American Thoracic Society
MEDICAL SECTION OF THE AMERICAN LUNG ASSOCIATION

# Adverse Effects of Crystalline Silica Exposure

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## INTRODUCTION AND MAGNITUDE OF THE PROBLEM

This American Thoracic Society Statement was prepared by a Committee of the Scientific Assembly on Environmental and Occupational Health at the request of the American Lung Association Occupational Health Expert Advisory Group to emphasize the continuing importance of silicosis as a major lung disease worldwide and the need for increasing efforts in prevention. Emphasis is placed on public health issues of silicosis rather than on important current research in pathologic mechanisms.

Diseases caused or contributed to by inhalation of free crystalline silica include silicosis, pulmonary tuberculosis, industrial bronchitis with airflow limitation, and several extrapulmonary diseases. Chronic simple silicosis, a diffuse fibronodular disease of the lung parenchyma, may be accompanied by subacute and chronic manifestations, including symptoms of dyspnea and cough; diffuse pulmonary nodulation sometimes progressing to conglomerate lesions on chest roentgenograms; and a ventilatory defect, usually obstructive, often with accompanying restrictive abnormality and reduced diffusing capacity. Acute and accelerated silicosis describe rapidly progressive forms, usually associated with intense silica exposure. These may be preceded or accompanied by silicoproteinosis, an alveolar filling process comparable to pulmonary alveolar proteinosis.

The most frequently occurring pathologic effects of silica are multiple nodular lesions in lung parenchyma, bronchial associated lymphoid tissue, lymph nodes, and other viscera and fibrotic lesions of the pleura. Nodular lesions in the lungs may develop by conglomeration into larger lesions of progressive massive fibrosis (1). Diagnostic approaches to the silica-related diseases are beyond the scope of this statement, but they are readily available elsewhere (2–4).

The three important crystalline forms of silica are quartz, tridymite, and cristobalite. These forms are also called "free silica" to distinguish them from the silicates, minerals containing silica bound to one or more metallic cations.

Silicosis is a major disabling occupational lung disease worldwide. In the United States, silicosis was a listed on death certificates as a primary or contributing cause in 4,313 deaths from 1979 to 1990. In four states participating in a special surveillance program, 447 confirmed cases were reported from 1988 to 1992 (5). Seven cases of silicosis in surface miners were reported from a single hospital in West Virginia during the period 1978 to 1988 (6). Among more than 17,000 black South African gold miners who died of unnatural causes while actively employed in mines between 1975 and 1991, the prevalence of autopsy silicosis was 9.7%. During the period of study, this prevalence rose of 12.8% in 1991, and it is expected to rise further (7). More than 4,000 cases of silicosis have been recorded in the national registry of Sweden since the early 1930s (8).

## HIGH RISK OCCUPATIONS

Whenever the earth's crust is disturbed and silica-containing rock and sand is used or processed, there are potential respiratory risks for workers. When modern technology is not accompanied by modern controls (such as rock drilling without appropriate dust suppression), it may lead to increased airborne concentrations of silica dust. Exposure to silica of a respirable size occurs in mining, quarrying, drilling, and tunneling operations. Silica exposure is also a hazard to sandblasters, stonecutters, and pottery, foundry, ground silica, and refractory brick workers. Finely ground silica sand is used as an additive in many manufacturing processes. Abrasive blasting using silica sand or other silica-containing materials has been found to be a particularly high-risk occupation. Clusters of new cases of silicosis in abrasive blasters continue to occur despite the development of respiratory protection more than 60 yr ago, and silica sandblasting still carries high risk for excessive exposure to silica even when respiratory protection is used (9–13). Less hazardous alternative abrasive materials without silica are available. Sandblasting has been banned in certain applications in some countries (14). New reports of silicosis in industries and work settings not previously recognized to be at risk still occur. Examples include tombstone sandblasters (15), silica flour mixers (16), construction workers, and surface strip miners operating mobile drill rigs (17, 18).

