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Request for Assistance in...

Preventing Silicosis and Deaths From Sandblasting

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[92-102sum.pdf \(Worker/Employer Summary Sheet Only\)](#) (233 kb) 1 page
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WARNING!

Abrasive blasting with sands containing crystalline silica can cause serious or fatal respiratory disease.

The National Institute for Occupational Safety and Health (NIOSH) requests assistance in preventing silicosis and deaths in workers exposed to respirable crystalline silica during sandblasting. Sandblasters, exposed coworkers, and their employers urgently need information about the respiratory hazards associated with sandblasting. Your assistance in this effort will help prevent silicosis and death, a national goal for health promotion and disease prevention stated in *Healthy People 2000* [DHHS 1990].

The Alert describes 99 cases of silicosis from exposure to crystalline silica during sandblasting. Of the 99 workers reported, 14 have already died from the disease, and the remaining 85 may die eventually from silicosis or its complications. NIOSH requests that editors of trade journals, safety and health officials, labor unions, and employers bring the recommendations in this Alert to the attention of all workers who are at risk.

BACKGROUND

Description and Uses of Abrasive Blasting

Abrasive blasting involves forcefully projecting a stream of abrasive particles onto a surface, usually with compressed air or steam. Because silica sand is commonly used in this process, workers who perform abrasive blasting are often known as sandblasters. Tasks performed by sandblasters include the following:

- Cleaning sand and irregularities from foundry castings
- Cleaning and removing paint from ship hulls, stone buildings, metal bridges, and other metal surfaces
- Finishing tombstones, etching or frosting glass, and performing certain artistic endeavors.

Description of Silicosis

When workers inhale the crystalline silica* used in abrasive blasting, the lung tissue reacts by developing fibrotic nodules and scarring around the trapped silica particles [Silicosis and Silicate Disease Committee 1988]. This fibrotic

condition of the lung is called silicosis. If the nodules grow too large, breathing becomes difficult and death may result. Silicosis victims are also at high risk of developing active tuberculosis [Myers et al. 1973; Sherson and Lander 1990; Bailey et al. 1974].

The silica sand used in abrasive blasting typically fractures into fine particles and becomes airborne (see Figure 1). Inhalation of such silica appears to produce a more severe lung reaction than silica that is not freshly fractured [Vallyathan et al. 1988]. This factor may contribute to the development of acute and accelerated forms of silicosis among sandblasters.



Figure 1. Sandblaster working in the dusty atmosphere created by airborne particles of silica sand.

Number of Exposed Workers

Estimates indicate that more than 1 million U.S. workers are at risk of developing silicosis and that more than 100,000 of these workers are employed as sandblasters [Shaman 1983]. Approximately 59,000 of the 1 million workers exposed to crystalline silica will eventually develop silicosis [Shaman 1983]. No published estimates indicate the number of sandblasters who will develop silicosis, but a 1936 study in Great Britain [Merewether 1936] reported that 5.4% of a population of sandblasters (24 of 441) died from silicosis or silicosis with tuberculosis in a 3.5-year period. The National Occupational Exposure Survey indicates that the construction industry employs the largest number of sandblasters, with the highest proportion in the special trades industries [NIOSH 1988b, c; 1990b].

Respiratory Protection Practices

Acute silicosis is less common today than it was in the 1930s because engineering controls are used to reduce exposure to respirable crystalline silica and because the use of alternative abrasives is increasing. However, data indicate that most abrasive blasters continue to work without adequate respiratory protection [NIOSH 1974a]. In addition, workers adjacent to abrasive blasting operations (for example, painters, welders, and laborers) often wear no respiratory protection [NIOSH 1990b].

Ventilation controls for reducing crystalline silica exposures are not used in most industries [NIOSH 1990b]. Samimi et al. [1974] found that even in short-term sandblasting operations (less than 2½ hours of blasting during an 8-hour workday), the average concentration of crystalline silica was 764 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), with an average silica content of 25.5%. This average dust concentration was twice the 1974 standard of the Occupational Safety and Health Administration (OSHA).

