Chapter 46
Mineral Dusts: Asbestos, Silica, Coal, Manufactured Fibers

Gregory R Wagner, Frank J Hearl

INTRODUCTION

Mineral is the term traditionally applied to naturally occurring inorganic substances having definable and consistent physical properties and chemical composition. By conventional contemporary usage, mineral also refers to similar substances of organic origin, such as coal, as well as manufactured materials of mineral origin, such as ceramic fibers or slag wool. Dusts generated in the mining, milling, manufacture, and use of minerals have long been recognized as causes of serious lung diseases. This chapter provides an overview of information useful in the recognition and prevention of diseases related to mineral dust exposure. As with any agents causing chronic disease, hazard recognition and environmental surveillance and control are key to disease prevention and are thus a primary focus of this section.

ASBESTOS

Exposure settings

Asbestos is a generic name used in referring to a group of fibrous minerals that have exceptional resistance to degradation by heat, acids, bases, or solvents. Asbestos is defined in US regulations as the minerals chrysotile, crocidolite, amosite, tremolite asbestos, actinolite asbestos, and anthophyllite asbestos, although there are other hazardous fibrous minerals such as erionite, richterite, and winchite with similar characteristics.

Asbestos fibers can be classified based on their physical and chemical structures as serpentine (curved) or amphiboles (straight and needle-like). The most commonly used asbestos fiber is chrysotile asbestos, which now comprises over 95% of worldwide production, and is the only serpentine asbestos of importance. Amphibole asbestos, used more in the past, includes commercially used amosite (mined in South Africa) and crocidolite, and non-commercially used anthophyllite, tremolite, and actinolite, which can potentially contaminate other mineral ores or cause environmental rather than occupational asbestos-related disease. The relative potency of the different forms of asbestos in causing both malignant and non-malignant asbestos-related conditions has been a source of controversy, complicated in part by the fact that each form is rarely pure, but may contain small amounts of other fiber types; anyone who has worked with asbestos for many years likely was exposed to several fiber types. Chrysotile is generally considered less potent for mesothelioma induction than certain amphiboles, but still may be a cause. Crocidolite is considered the most potent fiber for mesothelioma.

The asbestos minerals are not combustible and have a high melting point and low thermal and electrical conductivity. These and other useful properties resulted in the development of thousands of commercial uses for asbestos. Asbestos has been in widespread use for more than a century. Before 1900, less than 10,000 metric tons per year were used in the US. The use of asbestos for products including clutch and brake linings, packing and gaskets, asbestos flooring, and thermal and electrical insulation, led to increased demand. By 1950, the annual US demand for asbestos grew to over 660,000 metric tons, and increased to over 801,000 metric tons by 1970.[1-3] Since the early 1970s, regulatory action and liability concerns have reduced or eliminated many uses in the US and elsewhere. Complete or partial asbestos bans are in place in at least 20 countries, and asbestos use will be banned throughout the European Union in 2005.

There are six classes of products in which the use of asbestos is currently banned by the US Environmental Protection Agency (EPA):

- corrugated paper;
- rollboard;
- commercial paper;
- specialty paper;
- flooring felt; and
- new uses of asbestos.

Since the 1970s, the US demand for asbestos products has dropped to the current levels of 14,600 metric tons in 2000.[4]

Recent total US production of asbestos fell from 9550 metric tons in 1996, to an estimated production of 5260 metric tons in 2000. Worldwide production and usage have been relatively stable, declining from 2.1 million metric tons in 1996 to 1.9 million metric tons in 2000. In 2000, the major continuing uses for asbestos were roofing products, gaskets, and friction products. Nevertheless, millions of tons of asbestos insulation remain installed, and removal and disposal create continuing exposure potential. In some situations, asbestos-bearing materials may be sealed with a coating to minimize release of inhalable fibers. This only defers the problem of exposure until

major renovation or demolition is required. Today, most domestic exposure occurs in construction workers exposed to installed asbestos products or during asbestos abatement and removal activities. Exposure may also occur when asbestos is found as a contaminant in other commercially useful minerals, including, talc, clays, vermiculite, magnetite, marble, and metal ores.

The Occupational Safety and Health Administration (OSHA) estimated in its 1994 Asbestos Standard that about 700,000 US workers were potentially exposed to asbestos in general industry and shipyards.[5] Many more have been exposed in the past. Table 46.1 lists occupations where significant asbestos exposure has occurred and may continue to occur. Asbestos products have been so widely used that a complete list of potential exposure settings would include virtually all workplaces.

Acoustic product installers
Asbestos cement product makers and users
Asbestos grout makers and users
Asbestos millboard makers and users
Asbestos millers
Asbestos miners
Asbestos paper makers and users
Asbestos plaster makers and users
Asbestos insulators
Asbestos tile makers and installers
Asphalt mixers
Automobile repair workers
Boiler makers
Beer makers
Brake lining makers
Brake refabricators
Chemical workers
Clay workers
Construction workers
Demolition workers
Electric appliance workers
Electrical equipment workers
Electrical wire makers
Food processing workers
Gasket makers
Glass workers
Iron ore (taconite) miners and millers
Insulation workers
Loggers
Machinery makers
Maintenance and custodial workers
Nursery (agricultural) workers
Oil and gas extraction workers
Paint makers
Petroleum refinery workers
Primary metal industry workers
Plumbers and pipefitters
Railroad repair workers
Roofers
Rubber makers
Reinforced plastics makers
Shipyard workers
Stone workers
Talc miners and workers
Textile workers
Transportation equipment makers and repairers
Transportation workers
Vermiculite miners and workers

Table 46.1 Occupations with potential asbestos exposure

Environmental measurement

The techniques for environmental measurement of asbestos can be divided into two general categories. *Qualitative analysis* determines whether or not asbestos is present in a bulk material and estimates the relative amount and mineral type of asbestos present. Qualitative analysis can be done using polarized light microscopy, X-ray diffraction, or transmission electron microscopy. The results from qualitative analysis are usually given as percent asbestos on a projected area or a weight/weight basis. *Quantitative analysis* counts the number of fibers or structures present in a measured volume of air. Two principal techniques are generally used for quantitative analysis. Light microscopy with phase contrast enhancement is commonly used for the routine quantitative fiber measurements needed for comparison to occupational exposure limits. Transmission electron microscopy may be needed when thin fibers (<0.25 μm) are present. Fibers smaller than 0.25 μm are invisible using light microscopy techniques. When performing quantitative fiber analysis, the associated analytic capabilities of these microscopes limit the level of qualitative information (fiber-type identification) that can be gathered on individual fibers. Results from quantitative analysis are usually given as fibers per cubic centimeter or structures per cubic centimeter.[6,7]

Clinical effects

Specific asbestos-related diseases are discussed in detail in Chapters 19.8 and 30.2.

Acute effects

Aside from a non-specific mild respiratory and skin irritant effect, no clinically observable acute effects of asbestos exposure have been documented. Asbestos warts or corns have been described within weeks or months of initial skin exposure to asbestos fibers. These are small nodular lesions, sometimes on an inflammatory base, resulting from a granulomatous reaction to the presence of a foreign body.

Chronic effects

Non-malignant diseases such as pulmonary fibrosis, pleural fibrosis, benign pleural effusion, and chronic bronchitis, as well as a variety of malignancies, including cancers of the respiratory tract, malignant mesothelioma of the pleura or peritoneum, and gastrointestinal cancers, can result from occupational or environmental asbestos exposure. All are chronic conditions of insidious onset occurring after latencies of one or more decades. Each of the non-malignant or malignant effects of asbestos exposure may occur independently of the others. With the exceptions noted below, none is pathognomonic for asbestos dust exposure. Clinical manifestations are non-specific.

