan Publishing Co., New York, 1984.

POLYETHYLENE RESIN

Make sure the information provided by the physician regarding the injured worker's work history is accurate. In one case, a physician reported the injured worker worked with polyurethane products and described the work as melting down plastic resin and pouring it into a plastic bag. The physician suggested toluene diisocyanate (TDI) or trimethylamine (TMA) might be involved. This information was not accurate. Polyurethane resins, TDI and TMA were not used. In this case, the company only processed polyethylene. Melted resin was not poured into a bag, but blown into a film with highly automated equipment. A description of the blown film process can be found in the industrial hygiene text, Industrial Hygiene Aspects of Plant Operations, Vol. 2.

Also helpful is an article published in June, 1994 which describes a study of polyethylene extrusion processes. Several hundred samples were collected in worker breathing zones and from workplace air during the commercial-scale processing of polyethylene resins. The applications sampled included blown film and the analytes included formaldehyde and acrolein, other aldehydes, aromatics, carboxylic acids, ethers, and Ketones.

Of 440 personal exposure and representative workplace samples, only 10 had detectable levels. Only 2 of these 10 were from the blown film application; both of these were area samples. One showed concentrations of acetone at 0.01% of the permissible exposure limit (PEL) and another showed particulate levels were 2% of the PEL. The authors concluded polyethylene extrusion presents minimal hazards in an adequately ventilated environment.

Recent industry sponsored studies of polyethylene processing have shown detectable levels of aldehydes, including formaldehyde and acrolein, and other irritants inside the blown film bubbles during the blown film extrusion of polyethylene, but not in the operators' breathing zones.

Tikuisis, Tony; Michael F_ Phibbs. Kenneth L. Sonnenberg, "Quantitation of Employee Exposure to Volatile Emission Products Generated by Commercial-Scale Processing of Polyethylene," Novacor, June, 1994.

Industrial Hygiene Aspects of Plant Operations, Vol. 2 Unit Operations and Product Fabrication edited by Lewis I Cralley and Lester V. Cralley, Macmillan Publishing Co., New York, 1984.

POTTERIES

The National Institute for Occupational Safety and Health (NIOSH) reports workers in several industries, including ceramics and cement, have more chronic bronchitis than can be accounted for by such factors as age, smoking, and air pollution. NIOSH also reported a higher prevalence of breathlessness, chest illness, and chronic bronchitis among a group of pottery workers, including nonsmokers, who were presumably exposed to silica and glazing materials.[1] However, this reference did not describe the types of jobs studied.

1 Occupational Respiratory Diseases, US Department of Health and Human Services, DHHS (NIOSH) Publication No. 86-102, September, 1986.

REFRACTORY CERAMIC FIBERS

Pulmonary fibrosis has been reported in animal studies where high exposures of refractory ceramic fibers were generated. The manufacturer states x-ray changes indicative of pulmonary fibrosis have not been reported among workers involved in the manufacture of refractory ceramic fibers. Research is continuing in this area. There are also questions about the ability of zirconium compounds (used in refractory cement) to cause lung disease including granulomas and interstitial fibrosis.

SANDBLASTING

The primary hazard in abrasive blasting is dust, particularly the free crystalline silica dust produced in sandblasting. Prolonged inhalation of air containing large quantities of this dust causes silicosis.[1] Chronic silicosis occurs after many years of exposure to relatively low levels of dust. According to Dr. Anthony Seaton, the simple nodular form of the disease is not associated with any symptoms or physical signs. Subjects may complain of cough, sputum, or breathlessness, probably related to accompanying disease of the airways.[2] Occasionally, chronic silicosis is progressive and develops into progressive massive fibrosis, but this is becoming more rare.

Dr. Seaton also describes "accelerated silicosis" as occurring in workers in some occupations, including sandblasting, where exposure to high concentrations of silica over a relatively short period of a few years results in a more rapidly progressive form of the disease. The symptoms are those of the more chronic disease, but clinical and radiographic progression is rapid, producing a more diffuse and irregular fibrosis. Acute silicosis has also been described in subjects exposed to very high concentrations of silica over periods of just a few weeks to four or five years. This disease has also been described in unprotected sandblasters.[2,3]

The next section contains a brief discussion of some problems with the effectiveness of respiratory protection commonly worn by sandblasters. In addition to questions about the effectiveness of the personal protective equipment provided, a sandblaster, remaining in the work area, would be exposed to dust suspended in the air following removal of the hood. The blaster would be exposed to additional dust by shaking or blowing dust off equipment and other surfaces. It is probable, even with the approved respiratory protection equipment provided and a medical surveillance program in place, a sandblaster would still have some exposure to free crystalline silica.

Following a description of control measures including isolation of the operation by the use of abrasive blasting cabinets and the use of airline blasting helmets with scheduled maintenance for all the equipment, the authors of an article on sandblasting in the Encyclopedia of Occupational Health and Safety stated, "in spite of all these precautions, some of the dust will still be inhaled and consequently, medical examination and supervision of the workers is essential." [1]

In an article on silicosis for the NIOSH text, Occupational Respiratory Diseases Dr. John Peters wrote, " Since sandblasting generates so much dust and is so difficult to control, and since other substitute techniques are available, this practice should be prohibited." [3] In some countries the use of sand or other free silica-containing abrasives for abrasive blasting has already been prohibited due to the hazard.