In a 1974 study of respiratory protection practices during abrasive blasting [NIOSH 1974a], the protection factors for supplied-air respirators with helmets ranged from 1.9 to 3,750. This wide range was attributed to the varied conditions of the equipment rather than to the superiority of any brand. Maintenance was universally poor or nonexistent, and the persons responsible for selective respiratory protection for abrasive blasting were inadequately informed about the proper use and maintenance of such equipment. The higher protection factors were associated with high rates of helmet air flow, but these high flow rates increased noise levels as a result of air turbulence. The study also indicated that the air inlets were too noisy and that the blasters' helmets tended to fall from the wearers' shoulders when they stooped.

Prohibition of Silica in Abrasive Blasting

Because of the high risk for silicosis in sandblasters and the difficulty in controlling exposures, the use of crystalline silica for blast cleaning operations was prohibited in Great Britain in 1950 [Factories Act 1949] and in other European countries in 1966 [ILO 1972]. In 1974, NIOSH recommended that silica sand (or other substances containing more than 1% free silica) be prohibited as abrasive blasting material and that less hazardous materials be used in blasting operations [NIOSH 1974b].

CURRENT EXPOSURE LIMITS

The current OSHA permissible exposure limit (PEL) for respirable crystalline silica (quartz) is $100 \mu\text{g}/\text{m}^3$ as an 8-hour time-weighted average (TWA) [29 CFR** 1910.1000]. The NIOSH recommended exposure limit (REL) for respirable crystalline silica is $50 \mu\text{g}/\text{m}^3$ as a TWA for up to 10 hours/day during a 40-hour workweek [NIOSH 1974b]. This REL is intended to prevent silicosis. However, evidence indicates that crystalline silica is a potential occupational carcinogen [NIOSH 1988a; IARC 1987; DHHS 1991], and NIOSH is reviewing the data on carcinogenicity.

HEALTH EFFECTS OF CRYSTALLINE SILICA EXPOSURE

A worker may develop any of three types of silicosis, depending on the airborne concentration of crystalline silica:

- Chronic silicosis, which usually occurs after 10 or more years of exposure to crystalline silica at relatively low concentrations
- Accelerated silicosis, which results from exposure to high concentrations of crystalline silica and develops 5 to 10 years after the initial exposure
- Acute silicosis, which occurs where exposure concentrations are the highest and can cause symptoms to develop within a few weeks to 4 or 5 years after the initial exposure [Peters 1986; Ziskind et al. 1976].

Silicosis (especially the acute form) is characterized by shortness of breath, fever, and cyanosis (bluish skin); it may often be misdiagnosed as pulmonary edema (fluid in the lungs), pneumonia, or tuberculosis. Severe mycobacterial or fungal infections often complicate silicosis and may be fatal in many cases [Ziskind et al. 1976; Owens et al. 1988; Bailey et al. 1974]. Fungal or mycobacterial infections are believed to result when the lung scavenger cells (macrophages) that fight these diseases are overwhelmed with silica dust and are unable to kill mycobacteria and other organisms [Allison and Hart 1968; Ng and Chan 1991]. About half of the mycobacterial infections are caused by *Mycobacterium tuberculosis*, with the other half caused by *M. kansasii* and *M. avium-intracellulare* [Owens et al. 1988]. *Nocardia* and *Cryptococcus* may also cause lung infections in silicosis victims [Ziskind et al. 1976]. Investigations usually show the lungs to be filled with silica crystals and a protein material [Owens et al. 1988; Buechner and Ansari 1969].

CASE REPORTS

Case No. 1--One Death

In January 1992, the Ohio Department of Health responded to a physician's report of the death of a 55-year-old worker with accelerated silicosis and associated *M. kansasii* infection [ODH 1992]. The man was a sandblaster at a metal preparation shop and was reported to have been sandblasting for 10 years, possibly without adequate respiratory protection.

The Ohio Department of Health conducted a site visit at the metal preparation shop. Blasting had always been done manually in an enclosed room and was considered a necessary step to remove the "onion peel skin" that developed after heat stressing the metal.

The shop owner employed 17 workers and operated 3 shifts. All shifts had a designated sandblaster who was given a supplied-air respirator with a hood. Sandblasting was performed for about 6 hours on each shift. During the remainder of the shift, the sandblaster wore only a disposable particulate respirator and shoveled the used sand into a floor pit for recycling. Workers reported that coworkers had developed problems while working as sandblasters and that the

employer typically hired six to seven new sandblasters each year to replace those who quit.