Pulmonary fibrosis may be recognized as a result of mandated surveillance of current asbestos workers or through diagnostic evaluation of symptoms of persistent cough and exertional dyspnea of insidious onset. The chest

X-ray demonstrates small irregular opacities at the lung bases. Alternatively, fibrosis may be noted on pathologic examination of lung tissue removed during evaluation of a suspicious or malignant lung lesion, even when the preoperative chest X-ray shows no interstitial changes.[8]

Pleural fibrosis, in the form of discrete plaques or diffuse thickening of the pleura, can occur independently of pulmonary fibrosis. One or more discrete pleural plaques may be recognized along the diaphragm or along the lateral chest walls. Bilateral plaques and plaques with visible calcification seen in individuals with a credible history of asbestos exposure and sufficient latency are presumed to have resulted from the asbestos exposure, although unilateral plaques may result from asbestos exposure, and noncalcified plaques occur more commonly than calcified ones. Pleural plaques undetected by conventional radiographs are often visualized using computed tomography (CT). Diffuse pleural fibrosis often involving blunting or obliteration of the costo-phrenic angle on chest X-ray is also seen as a result of asbestos exposure. This may be associated with significant pulmonary function abnormality, although mild pulmonary dysfunction can also be associated with the presence of pleural plaques.[9]

Benign pleural effusions, lasting weeks to months before spontaneous resolution, occur in asbestos-exposed workers, often within the first two decades following first exposure. These are sterile and without malignant cells. Associated symptoms may be minimal. There are suggestive data associating the eventual development of extensive diffuse pleural fibrosis with antecedent pleural effusions. Chronic bronchitis, persistent cough with phlegm, in general occurs more often in people working in dust than in those who do not, when compared to others with similar smoking habits. This observation has been confirmed in some cohorts of asbestos workers.

Restrictive physiology with a reduced carbon monoxide diffusion capacity, characteristic of diffuse interstitial fibrosis, is typically seen in patients with asbestosis. Inflammation and fibrosis of the respiratory bronchioles can also occur as a manifestation of asbestos exposure, even in the absence of asbestosis visible on chest X-ray. Mild pure airflow obstruction from asbestos exposure (demonstrable as an increased loss of FEV_1 in the absence of other adverse exposures such as cigarette smoke) has been found in some asbestos-exposed workers. Some have observed FEV_1 deficits in asbestos workers with pleural fibrosis. Others have documented progressive loss of FEV_1 in asbestos-exposed workers as they develop symptoms.[10] Higher degrees of lung function loss are associated with increasing pulmonary function deficits.[11]

Respiratory tract cancers are the most common cancers associated with occupational asbestos exposure. These are pathologically indistinguishable from other respiratory tract cancers. Cancers of the lung and larynx are seen more frequently in workers occupationally exposed to asbestos than in others with comparable smoking habits. Latency averages about 25 years from first asbestos exposure. Combined exposure to asbestos and cigarette smoke puts workers at extremely high risk for development of respiratory tract cancers. There is evidence that smoking cessation results in diminished lung cancer risk over time in individuals who have had prior asbestos exposure. Some have asserted that asbestos only causes lung cancer in workers who have already developed pulmonary fibrosis, although evidence for this is limited and increased risk of lung cancer with or without visible fibrosis is well documented in a number of occupational groups.[12] Since both the development of fibrosis and the development of lung cancer are dose-related phenomena, unambiguous resolution of this controversy is challenging.

Malignant mesothelioma of the pleura, peritoneum, or mediastinum in the presence of a credible history of asbestos exposure, occurring after a reasonable latency, are considered 'sentinel health events' presumptively resulting from exposure to the mineral fibers, most often asbestos. Mesothelial cells are thought to be transformed through direct interaction with asbestos fibers. Malignant mesothelioma has been reported with minimal exposure to asbestos; cohorts with high levels of exposure experience high rates of this cancer. Latency between first exposure and mesothelioma development is, on average, longer than that for bronchogenic cancer, averaging 30–35 years. The tumors grow rapidly and are virtually universally fatal, with no effective curative surgical or chemotherapeutic interventions found to date.

Cancers of the gastrointestinal tract, including cancer of the esophagus, stomach, pancreas, colon, and rectum, occur more frequently in most occupational cohorts heavily exposed to asbestos, although the risk is substantially less than for lung cancer.

Other cancers, including certain lymphomas and kidney cancer, have been observed more frequently in some, but not all, asbestos-exposed cohorts. Complex interrelationships between multiple adverse occupational exposures, including asbestos, may in part account for the diversity of malignancies associated with exposure.

Clinical evaluation and management

Clinical management of patients with a history of asbestos exposure requires a combination of medical screening, health counseling, appropriate and timely medical interventions, and emotional support based upon a realistic appraisal of the risks resulting from the exposure. If exposure is continuing, exposure control is critical, and health surveillance consistent with that mandated by the Occupational Safety and Health Administration (OSHA) is warranted. Given current restrictions on asbestos use, exposure has often ceased by the time healthcare is sought. Nevertheless, the biologic persistence of the fibers and the long latency between exposure and disease justify special attention to former asbestos-exposed workers. Also, individuals with asbestos-related diseases may be involved in litigation that may place certain responsibilities on the healthcare provider.

Medical screening involves the performance of tests directed toward the identification of significant diseases or conditions before the diseases become clinically apparent.

Effective treatments or interventions should be available which can improve the outcome of the disease. Screening for disease in an individual is a secondary prevention strategy, distinguishable from public health or workforce surveillance efforts that also involve medical testing. Also, tests done exclusively for screening purposes differ in intent from those performed for diagnostic purposes or to provide information useful in health counseling.[13]

Theoretically, any of the diseases for which asbestos-exposed individuals are at risk would be potential targets for screening in the course of routine medical care. However, most fail to justify the conduct of screening tests. For example, screening for malignant mesothelioma will not result in improved outcome until effective interventions are available. Respiratory tract cancer screening among high-risk groups, using periodic chest X-ray and sputum cytology, has failed to reduce significantly the number of cancer deaths in those screened compared to others receiving routine care. Newer approaches to lung cancer screening using low-dose spiral CT scanning are under evaluation and have demonstrated promising results in selected circumstances.[14]

No effective intervention has been identified to modify the course of pulmonary or pleural fibrosis resulting from asbestos exposures which are no longer continuing. However, the identification of one or more of these conditions combined with appropriate health counseling may help motivate patients to control or eliminate exposure to other pulmonary toxins, particularly tobacco smoke.

When exposure is continuing, however, the presence of abnormalities on chest X-ray justifies recommendations to increase the level of respiratory protection or eliminate the exposure altogether, since the development of fibrosis appears to be dose related and continuing exposure might cause more rapid deterioration. Thus, periodic chest X-rays are indicated for active asbestos workers. Also, when there is ongoing exposure, the ability to use respiratory protective equipment should be monitored closely by periodic examinations including pulmonary function testing.

Gastrointestinal cancer screening, using tests for occult blood in the stool and periodic sigmoidoscopy or colonoscopy, has been recommended by the US Preventive Services Task Force, the American Cancer Society, and others, as being effective in the general population. Given the increased risk of gastrointestinal cancer in asbestos-exposed individuals, this practice should be encouraged. No studies have yet shown benefit from either earlier initiation or more frequent performance of these screening procedures because of increased risk in this group.

Health counseling for patients currently or formerly exposed to asbestos must be directed toward control or elimination of exposure to all current pulmonary hazards, particularly asbestos dust and tobacco smoke. The importance of smoking cessation in asbestos-exposed patients, whether or not they have manifested any of the stigmata of asbestos-related diseases, cannot be overemphasized. The combination of asbestos exposure and tobacco smoke is far worse than either alone. During the decade following smoking cessation, lung cancer risk gradually drops but remains significantly elevated as a result of the asbestos exposure. No healthcare encounter with a current or former asbestos worker should omit inquiry into tobacco use and reinforcement of a smoking cessation message to all current tobacco users.