Several authors have discussed the x-ray shadows created by particles of iron oxide retained in the lung, which may be indistinguishable from fibrotic pneumoconiosis.[1,4] According to one reference, the presence of iron oxide in the dust will produce x-ray appearances of pneumoconiosis very quickly and cause confusion.[1] An injured worker could have had some exposure to iron oxide dust generated from sandblasting mild steel products.

1. Encyclopaedia of Occupational Health and Safety, Third (Revised) Edition, edited by Dr. Luigi Parmcggiam, International Labour Organization, Geneva, Switzerland, 1989, pp. 1995 -1998.

2. Occupational Respiratory Diseases U. S. Department of Health and Human Services, DHHS (NIOSH) Publication No. April, 1987.

3. Morgan. W. Keith C., M. D., and Anthony Seaton, M.D., Occupational Lung Diseases, Second Edition, W. B. Saunders Co., 1984.

4. Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by G. J. Hathaway, N. R Proctor, J. P. Hughes, and M. L. Fischman, Van Nostrand Reinhold, New York, 199 1.

TALC

Although talc can contain asbestos and silica, most talc in use today is non-asbestiform and contains less than 1% crystalline silica, according to the manufacturer. Fibrous talc is classified as a fibrogenic dust, associated with nodular fibrosis (rarely conglomerate nodular fibrosis) and causing both restrictive and obstructive types of pulmonary impairment. The chief agent responsible for this fibrosis is thought to be tremolite asbestos. Fibrous talc has also been associated with increased incidence of cancer of the lungs and pleura. Non-fibrous talc is classified as an agent causing benign pneumoconiosis. [1,2]

The contamination of some talcs by asbestos causes confusion as to whether reports of adverse health effects are due to talc or contaminant. High levels of exposure to asbestos dust over long periods of time can result in the development of asbestosis, an interstitial fibrosis generally associated with a restrictive ave of respiratory impairment.[3]

Epidemiology studies of workers exposed to talc have included miners, millers, and rubber workers. Most studies showing an increased incidence of respiratory disease were of miners and millers; pneumoconiosis among users of talc is rarer.[4,5] Fine and Peters reported increased respiratory morbidity among rubber workers exposed to low fibrous content talc. They concluded 40 years of exposure to processing dusts would result in clinically apparent and disabling dyspnea. However, Seaton states this study of rubber workers using talc showed "no convincing evidence of pneumoconiosis.[4] The occupational health literature does not support a cause and effect relationship between talc dust and multiple chemical sensitivity.

1. Encyclopaedia of Occupational Health and Safety, International Labour Office. Geneva, Vol. 1.

2. Occupational Diseases: A Guide to Their Recognition US Dept. of Health. Education, and Welfare, 1977.

3. Occupational Respiratory Diseases US Dept. of Health and Human Services. Cincinnati, 01-L 1986.

4. Occupational Lung Diseases WKC Morgan, M.D.; A- Seaton, M.D., W. B. Saunders Company, 1984.

5. Hogue, WL and Mallette, "A Study of Workers Exposed to Talc and other Dusting Compounds in the Rubber Industry," Journal of Industrial Hygiene and Toxicology, Vol. 3 1, No. 6.

6. Fine, LJ, Peter, JM, Burgess, WA; DiBerardinis, LJ; "Studies of Respiratory Morbidity in Rubber Workers, Part IV: Respiratory Morbidity in Talc Workers," Arch Environ Health- July/August 1976.

TITANIUM TETRACHLORIDE

According to Hawley's Condensed Chemical Dictionary 11th Edition, titanium tetrachloride ($TiC1_4$) is a colorless liquid which fumes strongly when exposed to moist air, forming a dense and persistent white cloud. Concentrated solutions in water are corrosive. $TiC1_4$ is a strong irritant to skin and tissue. $TiC1_4$ is used to produce pure titanium and titanium salts, iridescent effects in glass, smoke screens, titanium pigments, and is used as a polymerization catalyst.[1] There is no entry for $TiC1_4$ in "Guide to Occupational Exposure Values - 1993"', published by the American Conference of Governmental Industrial Hygienists (ACGIH). [2]

1. Hawleys Condensed Chemical Dictionary 11th Edition, revised by N. Irving Sax and Richard J. Lewis, Sr., Van Nostrand Reinhold Co., New York, 1987, p. 1160.

2 "Guide to Occupational Exposure Values - 1993", American Conference of Governmental Industrial Hygienists, Cincinnati, OH 1993.

2, 4 -TOLUENE D111SOCYANATE (TDI) The ability of 2,4-toluene diisocyanate to cause asthma has been well established. It should be noted TDI, at a high enough concentration, can cause bronchospasm and subsequent sensitization in virtually any person. "Susceptibility to TDI-induced asthma does not require a prior history of atopy or allergic conditions, and sensitization may not be especially common in atopics." [1]

Repeated exposure to lower concentrations of TDI may produce the following symptoms in many people: coughing, wheezing, tightness or congestion in the chest, and shortness of breath. These symptoms appear to be related to hypersensitization. Once sensitized, individuals can react to concentrations of 5 parts per billion or less.