A full-shift personal sample collected outside the sandblaster's helmet indicated that the potential exposure to respirable crystalline silica was greater than 200 times the NIOSH REL of $50 \mu\text{g}/\text{m}^3$ [NIOSH 1974b]. The type of respirator worn during this blasting operation had an assigned protection factor (APF) of 25 [NIOSH 1987b]. The APF, which is discussed further in the section on respiratory protection (p. 9), is the minimum anticipated protection provided by a properly functioning respirator or class of respirators to a given percentage of properly fitted and trained users. Thus, wearing a respirator with an APF of 25 would theoretically provide adequate protection from hazardous concentrations up to 25 times the NIOSH REL--far less protection than needed for a potential exposure greater than 200 times the REL.

An area air sample collected inside the blasting room contained about 500 times the NIOSH REL for crystalline silica. An air sample collected immediately outside the blasting room contained 8 times the NIOSH REL, indicating poor containment of the dust by the blasting room (which was not sealed) and dangerous dust leakage from the sand-handling equipment.

Other problems were noted with regard to air-flow pressures at the helmet, improper ventilation, sporadic respirator use, and dust collection. The hopper outlet for the dust collector dumped fine dust directly onto the plant floor. This dust accumulated and exposed many workers as it was dispersed throughout the plant. A currently employed sandblaster stated that although the exposure was a nuisance, he considered the dust to be part of the job.

Case No. 2--One Death

In November 1988, a physician in western Texas reported three cases of sandblaster's silicosis to the Ector County Health Department [CDC 1990]. All three patients had been employed at a facility where they sandblasted oil-field drilling pipes. One of the workers, a 34-year-old man, subsequently died as a result of acute silicosis.

Following a later report by the physician in January 1989, the Ector County Health Department and the Texas Department of Health contacted local physicians and identified seven additional sandblasters who had suffered from silicosis since 1985. Of the 10 workers identified, 9 had worked at the same facility, which employed approximately 60 persons.

An investigation by the county and State health departments included a review of personal and occupational histories from each worker. Local radiologists evaluated chest X-rays. For four cases, a B reader*** also reviewed each patient's most recent chest X-ray for evidence of pneumoconiosis using the 1980 ILO guidelines [ILO Committee on Pneumoconiosis 1981]. The Texas Department of Health reviewed lung tissue pathology reports and conducted an environmental survey of the plant where nine of the workers had been employed.

Each of the 10 workers had histories of occupational exposure to silica and a chest X-ray consistent with pneumoconiosis; 8 had a lung tissue pathology report of silicotic nodules or acute silicosis [Silicosis and Silicate Disease Committee 1988]. All were Hispanic males aged 24 to 50 at the time of diagnosis. Seven workers were under age 30. Although tuberculosis was considered in all of the reported patients (three of whom had reactive tuberculin skin tests), all sputum and tissue samples from all patients were negative for *M. tuberculosis*.

All 10 workers had used sandblasting machinery. Duration of exposure to sandblasting ranged from 18 months to 8 years (mean: 4.5 years). Nine workers reported no previous silica exposure; the remaining worker had sandblasted oil-field drilling equipment for 3 years before working at the originally identified facility for 5 years.

The sandblasting process at this facility required that a blasting rod using an equal mixture of flint and garnet (20.5% crystalline silica) be passed through the drilling pipe to remove contaminants and to prepare the interior surface for a new protective plastic coating. Although the sandblasting operation was enclosed by blasting cabinets connected to exhaust systems, the cabinets were in poor repair and permitted clouds of dust to be released throughout the work area. Protective booths intended to reduce exposures drew air from areas with substantial silica contamination. Workers manually shoveled the used sandblasting material into the machinery for reuse.

In November 1988, air samples from personal breathing zones documented respirable crystalline silica exposures of 400 to 700 $\mu\text{g}/\text{m}^3$ for workers in the sandblasting area. These data were consistent with results reported by OSHA during a similar environmental inspection in which exposures substantially exceeded the current OSHA PEL (100 $\mu\text{g}/\text{m}^3$ for respirable silica [29 CFR 1910.1000]). Supplied-air respirators had not been used during sandblasting, and workers reported wearing only disposable particulate respirators.