To date, evidence that dietary modification may diminish cancer risk is unconvincing; however, research into this important question continues. Studies of former asbestos workers with X-ray abnormalities have failed to provide evidence justifying recommendations of specific dietary supplements or other dietary modifications for these patients. Given the risk status of exposed individuals and the limited number of interventions available, prudent advice based on the best available current recommendations from the National Cancer Institute should be offered.

Medical interventions depend upon the diseases manifested by the patient and do not differ appreciably from those that should be offered others with comparable diseases with other etiologies. For example, asbestos-associated malignancies have not been shown to respond any better or worse to conventional surgical techniques or chemotherapy than cancers of unknown or other etiologies. The course of pulmonary fibrosis caused by asbestos exposure is generally slowly progressive to indolent and has not been shown to be affected by any particular intervention. Some have tried decortication procedures to relieve restriction caused by the rare cases of massive diffuse pleural thickening. Such treatments must depend upon the particular circumstances of the individual patient.

Medical therapies should be directed toward early identification and treatment of infection and relief of any element of bronchoconstriction. Evidence of hypoxemia should be sought and treated in severely impaired patients. Desaturation with exercise or during sleep should stimulate consideration of intermittent or continuous oxygen therapy.

Comprehensive educational and support programs directed toward patients with chronic respiratory impairment and their families have been of benefit to some. These programs often direct attention toward significant changes resulting from the disease, such as the need for environmental controls, the onset of sexual dysfunction, or the discouragement resulting from involvement in protracted and incomprehensible litigation. Voluntary organizations such as the American Lung Association may offer referral to pulmonary rehabilitation programs and patient support groups.

Litigation seeking redress for injury from asbestos and finances to purchase necessary healthcare is common in the US among people with asbestos-associated diseases. Physicians caring for these patients must be prepared to provide timely reports when requested by the patient or the patient's representative. Standard diagnostic criteria may be useful in preparation of such reports.[15] The fact that the patient is involved in litigation should not affect diagnostic or therapeutic decisions. Legal issues are considered in detail in Chapter 56.

Prevention

Control or elimination of exposure to asbestos-containing materials is the only absolutely effective strategy for prevention of asbestos-related diseases. Although substitute materials are being used in many current settings, the asbestos already in place will result in ongoing exposure to maintenance, mechanical, and construction workers for decades to come. Disease prevention in these workers will depend in great measure on recognition of the potential for exposure and strict compliance with health and safety regulations. Comprehensive regulations promulgated under the Occupational Safety and Health Act, intended to prevent asbestos-associated diseases, focus not only on exposure control but also on issues of training and surveillance.

Exposure limits

In the US, occupational exposure is limited to 0.1 fiber/cm^3 as a time-weighted average (TWA) over an 8-hour workday for general industry. There is a 1.0 fiber/cm^3 excursion limit averaged over a 30-minute sampling period. If exposure exceeds either the TWA or the excursion limit during the year, periodic exposure sampling, training, and surveillance are required.[16]

Monitoring

Initial personal sampling is required for all workers who may reasonably be expected to be exposed at or above the TWA and/or excursion exposure limits. Additional monitoring is required with a frequency such that the exposure of the worker is adequately described.

Regulated areas

Respiratory protection is required in areas where exposures exceed the TWA and/or excursion exposure limits. Hazard communications requirements, including the posting of warning signs and other restrictions on access, apply in any area with asbestos-containing materials or potentially asbestos-containing materials.

Control

Engineering and work practice controls are the preferred methods for maintaining exposure levels below the TWA and/or excursion exposure limits.

Respiratory protection

A strict program of respiratory protection using NIOSH-certified respirators, periodic fit testing, and training is mandated. The use of 'disposable' filtering facepiece respirators is not permitted. The employer must provide a tight-fitting positive-pressure respirator that provides adequate protection, instead of any negative-pressure respirator, if the employee chooses to use this type of respirator.

Training

All workers exposed above the TWA and/or excursion exposure limits must be trained initially and at least yearly thereafter. Items to include are:

- the health hazards of asbestos exposure;
- the relationship between asbestos and smoking in producing lung cancer;
- operations which could result in exposure;
- the importance of control procedures;
- proper use and limitations of respirators; and
- details of the medical surveillance program.

Medical surveillance

In the US employers must provide a medical surveillance program for all workers exposed above the TWA and/or excursion exposure limits, at no cost to the workers. A baseline pre-placement examination must include a medical and work history; a complete physical examination with emphasis on the respiratory, cardiovascular, and digestive tract systems; a standardized respiratory questionnaire; a full-sized PA chest radiograph; spirometry including FEV_1 and FVC; and any other tests deemed appropriate by the examining physician.

After the baseline examination, periodic examinations including an abbreviated questionnaire, periodic chest X-ray, and spirometry are mandated. The questionnaire is administered at baseline, then in an abbreviated form annually. Spirometry and a physical examination are performed at baseline, then annually. A chest radiograph must be offered:

- every 5 years for workers under 35 years and/or with less than 10 years since first asbestos exposure;
- every 2 years for workers aged 36–45 with over 10 years since first asbestos exposure; and
- every year for workers over age 45 with over 10 years since first exposure.

Medical examinations and testing are performed or conducted under the supervision of a licensed physician and at a 'reasonable time and place'. Chest X-ray interpretation is done according to the International Labour Office (ILO) classification system by a reader trained, tested, and certified by NIOSH (B reader), a board eligible or certified radiologist, or 'an experienced physician with known expertise in pneumoconioses'. The examining physician is responsible for making a determination as to whether the worker has any medical condition that would put him or her at increased risk of 'material health impairment'. There is mandatory worker and employer notification of the examining physician's opinion resulting from the overall assessment of the test results. Records of the interpretation of the examination along with supporting information must be kept by the employer for 30 years, whether or not the test results are abnormal.

There are no data reported which permit evaluation of the effectiveness of the current regulations for prevention of all asbestos-associated diseases. NIOSH has recommended that exposure to asbestos fibers be limited to 0.1 fibers/cm^3 time-weighted averaged over a 100-minute sampling time, based on the technical limits of fiber monitoring rather than on absolute health protection.

Although compliance with current exposure standards should substantially diminish the burden of asbestos-associated disease in the future, there are still many settings

where significant asbestos exposure occurs before controls are put in place or where strict adherence to health protection programs is absent. When the possibility of asbestos exposure is determined after eliciting an occupational history, the healthcare provider should also inquire about the existence of a health protection program in the workplace.

SILICA

Exposure settings

Silicon is the second most common element in the earth's crust. The combination of silicon and oxygen, SiO_2, called silica, is found in amorphous or crystalline forms. Respirable crystalline silica causes silicosis. Heating can change amorphous silica into crystalline forms. The chief form of crystalline silica is α-quartz (hereafter simply 'quartz'). Quartz is a mineral found in most classes of rock, which is the principal source of exposure in many occupations (Table 46.2). Silica sand contains large amounts of quartz. Silica sand is used to produce glass, pottery, silica brick, mortar, and abrasives. Crystalline silica, as sand or aggregate, is added to cement to form concrete. Finely ground silica, also known as silica flour, is used in paints, porcelain, scouring soaps, and as wood filler. The clear rock crystal is of great value for electronic equipment. The colored varieties of crystalline silica are used as gems or ornamental materials.