## DOMESTIC AND ENVIRONMENTAL EXPOSURES

Individuals may also come into contact with respirable crystalline silica from domestic or environmental exposures even when they do not work in a dusty trade. Although pulmonary silicosis usually requires exposure to high dust levels for prolonged periods, public concern may be raised about potential health effects from brief exposure to airborne silica or residence in locations where prevailing winds carry silica particles from natural or industrial sites. There is little evidence to suggest that brief or casual exposure to low levels of crystalline silica dust produces clinically significant lung disease or other adverse health effects. Chronic simple silicosis has, however, been described after environmental exposures to silica in regions where soil silica content is high and dust storms are common (19). Mild mixed dust pneumoconiosis without silicotic nodules has also been reported in agricultural workers (20, 21).

## SILICOSIS, SILICA EXPOSURE, AND MYCOBACTERIAL DISEASE

The association between tuberculosis and silicosis has long been recognized (22–25), and a recent U.S. survey documents a substantially higher tuberculosis mortality associated with silicosis in the United States in the period 1979 to 1991 (26). The occurrence of tuberculous disease in silicotics is related to the underlying prevalence of prior latent tuberculous infection and risk of new exposure to tuberculosis of the population at risk for silicosis. Recent epidemiologic studies (27, 28) have confirmed that those with chronic silicosis have a 3-fold increase in incidence of tuberculosis (both pulmonary and extrapulmonary) compared with a similarly aged, silica-exposed group without silicosis. The incidence of tuberculosis and nontuberculous mycobacterial disease is highest in acute and accelerated silicosis (2). In chronic silicosis, the incidence of active tuberculosis increases in direct proportion with the increase in the profusion of silicotic nod-

ules, and with the highest category of profusion reaches levels of risk comparable with those reported for HIV-infected subjects. Some data suggest that subjects without silicosis but with long exposures to silica dust have an excess risk of developing tuberculosis compared with the non-silica-exposed population (28).

It is important to consider nontuberculous mycobacterial disease when investigating a subject with silicosis for "pulmonary tuberculosis." Nontuberculous mycobacteria (NTM) account for an increasing proportion of the mycobacterial disease in those with silicosis in the industrialized world. A 1974 study (29) found mycobacterial disease in 22 of 83 subjects with accelerated silicosis in the New Orleans area. Their disease was caused by *Mycobacterium tuberculosis* in 45%, by *M. kansasii* in 41%, and by *M. intracellulare* in 14%. NTM disease is relatively uncommon in developing countries with a high prevalence of tuberculosis, but in a study of a working population of black South African gold miners with a first episode of pulmonary tuberculosis, NTM only were cultured from 16.9% of the men with silicosis (30). The majority of the NTM cultured (78%) were *M. kansasii*, *M. intracellulare* accounted for 9%, and *M. scrofulaceum* accounted for only 4.3%. These NTM have been associated with dusty occupations, including mining, and they are more prevalent in those with occupational and other lung disease (31, 32).

The recommended management of tuberculosis in the setting of silicosis is the same as the management of tuberculosis in its absence (33, 34). Because of increased risk of tuberculosis in silicotics, it is important to consider it in any patient with silicosis and to make sure the silicotic patient does not have active tuberculosis before giving isoniazid prophylaxis alone.

The most effective way to prevent tuberculosis and NTM disease in those who are occupationally exposed to respirable crystalline silica is to limit silica exposure. Those who already have silicosis and those without silicosis but with exposure to crystalline silica for 25 yr or more should have a tuberculin test with an intradermal injection of 5 tuberculin units (TU) of purified protein derivative (PPD). If they react with an area of induration greater than 9 mm, irrespective of their status with regard to bacillus Calmette-Guérin (BCG), they should be offered tuberculosis chemoprophylaxis. The current recommendation is for chemoprophylaxis with isoniazid 300 mg daily for 1 yr (33). Problems with compliance with this long regimen, and with strains of *M. tuberculosis* resistant to isoniazid have stimulated studies of other, shorter, and multidrug regimens, but these have been of limited success (35, 36). Currently, chemoprophylaxis has not been evaluated for NTM infection in this situation.