Case No. 3--One Death

A 49-year-old nonsmoker who had worked as a sandblaster for 6 years came to a Louisiana hospital complaining of difficult breathing, a nonproductive cough, lack of appetite, fever, and a 20-lb weight loss [Owens et al. 1988].

A physical exam, chest X-rays, and sputum stain for bacteria led to a diagnosis of chronic silicosis and a bacterial pneumonia. Although the patient was treated with oxygen and antibiotics, he continued to deteriorate, and a breathing machine was necessary. A lung biopsy showed that the smallest cavities of the lungs were filled with a material composed of fat, protein, and silica particles. Further testing of the sputum revealed that the patient was suffering from tuberculosis, and an appropriate therapy was started. However, the patient continued to require a breathing machine and died on the 20th hospital day.

Case No. 4--Three Deaths

Acute silicosis developed in four men (aged 23, 38, 38, and 47) employed as tombstone sandblasters at a single factory for an average of 3 years. Three of the four men are known to have died of the disease [Suratt et al. 1977]. None of them showed any evidence of tuberculosis.

Investigations revealed that the sandblasters worked in enclosed but vented blasting chambers. Although supplied-air respirators were available to the workers, investigators indicated that they wore only negative-pressure, half-mask respirators with disposable filters. Workers in the blasting room were being exposed outside the mask to 98% crystalline silica sand at a concentration of 15 million particles per cubic foot (5 times the 1974 OSHA standard). A later investigation indicated that workers were using the supplied-air respirators but that they were being exposed to crystalline silica at a concentration of 3,400 $\mu\text{g}/\text{m}^3$ as a TWA (18 times the 1974 OSHA standard).****

Case No. 5--Eight Deaths

Eighty-three sandblasters in Louisiana were diagnosed as having silicosis [Bailey et al. 1974]. Twenty-two of the 83 had complicating mycobacterial infections. The average age of the patients was 44, with an average silica exposure time of fewer than 10 years. Eight of these patients are known to have died of respiratory failure caused by silicosis.

Almost all of the sandblasters prepared surfaces for painting and then painted them. Most of the workers wore supplied-air respirators, although the hoods were often unattached to an external air supply. When the sandblasting was completed, the workers removed their hoods and immediately began painting, even though large amounts of silica dust were still suspended in the air.

CONCLUSIONS

This Alert illustrates the continuing conditions in the American workplace that lead inevitably to the development of silicosis. Four conditions are characteristic of sandblasting worksites where silicosis is a problem:

- Failure to substitute less toxic abrasive blasting materials
- Inadequate engineering controls (such as ventilation) and work practices
- Inadequate respiratory protection for workers
- Failure to conduct adequate medical surveillance programs

RECOMMENDATIONS

NIOSH recommends the following measures to reduce crystalline silica exposures in the workplace and prevent silicosis and silicosis-related deaths:

1. Prohibit silica sand (or other substances containing more than 1% crystalline silica) as an abrasive blasting material and substitute less hazardous materials.
2. Conduct air monitoring to measure worker exposures.
3. Use containment methods such as blast-cleaning machines and cabinets to control the hazard and protect adjacent workers from exposure.
4. Practice good personal hygiene to avoid unnecessary exposure to silica dust.
5. Wear washable or disposable protective clothes at the worksite; shower and change into clean clothes before leaving the worksite to prevent contamination of cars, homes, and other work areas.
6. Use respiratory protection when source controls cannot keep silica exposures below the NIOSH REL.
7. Provide periodic medical examinations for all workers who may be exposed to crystalline silica.
8. Post signs to warn workers about the hazard and to inform them about required protective equipment.
9. Provide workers with training that includes information about health effects, work practices, and protective equipment for crystalline silica.
10. Report all cases of silicosis to State health departments and to OSHA or the Mine Safety and Health Administration (MSHA).

These recommendations are discussed briefly in the following subsections.

Use of Alternative Abrasives

The risk of silicosis is high in workers exposed to abrasive blasting with silica, and the hazard is difficult to control. NIOSH has therefore recommended since 1974 that silica sand (or other substances containing more than 1% crystalline silica) be prohibited as abrasive blasting material [NIOSH 1974b, NIOSH 1990a]. A variety of materials (corundum, glass beads, pumice, sawdust, slags, steel grit and shot, and walnut shells) are available as alternative blasting media [NIOSH 1974c; Mackay et al. 1980; Stettler et al. 1988]. However, no comprehensive studies have been conducted to evaluate the health effects of these substitute materials. Until comprehensive data are available, engineering controls and personal protective equipment should be used with any of the alternative abrasives.