Tridymite and cristobalite are chemically identical to quartz but differ in crystalline structure. These two minerals are used extensively as filtering and insulating media and as siliceous refractory materials for furnace linings and silica bricks. Flints contain crystalline silica and have been used for centuries because of their hardness and heat resistance.[17,18]

Abrasive blasting
Abrasives
Boiler scaling
Cement production workers
Ceramics
Coal mining and milling
Fillers (paint, rubber, etc.)
Foundry work (ferrous and non-ferrous)
Glass manufacture
Insulation production and installation
Metal mining and milling
Micaceous earth excavation
Mining, quarrying and tunneling
Non-metallic mining and milling
Plastic manufacturing
Pottery making
Refractory materials
Road working
Rubber manufacturing
Scouring soap manufacturing
Tile and clay production
Tunneling
Vitreous enameling

Table 46.2 Selected occupations and industries involving exposure to free crystalline silica

Description	Workers exposed
Masonry, plastering	13,800
Heavy construction	6300
Painting, paper hanging	3000
Iron and steel foundries	800
Metal services	400

Table 46.3 Estimation of workers exposed to at least 10 times the NIOSH REL for crystalline silica

In the US, occupational exposure to crystalline silica occurs in several large categories of industry. Using data from the National Occupational Exposure Survey (NOES) conducted from 1982 to 1983, and applying 1986 Bureau of the Census County Business Patterns, NIOSH estimated that 1.7 million workers are potentially exposed to one or more substances containing silica. Compliance data collected by OSHA inspectors indicate that within the exposed population, there are significant numbers of workers with continuing overexposures to crystalline silica. Table 46.3 lists the number of workers estimated to be exposed to at least 10 times the NIOSH recommended exposure limit (REL), which is approximately five times higher than the prevailing legal limit in the US, i.e., the permissible exposure limit or PEL.[19]

Occupational exposures to crystalline silica also occur throughout the mining industry. Over 300,000 coal, metal, and non-metal miners in stone or ground quarries are exposed to silica. Users of mined or quarried materials in activities such as road building, general construction, and railroad maintenance may be exposed to significant levels of quartz dust.

Environmental measurement

Silica often occurs in combination with other minerals whose composition may include silicon, aluminum, and varying amounts of magnesium, iron, calcium, and other elements. The term 'free silica' is sometimes used to describe SiO_2 that is not combined with other elements. Environmental measurement of silica involves two phases:

- collection of the sample using an appropriate sampling technique; and
- chemical analysis of the collected material.

Two types of samples are routinely collected: bulk dust and respirable dust. *Bulk samples* are collected to determine the average crystalline silica content of the dust in an environment. Once the average silica content is determined, an overall exposure limit for respirable dust can be computed in order to limit silica exposure. *Respirable dust samples* collect air-borne dust on a filter using a particle-size classifier such that the particle collection efficiency mimics the performance of the human respiratory system. It is possible to quantify individuals' exposure to low levels of silica using personal respirable dust samples.

Chemical analysis of both bulk dust and respirable dust samples may be done using X-ray diffraction (XRD) or infrared analysis (IR). A chemical technique involving acid digestion can also be used for bulk samples. The acid digestion technique was the basis for early (pre-1970) silica

analyses. Using phosphoric acid, the amorphous silica and silicate minerals from the bulk dust are digested, leaving the crystalline silica residue for quantification. The percent crystalline silica determined from the bulk analysis can be multiplied by air-borne respirable dust concentration to estimate worker exposure to air-borne crystalline silica. Using respirable dust personal samples, worker dust exposures are measured directly with subsequent crystalline silica analysis by IR or XRD on each individual respirable dust sample.[20]

Clinical effects

The health effects of silica exposure are discussed more fully in Chapters 19.1 and 30.2.

Acute effects

No acute effects of silica exposure have been documented.

Chronic effects

Silicosis is the pneumoconiosis resulting from fibrotic reactions to lung deposition of inhaled crystalline quartz dust. Disease latency and time course depend on the intensity and duration of dust inhalation. Particle size distribution and surface characteristics, as well as the crystalline structure of the quartz particle, may affect toxicity. A characteristic pathologic entity, the silica nodule, results from pulmonary response to inhaled and retained silica. These nodules may coalesce with resulting tissue destruction and disruption of the normal thoracic architecture. This is known as progressive massive fibrosis. Silicosis that occurs within a few years of the initiation of exposure is sometimes referred to as accelerated silicosis. Disease with a more indolent time course, often not identified for one or more decades from first exposure, is sometimes referred to as chronic silicosis. Accelerated and chronic silicosis most likely result from the same disease mechanisms, differing only in time course. A distinct condition, referred to as acute silicosis, may result from a different mechanism. It is a rapidly progressive disease which develops after inhalation of high concentrations of fine silica particles. Acute silicosis has pathologic characteristics similar to alveolar proteinosis.[21]

All forms of silicosis can progress in the absence of ongoing silica dust exposure, and people with occupational silica exposure may not manifest chest X-ray abnormalities until well after retirement. Although silicosis is irreversible, the likelihood of progressive impairment from accelerated or chronic silicosis is presumably diminished through early identification of disease and cessation of exposure. Once acute silicosis is present, interventions have not been shown to favorably influence disease outcome. Experimental therapies or interventions which are not widely available, such as bronchoalveolar lavage and lung transplantation, have been attempted for workers with acute silicosis and for some with total pulmonary incapacity due to progressive massive fibrosis.

Silicosis was listed as an underlying or contributing cause of death on 187 death certificates in the US in 1999. Surveillance data suggest that death certificate records significantly underestimate the extent of the problem.

The risk of tuberculosis is greater in workers with silicosis than in the general population. Tuberculosis is probably more common in silica-exposed workers even without diagnosed silicosis. Tuberculosis can cause significant morbidity and mortality, particularly if unrecognized and untreated. While effective treatments are available, treatment failures and relapses are reported to occur more commonly in people with silicosis.

Emphysema, bronchitis, and airways obstruction appear to occur more commonly in silica-exposed workers than in the general population. The risk is most likely dose dependent. In general, these conditions cause significant morbidity and can cause premature mortality. Early intervention with control or cessation of adverse pulmonary exposures (including dust and tobacco smoke) may result in reversal of bronchitis symptoms and presumably diminish the rate of progression of airways obstruction.

Lung cancer as a result of silica exposure is the subject of ongoing scientific investigation. After intensive review, the International Agency for Research on Cancer (IARC) has stated that there is sufficient evidence to conclude that inhaled crystalline silica from occupational sources is a human carcinogen,[18] and NIOSH has recommended control of exposure based on the importance of reducing silicosis risk and the conclusion that crystalline silica should be considered a potential occupational carcinogen.[17] Some investigators suggest that silicosis rather than silica exposure is the key risk factor. In any event, as with lung cancer from asbestos exposure, there is no indication that silica dust-associated lung cancers differ from other lung cancers. No effective methods of early identification leading to successful intervention have been identified. Because both silica exposure and lung cancer are so common, even a small increase in relative risk of disease resulting from the exposure could result in a significant number of excess cases.

Clinical evaluation and management

A silica-exposed worker with chest symptoms should be evaluated in the normal fashion with a comprehensive history, focused physical examination, and a logical sequence of medical testing. Medical history should concentrate on adverse pulmonary exposures, including occupational exposure to dusts and fumes and tobacco use. A history of significant chest trauma, pulmonary infections, and cardiovascular disease is relevant. The extent to which current pulmonary problems interfere with normal levels of functioning should be documented. In general, a diagnosis of silicosis is made on the basis of an abnormal chest radiograph along with a credible history of silica exposure.

General clinical management issues for treatment of silicosis are no different than for other chronic pulmonary diseases. Elimination of adverse exposures, patient and family support and education, and interventions directed at optimization of the level of function and minimizing

time lost to infection are appropriate. Because of the increased risk of mycobacterial infection in patients with silicosis, tuberculin skin testing and evaluation of risks and potential benefits of treatment should be pursued (see also Chapter 19.9).

Silicosis can progress in the absence of additional exposures, presumably through an iterative process of macrophage ingestion of retained silica particles followed by cell death (liberating inflammatory and fibrogenic mediators), and reingestion. In order to interrupt this process, some have suggested that bronchoalveolar lavage could diminish the body burden of silica. At this time, such therapies should be considered experimental and be performed only in the context of a clinical trial. Lung transplantation has also been attempted for far-advanced disease.