1 Proctor and Hughes' Chemical Hazards of the Workplace Third Edition, edited by Gloria J. Hathaway, Nick H. Proctor, James P. Hughes, Michael L. Fischman, Van Nostrand Reinhold, New York, 1991.

TRANSMISSION FLUID

According to the Material Safety Data Sheet (MSDS) for Dextron II, a transmission fluid manufactured by Pennzoil, this product contains 85% - 90% base lubricating oils, 10% -15% detergent/inhibitor system (trade secret), and less than 1% dye (trade secret). The manufacturer cautions users to prevent aerosolization or misting of this product and reports exposures below 5 milligrams per cubic meter appear to be without significant health risk. The manufacturer also reports on rare occasions, prolonged and repeated exposure to oil mist poses a risk of pulmonary disease such as chronic lung inflammation. The MSDS states this condition is usually without symptoms, but common symptoms are cough and shortness of breath.

Mr. Ken Rayford, Toxicologist, with Pennzoil said the main hazard with the product would be dermatitis and the oil would not be considered hazardous unless it was present as oil mist. He said the detergent/inhibitor system was 90% mineral oil, and this system made up 23% of the product. The red dye is present at 0.05%. According to Mr. Rayford, the ingredients of the detergent inhibitor system are "non-hazardous" but could cause allergic skin reactions in some people. He said there was no data suggesting these ingredients were respiratory sensitizers, but they could cause upper respiratory irritation.

WELDING

In general, **welding fumes and gases contain a mixture of bronchial and alveolar irritants which could give rise to obstructive lung disease**, especially in smokers. **The risk depends on the chemical composition of the materials used, the concentrations in the welder's breathing-zone, and the duration of exposure.** There have been numerous reports of chronic respiratory diseases including inflammatory changes of the mucosae, chronic bronchitis, and pneumoconiosis among welders.[1] **Siderosis, a form of pneumoconioses caused by the deposition of iron oxide particles in the lung, is the most common disease associated with welding.** [2]

Iron oxide by itself is not believed to cause fibrosis, but it has been shown to produce nodular densities which cause x-ray shadows that may be indistinguishable from those caused by other fibrotic pneumoconioses. It is not likely, however, that iron oxide would be the only air contaminant in a welder's environment. Welders are often exposed to other dusts, and a mixed-dust pneumoconiosis can occur.[3] In addition to these particulates, other metallic oxides; gases such as nitrogen dioxide, carbon monoxide, and ozone; and other contaminants are produced by welding.

The composition and concentration of the contaminants vary depending on the fluxes, shielding gases, welding rod or wire, surface treatment, metal being welded, etc. Using,, carbon dioxide as a shielding gas, MIG welding of mild steel typically produces iron oxide fumes, fumes of other metals contained in the alloy, ozone, oxides of nitrogen, and carbon monoxide. The amount of fume generated by MIG welding on mild steel can be higher than with some other types of welding, but with reasonable ventilation, the fumes and gases produced should not be hazardous at the breathing-zone.

Some welders who work in confined spaces are exposed to high concentrations of nitrogen dioxide, and some suggest that pulmonary edema may occur. Aluminum welders have also been exposure to increased concentrations of ozone, and while these may cause acute nasal and respiratory symptoms, there is little evidence to conclude that such exposures have a permanent effect. A recent study demonstrated that welders lose more time from work because of respiratory illness than expected (2.3 times). Absences due to other diseases were similar to the general population.

Another text states, "A cross-sectional study reported by Keimig, et. al. (1983), found that welders and controls who smoked had higher frequencies of reported respiratory symptoms (e.g., bronchitis, pneumonia, and cough) than corresponding non-smokers. Although welders who did not smoke reported higher frequencies of symptoms than nonsmoking controls, the differences were statistically significant only for symptoms of increased phlegm and episodes of cough and phlegm. The only statistically significant differences noted in pulmonary function tests were decreases in forced vital capacity at the end of the work shift from non-smoking welders, non-smoking controls and smoking controls. Similar findings were reported for a group of workers engaged in welding of medium- and high-alloy steel. Although no evidence of obstructive disease was found, 7 of 67 persons tested had restrictive lung impairment." Some of the contaminants generated during welding are also found in cigarette smoke.

According to Dr. Keith Morgan, co-author of the text Occupational Lung Diseases, the high incidence of bronchitis among welders is explained by smoking, welders tend to smoke more than the general population.[3] A study of absences attributed to respiratory illness among welders by Fawer et al. confirmed the findings of other studies that have shown that smokers tend to be more affected by welding fumes than non-smokers. The authors concluded that welders have more absences attributed to respiratory diseases, especially lower respiratory disease; this increase is enhanced by smoking. [1,4] Dr. Morgan states "when emphysema is present, a history of smoking invariably CO exists, unless the welder had been acutely exposed to cadmium fumes.[3]

An epidemiologic survey conducted by Emmett in 1976 found a significant increase in prevalence of cough and phlegm production among welders and those exposed to welding fume. There was no significant difference in the prevalence of other chest symptoms of major respiratory illness, of abnormalities on physical examination of the respiratory system and, of chest x-rays when this group was compared to the control group. The group exposed to welding fume, however, had poorer pulmonary function test results than the control group.[1]

1. Zakhari, Samir and John Strange, Effects of Welding on Health, M, prepared for Safety and Health Committee, American Welding Society, Miami, FL, 1980.