In addition to the health hazards of abrasive blasting materials, the finely fractured particles of material being removed (lead paint, for example) may also create health risks for workers [NIOSH 1991a].

Air Monitoring

Air monitoring should be performed to measure worker exposure to airborne crystalline silica and to provide a basis for selecting engineering controls. Air monitoring should be performed as needed to measure the effectiveness of controls. Air samples should be collected and analyzed according to NIOSH Method Nos. 7500 and 7602 [NIOSH 1984] or their equivalent.

Containment Methods

Blast-Cleaning Machines and Cabinets

Whenever possible, blasting should be done in enclosed blast-cleaning machines or cabinets. These devices permit operators to stand outside the cabinet and direct the stream of abrasive material inside with the hands and arms in gloved armholes.

Abrasive Blasting Rooms

Abrasive blasting rooms contain the hazard and protect adjacent workers from exposure. However, such rooms may increase the risk for blasters, since they must work inside the enclosure in high concentrations of hazardous blasting material. Blasting rooms must be ventilated to reduce these concentrations and to increase visibility. A supplied-air respirator is required for any blaster working inside a blasting room (see [Respiratory Protection](#) below).

Portable Blast-Cleaning Equipment

Portable blast-cleaning equipment presents particularly serious health problems because engineering controls are rarely used. Curtains can be used as temporary containment structures to reduce the hazard to adjacent workers and the general public. However, such temporary structures often leak and may allow large amounts of debris to escape. As with abrasive blasting rooms, these structures should be ventilated to reduce concentrations of hazardous materials and to increase visibility. During work inside the containment, a supplied-air respirator is required for the blaster.

Ventilation of Containment Structures

All containment structures should be ventilated to maintain a continuous air flow and prevent any leakage of dust to the outside. Exhaust air should be discharged to the outside through an appropriate dust collector. The dust collector should be set up so that accumulated dust can be removed without contaminating work areas. Detailed requirements are listed in the OSHA ventilation standard [29 CFR 1910.94].

Personal Hygiene

The following personal hygiene practices are important elements of any program for protecting workers from exposure to crystalline silica and other contaminants such as lead during abrasive blasting operations [NIOSH 1991a].

- All sandblasters should wash their hands and faces before eating, drinking, or smoking.
- Sandblasters should not eat, drink, or use tobacco products in the blasting area.
- Workers should shower before leaving the worksite.
- Workers should park their cars where they will not be contaminated with silica and other substances such as lead.

Protective Clothing

The following measures should be taken to assure that the blasters' dusty clothes do not contaminate cars, homes, or worksites other than the blasting area:

- Workers should change into disposable or washable work clothes at the worksite.
- Workers should change into clean clothes before leaving the worksite.

Respiratory Protection

[\[Return to "Abrasive Blasting Rooms"\]](#)

Respirators should not be used as the only means of preventing or minimizing exposures to airborne contaminants. Effective source controls such as substitution, automation, containment, local exhaust ventilation, and good work practices should be implemented to minimize worker exposure to silica dust. NIOSH prefers such measures as the primary means of protecting workers. However, when source controls cannot keep exposures below the NIOSH REL, controls should be supplemented with the use of respiratory protection during abrasive blasting.

When respirators are used, the employer must establish a comprehensive respiratory protection program as outlined in the *NIOSH Guide to Industrial Respiratory Protection* [NIOSH 1987a] and as required in the OSHA respiratory protection standard [29 CFR 1910.134]. Important elements of this standard are

- an evaluation of the worker's ability to perform the work while wearing a respirator,
- regular training of personnel,
- periodic environmental monitoring,
- respirator fit testing,
- maintenance, inspection, cleaning, and storage, and
- selection of proper NIOSH-approved respirators.

The respiratory protection program should be evaluated regularly by the employer.

