STATE-OF-THE-SCIENCE REVIEW OF THE OCCUPATIONAL HEALTH HAZARDS OF CRYSTALLINE SILICA IN ABRASIVE BLASTING OPERATIONS AND RELATED REQUIREMENTS FOR RESPIRATORY PROTECTION

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Excessive exposures to airborne crystalline silica have been known for over 100 years to pose a serious health hazard. Work practices and regulatory standards advanced as the knowledge of the hazards of crystalline silica evolved. This article presents a comprehensive historical examination of the literature on exposure, health effects, and personal protective equipment related to silica and abrasive blasting operations over the last century. In the early 1900s, increased death rates and prevalence of pulmonary disease were observed in industries that involved dusty operations. Studies of these occupational cohorts served as the basis for the first occupational exposure limits in the 1930s. Early exposure studies in foundries revealed that abrasive blasting operations were particularly hazardous and provided the basis for many of the engineering control and respiratory protection requirements that are still in place today. Studies involving abrasive blasters over the years revealed that engineering controls were often not completely effective at reducing airborne silica concentrations to a safe level; consequently, respiratory protection has always been an important component of protecting workers. During the last 15–20 yr, quantitative exposure-response modeling, experimental animal studies, and in vitro methods were used to better understand the relationship between exposure to silica and disease in the workplace. In light of Occupational Safety and Health Administration efforts to reexamine the protectiveness of the current permissible exposure limit (PEL) for crystalline silica and its focus on protecting workers who are known to still be exposed to silica in the workplace (including abrasive blasters), this state-of-the-science review of one of the most hazardous operations involving crystalline silica should provide useful background to employers, researchers, and regulators interested in the historical evolution of the recognized occupational health hazards of crystalline silica and abrasive blasting operations and the related requirements for respiratory protection.

Crystalline silica has been one of the most widely studied chemicals in the history of occupational disease and industrial hygiene. Crystalline silica is an abundant rock-forming mineral and is present in different forms or polymorphs in the environment. While each form is composed of units of silicon dioxide (SiO2), the forms differ in their atom spacing, lattice structure, and angular relation (NIOSH, 2002a). Quartz (also commonly referred to as “free silica”) is most prevalent in the environment and, consequently, in the workplace. Quartz or silica sand is used in abrasives, cleaners, ceramics, electronics, fillers, optics, polishes, and refractory materials. Cristobalite and tridymite, other polymorphs of crystalline silica less commonly found in rocks or soils, may occur in industrial operations involving the heating of quartz or amorphous silica, such as the calcining of diatomaceous earth or during brick manufacturing (NIOSH, 2002a). Although silica may also exist as a non-crystalline form (amorphous), crystalline silica continues to be of national interest. It is the most toxic form of silica, poses a major health hazard with estimates of approximately 1.7 million U.S. workers exposed to respirable crystalline silica in industries and occupations such as construction, sandblasting, and mining, and has been attributed to causing the over 15,000 silicosis deaths over the last three decades (NIOSH, 2005, 2006c).

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Crystalline silica is present in many occupational environments, including mines, metal foundries and mills, agricultural settings, construction, shipbuilding and ship repair, and manufacturing facilities for glass, paints, chemicals, soaps, cosmetics, rubber materials, and plastics (NIOSH, 2002a). Around the turn of the 20th century, changes associated with the industrial revolution led to greater airborne dust concentrations in many occupational settings (Air Hygiene Foundation of America, Inc., 1937b). This increase was primarily due to the use of machinery (e.g., drills, automatic grinders) in applications that had previously been performed by hand. The resulting increase in lung disease among workers in the “dusty trades” prompted studies that led to recognizing silica’s role in the development of what would eventually be called silicosis.

Because of silica’s abundance in the environment and its widespread use in consumer product and industrial manufacturing (Table 1), exposure occurs in a large variety of industries and occupational settings (NIOSH, 2002a). For example, silica that is naturally present in rocks can be released into the air during mining (e.g., rock drilling, dredging) and quarrying (e.g., crushing stone, stone

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**TABLE 1.** Industries and Operations in which Silica Exposure has been Reported

<table>
<thead>
<tr>
<th>Industry</th>
<th>Specific Operation/Task</th>
<th>Source Material</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abrasives</td>
<td>Silicon carbide production</td>
<td>Sand</td>
</tr>
<tr>
<td>Agricultural chemicals</td>
<td>Raw material crushing, handling</td>
<td>Phosphate ores and rock</td>
</tr>
<tr>
<td>Agriculture</td>
<td>Pottery firing, ceramics, clay mixing, kiln repairs, abrasive blasting, sand blasting, engraving, cutting, grinding, polishing, buffing, etching, engraving, casting, shaping, sharpening, sculpting</td>
<td>Soil</td>
</tr>
<tr>
<td>Arts, crafts, sculpture</td>
<td>Abrasive blasting</td>
<td>Clays, glazes, bricks, stones, rocks, minerals, sand, silica flour</td>
</tr>
<tr>
<td>Automobile repair</td>
<td>Abrasive blasting</td>
<td>Sand</td>
</tr>
<tr>
<td>Boiler scaling</td>
<td>Coal-fired boilers</td>
<td>Ash and Concretions</td>
</tr>
<tr>
<td>Cement</td>
<td>Raw materials processing</td>
<td>Clay, sand, limestone, diatomaceous earth</td>
</tr>
<tr>
<td>Ceramics (including bricks, tiles, sanitary ware, porcelain, pottery, refractories, vitreous enamels)</td>
<td>Mixing, moulding, glaze or enamel spraying, finishing</td>
<td>Clay, shale, flint, sand, quartzite, diatomaceous earth</td>
</tr>
<tr>
<td>Construction</td>
<td>Abrasive blasting of structures, buildings</td>
<td>Sand, concrete</td>
</tr>
<tr>
<td></td>
<td>Highway and tunnel construction</td>
<td>Rock</td>
</tr>
<tr>
<td></td>
<td>Excavation and earth moving</td>
<td>Soil and rock</td>
</tr>
<tr>
<td></td>
<td>Masonry, concrete work, demolition</td>
<td>Concrete, mortar, plaster</td>
</tr>
<tr>
<td>Dental material</td>
<td>Abrasive blasting, polishing</td>
<td>Sand, abrasives</td>
</tr>
<tr>
<td>Foundries</td>
<td>Casting, shaking out</td>
<td>Sand</td>
</tr>
<tr>
<td></td>
<td>Abrasive blasting, fettling</td>
<td>Sand</td>
</tr>
<tr>
<td></td>
<td>Furnace installation and repair</td>
<td>Refractory material</td>
</tr>
<tr>
<td>Glass (including fiberglass)</td>
<td>Raw material processing</td>
<td>Sand, crushed quartz</td>
</tr>
<tr>
<td>Iron and steel mills</td>
<td>Refractory preparation and furnace repair</td>
<td>Refractory material</td>
</tr>
<tr>
<td>Jewelry</td>
<td>Cutting, grinding, polishing, buffing</td>
<td>Semi-precious gems or stones, abrasives</td>
</tr>
<tr>
<td>Metal products (including structural metal, machinery, transportation equipment)</td>
<td>Abrasive blasting</td>
<td>Sand</td>
</tr>
<tr>
<td>Mining and milling</td>
<td>Most occupations (underground, surface, mill) and mines (metal and non-metal, coal)</td>
<td>Ores and associated rock</td>
</tr>
<tr>
<td>Paint</td>
<td>Raw materials handling</td>
<td>Fillers (tripoli, diatomaceous earth)</td>
</tr>
<tr>
<td>Quarrying and milling</td>
<td>Crushing stone, sand and gravel processing, monumental stone cutting and abrasive blasting, slate work, diatomite calcination</td>
<td>Diatomaceous earth</td>
</tr>
<tr>
<td>Roofing and asphalt felt</td>
<td>Filling and granule application</td>
<td>Sand and aggregate, diatomaceous earth</td>
</tr>
<tr>
<td>Rubber and plastics</td>
<td>Raw material handling</td>
<td>Fillers (tripoli, diatomaceous earth)</td>
</tr>
<tr>
<td>Shipbuilding and repair</td>
<td>Abrasive blasting</td>
<td>Sand</td>
</tr>
<tr>
<td>Silicon and ferro-silicon foundries</td>
<td>Raw materials handling</td>
<td>Sand</td>
</tr>
<tr>
<td>Soaps and cosmetics</td>
<td>Abrasive soaps, scouring powders</td>
<td>Silica flour</td>
</tr>
</tbody>
</table>

Data from IARC 1997 and NIOSH 2002a.
monument cutting, slate work) operations. Agricultural activities such as plowing and harvesting can disperse silica that is present in soil, and glass manufacturing (including fiberglass), cement, and ceramics may involve silica-containing sand. Various materials used in the construction industry that contain silica include sand, concrete, rock, soil, mortar, plaster, and shingles. Activities in the construction industry, including demolition, highway and tunnel construction, abrasive blasting, concrete work, excavation, jack hammering, and roofing, may result in exposure to respirable silica. Additionally, silica-containing sand was used widely in foundries for metal cast molding and abrasive blasting (NIOSH, 2002a).