Prior to the development of rifampin, treatment of pulmonary tuberculosis in subjects with silicosis was thought to be ineffective (37, 38). With the inclusion of rifampin, tuberculosis has been successfully treated using short-course regimens in subjects with silicosis (39–41). Treatment should include rifampin in regimens according to recommended standards.

When cultures of NTM are consistently grown from the sputum of patients with silicosis, treatment will need to be modified according to the type of mycobacterium grown. In this context, *M. kansasii* is generally responsive to therapy. A recent study has indicated that rifampin and ethambutol given for a period of 9 mo is sufficient therapy (42). Other NTM are not as responsive to chemotherapy and should be managed according to standard recommendations (33, 34).

## EXPOSURE-RESPONSE RELATIONSHIPS FOR SILICOSIS

The prevalence of silicosis increases with increasing silica dust exposure. A comprehensive review of this evidence has been recently published (43). Controversy exists concerning the precise quantitative relationship between dust inhalation and disease. The present U.S. Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL) for respirable crystalline silica in general industry is a respirable dust concentration of 10 mg/m³ divided by (% $SiO_2$ + 2) or 250 million particles per cubic foot divided by (% $SiO_2$ + 5), averaged over an 8-h workshift (44). (This standard superseded the similar permissible exposure limit of 0.1 mg/m³ respirable silica.) The OSHA standard was established largely on the basis of studies of Vermont granite workers (45–48). These and the following studies relied primarily on roentgenographic identification of silicosis to determine disease status. The efficacy of this standard was supported by a study of active Ontario gold and uranium miners (49–51). However, it is common for silicosis to be diagnosed after a worker has left the silica-exposed industry, and for it to continue to progress slowly over many decades of life after exposure has ended (52–56). Studies to establish the relationship between silica dust in air and the occurrence of silicosis are hampered by the long period between measurements of exposure and the detection of disease. Higher estimates of exposure risk have been published in studies including retired workers. A study of South African gold miners after they had left the mining industry documented a 25% cumulative risk of silicosis after 28 yr of mining at a 0.33 mg/m³ silica exposure level (52). A death certificate study of South Dakota gold miners predicted that a 45-yr cumulative exposure from 20 to 65 yr of age at 0.09 mg/m³ would result in a lifetime risk of silicosis of 47% (57). Study of Hong Kong granite quarriers indicated that cumulative silica exposure between 1 and 5 mg/m³ per year led to radiologic silicosis in 32% of men 50 yr of age and older (58). In a study of Colorado miners who had left the hard rock mining industry, estimated exposures using silica measurements (in contrast to dust measurements) were associated with even higher risks of radiologic silicosis (53). The continuing controversy regarding quantitative exposure-response relationships casts some doubt on the efficacy in preventing silicosis of a 0.1 mg/m³ respirable quartz standard. By contrast, the standard recommended by the National Institute for Occupational Safety and Health (NIOSH) is 0.05 mg/m³.

## CHRONIC BRONCHITIS AND AIRFLOW OBSTRUCTION

Chronic bronchitis, defined by chronic sputum production, is common among worker groups with exposure to dusty environments contaminated by silica. It cannot be clinically distinguished from chronic bronchitis caused by tobacco smoking or other factors. Epidemiologically it can be detected as bronchitis symptoms in excess of those expected from smoking alone in a cohort of workers exposed to dust, but who have no radiographic evidence of silicosis. Increased frequency of chronic bronchitis has been reported in U.S. coal miners (59), German coal workers (60), South African and Australian gold miners (61, 62), Indonesian granite workers (63), Indian agate workers (64), and other groups. In most studies chronic bronchitis was associated with airflow limitation.