NIOSH recommends that workers wear the type CE abrasive blasting respirator operated in the positive-pressure mode (APF of 2,000) during abrasive blasting operations that involve crystalline silica. For other operations, Table 1 lists the minimum respiratory equipment required to meet the NIOSH REL for crystalline silica under given conditions. Workers should wear the most protective respirator that is feasible and consistent with the tasks to be performed. For additional information about respirator selection, consult the NIOSH Respirator Decision Logic [NIOSH 1987b]. Workers should use only those respirators that have been certified by NIOSH and MSHA [NIOSH 1991b].

Table 1.--NIOSH-recommended respiratory protection for workers exposed to respirable crystalline silica

Condition	Minimum respiratory protection* required to meet the Condition NIOSH REL for crystalline silica (50 µg/m ³)**
Less than or equal to 500 µg/m ³ (10 x REL) ***	Any air-purifying respirator with a high-efficiency particulate filter
Less than or equal to 1,250 µg/m ³ (25 x REL)	Any powered, air-purifying respirator with a high-efficiency particulate filter, or Any supplied-air respirator equipped with a hood or helmet and operated in a continuous-flow mode (for example, type CE abrasive blasting respirators operated in the continuous-flow mode)
Less than or equal to 2,500 µg/m ³ (50 x REL)	Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter, or Any powered, air-purifying respirator with a tight-fitting facepiece and a high-efficiency particulate filter
Less than or equal to 50,000 µg/m ³ (1,000 x REL)	Any supplied-air respirator equipped with a half-mask and operated in a pressure-demand or other positive-pressure mode
Less than or equal to 100,000 µg/m ³ (2,000 x REL)	Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode (for example, a type CE abrasive blasting respirator operated in a positive-pressure mode)
Planned or emergency entry into environments containing unknown concentrations or concentrations less than or equal to 500,000 µg/m ³ (10,000 x REL)	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode,**** or Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive-pressure mode****
Firefighting	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode****
Escape only	Any air-purifying, full-facepiece respirator with a high-efficiency particulate filter, or Any appropriate escape-type, self-contained breathing apparatus

* Only NIOSH/MSHA-approved equipment should be used. [\[Return to top of table\]](#)

** These recommendations are intended to protect workers from silicosis; only the most protective respirators are recommended for used with carcinogens. [\[Return to top of table\]](#)

*** Assigned protection factor (APF) times the NIOSH REL. The APF is the minimum anticipated level of protection provided by each type of respirator. [\[Return to body of table\]](#)

**** Most protective respirators. [\[Return to body of table\]](#)

Medical Monitoring

Medical examinations should be available to all workers who may be exposed to crystalline silica. Such examinations should occur before job placement and at least every 3 years thereafter [NIOSH 1974b]. More frequent examinations (for example, annual) may be necessary for workers at risk of acute or accelerated silicosis. Examinations should include at least the following items:

- A medical and occupational history to collect data on worker exposure to crystalline silica and signs and symptoms of respiratory disease
- A chest X-ray classified according to the 1980 International Labour Office (ILO) Classification of Radiographs of the Pneumoconioses [ILO 1981]
- Pulmonary function testing (spirometry)
- An annual evaluation for tuberculosis [ATS/CDC 1986]

Warning Signs

Signs should be posted to warn workers about the hazard and specify any protective equipment required (for example, respirators). The sample sign in Figure 2 contains the information needed for a silica work area where respirators are required.

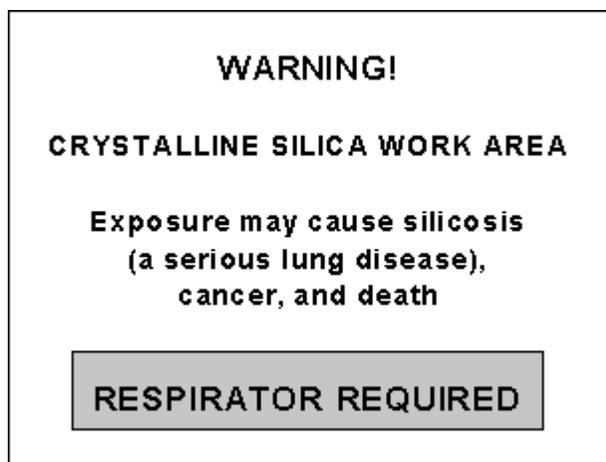


Figure 2. Sample of warning sign for work areas contaminated with crystalline silica.