Since the 1920s and through the present day, abrasive blasting has consistently been identified as one of the more hazardous operations with respect to potential exposure to airborne crystalline silica. During this process, an abrasive substance is propelled from a hose and nozzle using air or liquid. Abrasive blasting is frequently performed in foundry operations, where it is used to remove appendages from metal castings, during the construction and repair of buildings, bridges, and ships to remove rust or imperfections and to prepare surfaces for painting, and in auto body repair shops, arts and crafts, and dentistry (NIOSH, 2002a).

The potential for exposure to airborne crystalline silica and the associated risk of disease can be significant if abrasive blasting operations are conducted without adequate dust controls. This article presents a comprehensive historical examination of the literature on exposure, health effects, and personal protective equipment related to silica and abrasive blasting operations over the last 100 yr. This review is divided into four time periods: 1900 to 1939, 1940 to 1969, 1970 to 1989, and 1990 to the present (Figure 1). These periods were selected based on what were believed to be sentinel events with respect to the recognition of silicosis as an occupational disease, the development and standardization of respirators and workplace controls, and the promulgation of occupational exposure limits (OELs) for silica.

For each time period, the following subjects are addressed: (1) major developments in the recognition of the health hazards of crystalline silica, including key toxicology and epidemiology studies; (2) guidelines and regulations related to OELs for crystalline silica, as well as development of sampling and analytical methods for characterizing exposures; (3) exposure and health studies of workers performing abrasive blasting, as well as standard practices of various industries involving abrasive blasting; and (4) recommendations for personal protective equipment for abrasive blasting operations, with a particular emphasis on respirators.

The goal of this review is to identify when specific scientific knowledge about the health hazards of crystalline silica was established and communicated among the scientific and industrial hygiene communities. The use of this information in the development of guidelines and regulations for appropriate controls during abrasive blasting operations is also discussed. Given the breadth of silica-related research conducted across the world over the past century, it is not feasible to
describe in detail all of the studies in every industry. Rather, this article focuses on key industries (e.g., granite, metal mining, diatomite, foundries, shipbuilding), with a particular emphasis on abrasive blasting operations.

OVERVIEW OF SILICOsis

Silicosis was recognized as an occupational disease very early in the history of occupational medicine and industrial hygiene (Hunter, 1969). There are three forms of silicosis: chronic, accelerated, and acute (NIOSH, 2002a). Chronic silicosis is often considered the classic form of the disease, and can develop after 10 yr or more of sufficiently high exposure. Chronic silicosis may be described as simple silicosis, where individual fibrotic nodules are solitary and less than 1 cm in diameter, as conglomerate silicosis, wherein the nodules become confluent and eventually replace the lung parenchyma, or as progressive massive silicosis, an uncommon lesion composed of confluent silicotic nodules (Silicosis and Silicate Disease Committee, 1988). Patients with simple silicosis may not display any symptoms of pulmonary dysfunction. Symptoms of accelerated silicosis, which usually manifests within 5 to 10 yr from first exposure, are similar to chronic silicosis, but the onset of fibrosis is more rapid, irregular, and diffuse. Acute silicosis, also referred to as "alveolar lipoproteinosis," can occur within weeks to 5 yr after initial exposure to very high airborne concentrations of silica (Silicosis and Silicate Disease Committee, 1988; NIOSH, 2002a). With this form, the nodular pattern of fibrosis is completely absent.

Exposure to crystalline silica leads to tissue fibrosis through a cascade of cellular events. Silica is not readily degraded by macrophage lysosomal enzymes and is directly cytotoxic to macrophages and polymorphonuclear leukocytes. Macrophages that engulf silica particles become necrotic and release lysosomal contents and reactive oxygen species (ROS), leading to local recruitment of polymorphonuclear leukocytes and ultimate damage to the surrounding tissue (Blackford et al., 1997; Huffman et al., 1998; Kim et al., 1999; Kang et al., 2000; Zeidler et al., 2004). A cyclical process of macrophage recruitment, particle ingestion, and cell death can cause a focal point of immune activity and fibrosis in the lung, resulting in chronic inflammation (Huffman et al., 1998; Kim et al., 1999; Kang et al., 2000; Zeidler et al., 2004; Blackford et al., 1997).

A number of studies have shown that silica-induced toxicity appears to be most closely correlated with factors that can influence molecular or cellular interactions with the particle surfaces. Multiple lines of evidence suggest that the formation of ROS, reactive nitrogen species (RNS), or other highly reactive molecules may play key roles in the development of the cell injury, proliferation, apoptosis, and fibrogenesis associated with silica exposure (Blackford et al., 1994; Blackford et al., 1997; Hamilton et al., 2008; Huffman et al., 1998; Kim et al., 1999; Kang et al., 2000; Zeidler et al., 2004). These reactive species can be generated directly via chemical reactions with the silica particle surfaces or indirectly through interactions of silica with various cell types. In vitro studies have shown that freshly fractured silica particles generate more ROS and are more toxic to alveolar macrophages than aged silica particles (Vallyathan et al., 1988). This finding is supported by evidence that surface modifications or coatings have been shown to decrease the pathogenicity of silica particles in vivo (Albrecht et al., 2005). In addition, in vitro studies have demonstrated that hydroxyl or superoxide radicals are formed in the presence of freshly fractured silica, and that crystalline silica is a potent stimulant of the increase in ROS production in alveolar macrophages (Vallyathan et al., 1988; Vallyathan et al., 1992; Castranova, 1994). The sustained presence of ROS resulting from repeated attempts by alveolar macrophages to phagocytize silica particles can cause chronic cell injury or death and may promote fibrogenesis (Zeidler et al., 2004; Blackford et al., 1997; Huffman et al., 1998; Kim et al., 1999; Kang et al., 2000).

OVERVIEW OF ABRASIVE BLASTING OPERATIONS

Abrasive blasting is a process for cleaning metal and other surfaces to remove contaminants, rust, and paint by directing a high-pressure stream of abrasive materials against surfaces. Typically, when sand is used as an abrasive, it is sieved from its natural state to a uniform size and the quartz content is largely dependent on the source of sand. Depending on the abrasive blasting application,
industries may specify certain types of abrasive materials (NIOSH, 1976a; Dolley, 2003; Flynn & Susi, 2004). Technically, if sand is used as the abrasive material, the process is referred to as “sandblasting,” although this term is often used generically even when other materials such as coal and metal slags, steel shot, and garnet are used as abrasives.

Abrasives can be propelled in several different ways during abrasive blasting. Compressed air is used to project the abrasive during dry abrasive blasting. Other approaches include wet and airless blasting, in which the abrasives are propelled by water and centrifugal force, respectively. General equipment used for all types of abrasive blasting includes (1) a hopper, a reservoir for the abrasive material; (2) a source of pressure (i.e., air, water, gravity) to force the abrasive from the hopper through a hose; and (3) a nozzle, held by the abrasive blaster to direct the spray of abrasive material (Figure 2). To reduce exposure to airborne silica, abrasive blasting operations may be conducted inside enclosed blasting machines and rooms, an approach commonly used by foundries. Smaller enclosed units, such as cabinets, barrels, and tables, were developed for blasting of smaller castings. Both blasting rooms and small blasting units had exhaust ventilation to control airborne exposures to the worker, and included collection systems to collect the abrasive material for recycling. As discussed in this review, these measures were not always feasible or capable of adequately reducing airborne silica exposures, thereby necessitating an additional hierarchy of dust control by using respiratory protection as a secondary line of preventing exposure to abrasive blasters (NIOSH, 1976a). It should be noted, however, that current federal regulations require that substitution, isolation, and ventilation be the principle means of controlling exposure to airborne contaminants during operations such as abrasive blasting, and that respiratory protection should not be the primary method for preventing or minimizing worker exposures (OSHA, 1998a).

Abrasive blasting can present other occupational hazards to the blaster besides exposure to airborne contaminants. Because the abrasive material is projected at high speeds, ricochet of the abrasive material can be a serious physical hazard to the abrasive blasting operator. Protective gear that is generally worn to prevent injury from the ricochet of abrasive material includes a heavy canvas,
rubber, or leather ricochet shoulder cape or shroud, with or without a helmet that is worn over the head and shoulders. In addition, an apron or protective suit, leggings, steel-toed boots, and gloves are also worn to protect the blaster (Figure 2) (American National Standards Institute [ANSI], 1968). The head, neck, and shoulders can be protected by a hood or helmet that either is placed over an airline respirator or is designed as an integrated unit, where the respirator is built into the helmet or hood. Airline respirators and air-supplied hoods require the use of air compressors or blowers to deliver an outside source of breathable air, which can produce noise levels inside the hood that may be hazardous to the blaster. Under these circumstances, hearing protection is also used by the blaster.