Prevention

Primary prevention

Although the effects of exposure to free silica have been known for many years, silicosis continues to prevail among workers in the dusty trades. The critical strategy for prevention of silicosis is prevention of exposure. The OSHA PEL for general industry is based on the measurement of respirable dust (including silica and non-silica components) and comparing that measurement to the following formula:

$$\text{PEL} = 10\ \text{mg/m}^3\ /\ (\%\ \text{quartz} + 2)$$

The effect of the formula is to limit respirable dust containing 100% quartz below 0.1 mg/m^3, and to limit respirable dust below 5.0 mg/m^3 for quartz-free dusts. The OSHA limit for the two other forms of crystalline silica, cristobalite and tridymite, is half the limit allowed for quartz.[22] Both NIOSH and the American Conference of Governmental Industrial Hygienists (ACGIH) recommend exposure limits of 0.05 mg/m^3 for all crystalline forms of silica.[23,24] Complicating the situation, OSHA uses a different and older standard for the construction industry, nominally requiring a count of the number of particles collected. Today, few laboratories are equipped or trained to do the microscopic particle counting that is required, consequently respirable mass measurements are sometimes used in conjunction with mass-to-count conversion factors to compare measurements to the exposure limits. The exposure limit for all forms of crystalline silica in the construction industry is determined from the following formula:

$$\text{PEL} = 250\ \text{mppcf}\ /\ (\%\ \text{quartz} + 5)$$

where mppcf stands for million particles per cubic foot of air sampled.

The Mine Safety and Health Administration (MSHA) uses a crystalline silica standard equivalent to the OSHA standard for metal and non-metal mines. For underground coal mines, the respirable dust standard is 2.0 mg/m^3 for coal mine dust with less than 5% crystalline silica. When coal mine dust exceeds 5% quartz, the respirable dust standard (RDS) is calculated using the formula:

$$\text{RDS} = 10\ \text{mg/m}^3\ /\ (\%\ \text{quartz})$$

Silica dust is generated when silica-bearing soil, rock, or ore is drilled, blasted, cut, dug, or otherwise disturbed. There is no obvious way to determine if the material being processed contains silica other than to perform chemical analyses described earlier in this chapter. As a result, many exposures to crystalline silica go undetected simply because the exposure is not sampled and analyzed for silica. In mine settings, veins of silica may intertwine with veins of desirable ore. Crushed rock and gravel may have varying degrees of silica based on the specific location where the material originated. The best approach for protecting workers health is to sample and analyze the materials being mined or processed frequently.

Once the potential for exposure to silica is established, effective preventative measures are almost always available. These measures include substitution of materials and engineering controls. OSHA and MSHA have established legal standards for compliance, setting exposure limits at approximately 100 $\mu g/m^3$ described by the PEL and RDS formulae above; and a REL has been established by NIOSH at 50 $\mu g/m^3$. Any measure to control exposure should use these limits to determine the need for engineering controls and their adequacy. The selection of a specific control strategy that maintains exposures below these limits will be determined by the economic and technical feasibility of that particular strategy in each situation.

In abrasive blasting, where risk is high because of the generation of fine particles of 'freshly fractured' silica, it is often possible to replace the silica being used with a less toxic substitute. When using silica, even when the abrasive blaster operator is protected with an air-supplied respirator, blaster helpers and other process workers in the area may be exposed. There are alternative materials available that can be used as an abrasive agent in lieu of silica sand. Although those materials may also be hazardous, and the material liberated during blasting is often toxic, appropriate substitution combined with personal protective equipment can reduce the health risks for the blaster and other nearby workers.

The most common engineering control available in most settings is ventilation. In non-mining industries, ventilation is usually subdivided as blowing and exhausting ventilation and dilution ventilation. Particularly harmful dusts are often removed from the exhaust stream using bag-house filters to prevent general environmental contamination. Industrial applications where these controls have been successfully applied include, among others, foundries, ceramic industries, crushing, grinding, and screening.

In mining environments, the dust control problems are different. Drilling and grinding operations present high exposure potential at surface mines. Since these operations are typically open to the environment, ventilation is not a

practical solution. There are two alternative strategies that can be applied: enclosure and dust suppression. The operator's cab in equipment used at surface mines can be enclosed and designed to deliver air-conditioned filtered air to the operator; however, other workers who are not machine operators may continue to be exposed. It may be possible to enclose the point of dust generation. Water injection, water spraying, and the use of wetting agents are means of dust suppression which have been used at both surface and underground mines.

At underground mines, dust control can be achieved through forced ventilation systems. Underground operations may also use high-pressure water sprays, water injection drilling, and other wetting methods to suppress the generation of dust at its source.

Finally, there are a number of ways to do most jobs. Redesigning task performance, utilizing engineering equipment to reduce dust exposure, or altering work practices can be effective. Educational efforts, labeling of quartz-containing materials, and posting of hazardous work sites can assist workers in recognizing the dangers of exposure to dust and the benefits to be gained by altering work practices to reduce exposure to the minimum achievable level.

Secondary prevention

Workplace-based medical screening of silica-exposed workers is generally recommended, using periodic questionnaires, chest X-rays, and lung function tests. The effectiveness of screening efforts has not yet been published. Chest radiographs may detect silicosis in some workers at a point when the disease is asymptomatic, but X-rays may be insensitive to other silica effects. In most exposure settings, a PA chest radiograph repeated every 3 years for asymptomatic workers should be sufficient. More frequent examinations should be conducted if an abnormality is demonstrated and exposure continues. Spirometry is another tool for screening and surveillance. Spirometry is inexpensive, acceptable, and readily available. Lung function testing should be performed using equipment and methods meeting American Thoracic Society-recommended standards (see Chapter 19.1). Spirometry can demonstrate restrictive or obstructive abnormalities, either of which may result from silica dust exposure. Once patients test below population-based 'normal' values, disease is generally well established. Using the patient's own baseline spirometry values for comparison may provide an earlier indication of the development of a problem. In this case, a drop of 15 percentage points from the baseline 'percent predicted' value for either FEV_1 or FVC (e.g., from 105% of predicted FEV_1 to 90% of predicted) should suggest the need for additional evaluation.

A number of state health departments require reporting of cases of silicosis as 'sentinel events'. Investigations of reported cases can identify new cases and lead to prevention of disease in workers not yet affected. Case identification and reporting are no substitute for primary disease prevention through exposure control; they are, however, critical components of an overall program of prevention requiring the cooperation of primary healthcare professionals.

Although silicosis is often thought to be a disease of primarily historical interest, current exposure conditions continue to produce significant disease and even death from silicosis. Exposure is so widespread that workers, employers, and healthcare providers must maintain a high index of suspicion that any work in a dusty trade may result in the development of this serious pulmonary disease.

COAL

Exposure settings

Coal is organic material that has been fossilized after millions of years of pressure and temperature extremes. Coal is used as a basic fuel for numerous industries requiring a source of heat; some coal is also used for residential heating. A relatively small amount of coal is used for production of gaseous and liquid fuels.

The primary occupations that are exposed to coal dust are those associated with the actual mining and processing of coal. Other workers may have significant exposures through coal handling operations. The method of mining has an effect on the dust concentration level in which a miner may work. Usually, coal miners who are employed at the working face (the area of the mine where the actual extraction of coal occurs) of underground mines have the highest coal mine dust exposures. In 2000, there were approximately 76,000 workers in the coal mining industry in the US.

Environmental measurement

Coal dust health standards

In the US, respirable dust levels in both underground and above-ground coal mines have been regulated since 1969. The current US exposure limit for respirable coal mine dust is 2.0 mg/m^3.[25] A lower limit is applicable if more than 5% of the dust is quartz. Exposures to coal mine dust in general industry occur primarily in the steam generation of electricity.