Training

Workers should receive training [29 CFR 1926.21] that includes the following:

- Information about the potential adverse health effects of silica exposure
- Material safety data sheets for silica, alternative abrasives, or other hazardous materials [29 CFR 1926.59]
- Instruction about obeying signs that mark the boundaries of work areas containing crystalline silica
- Information about safe handling, labeling, and storage of toxic materials [30 CFR 56.20012, 56.16004, 57.20012, 77.208]
- Discussion about the importance of engineering controls, personal hygiene, and work practices in reducing crystalline silica exposure
- Instruction about the use and care of appropriate protective equipment (including protective clothing and respiratory protection)

Surveillance and Disease Reporting

NIOSH encourages reporting of all cases of silicosis to the State health departments and to OSHA or MSHA. To

enhance the uniformity of reporting, NIOSH has developed reporting guidelines and a surveillance case definition for silicosis (see Appendix). This definition and these guidelines are recommended for surveillance of work-related silicosis by State health departments and regulatory agencies receiving reports of cases from physicians and other health care providers [CDC 1990].

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We greatly appreciate your assistance in protecting the lives of American workers.

[signature]

J. Donald Millar, M.D., D.T.P.H. (Lond.)

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Director, National Institute for Occupational Safety and Health

Centers for Disease Control

NOTES

* Also referred to as free silica, or SiO₂; includes quartz, cristobalite, tridymite, and tripoli. [\[Return to main text\]](#)

** Code of Federal Regulations. See CFR in references. [\[Return to main text\]](#)

*** A physician certified by NIOSH to detect pneumoconiosis on X-rays using International Labour Office (ILO) guidelines. [\[Return to main text\]](#)

**** The 1974 OSHA standard refers to both million particles per cubic foot and $\mu\text{g}/\text{m}^3$. [\[Return to main text\]](#)

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APPENDIX

SURVEILLANCE GUIDELINES FOR STATE HEALTH DEPARTMENTS: SILICOSIS_{*,**}

Reporting Guidelines

State health departments should encourage physicians, including radiologists and pathologists, as well as other health-care providers, to report all diagnosed or suspected cases of silicosis. These reports should include persons with:

- A. A physician's provisional or working diagnosis of silicosis, OR
- B. A chest radiograph interpreted as consistent with silicosis, OR
- C. Pathologic findings consistent with silicosis.

State health departments should collect appropriate clinical, epidemiologic, and workplace information on persons reported with silicosis as needed to set priorities for workplace investigations.

Surveillance Case Definition

- A. 1. History of occupational exposure to airborne silica dust,_{**} AND
 - A. 2. Chest radiograph or other imaging technique interpreted as consistent with silicosis,_{***}
- OR
- B. Pathologic findings characteristic of silicosis. _{****}

* Reprinted from CDC [1990], p. 436. [[Return to top of Appendix](#)]

** Exposure settings associated with silicosis are well characterized and have been summarized in several reviews [Ziskind et al. 1976; Peters 1986]. The induction period between initial silica exposure and development of radiographically detectable nodular silicosis is usually >10 years. Shorter induction periods are associated with heavy exposures, and acute silicosis may develop within 6 months to 2 years following massive silica exposure. [[Return to top of Appendix](#)]

*** Cases can be classified as simple or complicated. Simple silicosis is present if the largest opacity is <1 cm in diameter. Complicated silicosis (also known as progressive massive fibrosis [PMF]) is present if the largest opacity is greater than or equal to 1 cm in diameter. Common radiographic findings of nodular silicosis include multiple, bilateral, and rounded opacities in the upper lung zones; other patterns have been described. Since patients may have had mixed dust exposure, irregular opacities may be present or even predominant. Radiographs interpreted by NIOSH-certified "B" readers should have profusion categories of 1/0 or greater by the International Labour Organization classification system [ILO Committee of Pneumoconiosis 1981]. A bilateral alveolar filling pattern is characteristic of acute silicosis and may be followed by rapid development of bilateral small or large opacities. [[Return to body of Appendix](#)]

**** Characteristic lung tissue pathology [Silicosis and Silicate Disease Committee 1988] in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent

particles seen under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate of the alveolar walls. [\[Return to body of Appendix\]](#)

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