Studies from many different work environments suggest that exposure to working environments contaminated by silica at dust levels that appear not to cause roentgenographically visible simple silicosis can cause chronic airflow limitation and/or mucus hypersecretion and/or pathologic emphysema. At low doses, this effect may not be substantial or disabling. In moderate to severe silicosis, nodules occur in close proximity to small and medium airways causing narrowing and distortion of the lumen. Hypertrophy and scarring in bronchial-associated lymphoid tissue and intrapulmonary lymph nodes may compress larger airways (1). In patients with more advanced silicosis, pulmonary function tests usually reflect a mixed pattern of nonreversible airflow obstruction as well as the features of volume restriction and impaired gas exchange expected with diffuse interstitial lung disease.

Among South African gold miners an association was observed between the extent of emphysema at autopsy and the degree of silicosis (65, 66), as well as with dust exposure intensity independent of silicosis (67). Physiologic studies of miners have shown associations between the extent of radiographic silicosis on the one hand, and the degree of mine dust exposure and physiologic airflow obstruction on the other hand (68, 69). Higher rates of radiographic emphysema by computed tomography (CT scan) have also been found among gold miners with silicosis (70).

Granite quarry workers in Hong Kong also experienced a loss of $FEV_1$ in excess of vital capacity, suggesting airflow limitation, in association with radiographic progression of simple silicosis (17). Canadian granite workers showed an association between the extent of radiographic silicosis by CT scan and the degree of both reduced vital capacity and airflow limitation (72). Comparison of matched groups of Scottish oil shale workers revealed worse restrictive and obstructive deficits among those with pneumoconiosis than among those without (73).

Pathology studies of workers exposed to nonasbestos mineral dusts have revealed pigmentation and fibrosis surrounding small airways (74, 75). This abnormality, to which the term mineral dust small airways disease has been applied, rarely occurred among subjects without dust exposure, and it was associated with airflow obstruction in excess of that found among dust-exposed control subjects (76). Other series of workers with mineral dust exposure did not detect an association between silicosis and airflow obstruction, although emphysema by CT scan and abnormal lung function were common findings (77, 78). The majority of reports describe airflow limitation (in the clinical/physiologic studies), peribronchiolar fibrosis (in pathologic studies), and/or emphysema (primarily in association with progressive massive fibrosis lesions) as aspects of silicosis.

Small airways disease was detected in Indonesian granite workers in the absence of radiographic silicosis or large airways disease (79). Among Colorado hard rock miners dust exposure was associated in smokers with lower airflow rates than accounted for by smoking, whereas in never-smokers a distinct restrictive pattern was observed (80). A meta-analysis of 13 studies among coal and gold miners confirmed an excess of bronchitic symptoms and obstructive physiology, even among nonsmokers (81). There may be an additive effect between tobacco smoke and occupational pollutants in producing chronic bronchitis and air flow obstruction.

The data supporting quantitative relationships between silica exposure (or exposure to dusty environments containing silica dust) and airway disease are more limited than for silicosis and airway disease, although several studies have shown relationships for chronic airflow limitation (68, 82–86) for chronic bronchitis (68), and for emphysema (67). Other studies have found no relationship (87, 88). In addition, quantitative estimates exist showing increased risk for clinically significant loss of lung function among South African gold miners in relation to dust exposure (53); for airflow limitation in Colorado hard rock miners (80) in relation to dust exposure; and for emphysema and COPD mortality in South African gold miners (66, 89), in relation to dust exposure.

## SILICOPROTEINOSIS

This alveolar filling disease is associated with heavy and intense silica dust exposure and a high mortality rate. Silicoproteinosis may present with apparent repetitive attacks of pneumonia (90). Silicoproteinosis is associated particularly with the sandblasting process because of the high exposures. This entity requires special emphasis because of the difficulty in diagnosis and the potential for misdiagnosis.