The fundamental basis of respiratory protection and personal protective equipment for controlling the physical and respirable hazards of abrasive blasting operations in place today was established in the 1920s and 1930s (Bloomfield & Greenburg, 1933; Greenburg & Winslow, 1932; Winslow et al., 1919, 1920). Despite the early development and use of respiratory and personal protective equipment, engineering and administrative controls and substitution of hazardous materials are the primary means of protection against dust hazards (as applied in the hierarchy of controls), a practice fundamental to historical and contemporary industrial hygiene and a requirement by current federal regulations (OSHA, 1998a). When these measures are not effective, respiratory protection is also used and environmental monitoring should be performed regularly to ensure that the Applied Protection Factor (APF) of the respiratory protection is sufficient to keep exposure levels below the permissible exposure limit (PEL) (NIOSH, 1996b).

THE EARLY YEARS (1900–1939) — ERA OF RECOGNIZING THE HAZARDS OF SILICA

Studies of Health Effects

Lung Disease in the “Dusty Trades” The relationship between exposure to dust and lung disease has long been recognized. In the first century, the Greeks described a lung disease that appeared to cause a wasting away of the body, and in Pliny’s *Natural History*, devices used by refiners were described as a means of reducing dust inhalation (Mavrogordato, 1929; Lanza, 1938). By the 16th and 17th centuries, lung diseases among miners, smelters, and stone cutters were described by a number of physicians, and occasionally autopsies were performed on workers, revealing the presence of dust in the lungs (Lanza, 1938).

Around the late 1800s, the increasingly widespread use of pneumatic tools and automatic machinery, particularly in the mining and stone-cutting industries, created very dusty work environments. The dust generated by power tools and machinery was finer in consistency and greater in quantity than that created by hand tools. Because few dust-control measures were used and respiratory protection was not worn by workers, disease and mortality rates during these years were significantly higher among workers in “dusty trades” compared to other occupations (Air Hygiene Foundation of America, Inc., 1937b).

Early occupational studies generally did not distinguish among lung diseases caused by different types of dust (e.g., coal, silica, metal, asbestos), chemicals, or bacterial agents. Originally, nearly all respiratory diseases were termed “phthisis” or “consumption,” which encompassed both silicosis and tuberculosis (Lanza, 1938). Some of the first reports to correlate disease prevalence with silica content were those published by the Miner’s Phthisis Prevention Committee, the Miner’s Phthisis Medical Bureau, and the South African Institute for Medical Research between 1903 and 1920. In their 1916 report, it was noted that 26% of approximately 3000 miners showed definite signs of silicosis, with another 5.5% considered probable cases of silicosis, as a result of working in South African gold mines with ore reportedly containing 80 to 90% quartz (Miners’ Phthisis Prevention Committee, 1916; Lanza, 1938). These, as well as studies of tin miners in Cornwall and metal, quartz, slate, and sandstone miners in the United Kingdom and Australia that also documented high rates of lung disease, were among the first to provide evidence suggesting that crystalline silica was the causative agent for the lung disease (Lanza, 1938).

Recognition of Silicosis as a Distinct Disease The recognition of silicosis as a distinct disease was confounded by the fact that early medical techniques could not distinguish silicosis from
tuberculosis, which was often prevalent in the same populations. During the late 1800s, microbiological methods were developed that allowed researchers to identify *Mycobacterium tuberculosis*, the causative agent of tuberculosis (Ingraham & Ingraham, 1995). Despite this advance, tuberculosis was not differentiated from silicosis in occupational studies until diagnostic techniques, such as the x-ray, were used in combination with sputum analysis. Although chest x-rays were being used frequently by the 1930s, the detection of early-stage silicosis by this method was limited, underscoring the importance of acquiring a complete occupational and medical history (Russell et al., 1929). Because the classification of silicosis by chest x-rays proved to be difficult and required that sputum collection and review be performed by experts, dyspnea, especially upon exertion, was one of the most common criterion by which the disease was identified. Respiratory function tests, sputum tests, and blood pressure monitoring were also used in the medical monitoring of workers, although these were not considered reliable indicators of silicosis (Kuechle, 1934; National Silicosis Conference, 1938c). As a result of worker’s compensation acts developed during this time, preemployment and regular follow-up medical examinations were often used in industries that were shown to pose a high risk for silicosis (Russell et al., 1929; Kuechle, 1934; Lanza & Vane, 1934; Air Hygiene Foundation of America, Inc., 1937b; Kammer, 1939).

After the development of medical techniques that allowed physicians to diagnose tuberculosis infections and distinguish them from silicosis, it was reported that rates for both diseases were often increased in the same occupational groups. These observations led scientists to believe that exposure to crystalline silica increased one’s risk of developing tuberculosis. In Australia, studies of quartz, slate, and sandstone miners exposed to 20–80% quartz showed tuberculosis rates significantly higher than those found in the general population. In addition, workers from the Bendigo mines, which contained 80% quartz, showed higher rates of tuberculosis than miners working in Western Australia, where the ore contained 20–45% quartz (Air Hygiene Foundation of America, Inc., 1937b). Higher rates of silicosis were also observed in industries where dust contained a high percentage of silica, including the tin mining, slate, refractory, sandstone, pottery, and coal mining industries in Great Britain, sewer workers and metal, quartz, slate, and sandstone industries in Australia (Air Hygiene Foundation of America, Inc., 1937b), and gold mines in South Africa (Miners’ Phthisis Prevention Committee, 1916). The significance of this correlation was not overlooked by the scientific community. For example, in 1915, Dr. Collis noted in his Milroy Lectures to the Royal College of Physicians that several studies supported his theory that “free” crystalline silica was the causative agent in nearly all dusts that produced serious lung injury or increased susceptibility to tuberculosis (Lanza, 1938).

**Case Reports and Initial Studies in the United States**

It was not until about 10–15 yr later that similar investigations were carried out in the United States (Figure 3). Although case reports documenting silica-related disease in mines or foundries were published as early as 1900, these reports generated little attention (Betts, 1900). Some of the first large-scale studies of silicosis in the United States were conducted by Dr. Frederick Hoffman, a statistician at the Prudential Insurance Company. Using mortality records and occupational information collected every year by Prudential, Hoffman estimated mortality rates for the general population and for workers in various trades. In 1907, he published his first compilation of records, “Mortality From Consumption in Dusty Trades,” in which he noted distinct patterns of mortality from consumption among those working around dust with a marked predisposition to phthisis in industries involving hard stones, such as flint, granite, or sandstone. (Hoffman, 1908). In later studies, Dr. Hoffman’s analyses focused on respiratory diseases in the “dusty trades,” including occupations that involved exposure to both metallic and mineral dusts (e.g., metal mines, quarries, metal industry, iron and steel foundries) (Hoffman, 1918). In nearly all of his studies, workers employed in dusty trades exhibited higher rates of mortality or lung disease than in the general population.

It was several years after Dr. Hoffman’s publications appeared that the U.S. Public Health Service (USPHS) and the U.S. Bureau of Mines (USBM) began to formally study silicosis and other lung diseases among American workers. During the first half of the 20th century, the USPHS expanded its role from monitoring the health of sailors and ship passengers to broader health issues related to sanitation and work conditions (Parascandola, 1998). Similarly, the USBM, which was created in 1910 in response to a large number of coal mine disasters, was authorized to conduct investigations
FIGURE 3. Timeline of key studies and events that led to the recognition of the hazards of silica (1900–1939).
of mine safety and health of miners (U.S. Department of Labor, 2008). As such, both agencies played major roles in many large-scale silicosis studies, often in collaboration with other research agencies, industry, and labor, to evaluate mortality, tuberculosis, and pneumoconiosis rates in hazardous operations or industries.

One of the first collaborative investigations between the USPHS and USBM involved miners in Joplin, Missouri, where a high rate of tuberculosis among miners in the area had been noted for years (Lanza & Higgins, 1915; Lanza, 1917; Higgins et al., 1917). Of the 720 workers evaluated in this study, 60.1% had silicosis or “miner’s consumption” (as it was called at that time). Of the 433 men with silicosis, 103 (23.8%) also had tuberculosis. These results from the Joplin mines, which contained a high amount of quartz (70–95%), were similar to what had been observed in the South African miners (Lanza, 1917; Higgins et al., 1917).