The basic sampling unit used in the US for measuring respirable dust consists of a battery-powered personal sampling pump which draws dust-laden air through a particle pre-separator (generally a cyclone) and onto a pre-weighed filter at a controlled flow rate for an 8-hour shift. The large particles are removed by the pre-separator, and the smaller respirable dust is collected on the filter. Because the exposure limit is for 'respirable coal mine dust', no special chemical analysis is needed; the filters are weighed and the measured mass collected is divided by the volume of air sampled, to calculate the worker's daily exposure.

Coal dust particle size distributions and regional effects

The current coal mine dust standard was intended to prevent deposition of fine particles in the non-ciliated alveolar air spaces. A study of particle size distributions in

underground coal mines found that a substantial fraction of mine dust consists of larger particles capable of deposition in the upper airways. Chronic bronchitis is also prevalent in coal miners and may be related in part to the deposition of larger particles in the ciliated, conducting airways. Dust control techniques designed to obtain compliance with the respirable dust standard do not necessarily reduce the thoracic dust exposures.

Clinical effects

The health effects of coal mining are discussed further in Chapter 19.10.

Acute effects

A few studies have identified a relatively acute drop in FEV_1 among some, but not all, miners during the first months to years following first exposure to coal mine dust. The clinical and prognostic significance of this drop is unclear and is the subject of ongoing investigations.

Chronic effects

Coal workers' pneumoconiosis (CWP) is one of the lung diseases arising from inhalation and deposition of coal dust in the lungs and the reaction of the lungs to the dust. CWP is a chronic, irreversible disease of insidious onset, usually requiring 10 or more years of dust exposure before becoming apparent on routine chest radiographs. It is characterized by macular and nodular pigmented lesions in the lungs, which may be visible radiographically as small or large opacities. When only small opacities are present, the condition is called simple CWP or chronic CWP. Progressive massive fibrosis (PMF) or complicated CWP are terms used when radiographic shadows greater than 1 cm, attributable to coal dust exposure, are present. CWP, particularly in advanced stages or when PMF is present, has been associated with pulmonary incapacity and increased morbidity and mortality. The occurrence of CWP is dependent on the intensity and duration of coal mine dust exposure. CWP, in some instances, progresses after cessation of exposure, although this seems to be less common and the progression less aggressive than that resulting from retained silica dust.

CWP was listed as an underlying or contributing cause of death on over 1417 death certificates in the US in 1996. As exposure conditions have improved, the average age at death among men with CWP has increased and approximates the average age at death of all men in the US. Since chest X-ray abnormalities may be visible in the absence of clinical disease in some individuals, there may be a 'preclinical' stage during which CWP can be identified. Although reduction or elimination of exposure to coal mine dust in workers with chest X-rays showing category 1 abnormalities, by ILO criteria, would presumably result in decreased progression of disease and in fewer cases of PMF, statistical modeling based on large data sets in the UK and the US suggests that this benefit may not prove very great.

Dust control strategies in the US, initiated in the early 1970s, were directed at preventing miners from developing ILO category 2 CWP. Miners with category 1 CWP were offered work in jobs with a maximum dust exposure of 1 mg/m^3. It was anticipated that this would result in the virtual elimination of PMF. This strategy has not been completely successful, in part because PMF can appear on a background of category 1 CWP or, rarely, category 0.

Chronic bronchitis, emphysema, and airways obstruction (accelerated loss of FEV_1) are more common in workers exposed to coal mine dust than in others with comparable tobacco use habits. All of these conditions appear to be dose related. Chronic bronchitis symptoms may be reversible if adverse exposures are controlled early. Presumably, the rate of loss of FEV_1 could be diminished through reduction or elimination of dust exposure, although the rate of loss that should trigger intervention and the optimal timing for intervention remain the subject of investigation. Chronic bronchitis, emphysema, airways obstruction, and CWP all result from exposure to coal mine dust and may occur in various combinations. The chest X-ray is not a reliable predictor of the likelihood of airways obstruction associated with dust exposure.[26] In general, diminished FEV_1 is associated with morbidity and premature mortality. Dust exposure unlikely to result in the development of CWP or PMF may still cause or contribute to clinically significant airways obstruction.[27]

Coal mine dust may contain varying proportions of crystalline quartz. CWP may be indistinguishable from silicosis on chest X-ray.

Excess risk of stomach cancer has been reported with coal mine dust exposure, although the level of increased risk is not great and the population prevalence of this condition is generally low (see Chapter 30.5).

Clinical evaluation and management

Miners with lung disease generally present for evaluation either of progressive dyspnea or an acute chest illness. Because a federal benefits program compensates miners with severe impairment, some miners present for consultation or evaluation of their qualification for these benefits. In other instances, the possibility of disease from coal mine dust exposure is an incidental finding in a routine health evaluation.

A clinical evaluation should include a comprehensive medical and occupational history, with a focus on the pulmonary and cardiovascular systems. Since miners with lung disease often become involved in benefits applications and significant weight is given to the assessment of the primary care physician, the history should be carefully taken and well documented. The onset and evolving course of symptoms such as dyspnea, cough, and sputum production should be described. Any changes in normal life activities should be explored in detail. For example, many miners describe the need to abandon hobbies such as hunting, or may report the modification of sexual activity as a result of increasing pulmonary impairment. Work decisions, such as a job transfer from a strenuous or dusty job to one that is less demanding physically, are significant. Since disease development is frequently insidious,

it is often helpful to ask about the abilities of the patient in comparison with non-exposed contemporaries. Careful inquiry should also be made into adverse pulmonary exposures on and off the job, including tobacco use.

A comprehensive physical examination is appropriate, although findings are not specific. It is, however, often helpful to determine if other conditions associated with work exposures are present, such as noise-induced hearing loss or musculoskeletal disorders, which should be evaluated and treated.

Testing may include chest radiography, which should be interpreted by someone skilled in recognition of the pneumoconioses, preferably a certified B-reader. Spirometry should be performed using methods and equipment meeting American Thoracic Society recommendations. Other measures of lung function may be helpful in certain clinical settings but do not need to be performed routinely.

Care for miners with chronic lung disease should attempt to maximize their level of functioning and minimize life disruption. Ongoing dust exposure should be controlled to the extent possible, with transfer to a low-dust job recommended for working miners. Where other adverse exposures such as tobacco smoke can be identified, they should be eliminated. Bronchodilators provide symptomatic relief for some. Infections should be treated early and monitored carefully. Immunization against influenza and pneumococcal disease should be encouraged. Tuberculosis does not complicate CWP as it does silicosis.

In coal mining areas, the US Department of Labor has assisted in the development of clinic- or hospital-based respiratory disease treatment programs for miners and their families. Patient and family education can reinforce the therapeutic interventions of the primary care provider and provide emotional support.

Prevention

Primary prevention

The primary means of preventing dust-related illnesses is to limit the exposure of all workers through the use of engineering controls.[28] These include dilution ventilation and dust suppression. The MSHA prescribes minimum ventilation requirements for face operations, primarily to control the emission of methane from freshly fractured coal. The fresh air also reduces the dust concentration through mixing with the dust-laden air exhausting out of the return air tunnels. Dust suppression usually involves a system of water sprays near the cutting head of continuous mining machines or longwall shears. Dust exposures can also be reduced on an individual basis through the use of approved particulate air-purifying respirators. Respirator usage is usually voluntary and is not considered by MSHA to be an acceptable means for the operator to reduce exposures to respirable dust. However, many miners choose to wear respirators, particularly during the dust-producing mining operations. There is some evidence that use of respirators is associated with preservation of lung function.

