

OSHA Instruction – National Emphasis Program – Crystalline Silica

Appendix A: Background Information on Silica

This Appendix provides an overview of the following silica-related topics:

- The forms and sources of silica;
- Common industrial uses of silica and workplaces with silica exposure;
- History of silicosis; and health effects associated with exposure.

The reference list at the end of this document provides many sources that may prove useful to those interested in a more in-depth treatment of these topics.

Introduction

“Silica,” is a term which refers broadly to the mineral compound silicon dioxide (SiO₂). Silica can be crystalline or amorphous. Crystalline silica is significantly more hazardous to employees than amorphous silica. In addition to causing the disabling and irreversible lung disease known as silicosis, crystalline silica has been classified as a human carcinogen by the International Agency for Research on Cancer (IARC) [IARC, 1997]. As it is typically used in this document, “silica” refers specifically to crystalline silica. Crystalline silica is characterized by a large scale, repeating pattern of silicon and oxygen atoms, as distinguished from the more random arrangement found in amorphous silica. Abundant in the earth’s crust, crystalline silica is a basic component of most classes of rock. Naturally-occurring forms of amorphous silica include diatomaceous earth (the skeletal remains of marine organisms) and vitreous silica or volcanic glass [Markowitz and Rosner, 1995; Davis, 1996].

Forms and Sources of Crystalline Silica

Crystalline silica occurs in three primary mineralogical forms, or polymorphs—quartz, cristobalite, and tridymite. Silica is also called “free silica,” to distinguish it from the silicates, which are minerals containing silicon dioxide bound to one or more cations [Beckett et al., 1997].

Quartz is by far the most common form of naturally-occurring silica [Davis, 1996; IARC, 1997]. Cristobalite and tridymite, which are molecularly identical to quartz, are distinguishable by their unique crystalline structures. They are less stable than quartz, thus accounting for the dominance of the quartz form. Quartz itself exists as either of two sub-polymorphs, alpha-quartz (also known as low quartz), and beta-quartz (high quartz). Alpha-quartz is the thermodynamically stable form of crystalline silica and accounts for the overwhelming portion of naturally-occurring crystalline silica [IARC, 1997].

Quartz is a major component of soils and is readily found in both sedimentary and igneous rocks, although the quartz content varies greatly from one rock type to another. For instance, granite contains on average about 30 percent quartz, and shales contain about 20 percent quartz. Natural stone, such as beach sand or sandstone, may be nearly pure quartz [IARC, 1997; Davis, 1996].

Cristobalite and tridymite are natural constituents of some volcanic rock, and man-made forms result from direct conversion of quartz or amorphous silica that has been subjected to high temperature or pressure. Diatomaceous earth, composed of amorphous silica, crystallizes during heating (calcining), yielding a calcined product that contains as much as 75 percent cristobalite.

Cristobalite is also found in the superficial layers of refractory brick that has been repeatedly subjected to contact with molten metal [Markowitz and Rosner, 1995; Ganter, 1986; Cheng et al., 1992; Bergen et al., 1994].

Major Industrial Sources of Crystalline Silica Exposure

Crystalline silica is an important industrial material and occupational exposure occurs across a broad range of industries, including mining, manufacturing, construction, maritime, and agriculture (see Appendix B for a listing of industries and Standard Industrial Classifications with potential for significant occupational exposure). Processes associated historically with high rates of silicosis include sandblasting, sand-casting foundry operations, mining, tunneling, and granite cutting.

Crystalline silica, in the form of finely ground quartz sand as an abrasive blasting agent, is used to remove surface coatings prior to repainting or treating, a process that typically generates extremely high levels of airborne respirable crystalline silica. A 1992 report published by the National Institute for Occupational Safety and Health (NIOSH) estimates that there are more than one million U.S. employees who are at risk for developing silicosis, and of these employees, more than 100,000 are employed as sandblasters. Abrasive blasting is performed in a wide variety of different industries; the construction industry employs the largest number of employees as abrasive blasters, concentrated in the special trades [NIOSH 92-102; CDC, 1997].