## LUNG CANCER ASSOCIATED WITH SILICA EXPOSURE

In 1987 the International Agency for Research on Cancer (IARC) reviewed the evidence for carcinogenicity of crystalline silica and concluded that there was *sufficient* evidence of carcinogenicity in experimental animals and *limited* evidence for carcinogenicity in humans (91). In October 1996, a committee of IARC reclassified silica as a Group I substance described as "carcinogenic to humans," concluding that there is "sufficient evidence of carcinogenicity in humans." The problems of confounding lung carcinogens such as smoking and radon exposure, and of selection bias in the detection of cases of pneumoconiosis, complicate the analyses (92). One review of the subject has emphasized that studied metal miner groups may be exposed to other lung carcinogens (93). The balance of evidence indicates that silicotic patients have increased risk for lung cancer. It is less clear whether silica exposure in the absence of silicosis carries increased risk for lung cancer.

Rats exposed to silica by intratracheal instillation of inhalation develop respiratory tract tumors, some of which resemble human bronchogenic carcinoma (94). Relatively high doses of silica and prolonged observation approaching the lifetime of the rats were needed before tumors appeared.

The association between human exposure to silica and risk for bronchogenic carcinoma has been examined in autopsy series, case-control series drawn from workers with silicosis, or from patients with lung cancer, and in population-based groups of silica-exposed workers. Elevated standardized mortality ratios (SMR), approximately 150 (1.5 times the expected rate of cancer deaths), were detected in large population-based studies in Massachusetts (95) and Canada (96). Workers exposed to silica in Italy (97), and in some, but not all, Nordic countries showed 3- to 5-fold increased lung cancer risk (98).

Reports from many countries have identified lung cancer with increased frequency among workers compensated for silicosis (99–105), with a relative risk for lung cancer, compared with that in the general population, from 1.3 to 6.9. A slight excess cancer mortality was found after adjusting for the effects of smoking, and cancers were found in never-smokers in two series (106, 107).

Metal ore miners with silica exposure in the United States (108), the United kingdom (109), China (110–112), and South Africa (113) experienced a significant increase in mortality because of lung cancer, with 2- to 5-fold increases in risk. Several of these studies adjusted for the effects of smoking (66, 108, 111, 112). Miners may also be exposed in the underground work environment to radon, arsenic, and diesel exhaust (114, 115).

In addition, an increased lung cancer risk (SMR, 150 to 200) has been demonstrated in a variety of other nonmining dusty trades, including granite workers in Vermont (116) and China (117), foundry workers (118), German slate workers (119), North Carolina workers in dusty trades (120), and California diatomaceous earth workers (exposed to amorphous silica and cristobalite) (121). Ceramic or pottery workers demonstrated increase cancer risk in Sweden (122) and Italy (123), but not in China (107). Workers in foundries, quarries, and manufacturing industries do not experience radon exposure, although some may be exposed to polycyclic aromatic hydrocarbons or other lung carcinogens.

The available data support the conclusion that silicosis produces increased risk for bronchogenic carcinoma. The cancer risk may also be increased by smoking and other carcinogens in the workplace. Epidemiologic studies provide convincing evidence for increased cancer risk among tobacco smokers with silicosis. Less information is available for never-smokers and for workers exposed to silica but who do not have silicosis. For workers with silicosis, the risks for lung cancer are relatively high and consistent among various countries and investigators.

Silicosis should be considered a condition that predisposes workers to an increased risk of lung cancer. The concern about cancer should enter into decisions about permissible exposure limits.