2. Effects of Welding on Health, prepared for Safety and Health Committee, American Welding Society, Miami, FL, 1980.

3. Occupational Lung Diseases WKC Morgan, M.D.; A Seaton, M.D.; W.B. Saunders Company, 1984.

4. Fawer, R_ F., A. Ward Gardner, and D. Oakes, "Absences attributed to respiratory diseases in welders," British Joumal of Industrial Medicine 1982-,39: 149-152.

5. Developments in Occupational Medicine ed. by Carl Zenz, M. D. Year Book Medical Publishers. Inc., Chicago, 1980

ZIRCONIUM

The American Conference of Governmental Industrial Hygienists (ACGIH) established a Threshold Limit Value (TLV) of 2 milligrams per cubic meter of air for zirconium. In their Documentation for the TLVs, the ACGIH did not mention any sarcoidosis-type pulmonary illness as resulting from inhalation of zirconium compounds. The ACGIH did report skin granulomas of an allergic nature from the use of deodorants or poison-ivy remedies containing zirconium lactate or zirconium dioxide. In a chapter in the NIOSH text Occupational Respiratory Diseases Dr. Stuart Brooks suggests zirconium compounds might cause chest x-ray opacities and perhaps diffuse granulomatous pneumonitis and interstitial fibrosis.[1] However, the authors of Occupational Lung Diseases report, "The weight of evidence is against the association of zirconium with the development of pneumoconiosis in man, except under rare circumstances. "[2]

1. Occupational Rest3iratory Diseases, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 86-102.

2. Morgan. W. Keith C., and Anthony Seaton, Occupational Lung Diseases, W. B. Saunders, Philadelphia. 1984.

ADDITIONAL INFORMATION

MATERIAL SAFETY DATA SHEETS

It is very important when reviewing MSDSs to find out if the claimant used the particular product for which the MSDS has been provided. Sometimes the company, but more often the claimant will provide a copy of MSDSs for all the products used in their department. This does not mean the individual was exposed to all the ingredients listed on the MSDS or experienced all or any of the symptoms listed in the discussion of health hazards. You should request an industrial hygienist to help you interpret how the information on the MSDS related to the claim you are investigating.

RESPIRATORY PROTECTION

The use of such a mask does not eliminate the possibility of exposure. It was common in the past for employers to provide dust masks which were not protective against toxic dusts, such as silica. Often the masks were provided with no attempts to ensure proper fit. Training, storage, and handling procedures were often inadequate and lessened any protection provided.

Some questions to ask concerning the effectiveness of supplied air respirators, such as worn typically by sandblasters, are:
Was an approved supplied-air abrasive blasting hood worn?
Was the helmet equipped with "turtleneck-type" collar and shroud to minimize dust leakage inside the equipment?
Was the shroud in good condition?
Upon inspection, if appropriate, was the inside of the helmet clean, not gritty?
Was the air intake located away from the blasting area?
Was the air filtered for oil, grease, and particulates?
Was a carbon monoxide alarm provided?
Did the sandblaster adjust the flow of air inside the hood for comfort?

An article in the Journal of Occupational Medicine raises some serious questions about the effectiveness of supplied-air respirators. These respirators are routinely worn by abrasive blasters or sandblasters, who wiI1 frequently adjust the flow rate of air to an air-supplied hood for comfort. Problems are created when not enough air flows into the respirator. The authors recommend against the practice of adjusting the flow rate to a comfortable level.

Glindmeyer, Henry W. and Yehia Y. Harnmad, Contributing Factors to Sandblasters' Silicosis: Inadequate Respiratory Protection Equipment and Standards, Journal of Occupational Medicine Vol. 30, No. 12, 12/88.

SMOKING

Smoking is a very important factor in many occupational lung diseases. Smoking hinders the lungs' natural ability to clear themselves. This allows more particles to stay longer, increasing the opportunity for lung damage. One author reports 75% of all chronic bronchitis and 80% of all emphysema as associated with smoking.

The National Institute for Occupational Safety and Health (NIOSH) lists several agents known to cause chronic obstructive lung disease.[2] This reference also states:

"Since chronic bronchitis and emphysema result from the inhalation of environmental agents associated with particles, these disorders could be prevented by reducing inhalational exposures. The major exposure to reduce is mainline cigarette smoke. A possible etiologic role of viruses in chronic bronchitis, or the possibility that emphysema may be partly due to a failure of antiprotease defenses or to faulty developmental alveolarization, are not to be ignored. But without reduction of the particulate burden of cigarette smoke (2 billion particles per milliliter per puff), prevention of all but the most enormous environmental exposures is likely to have a small effect upon these diseases. However, operations in many industries, such as the rock digging and crushing involved in obtaining coal, ore, paving, and building material and smelting, evolved without regard to minimizing generation of dust. Such exposure was regarded only as a nuisance dust until studies showed excessive prevalence of chronic bronchitis. " [2]