In addition to abrasive blasting, construction employees perform numerous other activities that may result in significant silica exposure, including tunnel and road construction, excavation and earth moving, masonry and concrete work, and demolition [IARC, 1997]. Foundry employees, primarily in iron and steel foundries, may be exposed to crystalline silica throughout the metalcasting process, including the production of sand-based molds and cores, shakeout and knockout, and finishing and grinding operations.

Crystalline silica, primarily as quartz, is a major component of the sand, clay, and stone raw materials used to manufacture a variety of products, including concrete, brick, tile, porcelain, pottery, glass, and abrasives. The powdered form of quartz, also called silica flour, is used in the manufacture of fine china and porcelain. Finely ground crystalline silica is also used as a functional filler in the manufacture of paints, plastics, and other materials. The rock crystal form of quartz is of great value to the electronics industry.

Agricultural employees perform activities, including plowing and harvesting, that may generate elevated silica levels. However, OSHA does not regulate crystalline silica exposure on farms with fewer than ten employees and exposure data for this population is lacking [Linch et al., 1998]. On the other hand, OSHA does regulate crystalline silica exposure in the agricultural services sector, and crystalline silica exposures have been documented in the sorting, grading, and washing areas of food processing operations for crops such as potatoes and beans.

Cristobalite, as calcined diatomaceous earth, is used as a filler in materials such as paints and as a filtering media in food and beverage processing. Maintenance and trades personnel who repair and replace refractory brick linings of rotary kilns and cupola furnaces may be exposed to significant levels of quartz, as well as cristobalite and tridymite. These kilns and furnaces are found in glass, ceramics, and paper manufacturing facilities as well as foundries [Markowitz and Rosner, 1995].

The industries described above, (see Appendix B) represent the major industrial sources of crystalline silica exposure. However, there are numerous other operations in which silica may be used or otherwise encountered, and it is important to be aware of the risk of silicosis in industries not previously recognized to be at risk.

History of Silicosis

Silicosis is one of the world's oldest known occupational diseases; reports of employees with the disease date back to ancient Greece. By 1800, there were numerous common names for the lung disease now known as silicosis. The names frequently referred to the affected laborers' trade, such as grinders' asthma, grinders' rot, masons' disease, miners' asthma, miners' phthisis, potters' rot, sewer disease, and

stonemasons' disease. Despite its different names through the centuries, silicosis is a single disease with a single cause—exposure to respirable crystalline silica dust.

During the 1920s, the health risks of the “dusty” trades, in particular the granite industry, emerged as a significant public health concern, and by 1930 silicosis was considered the most serious occupational disease in the United States. During the 1930s and 1940s, the granite industry was the focus of a major effort to alleviate dusty conditions and create a safer working environment [Rosner and Markowitz, 1994]. However, as the more extreme silica hazards were brought under control, attention shifted away from silica to other occupational health hazards. Nonetheless, as the studies described below indicate, in recent decades silicosis has continued to pose a significant health threat to employees in a variety of occupations, including but not limited to construction, foundries, and sandblasting. It is important to be aware of the possible risk of silicosis in workplaces not previously recognized to be at risk.