**Large-Scale Studies in the U.S. Mining Industry** By 1923, mining operations had expanded due to the increasing need for energy and materials to manufacture goods. In 1924, the USBM established a clinic in the heart of the lead and zinc mining district (Missouri, Kansas, and Oklahoma) and later collaborated with the USPHS and the Metropolitan Life Insurance Company to monitor worker health (Figure 3). More than 60,000 physical examinations were conducted over a 9-yr period, during which the rate of silicosis among metal mine workers in the United States ranged from 20 to 25% (Sayers et al., 1933; Meriwether et al., 1933; Lanza, 1938). It was also noted that the effects of dust exposures could take years to manifest into clinical symptoms, and that removal from exposure did not always prevent later development of disease. Additional studies of metal miners in other states, notably in Butte, Montana, around the same time period also indicated higher rates of tuberculosis and silicosis among the miners (Harrington & Lanza, 1921; National Silicosis Conference, 1938c).

Studies of workers in the bituminous (soft coal), anthracite (hard coal), and hematite mines were among the first to provide evidence among American workers that quartz content was an important determinant of disease risk in the work environment (Brundage & Frasier, 1933). Although lung disease had been reported in nonmetal miners, the prevalence of silicosis and tuberculosis varied by location and industry. The study of the anthracite mines in Pennsylvania was a cooperative effort between the USPHS, the coal mines, the United Mine Workers of America, and the Pennsylvania Department of Labor and Industry that involved the evaluation of approximately 3000 workers (Sayers, 1935). Miners in bituminous mines, which contained low levels of quartz in the surrounding rock, did not show elevated rates of lung fibrosis or tuberculosis, whereas workers in the anthracite mines, which contained about 43% quartz, showed evidence of fibrosis in 23% of those tested (Sayers, 1935; Lanza, 1938). The importance of exposure duration was noted, in that the prevalence of tuberculosis in anthracite workers under the age of 35 was approximately the same as the general population, but about 10-fold higher in workers over the age of 55. It was also reported in this study that the highest rates of disease were observed in rock drillers (Sayers, 1935).

Similar findings were reported by Saranac Laboratories in a study of hematite miners in northern Michigan and Wisconsin that showed that miners who had worked in areas where the rock contained silica developed fibrosis following periods greater than 20 yr of exposure, on average, whereas workers exposed to ore bodies containing little silica for as long as 30 yr of exposure rarely developed nodular fibrosis (Air Hygiene Foundation of America, Inc., 1937b). Taken together, studies conducted in the coal and iron mines indicated that the health hazards experienced by miners were proportional to the amount of silica in the rock being mined. In fact, many of the initial studies, which reported conflicting rates of disease, actually reflected the fact the dust in some mines had higher concentrations of silica than others. It was also apparent from these mining studies that certain operations (i.e., drilling) posed a greater hazard to workers due to the high airborne dust concentrations created during these activities (Lawson et al., 1931; Air Hygiene Foundation of America, Inc., 1937b).

**Large-Scale Studies in the U.S. Granite Industry** Perhaps some of the most influential early studies were those concerning New England’s granite industry (Figure 3). These studies solidified the understanding that not only silica content was important in the development of lung disease, but, also that the intensity and duration of exposure to silica were significant factors. Both the
USPHS and Dr. Fredrick Hoffman conducted separate analyses of granite workers in New England. During the period 1915–1918, the death rate from tuberculosis among the general population in Massachusetts averaged 203.2 per 100,000, whereas the rate for granite workers during the same period was 1056.7 per 100,000. It was further noted that the death rates among stone cutters, who used pneumatic tools, were the highest of all the workers investigated. Additionally, the rate of tuberculosis among the granite workers had increased while tuberculosis rates decreased in the general population (Hoffman, 1922). Dr. Hoffman concluded that the hazardous nature of the dust was directly related to the silica content of dust inhaled.

The USPHS also evaluated the largest granite-cutting center in the United States, in Barre, Vermont. Approximately 1000 granite workers, including stone cutters, carvers, polishers and blasters, were included in a morbidity study, with half involving physical examinations (Russell et al., 1929). This evaluation was one of the most comprehensive studies of its time concerning the exposure-response relationship between airborne silica concentrations and prevalence of silicosis. Findings from this study ultimately served as the basis for the first OELs for silica, which remained in place for decades. Exposures of granite workers to silica were assessed by collecting airborne dust samples and conducting medical surveys using physical exams and chest x-rays to track the health status of workers. After accounting for the employment duration, the results showed a consistent dose-response pattern. Four exposure groups (average plant dustiness of approximately 60, 27–44, 20, and 3–9 million particles per cubic foot [mppcf], respectively) were identified, and the highest prevalence of silicosis (nearly 100% of workers) was observed in the highest exposure groups with 9 yr of service. For example, the majority (90%) of pneumatic tool operators working for 9 yr in an environment containing 60 mppcf of dust had advanced silicosis, whereas only 3 cases of early silicosis were identified in the lowest exposure group among workers with up to 10 yr of employment. Based on the findings of this study, the authors suggested in 1929 that the “safe” limit of dustiness for work with granite dust containing around 35% free silica was somewhere between 9 and 20 mppcf (Russell et al., 1929).

Large-Scale Studies in the Foundries A number of large-scale studies were conducted throughout the 1920s and 1930s in which the incidence of silicosis and other respiratory diseases in 10 to 30 different foundries across several U.S. states was evaluated (Figure 3) (McConnell & Fehnel, 1934; Sander, 1938). This approach allowed for characterization of the industry as a whole, even though conditions varied substantially among foundries depending on industrial practices and the size of the operation. An increased incidence of silicosis was reported among foundry workers (Sander, 1938; McLaughlin et al., 1950). It was also noted in several studies that foundry workers had higher rates of lung disease, in particular pneumonia (Dublin, 1917; McConnell & Fehnel, 1934).

Many of the studies conducted through the 1930s identified mold and core making, shake-out, fettling, cleaning and finishing, and, in particular, abrasive blasting as operations that presented the greatest potential for silica exposure and disease (Sander, 1938; McLaughlin et al., 1950). Local exhaust ventilation and other engineering controls were installed in many facilities, consistent with what was considered to be the hierarchy of control by industrial hygienists (Raterman, 1996, Soule, 2000). If engineering controls were not feasible or were not sufficiently effective, then an industrial hygienist might also implement administrative controls. If both of these control measures failed to reduce exposure to a safe level, the use of respiratory protection, which became more widespread around this time, would also be implemented (Colton & Nelson, 1997; Soule, 2000).

Experimental Animal Studies During the 1920s and 1930s, much of the experimental research related to silica was conducted at the Saranac Lake Laboratory (Figure 4) (Gardner, 1932, 1938a). Many of the findings from these studies were shared in a series of symposia known as the “Saranac Lake Symposia” at the Trudeau School of Tuberculosis (Kuechle, 1934), the proceedings of which were published in medical and industrial hygiene journals. In the late 1920s, studies demonstrated that leukocytes were important for removing bacteria and particles from the lungs, and that exposure to silica somehow depressed the function of these cells (Mavrogordato, 1929). It was later discovered that silica was directly toxic to macrophages, which were the primary immune cell type responsible for clearing bacteria from the lungs. Silica toxicity to these cells resulted in an increased susceptibility to tuberculosis infection. Gardner demonstrated that animals exposed to
FIGURE 4. Timeline of key animal studies that contributed to the recognition of the hazards of silica (1900–2008).
silica succumbed to infection by an attenuated strain of tuberculosis with low virulence, whereas unexposed animals failed to develop infection (Air Hygiene Foundation of America, Inc., 1937b).

In the late 1930s, Dr. Leroy Gardner was the first to report that different forms of silica caused varying degrees of fibrosis and that the rate and extent of tissue reaction was inversely proportional to particle size, with silica particles greater than 10 μm not producing appreciable reactions (Air Hygiene Foundation of America, Inc., 1937b; Gardner, 1938b). To address the influence of crystalline silica content on fibrogenicity, a number of animal experiments were conducted by administering dusts with varying silica concentrations to guinea pigs. Results showed a dose-response relationship between the concentration and duration for quartz exposure and the extent and rate of onset of fibrosis. While most of these animal studies were conducted by intravenous injection of different silica suspensions, some inhalation studies of crystalline silica in guinea pigs and rabbits reported nodular fibrosis analogous to silicotic lesions in humans (Gardner, 1932). It was also shown that tuberculosis infection prior to exposure to silica dust resulted in exacerbated pulmonary lesions compared to animals with infection or dust exposure alone (Air Hygiene Foundation of America, Inc., 1937b). Collectively, these studies indicated that silica content was important not only for determining the severity and rate of onset of fibrosis, but also for assessing susceptibility to tuberculosis infection.