The American Lung Association (ALA) states, "Cigarette smoking is the most important cause of chronic obstructive bronchopulmonary disease (COPD) in the United States. It increases the risk of dying from pulmonary emphysema and chronic bronchitis. Smokers show an increased prevalence of respiratory symptoms, including cough, sputum production, and breathlessness, when compared with non-smokers. For the bulk of the population of the US, the importance of cigarette smoking (as a cause of COPD) is much greater than that of atmospheric pollution or occupational exposure." [3]

In a more recent publication, the ALA states, "Cigarette smoking causes lung disease by itself without any help from other lung hazards. But when smokers are also exposed to occupational lung hazards, their protective mechanisms are already handicapped and disease develops more rapidly and progresses more quickly than in nonsmokers." [4]

The following discussion of smoking and lung cancer is from the same reference: "The role of cigarette smoking in causing lung cancer cannot be overstated. Alone and unaided cigarette smoke is a potent cause of lung cancer and cancer of other sites. When the effect of cigarette smoke is combined with the effects of occupational carcinogens, the incidence of lung cancer rises dramatically. In several of the jobs, trades, and professions found to be associated with a high risk of lung cancer, there is considerable debate among experts as to whether the occupational carcinogen can, in fact, be proved to cause lung cancer in the absence of cigarette smoke as a co-carcinogen.[1,4]

NIOSH states, "The overwhelming majority of lung cancers occur in smokers, and in the United States squamous cell carcinoma (non-small cell) is known to be the most prevalent

histologicial type of tumor in males followed by adenocarcinoma, oat cell carcinoma, and large cell carcinoma, respectively. Histologically, lung cancers can be divided into four major categories: squamous cell carcinoma, small cell carcinoma, adenocarcinoma and large cell carcinoma. All types of lung tumor show a dose-response relationship with cigarette smoking. Several studies indicate this effect is greatest for squamous cell carcinomas. " [2]

The American Cancer Society (ACS) notes lung cancer rates are insignificant when smoking factors are removed, even when occupational factors are considered. [5] The ACS also notes the single, most prevalent and important factor in cancer of all types is age. The longer you live, the more likely you are to get cancer.

1. Patty's Industrial Hygiene and Toxicology Vol. 3A, 2nd Edition, cd. by Lewis J. Cralley and Lester V. Cralley, John Wiley & Sons, New York, 1985, pp. 29, 47.

2. Occupational Respiratory Diseases. US Dept. of Health and Human Services, DHHS(NIOSH) Publication No. 86-102, September, 1986.

3. Chronic Obstructive Pulmonary Disease American Lung Association, N. Y., 1977.

4. Occupational Lung Diseases, An Introduction, American Lung Association, 1983.

5. Ca-A Cancer Journal for Clinicians Vol. 35, No. 1, American Cancer Society, N. Y., Jan./Feb., 1985.

INTRODUCTION TO THE
CHEMICAL SUBSTANCES

Threshold Limit Values (TLVs) refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse health effects. Because of wide variation in individual susceptibility, however, a small percentage of workers may experience discomfort from some substances at concentrations at or below the threshold limit; a smaller percentage may be affected more seriously by aggravation of a pre-existing condition or by development of an occupational illness. Smoking of tobacco is harmful for several reasons. Smoking may act to enhance the biological effects of chemicals encountered in the workplace and may reduce the body's defense mechanisms against toxic substances.

Individuals may also be hypersusceptible or otherwise unusually responsive to some industrial chemicals because of genetic factors, age, personal habits (e.g., smoking, alcohol, or other drugs), medication, or previous exposures. Such workers may not be adequately protected from adverse health effects from certain chemicals at concentrations at or below the threshold limits. An occupational physician should evaluate the extent to which such workers require additional protection.

TLVs are based an available information from industrial experience; from experimental human and animal studies; and when possible, from a combination of the three. The basis on which the values are established may differ from substance to substance; protection against impairment of health may be a guiding factor for some, whereas reasonable freedom from irritation, narcosis, nuisance, or other forms of stress may form the basis for others. Health impairments considered include those that shorten life expectancy, compromise physiological function, impair the capability for resisting other toxic substances or disease processes, or adversely affect reproductive function or developmental processes.

The amount and nature of the information available for establishing a TLV varies from substance to substance; consequently the precision of the estimated TLV is also subject to variation and the latest TLV Documentation should be consulted in order to assess the extent of the data available for a given substance.

These limits are intended for use in the practice of industrial hygiene as guidelines of recommendations in the control of potential health hazards and for no other use. e.g., in the evaluation or control of community air pollution nuisances; in estimating the toxic potential of continuous uninterrupted exposures or other extended work periods; as proof or disproof of an existing disease or physical condition; or adoption or use by countries whose working conditions or cultures differ from those in the United Slates of America and where substances and processes differ. These limits are *not* **line lines between safe and dangerous concentration not are they a relative index of toxicity. They should** ***not*** **be used by anyone untrained in the discipline of industrial hygiene.**

The TLVs as issued by the American Conference of Governmental Industrial Hygienists (ACGIH) are recommendations and should be used as guidelines for good practices. In spite of the tact that serious injury is not believed likely as a result of exposure to the threshold limit concentrations, the best practice is to maintain concentrations of all atmospheric contaminants as low as is practical.