- Silicosis was listed as the underlying cause of death in 6,322 fatalities in the United States from 1968 through 1990, according to a study reviewing multiple-cause-of-death data from the National Center for Health Statistics. The total number of U.S. deaths with mention of silicosis for that period was 13,744. The study found that 69 percent of the deaths due to silicosis were concentrated in 12 states: California, Colorado, Florida, Illinois, Michigan, New Jersey, New York, Ohio, Pennsylvania, Virginia, West Virginia, and Wisconsin. The construction industry accounted for more than 10 percent of the total silicosis-related deaths, and iron and steel foundries accounted for another 5.4 percent [Bang et al., 1995].
- Death certificates for approximately 868 men and 46 women listed silicosis as the underlying cause of death in non-mining occupations, according to a study that reviewed death certificates for the period 1985 to 1992. The researchers focused on death certificates that provided an entry for indicating the potential for substantial silica exposure, reviewing a total of 411,404 death certificates for men and 30,563 for women [Walsh, 1999].
- A ten-year study (1985 to 1995) of Michigan employees found that nearly 80 percent of the 577 confirmed cases of silicosis occurred in industries in the Standard Industrial Classification (SIC) 3300, Primary Metals, which encompasses iron and steel foundries [Rosenman et al., 1997]. In another study, foundry employees whose lungs exhibited radiographic changes consistent with silicosis were concentrated in four primary job assignments: core making, mold making, core knockout, and cleaning/finishing. The study was conducted at a Midwestern gray iron foundry that has produced automotive engine blocks since 1949; the researchers analyzed medical records and silica exposure data for 1,072 current and retired employees with at least five years of employment as of June 1991. Radiographic readings consistent with silicosis were also correlated with the number of years at the foundry, smoking habits, and silica exposure levels [Rosenman et al., 1996].
- In the mid-1990s, there were two cases of accelerated silicosis in relatively young sandblasters following short periods of extremely high crystalline silica exposures. In 1995, a 36-year-old man who had sandblasted oil field tanks in Western Texas for 36 months died from respiratory failure, eleven years after his initial exposure to crystalline silica. A second sandblaster at the same facility, a 30-year-old man who had worked as a sandblaster from 1986 to 1990, died in 1996, ten years after his initial exposure [CDC, 1998]. Both of these sandblasters died from progressive massive fibrosis, an advanced stage of silicosis.

Adverse Health Effects of Crystalline Silica Exposure

Pulmonary silicosis has historically been the disease most well-known as being caused by the inhalation of respirable crystalline silica particles. Additionally, there is evidence that exposure to crystalline silica-containing dusts causes or is associated with the following conditions: lung cancer, tuberculosis, chronic obstructive pulmonary disease (including emphysema and bronchitis), autoimmune diseases or immunologic disorders, chronic renal disease, and subclinical renal changes [NIOSH, 2002].

Silicosis

Silicosis is a fibrotic disease of the lungs caused by the inhalation of crystalline silica dust. It is a type of pneumoconiosis, which is a general term for chronic lung disease that occurs when certain particles are inhaled and deposited deep in the lung. There are two main types of silicosis, *chronic silicosis* (also called “classical” or “nodular” silicosis) and *acute silicosis*, medically referred to as silico-proteinosis or alveolar lipoproteinosis-like silicosis. Chronic silicosis, by far the most common form of the occupational disease, typically appears 20 to 40 years after initial exposure and tends to progress even after exposure ceases. *Accelerated silicosis* is a variant of chronic silicosis but develops after more intense exposure to crystalline silica; it is characterized by earlier onset (within 5 to 15 years of initial exposure) and more rapid progression of disease than chronic silicosis [Weill et al., 1994].

Acute silicosis results from an overwhelming exposure to silica and the symptoms become manifest in a little time as a few weeks after exposure. Acute silicosis appears to be distinct from the other forms of silicosis, possibly involving an immune mechanism not associated with either accelerated or chronic silicosis. This disease, though rare, is invariably fatal. Outbreaks of acute silicosis have occurred among sandblasters and silica flour mill employees [Peters, 1986].

The development of silicosis is dependent on the size of the crystalline silica dust particle, the dust concentration, and the duration of exposure. Crystalline silica particles smaller than 10 micrometers (μm) in diameter, so-called *respirable* particles, are particularly hazardous, because they easily pass through the tracheobronchial tree and are deposited in the deepest recesses of the lungs, the alveolar structures. Particles larger than 10 μm in diameter are trapped in the nose or the mucous lining of the airway and are removed by the mucociliary escalator. Chronic silicosis has an early manifestation of a dry or non-productive cough when there is continued exposure to the inhaled irritant. The cough then becomes prolonged and distressing, with sputum production as the disease advances. Initially, breathlessness occurs while exercising, but progresses to shortness of breath during normal activity [Porth, 1994]. Wheezing typically only occurs when conditions such as chronic obstructive bronchitis or asthma are also present. Advanced states of silicosis include pneumothorax and respiratory failure. Respiratory symptoms increase with the progression of silicosis [Wang, 1999].