Exposure limits in the US for coal mine dust are enforced by MSHA. Mine operators must take periodic dust samples during normal working conditions and send them to MSHA for weighing and analysis. The integrity of this program has been brought into question after MSHA's allegations of widespread tampering with dust sampling. The mandatory operator sampling program is supplemented by periodic inspections by MSHA personnel. Recent information suggests that the current dust exposure limit might not be fully protective, and NIOSH has recommended a lowering of the dust limit along with changes in the program of inspection and enforcement.[29]

Secondary prevention

Medical surveillance, directed toward the early identification of CWP through periodic radiographic examinations, is available for all underground coal miners in the US through the Coal Workers' X-ray Surveillance Program administered by NIOSH. Miners with CWP may exercise a right to work in a reduced dust environment of 1 mg/m^3 and have their exposures sampled more frequently. There is currently no screening or surveillance program directed toward the prevention of airways obstruction, which can cause significant morbidity among miners. Primary healthcare providers should consider performing periodic spirometry using techniques and equipment meeting the American Thoracic Society recommendations as a guide to care and education of patients exposed to coal mine dust. A drop of 15 percentage points or more from baseline 'percent predicted' values of FEV_1 should suggest the need for more intensive investigation and additional preventive interventions.

In the years following adoption of the current coal mine dust exposure regulations, an average of over 2000 former miners have continued to die each year in the US with CWP as an underlying or contributing cause of death. Thousands of others suffer from significant airways disease caused, at least in part, by their coal mine dust exposure. There must be continued attention to early disease identification and preventive intervention in order to supplement dust control efforts and prevent the continuing progression of these diseases.

MAN-MADE MINERAL FIBERS

Exposure settings

Man-made mineral fibers (MMMF), also called synthetic or man-made vitreous fibers, include a variety of manufactured materials with excellent insulating properties. Not true minerals, they are amorphous silicates manufactured into a fibrous form. MMMF are grouped according to their origin into glass fiber (from glass), ceramic fiber (from kaolin clay), and mineral wool (from rock or slag). Increasingly used as asbestos substitutes, they have thousands of applications in industrial and non-industrial settings. They are used for thermal and acoustic insulation and plastic reinforcement in everything from buildings, ships, and automobiles, to home appliances, industrial kilns, and furnaces.

Managers and administrators
Civil engineers
Mechanical engineers
Chemists (except biochemists)
Electrical and electronic technicians
Chemical and science technicians
Production coordinators
Warehouse clerks (receiving, recording, and issuing)
Janitors and cleaners
Supervisors (mechanics and repairers)
Engine mechanics (aircraft, bus, truck, and stationary)
Heavy equipment mechanics
Industrial machinery repairers
Mechanics (heating, air conditioning, and refrigeration)
Brick masons and stonemasons
Tile setters
Carpenters
Drywall installers
Painters (construction and maintenance)
Plasterers
Plumbers (pipe fitters and steam fitters)
Insulation workers
Roofers
Sheet metal duct installers
Structural metal workers
Boilermakers
Sheet metal workers
Plant and system operators
Machine operators (grinding, abrading, buffing/polishing)
Machine operators (metal, plastic, stone, and glass working)
Packing and filling machine operators
Slicing and cutting machine operators
Crushing and grinding operators
Oven operators (furnace and kiln)
Assemblers
Production manager (inspectors, checkers, and examiners)
Production testers, samplers, weighers, and helpers
Truck drivers (heavy and light)
Crane and tower operators
Industrial truck and tractor equipment operators
Material moving equipment operators
Construction laborers
Stock handlers and baggers
Vehicle washers and equipment cleaners
Hand packers and packagers

Table 46.4 Workers potentially exposed to man-made fibers

Exposure to MMMF occurs during production, fabrication, and application of materials. Over 500,000 US workers are potentially exposed to MMMF (Table 46.4), and that number is likely to increase.

Environmental measurement

The sampling protocol for MMMF is virtually identical to that for asbestos. The sample is collected on a filter for microscopic examination. The current literature indicates that in addition to biopersistence and durability, health effects relate to the length and diameter of the fibers; therefore, some size selectivity must be employed in either the sampling or analysis. The technique for asbestos sampling, as described earlier in this chapter, is the most common measurement technique.

Clinical effects

The health effects of MMMF are discussed in greater detail in Chapters 19.11 and 30.2.

Acute effects

Acute respiratory and skin irritation occur frequently among workers newly exposed to mineral fibers. Irritation has been reported most often in glass wool workers, but it can result from contact with other fibers. Although usually self-limited, these problems may cause workers to leave employment.

Chronic effects

Some studies have shown excess lung cancer risk in glass fiber and mineral wool production workers. Synthetic fibers vary widely in their physicochemical characteristics, and carcinogenic potential most likely relates to the biopersistence and solubility of inhaled fibers, with the most persistent and durable fibers – those most like asbestos – posing the greatest risk. IARC has recently reviewed the relevant scientific literature and concluded that the more biopersistent materials such as refractory ceramic fibers and some special-purpose glass wools are possible human carcinogens (IARC Group 2B). IARC has reclassified the more commonly used vitreous fiber glass wool and slag wool as not classifiable as to their carcinogenicity (IARC Group 3).[30] The US National Toxicology Program classifies ceramic fibers of respirable size as reasonably anticipated to be a human carcinogen.[31]

Epidemiologic investigations have suggested that exposure to mineral fibers may be associated with the development of low profusions of chest X-ray opacities consistent with a diagnosis of pneumoconiosis in some workers. In addition, refractory ceramic fiber production workers have been found to develop pleural plaques and pleural thickening which increases with increased latency.[32] These asbestos-like effects suggest the possibility that other asbestos-like health effects, particularly lung cancer, could result from refractory ceramic fibers. Mineral fiber exposure may also increase the risk of chronic bronchitis.

Clinical evaluation and management

When workers newly exposed to MMMF complain of intense pruritus with a minimum of visible dermatologic changes, the etiology is readily apparent. At times, fibers can be seen microscopically after cellophane tape is pressed against exposed skin. Workers presenting with skin irritation from mineral fiber exposure should be encouraged to minimize exposure through use of appropriate protective equipment and should be reassured that the acutely troublesome symptoms are generally self-limited. Anti-pruritic medications may be of benefit to some.

The approach to evaluation for chronic conditions associated with mineral fiber exposure is similar to the assessment of these conditions where there is no occupational exposure. A careful medical history, including a history of occupational and environmental exposures, forms the basis for care. Chest imaging to exclude other acute or reversible disease processes is indicated. Spirometry and possibly other measures of pulmonary function provide diagnostic and prognostic information.

There are no specific interventions uniquely beneficial for individuals with cancers that may be associated with mineral fiber exposure. Chronic bronchitis and airways obstruction should be approached in the usual fashion. Management should include reduction or elimination of adverse pulmonary exposures, including workplace dusts and tobacco smoke. Symptomatic treatment, reversal of reversible or intermittent airways obstruction, and infection control are also important. In the case of moderate-to-severe disease, referral to patient and family support and education programs may be helpful.

Disease prevention

Primary prevention

Given the classification of MMMF as potential human carcinogens by IARC, mineral fiber exposure should be limited to the extent feasible by technology. This can be done by several standard techniques. Where possible, substitutes should be used in place of the MMMF. This is often difficult since these products were, in many cases, replacements for asbestos. Lacking alternative materials, engineering controls to contain the source of exposure become the next best approach. When the process cannot be fully controlled, the worker must be isolated from the process generating the dust. Finally, the worker may be isolated by use of respiratory protection equipment. For uncontrolled exposures to RCF, as with asbestos, the appropriate type of respirator could be an air purifying or self-contained breathing apparatus (SCBA) with a full facepiece, operated in a pressure demand or other positive pressure mode. Respirators should be used with a complete respiratory protection plan. Administrative controls can also be used to reduce exposures by limiting the amount of time a worker spends in a contaminated environment. Administrative controls need to be coupled with personal samplers on the workers to closely monitor exposures and assure the maximum level of exposure is not surpassed.