## EXTRAPULMONARY DISEASE

Transport of silica particles after they have been inhaled or ingested can result in their widespread dissemination. Silica particles have been demonstrated in brain and kidney. Silicotic nodules have been found in liver, spleen, and bone marrow (1). The silicon content of organs has sometimes been elevated in patients dying of silicosis. Silica also affects humoral and cellular immune responses, and it may have systemic effects while remaining in the lungs and regional lymph nodes. Extrapulmonary effects are thus biologically plausible. Regional lymph node disease is the commonest extrapulmonary effect, common enough in silicosis to be considered part of the disease, but it occurs in some exposed workers who have no evidence of lung disease, even by histopathologic examination (1, 66). Extrathoracic nodes may be involved, including the anterior cervical and aortic chains and the groups at the celiac axis and porta hepatis. The process is a granulomatous inflammation leading to formation of typical silicotic nodules within lymph nodes. Capsular scarring can produce the radiographic pattern of peripheral or "eggshell" calcification of lymph nodes.

The observation of other extrapulmonary conditions comes mainly from occupational settings in which exposures have been heavy enough to result in silicosis. No increased prevalence of these conditions has been convincingly described at low exposure levels. There are still divisions of opinion as to whether true cause and effect relationships exist between silica and the listed conditions.

### Scleroderma

Association of scleroderma with dusty work dates from a 1914 description of "diffuse sclerodermia" in Scottish stonemasons (124). Subsequent reports (125, 126) have described increased prevalences of acrosclerosis (sclerodactyly), progressive systemic sclerosis (PSS), or both in patients from dusty trades or in patients with silicosis. The association is complicated by the occurrence of Raynaud's phenomenon and acrosclerosis in vibration injury (127), to which many manual workers, including miners who drill rock, are subject. A recent review has concluded that "silica-associated systemic sclerosis" is indistinguishable from idiopathic PSS (128). Increased incidence of PSS has been found in South African miners (129). In another study of South African miners, there were more cases of PSS than systemic lupus erythematosus (SLE), a reversal of the ratio observed in the general population; an association with higher average silica exposures but not with silicosis was also observed (130). There is persuasive evidence relating scleroderma to occupational silica exposures in settings where there is an appreciable silicosis risk.

### Rheumatoid Arthritis

The evidence of association between rheumatoid arthritis and silica exposure or silicosis is less clear, and the potential for misclassification is greater. It is often hard to be sure that an indolent polyarthritis is rheumatoid arthritis (RA), and silicosis and most other pulmonary fibroses can produce positive serum tests for antinuclear antibodies (131) and rheumatoid factor. A Finnish cohort of 1,026 current and former granite workers had excessive numbers disabled by or taking medication prescribed for RA compared with age-specific rates in the general male population. Fifteen cohort members with RA had died before 1982, and chest radiographs showed silicosis in three at the time of onset of their arthritis (132). South African miners with RA were more likely to have silicosis than were miners not exhibiting RA, and their silicosis was more progressive. The study design did not allow determination of whether silica or silicosis increased the risk of RA, but the investigators thought that if an association exists "it is unlikely to be a strong one" (133). A causal association between rheumatoid arthritis and silica exposure is thus plausible but unproved.

### Other Connective Tissue Diseases

Systemic lupus erythematosus (SLE) or a similar illness has been reported in sandblasters with silicosis (2, 134). Some cases exhibit features of scleroderma and of RA, and may represent mixed connective tissue disease or "overlap syndrome." PSS, RA, and "undifferentiated" findings were reported from a work force exposed to ground silica, a substantial proportion of whom had silicosis (135). They handled and ground silica for scouring powder. On the basis of the evidence cited, a causal association between SLE and silica exposure should be suspected only in the presence of acute or accelerated silicosis.

### Renal Disease

An interest in the possible relationship between renal disease and silicosis dates from the findings in a 1933 study of the causes of death in men in England and Wales. There were 3,158 deaths in those with occupations implying silica exposure, and the death rate from chronic nephritis was 45% greater than expected from age-specific rates in the reference population (136). A case-control study of men with end-stage renal disease found elevated odds ratios for "regular occupational exposures to solvents or silica" but characterized evidence of other silica-related renal disease as limited to case reports (137). Case reports have also shown an association between acute silicoproteinosis and a glomerular injury (138, 139).