**Occupational Exposure Limits for Silica**

Hawk’s Nest Incident Prompts Improvement of Occupational Conditions. Research conducted through the 1930s revealed the enormity of the silicosis problem in American industry. One of the underlying themes of this work was defining a “safe” level of dust exposure. For many scientists and public health agencies, this goal continued to drive additional research. However, a pivotal event occurred in 1935 that led to an even greater effort to understand the hazards of silica and derive an occupational exposure limit that would protect workers. Even today, it remains one of the greatest industrial disasters involving silica: the Hawk’s Nest incident (also referred to as “Gauley Bridge”) (Cherniack, 1986). It occurred near Gauley, West Virginia, where thousands of workers were exposed to extremely high levels of silica while drilling a tunnel. Not only was the rock itself nearly pure quartz, but many of the workers drilling in the rock did not use respiratory protection. While an exact death toll from silicosis was never reported, estimates ranged from hundreds to several thousand; however, most reports approximated the number to be around 700 workers (Lucas & Paxton, 2005; Cherniack, 1986). This event was highly publicized and silicosis became an important national health issue.

In the year or two following Hawk’s Nest/Gauley Bridge, there was considerable activity on the part of industry and Congress to address silica as an occupational health hazard. Shortly thereafter, the Air Hygiene Foundation (AHF), an industry-based group of industrial hygienists, physicians, and scientists, was formed and held their first meeting, which was devoted to silicosis and finding ways to reduce dust exposures in the workplace (Air Hygiene Foundation of America, Inc., 1937a, 1937b). Around the same time, in 1936, Congressional hearings were held in response to Gauley Bridge. The inquiry ultimately prompted the convening of the National Silicosis Conference, during which labor, health, and industry organizations met to discuss the impact of silicosis in American industry. The hearings and the conference were widely covered in the popular press. The conference convened on April 14, 1936, and was attended by several hundred individuals, representing labor, industry, the public, insurance carriers, and prominent members of the medical, engineering, and legal professions (National Silicosis Conference, 1937). Four committees of experts (medical, engineering, economic/legal/insurance, and regulatory/administrative) were appointed to carry out detailed investigations and to make recommendations for control of the silicosis problem. Meetings were held throughout 1936, and in 1937, the committees jointly presented summary reports of their findings (National Silicosis Conference, 1937, 1938a, 1938b, 1938c, 1938d). At the time, it was estimated that around 1,000,000 U.S. workers were exposed to silica, of which approximately 110,000 had some stage of silicosis (National Silicosis Conference, 1937). Hence, the designation of an OEL was a predominant topic at the meeting, and the occupational studies upon which the first recommendations would be based were presented and discussed.
Establishing the First Occupational Exposure Limits  The first recommendations for an OEL in the United States were based on studies of granite workers in Barre, Vermont, as well as those of the anthracite mines in Pennsylvania and gold mines in Ontario (Table 2). The 1924–1926 survey of the granite industry revealed little or no disease among workers who were exposed to 9–20 mppcf of dust (particles less than 10 μm) (Russell et al., 1929). Quartz content in the granite dust was estimated to be approximately 35%. In a reexamination of the same worker population in 1941, it was again concluded that concentrations between 9 and 20 mppcf for dusts containing 25–35% quartz were observed to not cause harmful effects. It was therefore recommended that a maximum dust exposure of about 10 mppcf for dust making occupations involving 25–30% free silica in the form of quartz, was a desirable occupational limit. (Russell, 1941). Additional studies conducted around the same time period in the Pennsylvania anthracite mines and the Ontario gold mines, which had quartz content similar to that of the granite dust, provided further support for the recommendations

### Table 2. Occupational Exposure Limits for Silica (Quartz), 1900–2008

<table>
<thead>
<tr>
<th>Year</th>
<th>Organization</th>
<th>Recommended OEL (particle size)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1900–1939</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1929</td>
<td>USPHS (Vermont Granite Industry)</td>
<td>9–20 mppcf (&lt;\text{10}\mu\text{m}) (\text{a})</td>
</tr>
<tr>
<td>1934</td>
<td>Saranac Lake Symposium</td>
<td>5 mppcf (\text{b})</td>
</tr>
<tr>
<td>1936</td>
<td>DOL (National Silicosis Conference)</td>
<td>5 mppcf (\text{c})</td>
</tr>
<tr>
<td>1937</td>
<td>AHF</td>
<td>5 mppcf (\text{d})</td>
</tr>
<tr>
<td>1938</td>
<td>NBS</td>
<td>50 mppcf (\text{e})</td>
</tr>
<tr>
<td>1940–1969</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1946</td>
<td>ACGIH</td>
<td>For dusts containing (x) percentage of free silica (\text{f})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(x = &lt;5% \rightarrow 50 \text{mppcf})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(x = 5 - 50% \rightarrow 20 \text{mppcf})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(x = &gt;50% \rightarrow 5 \text{mppcf})</td>
</tr>
<tr>
<td>1966</td>
<td>ACGIH</td>
<td>(250 / (% \text{quartz} + 5) \text{mppcf} (\text{g})</td>
</tr>
<tr>
<td>1970–1989</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>ACGIH</td>
<td>10 / (% quartz + 2) mg/m(^3) (\text{h})</td>
</tr>
<tr>
<td>1971</td>
<td>OSHA</td>
<td>10 / (% quartz + 2) mg/m(^3) (TEL) (\text{i,j})</td>
</tr>
<tr>
<td>1975</td>
<td>NIOSH</td>
<td>0.05 mg/m(^3) (REL) (\text{ij})</td>
</tr>
<tr>
<td>1983</td>
<td>ACGIH</td>
<td>0.1 mg/m(^3) (\text{k})</td>
</tr>
<tr>
<td>1986</td>
<td>ASTM</td>
<td>0.1 mg/m(^3) (\text{l})</td>
</tr>
<tr>
<td>1986</td>
<td>WHO</td>
<td>0.04 mg/m(^3) (\text{m})</td>
</tr>
<tr>
<td>1990–2006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>ACGIH</td>
<td>0.05 mg/m(^3), suspected human carcinogen (\text{f})</td>
</tr>
<tr>
<td>2006</td>
<td>ACGIH</td>
<td>0.025 mg/m(^3), suspected human carcinogen (\text{l})</td>
</tr>
</tbody>
</table>


\(\text{a}\) Millions of particles per cubic feet for granite containing 35–70% quartz.
\(\text{b}\) Represents a “primary” threshold for working in environment of 100% quartz.
\(\text{c}\) Considered safe for dust concentration containing a high percentage of free silica.
\(\text{d}\) Stated that concentrations above this limit were dangerous.
\(\text{e}\) Limit of exposure to mineral dusts for workers (total dust concentration).
\(\text{f}\) ACGIH recommendations called MAC (Maximum Allowable Concentrations) until 1956 when TLV Threshold Limit Value) was substituted. The documentation of TLVs for silica is based on analysis of particles 0.5 – 5 μm in size.
\(\text{g}\) Adapted by the U.S. Department of Labor under the Walsh-Healey Public Contracts Act.
\(\text{h}\) Ayer et al., 1969 found gravimetric method to be consistent with the particle count method; the exposure limit of 9–10 mppcf was equivalent to 0.1 mg/m\(^3\).
\(\text{i}\) Permissible Exposure Limit, remains as current value today.
\(\text{j}\) Time-Weighted Average, averaged over an 8 hour shift.
\(\text{k}\) Recommended Exposure Limit, remains as current recommendation today.
\(\text{l}\) Occupational exposure limit for respirable dust.

\(\text{OSHA PEL can also be calculated for mppcf using the same equation as noted for the 1966 ACGIH TLV.}\)
set forth at the National Silicosis Conference (National Silicosis Conference, 1938c; Sayers, 1935). International studies of workers exposed to dust containing upward of 80% quartz were also considered in the initial discussions of appropriate OELs (National Silicosis Conference, 1938c). Based on the prevalence of silicosis observed in Australian and South African workers, exposure limits from 3 to 6 mppcf were viewed as consistent with the experience of the Barre granite workers.

Based on the understanding that there was increased disease risk in environments with higher quartz concentrations, early recommendations of OELs took into consideration % of quartz in dusts (National Silicosis Conference, 1937). At the Saranac Lake Symposium on silicosis in 1934, it was stated that the current research suggested that exposure to pure crystalline silica at a concentration of 5 mppcf or less would not produce physical impairment to workers within 5 yr, but after that time, “demonstrable” silicosis could appear, depending on exposure levels and individual susceptibilities (Kuechle, 1934). It was emphasized that OELs could be modified for various industries to account for differing concentrations of silica in the dust, or for the presence of “mixed” dusts (e.g., coal). The 1937 National Silicosis Conference echoed the recommendations of the Saranac Lake Conference of a safe limit of 5 mppcf for dusts with a high % of silica, and the reports provided a discussion of how to calculate an exposure limit by multiplying the percentage of quartz by the total particle count of the dust (National Silicosis Conference, 1938c). This recommendation was reiterated at the 1937 Symposium on Silicosis at the Annual Meeting of the California Tuberculosis Association (Sayers, 1937).