Secondary prevention

There is no consensus approach to screening or surveillance of MMMF-exposed workers. However, in light of concern about asbestos-like effects, it would be prudent to closely monitor these workers for pulmonary health effects. The level of health risk from MMMF exposure most likely varies depending on the intensity and duration of exposure as well as with the specific physical and chemical characteristics of the fiber. Until the specific risks are better defined, a generic approach to surveillance of 'fiber-exposed' workers is reasonable. Baseline and periodic respiratory symptom questionnaires, spirometry, and chest radiographs should be offered. Since the diseases of concern have long latency, the frequency of observation should increase in each decade following first exposure. Careful collection and retention of exposure data should make future screening more productive as new knowledge from ongoing systematic observation of occupational cohorts becomes available.

References

1. Virta RL. Asbestos: geology, mineralogy, mining, and uses. US Department of Interior, US Geological Survey, Open-File Report 02-149 Version 1.0, URL: pubs.usgs.gov/of/2002/of02-149/index.html.
2. Virta RL. Worldwide asbestos supply and consumption trends from 1900 to 2000. US Department of Interior, US Geological Survey, Open-File Report 03-83, URL: pubs.usgs.gov/of/2003/of03-083/of03-083.pdf.
3. Mlynarski KW. US Geological Survey Minerals Yearbook. Washington, DC: Government Printing Office; 1998.
4. Environmental Protection Agency (EPA). EPA's asbestos and vermiculite home page. www.epa.gov/opptintr/asbestos/index.html. Accessed July 14, 2003.
5. Occupational Safety and Health Administration (OSHA). Preamble to final Rule: Occupational Exposure to Asbestos, Section 4-IV. Final regulatory impact and regulatory Flexibility Analysis. Federal Register 59: 40964-41162. Washington, DC: Government Printing Office; 1994.
6. McCrone WC. Asbestos identification. Chicago: The McCrone Research Institute; 1987.
7. Cassinelli ME, O'Connor PF, eds. NIOSH manual of analytical methods (NMAM®), 4th edn. Methods nos. 7400, 7402, 9000, and 9002. Washington, DC: Government Printing Office; DHHS (NIOSH) Publication No. 94-113; 1994.
8. Kipen HM, Lilis R, Suzuki Y, et al. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. Br J Ind Med 1987; 44:96-100.
9. Oliver LC, Eisen EA, Green R, Sprince NL. Asbestos-related pleural plaques and lung function. Am J Ind Med 1988; 14:649-56.
10. Brodkin CA, Barnhart S, Checkoway H, Balmes J, Omenn GS, Rosenstock L. Longitudinal pattern of reported symptoms and accelerated ventilatory loss in asbestos-exposed workers. Chest 1996; 109:120-6.
11. Miller A, Lilis R, Godbold J, Chan E, Selikoff IJ. Relationship of pulmonary function to radiographic interstitial fibrosis in 2,611 long-term asbestos insulators. An assessment of the International Labour Office profusion score. Am Rev Respir Dis 1992; 145:263-70.
12. Wilkinson P, Hansell DM, Janssens J, et al. Is lung cancer associated with asbestos exposure when there are no small opacities on the chest radiograph? Lancet 1995; 345:1074-8.
13. Wagner GR. Screening and surveillance of workers exposed to mineral dusts. Geneva: World Health Organization; 1996.
14. Henschke CI, McCauley DI, Yankelevitz DF, et al. Early Lung Cancer Action Project: overall design and findings from baseline screening. Lancet 1999; 354:99-105.
15. Anonymous. Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health 1997; 23:311-16.
16. US Department of Labor (DOL). Title 29 Code of Federal Regulations, Part 1910.1001. Occupational Safety and Health Administration, Asbestos Standard for General Industry, June 20, 1986 (as amended 1988, 1989, 1990, 1992, 1994, 1995, 1996, and 1998).
17. National Institute for Occupational Safety and Health (NIOSH). NIOSH hazard review: health effects of occupational exposure to respirable crystalline silica. Washington, DC: Government Printing Office; DHHS (NIOSH) Publication No. 2002-129; 2002.
18. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Silica, some silicates, coal dust and para-aramid fibrils. Lyon, 15-22 October 1996. IARC Monogr Eval Carcinog Risks Hum 1997; 68:41-242.
19. Linch KD, Miller WE, Althouse RB, Groce DW, Hale JM. Surveillance of respirable crystalline silica dust using OSHA compliance data (1979-1995). Am J Ind Med 1998; 34:547-58.
20. Cassinelli ME, O'Connor PF, eds. NIOSH manual of analytical methods (NMAM®), 4th edn. Methods nos. 7500, 7501, 7602,

and 7603. Washington, DC: Government Printing Office; DHHS (NIOSH) Publication No. 94-113; 1994.
21. American Thoracic Society. Adverse effects of crystalline silica exposure. Am J Respir Crit Care Med 1997; 155:761-5.
22. US Department of Labor, Occupational Safety and Health Administration. Title 29 Code of Federal Regulations, Part 1910.1000, Table Z-3. Mineral Dusts. Published at 39 FR 23502, June 27, 1974.
23. National Institute for Occupational Safety and Health (NIOSH). Criteria for a recommended standard. Occupational exposure to crystalline silica. Washington, DC: Government Printing Office; DHEW Publication No. (NIOSH) 75-120; 1974.
24. American Conference of Governmental Industrial Hygienists (ACGIH). 2003 TLVs® and BEIs®: based on the documentation of the threshold limit values for chemical substances and physical agents & biologic exposure Indices. Cincinnati: ACGIH Worldwide; 2003.
25. Federal Mine Safety & Health Act of 1977, Public Law 91-173, as amended by Public Law 95-164, Section 101.
26. Wagner GR, Attfield MD, Parker JE. Chest radiography in dust-exposed miners: promise and problems, potential and imperfections. Occup Med 1993; 8:127-41.
27. Marine WM, Gurr D, Jacobsen M. Clinically important respiratory effects of dust exposure and smoking in British coal miners. Am Rev Respir Dis 1988; 137:106-12.
28. Kissell FN. Handbook for dust control in mining. Washington, DC: Government Printing Office; DHHS (NIOSH) Publication No. 2003-147; 2003.
29. National Institute for Occupational Safety and Health (NIOSH). Criteria for a recommended standard. Occupational exposure to respirable coal mine dust. Washington, DC: Government Printing Office; DHHS (NIOSH) Publication No. 95-106; 1995.
30. International Agency for Research on Cancer. Man-made mineral fibres and radon. IARC Monogr Eval Carcinog Risks Hum 1988; 43.
31. National Toxicology Program on Carcinogens, tenth edition: carcinogen profiles 2002. Research Triangle Park, NC: NIEHS; 2002: III-46-47.
32. Lockey J, Lemasters G, Rice C, et al. Refractory ceramic fiber exposure and pleural plaques. Am J Respir Crit Care Med 1996; 154:1405-10.

Textbook of Clinical Occupational and Environmental Medicine

Second Edition

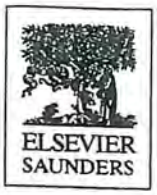
Edited by

Linda Rosenstock MD MPH
Dean, School of Public Health
Professor of Medicine and Environmental Health Sciences
University of California, Los Angeles
Los Angeles, CA
USA

Mark R Cullen MD
Professor of Medicine and Public Health Director
Occupational and Environmental Medicine Program
Yale University School of Medicine
New Haven, CT
USA

Carl Andrew Brodkin MD MPH
Clinical Associate Professor of Medicine and
Environmental and Occupational Health Sciences
University of Washington
Seattle, WA
USA

Carrie A Redlich MD MPH
Professor of Medicine
Occupational and Environmental Medicine Program
and Pulmonary Critical Care Section
Yale University School of Medicine
New Haven, CT
USA



Philadelphia · Edinburgh · London · New York · St Louis · Sydney · Toronto 2005