## RECOMMENDATIONS OF EDUCATION, PREVENTION, RESPIRATORY PROTECTION, AND FURTHER RESEARCH

In the absence of effective specific treatment for silica-related diseases, the only approach remains primary prevention, i.e., control of exposure to respirable silica (140). Public awareness of the hazards of silica is currently low in many countries, including the United States, and improved preventive and educational measures are needed (141, 142). Many workers and employers have not heard of silicosis and are unaware of how it is acquired. Prevention involves anticipation that a hazardous exposure might occur, the evaluation of the circumstances leading to exposure to crystalline silica, and the use of effective controls. Proved methods of control include engineering controls such as dust suppression, process isolation, and ventilation; administrative controls include substitution of alternative abrasives in blasting; and, as a temporary and last resort, the use of personal respiratory protection. Education plays a critical role in alerting employers and workers to the potential for a problem, selection of appropriate workplace controls, and in assisting ongoing surveillance efforts. Education should begin in vocational programs for high-risk occupations (such as apprenticeship programs for heavy equipment operators) and continue at the work site as part of training for hazardous jobs and ongoing prevention programs. Yet in the absence of a comprehensive preventive effort, education alone may not be effective.

Because of the widespread presence of naturally found silica-containing minerals, it is not practicable to label silica everywhere it occurs. It may, however, be practicable to label equipment designed to cut and drill rock, and to set industrywide standards for work practices (such as those currently in operation for exposure to coal dust in coal mining) (6). High-risk materials (such as ground silica for industrial use and other respirable forms of crystalline silica) should always be labeled with easily comprehended messages as to the hazard. In many cases, safer materials can be substituted for silica-containing materials. Because silica

sandblasting has been such a persistently high-risk occupation, and because alternative abrasive materials without silica are in wide use, available, and of equivalent cost, restricting the use of silica-containing abrasives for abrasive blasting is strongly recommended.

Reporting of all cases of silicosis is a legal responsibility of physicians in many countries, states, and provinces, and active investigation of all cases may be important in detecting workplaces with excessive exposures (143). In the United States, the appropriate agency to which to report new cases is usually the local Health Department. If ongoing overexposure is suspected in others at the same workplace, a health inspection by the local office of the federal or state Occupational Safety and Health Administration may be requested by the physician. In addition, a health hazard evaluation by NIOSH may be requested by workers or employers (144).

The technology to control silica dust exposure is simple and relatively inexpensive. In mining, cutting, and drilling, the addition of water to the cutting surface effectively reduces dust levels in the breathing air of those working nearby. Where a dry process is necessary, enclosures or local exhaust ventilation can be designed to carry dust to a filtering apparatus where it can be recycled or disposed of safely. Although they are the least efficacious preventive measure, industrial respirators may effectively lower individual exposures. The use of respirators requires that silica levels in the air have been measured to establish what type of respirator is needed to provide effective protection. Respirators with a variety of protection factors appropriate to progressively higher air levels of silica dust have been certified by NIOSH laboratories in conditions of inadequately controlled air levels (145). Physicians who certify workers for respirator use should be aware of the kind of device to be used in determining whether the individual can perform the requirements of the job both without and with the respirator (146).

The paucity of scientific investigation of exposure-response relationships is striking for an occupational disease with such worldwide morbidity and mortality. The conflicting exposure-response estimates in the literature point to the need of further study of groups whose exposures have been well-characterized and who are followed up after they have left the silica-exposed industries as a basis for reconsideration of existing standards. Such research will provide a better scientific basis for establishing preventive exposure regulations. Under circumstances where preventive approaches do not succeed, a better understanding of the biologic mechanisms involved in causing and sustaining silicosis may also lead to effective means of halting or slowing the progression of established disease in the future.

*This statement was prepared by an Ad-Hoc committee of the Scientific Assembly on Environmental and Occupational Health. The members of the committee are:*

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