The U.S. government provided similar recommendations in 1938 in the Department of Commerce National Bureau of Standards publication “American Standard Safety Code for the Protection of Heads, Eyes and Respiratory Organs.” This issue stated that workers should not be exposed to silica-containing dusts greater than “the limit of tolerance” for granite dust that contained 35% quartz; for dusts containing 75% to 100% quartz, it was noted that workers should be exposed to less than this limit (National Bureau of Standards, 1938). The limits themselves were not stated explicitly; however, the purpose of this book was to provide standards regarding personal protective equipment and engineering controls, which the scientific community addressed as key to limiting exposures. A government standard for occupational exposure to quartz would not be established for another 30 yr; in the meantime, industry relied on the USPHS reports and the emerging field of industrial hygiene for expertise and knowledge in how to best protect workers.

By the mid-1930s, it was generally accepted by the scientific community that particles less than 10 μm were the cause of most occupational lung disease in the dusty trades. It was also recognized that the majority of particles in a sample taken from the dusty trades were less than 3 μm in size and the capacity of particles of this size to cause disease was duly noted in animal studies (National Silicosis Conference, 1937; Kuechle, 1934). Therefore, the recommendations for an OEL took into consideration particles less than 10 μm in size. Several methods were available for both sampling and analysis, and it was evident that certain methods were more useful than others and should be chosen based on the environment and sampling objective (National Silicosis Conference, 1938b). In South Africa, the tool predominantly used for sampling was the konimeter. However, it was not efficient for sampling in conditions with a dust content above 35 mppcf, and the sample could not be analyzed chemically or gravimetrically (National Silicosis Conference, 1938b; Bloomfield & Dalla Valle, 1935).

The impinger method proved to be the most efficient and most common sampling method used during the 1920s and 1930s in the United States. Introduced in 1922 by Greenburg and Smith, the impinger was a small device that was used to draw air through a liquid, which would trap the particles for analysis (Drinker & Hatch, 1954). Early studies based on impinger sampling noted that particle counts were highly variable from one operator to the next, and that, overall, the method was labor intensive. However, due to the ease of measuring the particles captured in a fluid, the impinger method remained the most commonly used method of measuring dust concen-

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1 With the konimeter, dust samples were collected via the use of a spring-activated pump and deposited on a plate covered with a film of petroleum or glycerin jelly; particles were then counted under the microscope.

2 A sample from the liquid was taken to count the particles under light-field microscopy with 10x magnification. Light-field microscopy enabled the counting of particles as small as 1 μm (Drinker & Hatch, 1954).
trations for decades (Drinker & Hatch, 1954; Institution of Mining Engineers, 1947). Although variable, a correlation was noted in the literature between dust levels measured by impinger in mines and the implementation of engineering controls, in addition to decreases in the prevalence of silicosis among workers (Institution of Mining Engineers, 1947; NIOSH, 1974).

Abrasive Blasting Studies and Developments in Industry

One of the earliest studies specific to abrasive blasting was conducted in 1920 by Dr. C. E. A. Winslow at a large automobile factory in Connecticut (Table 3 and Figure 5). This evaluation was a follow up of a previous study by the same author in the abrasive manufacturing industry, in which engineering controls, such as confinement of dust-producing work to enclosed spaces and the use of exhaust fans or hoods, were evaluated (Winslow et al., 1919). One conclusion of this earlier study was that the exhaust systems were incapable of reducing dust concentrations to a level that would not induce disease. Thus, the 1920 study addressed various engineering controls and the feasibility of requiring air-supplied helmets and respirators to protect workers during blasting (Winslow et al., 1920). Dust concentrations during abrasive blasting were found to be high (Table 3), and the efficacy of various combinations of a respirator (described as an ordinary “muzzle” type with a rubber body fitting over the nose and mouth, an air filter composed of two layers of muslin, and a piece of sponge fitted with an air outlet valve) and helmet (made of cloth-covered cardboard with an inlet tube on top for fresh-air supply) was evaluated. It was reported that respirator use improved worker protection, but that the supply of positive-pressure air to the face mask afforded the greatest protection (Winslow et al., 1920). These results were consistent with a later study carried out by the USPHS in the granite industry, where the efficacies of positive-pressure air supply helmets were evaluated (Table 3) (Russell et al., 1929; Bloomfield, 1929). Between 1929 and 1931, J. J. Bloomfield and Leonard Greenburg of the USPHS, in cooperation with the National Safety Council (NSC), surveyed plants across the United States—primarily foundries—with abrasive blasting operations and evaluated the efficacy of protective equipment in abrasive blasting rooms (positive-pressure air-supplied helmet, respirator and non-air-supplied helmet, and helmets only without separate air supply) (Bloomfield & Greenburg, 1933). The results were very similar to what was observed nearly a decade earlier by Winslow et al. (1920): the use of an air-supplied helmet significantly reduced dust concentrations relative to the other combinations of protective equipment. It was noted by Bloomfield and Greenburg (1933) that dust concentrations in foundries were high, and that the use of positive-pressure air-supply respiratory devices was often the primary means of adequately protecting workers.

An additional study deserving mention was conducted by E. R. A. Merewether in the early 1930s in the sandblasting industry in England (Merewether, 1936). This study reported that the duration of employment of abrasive blasters who died from silicosis was much shorter than that of other trades known to involve high silica exposure (10.3 vs. 40.1 yr, respectively), and that abrasive blasters experienced higher rates of silicosis and silicosis with tuberculosis. In his discussion, Dr. Merewether noted that the severity of silica exposures during abrasive blasting could be due to the creation of extremely fine particles as the abrasive material came into contact with the object being blasted. His report also indicated that the only adequate protection for an abrasive blaster was the use of a helmet with a positive fresh-air supply.

Respiratory Protection Guidelines for Abrasive Blasting

Recommendations for Engineering Controls in Dusty Operations After it became clear to the scientific community and to industry that abrasive blasting operations were among the most hazardous work practices involving crystalline silica, the focus shifted to developing engineering controls to reduce exposures in the workplace. Many of the recommendations during this time period focused on making major changes to plant facilities, such as installing ventilation systems or building separate blast rooms or cabinets. For example, the foundry industry developed ways to

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3A survey of abrasive blasting equipment manufacturers revealed that approximately two-thirds of plants used sand as an abrasive material (metal shot was the most commonly used alternative).
<table>
<thead>
<tr>
<th>Study (year)</th>
<th>Industry</th>
<th>Measurement Technique</th>
<th>Ambient Dust (mppcf)</th>
<th>Respiratory Protection</th>
<th>Average Dust (mppcf) under PPE</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Winslow et al (1920)</td>
<td>Auto parts factory</td>
<td>Palmer water-spray apparatus</td>
<td>60.9</td>
<td>Respirator only</td>
<td>4.5</td>
<td>Combination of respirator and helmet with positive pressure provided greatest protection</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Helmet + respirator (no air-supply)</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Helmet with positive-pressure only</td>
<td>0.37</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Helmet with positive-pressure + respirator</td>
<td>0.15</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Russell et al (1929)</td>
<td>Granite</td>
<td>Greenburg-Smith impinger</td>
<td>157.1</td>
<td>Helmet + no air-supply</td>
<td>11.7</td>
<td>Use of positive air supplied hoods provides a greater advantage to the abrasive blaster</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Helmet + positive-pressure</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Greenburg and Winslow (1932)*</td>
<td>Abrasive blasting operations</td>
<td>Greenburg-Smith impinger</td>
<td>2392</td>
<td>Helmet + respirator (no air-supply)</td>
<td>1912</td>
<td>Combination of respirator and helmet with positive pressure provided greatest protection</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Helmet + positive-pressure</td>
<td>25</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bloomfield and Greenburg (1933)*</td>
<td>Abrasive blasting operations (mostly foundries)</td>
<td>Greenburg-Smith impinger</td>
<td>2000</td>
<td>Helmet + positive-pressure + respirator</td>
<td>1317</td>
<td>Combination of respirator and helmet with positive pressure provided greatest protection</td>
</tr>
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<td></td>
<td></td>
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<td>25</td>
<td></td>
</tr>
</tbody>
</table>

Note: Ambient dust (mppcf) represents concentrations inside abrasive blasting room during abrasive blasting operations.

*Values reported for operations associated with maximum ambient dust concentrations.
FIGURE 5. Timeline of key events and studies related to abrasive blasting (1900–2008).

1900

1925

1950

1975

2000

1970 Council of Europe publishes report on materials that could be used as alternatives to abrasive sand blasting operations

1949 Silica as abrasive material banned in UK (Council of Europe, 1970)

1933 Silica particles (0.5-5 mm) identified as most hazardous (Bloomfield & Greenburg)

1920 Exposure studies demonstrated that positive pressure air-supplied helmet provided best respiratory protection during abrasive blasting (Winslow et al.)