A rapid increase in the rate of synthesis and deposition of lung collagen has also been seen with the inhalation of crystalline silica particles. The collagen formed is unique to silica-induced lung disease and is biochemically different from normal lung collagen [Olishifski and Plog, 1988]. Silicosis in all its forms is incurable and causes significant impairment or death. Therefore, eliminating or controlling occupational exposure to respirable crystalline silica is critical to prevention of the disease.

Lung Cancer

The International Agency for Research on Cancer [IARC, 1997] classifies crystalline silica inhaled in the form of quartz or cristobalite from occupational source as “carcinogenic to humans (Group 1).” However, in making the overall evaluation, the IARC Working Group noted “that carcinogenicity in humans was not detected in all industrial circumstances studied.” The Working Group also stated: “Carcinogenicity may be dependent on inherent characteristics of the crystalline silica or on external factors affecting its biological activity or distribution of its polymorphs.”

The IARC analysis included studies of U.S. gold miners, Danish stone industry employees, U.S. granite shed and quarry employees, U.S. crushed stone industry employees, U.S. diatomaceous earth employees, Chinese refractory brick makers, Italian refractory brick makers, U.K. pottery makers, Chinese pottery makers and cohorts of registered silicotics from North Carolina and Finland. Most of these studies found a statistically significant association between occupational exposure to crystalline silica and lung cancer.

Tuberculosis

Epidemiologic studies have firmly established the association between TB and silicosis. Some studies have indicated that employees who do not have silicosis but who have had long exposures to silica dust may also be at increased risk of developing TB [NIOSH, 2002].

Individuals with chronic silicosis are more susceptible to developing active tuberculosis than the general population. However, it is not clear whether low-level exposure to silica, in cases where silicosis has not developed, also predisposes employees to tuberculosis [Davis, 1996].

Chronic Obstructive Pulmonary Disorder

Epidemiologic studies have shown that occupational exposure to respirable crystalline silica is associated with chronic obstructive pulmonary disease, including bronchitis and emphysema. The findings from some of these studies suggest that emphysema and bronchitis may occur less frequently or not at all in nonsmokers. Epidemiologic studies have also found significant increases in mortality from nonmalignant respiratory disease, a category that includes silicosis, emphysema, and bronchitis, as well as some other related pulmonary diseases [NIOSH, 2002].

Immunologic Disorders and Autoimmune Diseases

Several epidemiologic studies have found statistically significant increases in mortality from or cases of immunologic disorders and autoimmune diseases in employees exposed to silica. These disorders and diseases include scleroderma (a rare multisystem disorder characterized by inflammatory, vascular, and fibrotic changes usually involving the skin, blood vessels, joints, and skeletal muscle), rheumatoid arthritis, systemic lupus erythematosus (lupus), and sarcoidosis (a rare multisystem granulomatous disease characterized by alterations in the immune system) [NIOSH, 2002].

Renal Disease

Epidemiological studies report statistically significant associations between occupational exposure to silica dust and several renal diseases or effects, including end-stage renal disease morbidity (including that caused by glomerular nephritis, chronic renal disease mortality, and Wegener's granulomatosis (systemic vasculitis often accompanied by glomerulonephritis) [NIOSH, 2002].

Stomach and Other Cancers

There is some evidence from studies of various occupational groups exposed to crystalline silica of statistically significant excesses of mortality from stomach or gastric cancer. However, most of these studies did not adjust for confounding factors and possible exposure-response relationships were not assessed. Similar issues with confounding and lack of exposure-response assessment exist for the infrequent reports of statistically significant numbers of excess deaths or cases in silica-exposed employees of other nonlung cancers such as nasopharyngeal or pharyngeal, salivary gland, liver, bone, pancreatic, skin, esophageal, digestive system, intestinal or peritoneal, lymphopoietic or hematopoietic, brain, and bladder [NIOSH, 2002].

Summary

As these health findings indicate, crystalline silica exposure is associated with a number of diseases, in addition to silicosis. Silica exposure continues to pose substantial risks to employees, centuries after it was first identified as an occupational hazard. The only way to prevent disease is to eliminate exposure to crystalline silica or reduce crystalline silica exposure to safe levels.

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