The ACGIH disclaims liability with respect to the use of TLVs.

Notice of Intended Changes. Each year, proposed actions of the Chemical Substances TLV Committee for the forthcoming year are issued in the form of a "Notice of Intended Changes.- This Notice provides an opportunity for comment and *solicits suggestions of substances to be added to the list. The suggestions should be accompanied by substantiating evidence.* The *Notice of Intended Changes" is presented after the Adopted Values in this section. Values listed in parentheses in the "Adopted" list are to be used during the period in which a proposed change for that Value is listed in the Notice of Intended Changes.

Definitions. Three categories of Threshold Limit Values (TLVs) are specified herein as follows:

a) Threshold Limit Value- Time-Weighted Average (TLV-TWA) - the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed. day after day, without adverse effect.

b) Threshold Limit Value-Short-term Exposure Limit (TLV-STEL) - the concentration to which workers can be exposed continuously for a short period of time without suffering from 1) irritation. 2) chronic or irreversible tissue damage, or 3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency, and provided that the daily TLV-TWA is not exceeded. It is not a separate independent exposure limit, rather, it supplements the time-weighted average (TWA) limit **where there are recognized acute effects from a substance whose toxic** effects are primarily of a chronic nature. STELs are recommended only where toxic effects have been reported from high short-term exposures in either humans or animals.

A STEL is defined as a 15-minute TWA exposure which should not be exceeded at any time during a workday even if the 8-hour TWA is within the TLV-TWA. Exposures above the TLV-TWA up to the STEL should not be longer than I5 minutes and should not occur more than four times per day- There should be at least 60 minutes between successive exposures in this range. An averaging period other than 15 minutes may be recommended when this is warranted by observed biological effects.

c) Threshold *Limit Value-Ceiling* (TLV-C) - the concentration that should not be exceeded during any part of the working exposure.

In conventional industrial hygiene practice if instantaneous monitoring is not feasible, then the TLV-C can be assessed by sampling over a 15-minute period except for those substances that may cause immediate irritation when exposures are short.

"Skin" Notation. The designation "skin" in the "Notations" column refers to the potential significant contribution to the overall exposure by the cutaneous route, including mucous membranes and the eyes, either by contact with vapors or, of probable greater significance by direct skin contact with the substance. Vehicles present in solutions or mixtures can also significantly enhance potential skin absorption. It should be noted that while some materials are capable of causing irritation, dermatitis, and sensitization in workers, these properties are not considered relevant when assigning a skin notation. It should be noted, however, that the development of a dermatological condition can significantly affect the potential for dermal absorption.

While limited quantitative data currently exist with regard to skin absorption of gases, vapors, and liquids *by* workers, the Chemical Substances TLV Committee recommends that the integration of data from acute dermal studies and repeated dose dermal studies in animals and/or humans, along with the ability of the chemical to be absorbed, be used in deciding on the appropriateness of the skin notation. In general, available data which suggest that the potential for absorption via the hands/forearms during the workday could be significant, especially for chemicals with lower TLVs could justify a skin notation. From acute animal toxicity data, materials having a relatively low dermal LD_{50} (1000 mg/kg of body weight or less) would be given a skin notation. Where repeated dermal application studies have shown significant systemic effects following treatment, a skin notation would be considered, When chemicals penetrate the skin easily (higher octanol-water partition coefficients) and where extrapolations of systemic effects from other routes of exposure suggest dermal absorption may be important in the expressed toxicity, a skin notation should be considered.

Substances having a skin notation and a low TLV may present special problems for operations involving high airborne concentrations of the material, particularly under conditions where significant areas of the skin are exposed for a long period of time. Under these conditions, special precautions to significantly reduce or preclude skin contact may be required.

Biological monitoring should be considered to determine the relative contribution of exposure via the dermal route to the total dose. The TLV/BEI Booklet contains a number at adopted Biological Exposure Indices, which provide an additional tool when assessing the worker's total exposure to selected materials. For additional information, refer to "Dermal Absorption" in the "Introduction to the Biological Exposure Indices," *Documentation of Threshold Limit Values and Biological Exposure Indices,* and to Leung and Paustenbach. [(2)]

Use of the skin designation is intended to alert the reader that air sampling alone is insufficient to accurately quantitate exposure and that measures to prevent significant cutaneous absorption may be required.

Mixtures. Special consideration should be given also to the application of the TLVs in assessing the health hazards that may be associated with exposure to mixtures of two or more substances. A brief discussion of basic considerations involved in developing TLVs for mixtures and methods for their development, amplified by specific examples, are given in Appendix C.

Respirable and Inhalable Dust. For solid substances and liquified mists, TLVs are expressed in terms of inhalable dust, except where the term 'respirable dust' is used. See Appendix D, Particle Size-Selective Sampling Criteria for Airborne Particulate Matter, for the definition of respirable dust (respirable particulate mass).