1950 USPHS and Illinois State Department of Health evaluation of ferrous foundries in Illinois showed 9.2% of 1,937 surveyed foundry workers had pulmonary fibrosis, despite improvement in conditions

1972 Longitudinal study of silicotics initiated in Louisiana Gulf area (Hughes et al., 1982)

1974 NIOSH study of industries with abrasive blasting (Blair)

1974 • NIOSH study of industries with abrasive blasting (Blair)


• Case reports of acute silicosis and systemic lupus erythematosus in abrasive blasters (Ziskind et al.)

1977 Acute silicosis reported in abrasive blasters of tombstones (Suratt et al.)

1974 NIOSH publishes Industrial Health and Safety Criteria for Abrasive Blast Cleaning Operations (Goodier)

1990 CDC reports acute silicosis cluster in abrasive blasters
isolate abrasive blasting operations, including enclosed blasting machines and rooms devoted to these operations (NIOSH, 1976a). Smaller enclosed units, such as cabinets, barrels, and tables, were developed for blasting of smaller casting. Both blasting rooms and small blasting units were developed with exhaust ventilation to control airborne exposures to the worker, and with a collection system to collect the abrasive material for recycling. In 1937, engineering controls (e.g., use of wet methods and ventilation controls) and good housekeeping practices were recommended for reducing dust exposures by the Committee on the Prevention of Silicosis through Engineering Control, which was formed out of the National Silicosis Conference. The committee also noted that, when these dust suppression and control methods were ineffective at reducing dust levels, respiratory protection for workers was essential (National Silicosis Conference, 1938b).

Implementing effective engineering controls to reduce silica exposure often represented a major cost or effort for small employers, and compliance with these recommendations varied considerably throughout industry during this time period. It was often acknowledged in studies and reports that it was nearly impossible to control dust concentrations to an acceptable level during some foundry operations. As a result, many employers imposed restrictions on the amount of time that workers spent doing the most hazardous jobs. It was not an uncommon practice to restrict workers to abrasive blasting for shorter time periods or to require that they divide their time equally between the positions of blaster or pot tender (i.e., maintaining the hopper during abrasive blasting). However, while it was believed that reducing the amount of time employees worked in dusty conditions would lower their overall lifetime exposure, high short-term exposures could still pose a health hazard. Thus, as the last step in the hierarchy of controls, research focused on developing and using respiratory protection in addition to other controls to ensure worker protection in some of the most hazardous operations, including abrasive blasting.

Earliest Recommendations for Respiratory Protection during Abrasive Blasting

When the United States entered World War I in 1917, the threat of chemical warfare increased the demand for American-made respirators; with this demand came a need to ensure that the respirators were providing the protection that they claimed. The USBM published the first requirements for respirator approval in 1919, Schedules 13 and 14, which were specifically established for self-contained breathing apparatus (SCBA) and gas masks, respectively (Held, 1977). Through these schedules, respirator manufacturers could send their product to the USBM for testing and approval. In 1932, in response to the growing awareness of occupational illnesses, the USBM expanded its respirator approval schedule to include Schedule 19 for supplied-air respirators (as would be used for abrasive blasting) (U.S. Bureau of Mines, 1937). The respirator approval schedule was again expanded in 1934 to include dust, mist, and fume respirators (Schedule 21), and a few years later, in 1944, it published Schedule 23 for chemical cartridge respirators (Held, 1977).

While much of the equipment for abrasive blasting was the same across different industries, the means of controlling airborne dust through engineering controls and personal protective equipment often differed. Prior to the 1920s, there were no federal regulations for respiratory protection for abrasive blasting operations in the workplace (Figure 6). Foundries were the first to implement engineering controls and methods of respiratory protection during abrasive blasting. In 1916, the American Foundrymen’s Association (AFA) published safety codes for respiratory protection in foundries. The code stated that “Sand-blasting by hand-operating apparatus shall be carried on in suitable sand-blast rooms. Sand or shot blast operatives shall be provided with suitable helmets or masks, respirators, approved safety googles, gloves and leggings.” (American Foundrymen’s Association, 1916).

In 1923, the first U.S. governmental safety code was published by the Department of Labor (DOL) Bureau of Labor Statistics for foundry workers, which recommended that appropriate helmets or hoods should be provided by the employer to workers engaged in abrasive blasting operations (U.S. Bureau of Labor Statistics, 1923). The AFA and DOL recommendations were derived from many of the early studies on exposure and control of silica during abrasive blasting in foundries. Specifically, Winslow et al. (1920) recommended specifications for appropriate air-supply volumes and length of tubing for the air supply, although it was acknowledged that in environments where the worker would be mobile over large areas, an air-line system delivering positive pressure might not be feasible. A later study conducted by Bloomfield and Greenburg (1933) determined the
FIGURE 6. Timeline of recommendations and regulations of respiratory requirements and engineering controls for abrasive blasting (1900–2008).
optimal rate of supplied air, six cubic feet per minute, that was most effective in reducing dust levels in the breathing zone of the worker; it was eventually incorporated in the US Bureau of Mines criteria for effective operation of air-supplied respirators (US Bureau of Mines, 1937). These findings were also incorporated in recommendations made by the AFA in 1935 that all helmets were to be supplied with positive-pressure air flow wherever the concentration of dust was thought to be a health hazard (American Foundrymen’s Association, 1935).

Positive-pressure respiratory devices were officially deemed by the US Bureau of Mines Schedule 19, in which, supplied-air respirators are categorized as follows: Type A—hose mask with blower; Type B—hose mask without blower; Type C—air-line respirator (to be used in conjunction with a pressurized air system). Additional categories were also defined for supplied-air respirators that could be modified with protective covering for the head and neck for use in abrasive blasting. These modified respirators were referred to as Type AE, BE, and CE, where the letter “E” designation indicated that the respirator had been altered to provide physical protection during abrasive blasting operations (Leitch, 1935).

The research on respiratory protection was incorporated into discussions at the national level during the Second National Silicosis Conference of 1937 (National Silicosis Conference, 1937). In the final report of the Committee on the Prevention of Silicosis through Engineering Control, an abrasive blasting respirator was defined as an air-supplied device or respirator with a covering for the head, neck, and shoulders, designed to protect against rebounding particles. Either a hood with an air-supplied respirator or a helmet with a continuous flow of supplied air was stated to be sufficient for this purpose (National Silicosis Conference, 1938b).

THE MIDDLE YEARS (1940–1969) —ERA OF DEVELOPING WORKPLACE CONTROLS

Studies of Health Effects

By the 1950s, continuing worker compensation claims for silicosis demonstrated that it was still a significant industrial problem in the United States (Doyle et al., 1958). Based on compilations of available data from 1950 to 1955, the USPHS was able to collect information on 12,763 cases of silicosis that had been identified during that 6-yr period (Trasko, 1958). These figures were obtained from x-ray examinations, physician reports, and death records of workers in dusty trades, and it was believed that the true number of silicosis cases were grossly underestimated due to incomplete data and lack of reporting. Two-thirds of the cases were associated with mining industries (i.e., metal, and nonmetallic mining and quarrying). The remaining cases were from manufacturing industries, including foundries, pottery, stone, silica-brick, tile, clay, and glass industries. Most notable, however, was the finding that silicosis was diagnosed in workers who began employment after the mid-1930s, when the majority of dust controls were implemented across most industries.

Reevaluation of the Metal Mining Industry

In December of 1956, Congress appropriated funds to the USPHS and the USBM to reevaluate the exposure and health status of workers in the metal mining industry. A direct outcome of this funding was a large-scale environmental and medical survey that was conducted in 1958–1961 in 67 underground mines employing approximately 20,500 persons, which represented over 50% of the U.S. work force within this industry (Flinn et al., 1963). Methods used to reduce airborne dust at the time included the application of water during dusty operations (i.e., drilling, blasting), restriction of dusty operations to certain times of the day, implementation of local and general exhaust ventilation, and use of respiratory protection (i.e., filter-type, air-line). The USPHS and USBM investigations showed that, while dust levels were

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4In 1949, the International Labor Office (ILO) published guidelines for classifying radiographs of pneumoconiosis based on the size and quantity of the nodules in the lungs. According to medical textbooks at the time, the appearance of nodules surrounded by concentrically arranged bundles of collagen between 1 and 10 mm in diameter on the chest x-ray was generally considered indicative of silicosis (NIOSH, 1974). In addition, a work history was usually required for diagnosis. A standardized approach for diagnosing silicosis had important implications in workplace surveillance programs, as well as worker’s compensation claims.
reduced by 80–90% compared to those measured in 1939, some of the engineering controls were ineffective at reducing respirable-sized particles (Flinn et al., 1963). Approximately 75.6% of the samples collected showed airborne dust concentrations less than 5 mppcf, 19.3% in the range of 5–20 mppcf, 3.9% in the range of 20–50 mppcf, and 1.2% greater than 50 mppcf (Flinn et al., 1963). The investigations concluded that the majority of high dust measurements were attributable to insufficient engineering controls in several mines.