Particulates Not Otherwise Classified (PNOC). There are many substances on the TLV list and many more that are not on the list, for which there is no evidence of specific toxic effects. Those that are particulates have frequently been called "nuisance dusts." Although these materials may not cause fibrosis or systemic effects, they are not biologically inert. At high concentrations, otherwise nontoxic dusts have been associated with the occasionally fatal condition known as alveolar proteinosis. At lower concentrations, they can inhibit the clearance of toxic particulates from the lung by decreasing the mobility of the alveolar macrophages. Accordingly, the Chemical Substances TLV Committee recommends the use of the term "Particulates (Insoluble) Not Otherwise Specified (PNOS)" to emphasize that all materials are potentially toxic and to avoid the implication that these materials are harmless at all exposure concentrations. Particulates identified under the PNOS heading are those containing no asbestos and *<1%* crystalline silica. To recognize the adverse effects of exposure to otherwise nontoxic particulate matter, a TLV-TWA of 10 mg/m3 for inhalable particulate and a TLV-TWA of 3 mg/m3 for respirable particulate have been established and are included in the main TLV list. Refer to the PNOS documentation for a complete discussion of this subject.

Simple Asphyxiants- "Inert" Gases or Vapors. A number of gases and vapors, when present in high concentrations in air, act primarily as simple asphyxiants without other significant physiologic effects. A TLV may not be recommended for each simple asphyxiant because the limiting factor is the available oxygen. The minimal oxygen content should be 18% by volume under normal atmospheric pressure (equivalent to a partial pressure, pO2 of 135 torr). Atmospheres deficient in 02 do not provide adequate warning and most Simple asphyxiants are odorless. Several simple asphyxiants present an explosion hazard. Account should be taken of this factor in limiting the concentration of the asphyxiant.

Biological Exposure Indices (BE1s). The note "BEI" is listed in the "Notations" column when a BEI is also recommended for the substance listed. Biological monitoring should be instituted for such substances to evaluate the total exposure from all sources, including dermal, ingestion, or non-occupational. See the BEI section in this Book and the Documentation of the TLVs and BEIs for the substance.

Physical Factors. It is recognized that such physical factors as heat, ultraviolet and ionizing radiation, humidity, abnormal pressure (altitude), and the like may place added stress on the body so that the effects from exposure at a TLV may be altered. Most of these stresses act adversely to increase the toxic response of a substance. *Although most TLVs have built-in uncertainty factors to guard against adverse effects to moderate deviations* from normal environments, the safety factors of most substances are not of such a magnitude as to take care of gross deviations. For example, continuous, heavy work at temperatures above 25°C WBGT, *or* overtime extending the workweek more than 25%, might be considered gross deviations. In such instances, judgment must be exercised in the proper adjustments of the TLVs.

TLVs exist for some certain substances of biological origin, and for cotton dust, which is at least in part biological. There are no TLVs for interpreting environmental measurements of a) total culturable or countable bioaerosols (e.g., total bacteria or fungi); b) specific culturable or countable bioaerosols (e.g., *Aspergillus fumigatus);*c) infectious agents (e.g., *Legionella pneumophila* or *Mycobacterium tuberculosis);* or d) assayable biological contaminants (e.g., endotoxin, mycotoxin, antigens, or microbial volatile organic compounds) for the following reasons.

A. Total culterable or countable bioaerosols. Culterable bioaerosols are those bacteria and fungi that can be grown in laboratory culture. Such results are reported as the number of colony-forming units (CFU). Countable bioaerosols are those pollen grains, fungal spores, bacterial cells, and other material that can be identified and counted by microscope. A general TLV for culterable or countable bioaerosol concentrations is not scientifically supportable because of the following:

1. Culturable organisms or countable biological particles do not comprise a single entity, i.e., bioaerosols in occupational settings are generally complex mixtures of many different microbial, animal, and plant particles.
2. Human responses to bioaerosols range from innocuous effects to serious disease and depend on the specific agent and susceptibility factors within the person.
3. Measured concentrations of culturable and countable bioaerosols are dependent on the method of sample collection and analysis. It is not possible to collect and evaluate all of these bioaerosol components using a single sampling method.
4. At present, information relating culterable or countable bioaerosol concentrations to health effects is generally insufficient to describe exposure-response relationships.

B. Specific culturable or countable bioaerosols other than infectious agents. Specific TLVs for individual culturable or countable bioaerosols have not been established to prevent hypersensitivity, irritant, or toxic responses. At present, information relating culturable or countable bioaerosol concentrations to health effects consists largely of case reports containing only qualitative exposure assessments. The data available are generally insufficient to describe exposure--response relationships. Reasons for the absence of good epidemiologic data on exposure-response relationships include:

1. Most data on concentrations of specific bioaerosols are derived from indicator measurements rather than from measurement of actual effector agents. For example, culturable fungi are used to represent exposure to allergens. In addition, most measurements are either from reservoir or from ambient air samples. These approaches are unlikely to accurately represent human exposure to actual effector agents.
2. The components and concentrations of bioaerosols vary widely. The most commonly used air sampling devices collect only grab samples over short periods of time and these single samples may not represent human exposure. Short-term grab samples may contain an amount of a particular bioaerosol that is orders of magnitude higher or lower than the average environmental concentration. Some organisms release aerosols as “concentration bursts" and can be detected only rarely using grab samples. Yet, such episodic bioaerosols may produce significant health effects.
3. In studies of single workplaces, the number of persons affected by exposure to biological agents may be small if contamination is localized, thereby affecting only a fraction of the building occupants. However, data from different studies can seldom be combined to reach meaningful numbers of test subjects because the specific types of biological agents responsible for bioaerosol-related illnesses are diverse and often differ from study to study. These factors contribute to the low statistical power common in evaluations of cause-effect relationships between exposures to specific biological agents and building-related health complaints.

GLOSSARY

ACGIH - American Conference of Governmental Industrial Hygienists. This is a trade association of industrial hygienists that has recommended exposure limits for chemicals and noise called threshold limit values (TLVs) which they update on a yearly basis. In addition, they make recommendations on how to minimize occupational exposures through use of such things as ventilation controls and biological monitoring.

Acute - Of a short term, immediate nature; e. g., acute exposure, acute health effect.

Asbestos Bodies - Asbestos fibers found in the lung that have been coated with iron-protein complexes.

Asbestos Related Disease - Any of a number of diseases associated with exposure to asbestos fibers; e. g., asbestosis, lung cancer, mesothelioma.

Atopic - Allergic

B-Reader - An individual specifically trained to read chest X-rays to diagnose pneumoconiosis type lung diseases; e. g., asbestosis, silicosis.

Chronic - Of a longer term, slower in development; i.e., chronic exposure, chronic health effect.

Encephalopathy - Any degenerative disease of the brain. Lead encephalopathy is a brain disorder caused by lead poisoning, also call saturnine encephalopathy.

Epidemiology - The study of health effects on specific human populations.

Exposure Limits - Any of a set of permissible or recommended limits established to prevent or minimize harmful physical effects. OSHA's permissible exposure limit (PEL) is required by law not to be exceeded. The ACGIH TLV and the NIOSH REL (recommended exposure limit) are recommended levels. The PEL, TLV and REL are the three most commonly cited exposure limits.

Ferruainous Bodies ' - Minerals found in the lung, e. g., carbon, ceramic aluminum silicate fibers and fiberglass, that have a similar type coating as an asbestos body (iron-protein complexes).

Fumes - Very small airborne particles formed by the evaporation and oxidation of metals, most commonly used in reference to welding operations.

General Ventilation - The use of ceiling and/or wall fans, or open doors to provide a general dilution effect of chemicals in a work area.

HVAC - Heating, Ventilation, Air Conditioning

Industrial Hygiene Data - Typically, this phrase refers to the findings of an industrial hygiene survey, which might include air monitoring or noise monitoring results. Personal sampling is usually done, with the collection of the sample taken from the breathing zone (a restricted area around the head of the individual) for chemical monitoring and at shoulder level for noise monitoring. Long term sampling can be done, which means as much of the worksheet is sampled as possible. Short term sampling covers a represent instantaneous or short term exposures. Area sampling is sometimes conducted to evaluate a background level of a particular chemical in a given area. By OSHA law, records of industrial hygiene sampling must be retained by the employer for thirty years.

Latency Period - The period of time between the exposure to a chemical and the initiation of response or time when response is commonly first detected.

Local Exhaust Ventilation - The placement of a ventilation unit at the immediate source of emission of a particular chemical contaminant. The purpose of local ventilation is to prevent or minimize chemical exposures by removing the hazard before it gets into the breathing zone of the worker.

MSDS' - Material Safety Data Sheet. This sheet provides specific information of the ingredients of a chemical mixture, as well as information on health effects, physical characteristics, precautions to be taken during use, in a spill, -etc. The Hazard Communication Standard of 1986 made it a requirement that employers have a copy of an updated MSDS for each chemical they have on site.

Nephropathy - Disease of the kidneys

NIOSH - National Institute for Occupational Safety and Health. This non-enforcement governmental agency primarily conducts research resulting in the publication of literature on occupational safety and health issues. NIOSH also approves certain personal protective equipment, such as respirators.

Odor Threshold - The level at which a particular chemical can be detected by one's sense of smell. However, this level has no direct relationship with the chemical's toxicity.

Pack Years - A quantitative measure of an individual's smoking history, determined by multiplying the number of packs smoked per day by the number of years the person smoked.

PEL - Permissible exposure limit.

Pneumoconiosis - Lung disease resulting from exposure to certain types of dust.

REL - Recommended exposure limit.

Respirable Dust - Particles in the size range that permits them to penetrate deep into the lungs upon inhalation.

Results Tables - A table which fists the level of exposure for the chemical or noise.

Sensitizer - A material that on first exposure causes little or no reaction in man or test animals, but which on repeated exposure may cause a marked response not necessarily limited to the contact site. Skin sensitization is the most common form. Respiratory sensitization to a few chemicals is also known to occur. (Example: Diisocyanates can cause skin or respiratory sensitization.)

Synergistic Effect - When the exposure to two or more substances results in an effect on the body that is greater than the additive effect of each substance alone.

TLV - Threshold limit value.

Toxicity - The ability of a substance to cause a harmful effect on a biologic mechanism and the condition under which this occurs.

ZPP - Zinc Protoporphorin, a compound found in the blood that is measured to determine whether an individual has had an excessive exposure to lead.