In the medical study, chest x-rays, medical exams, and medical and occupational histories of 14,076 metal miners revealed silicosis in 3.4% of the current worker population (Flinn et al., 1963). These rates were significantly lower than previous studies of miners, which had rarely reported a prevalence of silicosis less than 25%. In addition, it was observed in the follow-up study that the occurrence of silicosis was confined to workers with over 15 yr of mining experience, with no disease observed in miners younger than 35 yr or with less than 5 yr of employment.

**Reevaluation of the Granite Industry** Despite the continued occurrence of silicosis in some industries, follow-up investigations confirmed earlier findings among the Vermont granite workers that silicosis was not observed when exposures were maintained below 10 mppcf (Figure 7). In 1955, an environmental survey by the Industrial Hygiene Division of the Vermont Department of Health showed that dust concentrations were much lower than what was reported in the 1924–1926 and 1937–1938 surveys (Hosey et al., 1957). Average counts were well below 10 mppcf, with only 10% of the total measurements exceeding this level. Health surveys indicated that the overall rate of silicosis steadily decreased throughout the 1940s and 1950s, with 45% of surveyed workers being diagnosed with silicosis in 1937–1938, 20.3% in 1952, and 15.1% in 1956 (Hosey et al., 1957). Striking differences in silicosis rates were observed among men who had worked prior to the installation of engineering controls compared to those who had been hired after 1937. Nearly half of the 1112 men employed prior to 1937 had silicosis, whereas only one of 1134 men hired after the installation of dust-control measures exhibited symptoms of disease. Follow-up studies of these granite workers through the 1960s supported prior data indicating that exposures to dust concentrations of less than 5 mppcf did not result in disease (Ashe & Bergstrom, 1964).

**Early Studies Involving Cristobalite and Tridymite** It was not until the first half of this second era that researchers and industrial hygienists began to appreciate the varying fibrogenic potential of the different crystalline forms of silica. Occupational studies in the brick manufacturing and diatomaceous earth industries provided evidence that different polymorphs of crystalline silica, specifically cristobalite and tridymite, had greater fibrogenic potencies than quartz. The development of x-ray diffraction in the early 1940s provided a means for distinguishing and quantifying the polymorphs of crystalline silica in airborne dust samples, which facilitated characterizing the toxicity of each form in subsequent human and animal studies (Berkelhamer, 1941).

Some of the earliest studies of silicosis involving nonquartz forms of crystalline silica were those in the Pennsylvania brick industry (Figure 7). During brick manufacturing, rock containing an average of 97% quartz was heated to temperatures of 1482°C. Dust associated with the finished bricks was composed of approximately 90% cristobalite and tridymite, with only trace amounts of quartz. In a study of 4 brick manufacturing plants in Pennsylvania, Fulton et al. (1941) compared exposures and silicosis prevalence among workers in manufacturing departments where exposures were primarily to quartz versus those involving cristobalite and tridymite. Comparison of silicosis rates among workers in the green (primarily quartz exposure) and burned (primarily cristobalite and tridymite exposure) brick departments revealed similar rates (Fulton et al., 1941). It was observed, however, that silicosis in the burned brick department progressed faster than that observed among men who were primarily exposed to quartz-containing dust.

**Cristobalite in the Diatomite Industry** Diatomaceous earth consists of porous and friable sedimentary rock that is composed of silicate cell walls of diatoms, a type of single-celled algae. It has been mined since the end of the 19th century and used as a liquid filtering medium and as a filler material in a variety of products such as paper, paint, brick, tile, and ceramics. Cristobalite is formed when diatomaceous earth is heated during calcining and flux-calcining processes. During World War II, the volume of diatomaceous earth mining increased dramatically, resulting in an increased incidence of silicosis within this industry (Vigliani & Mottura, 1948; Cooper & Crabley, 1958).
FIGURE 7. Timeline of key studies and events that led to the recognition of the hazards of silica (1940–1969).

- **1948** Cristobalite more pathogenic than quartz in Italian diatomite filter candle manufacturing plants (Vigliani & Mottera)
- **1952–1958** Comprehensive study of diatomite mining and processing industry in California, Nevada, and Oregon shows exposure to cristobalite associated with greater prevalence of silicosis than amorphous silica or quartz (Cooper & Cralley)
- **1956-1961** USPHS and USBM conducts environmental and medical surveys of 20,500 workers in 67 underground mines. Silicosis reported in 3.4% of workers (Flinn et al., 1963)
- **1964** Dust controls improve silicosis in VT granite workers (Ashe & Bergstrom)
- **1957** First AIHA Hygienic Guide for quartz
- **1949** Factories Act banned silica for abrasive blasting in Great Britain (Council of Europe, 1970)
- **1946** First ACGIH MAC for quartz
- **1943** US Navy Minimum Requirements for Safety and Industrial Health in Contract Shipyards
- **1957** Decreased incidence of silicosis and dust levels in Vermont granite mines (Hosay et al.)
- **1950** 1950
- **1940** 1940
- **1930** 1930
- **1952** 1952
- **1958** 1958
- **1952** 1952
- **1958** 1958
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- **1952** 1952
Vigliani and Mottura (1948) studied two Italian factories that manufactured filter candles from crude diatomite mined in Tuscany and calcined, ultimately forming cristobalite (Figure 7). Although it was not always reported whether the measured dust concentrations reflected amorphous or crystalline forms of silica, or quartz versus cristobalite, a number of silicosis cases were identified, with illness developing relatively rapidly, often within a few years. The authors noted that this observation of disease was in contrast to prior studies which indicated diatomite did not produce silicosis. The authors compared the x-ray diffraction patterns of raw versus calcined diatomite and concluded that cristobalite formed by calcining at 1250°C contributed to the development of silicosis within these workers.

In 1952, the California State Department of Public Health and the USPHS conducted a comprehensive environmental and medical study of the diatomite industry, involving 5 plants in California, Nevada, and Oregon (Cooper & Cralley, 1958). Four of the plants produced natural, high-temperature (1800–2000°F) straight-calcined and flux-calcined products, while the fifth produced only natural and low-temperature (approximately 1600°F) straight-calcined products. In the medical survey, x-rays of 869 workers showed changes consistent with pneumoconiosis in 9% of the workers, with the highest rates observed among employees who worked in the mills for 5 yr or more (48 out of 101); mill dust contained a high percentage of cristobalite. In contrast, quarry workers, who were exposed primarily to amorphous silica, did not show abnormal x-rays. Taken together, studies of the diatomite industry provided evidence that exposure to cristobalite was associated with greater prevalence and faster progression of silicosis than exposure to quartz.

**Experimental Animal Studies** The expansion of the diatomite industry and the recognition of the silicosis hazards associated with worker exposure to cristobalite led to experimental studies involving different forms of silica, which showed that cristobalite and tridymite were more fibrogenic than quartz. A hallmark paper on this topic, published by King and colleagues (1953a), showed that, following intratracheal installation of different crystalline forms of silica with similar particle size ranges, tridymite produced the most rapid and severe fibrotic responses in the rat lung, followed by cristobalite and quartz. A later study conducted in the late 1960s attempted to model potential human exposures to cristobalite by exposing animals to airborne concentrations that were representative of potential working environments (Wagner et al., 1968). These experimental animal studies provided support for the development of OELs for the different crystalline forms of silica.

Additional studies in this era provided further evidence of the importance of particle size in determining fibrogenicity (Goldstein & Webster, 1966; Gardner, 1938a; King et al., 1953b). These studies were also conducted using more relevant routes of exposure (i.e., delivery by intratracheal installation or inhalation rather than intravenous injection) (Figure 4). In 1953, King demonstrated that intratracheal installation of silica particles ranging from less than 0.5 μm to 8 μm in diameter all produced fibrosis in the rat lung (King et al., 1953b). A dose-response relationship between particle size and the time required to produce each stage of fibrosis was also observed. Particles 1–2 μm in diameter produced the greatest extent of fibrosis within the shortest period of time following exposure. This observation was in contrast to a 1966 study, which reported a higher grade of fibrosis in rats that were exposed to large (4–5 μm) silica particles compared to those less than 1–3 μm in size (Goldstein & Webster, 1966). In the 1966 study, rats were exposed by intratracheal installation to doses of silica with varying particle size but equal total surface area. The authors noted that the results might be attributed to either higher silica exposures or the direct effect of the larger sized particles.

**Occupational Exposure Limits for Silica**

Prior to the 1940s, a number of governmental, industrial, and research-based organizations provided their views of a safe limit of exposure for airborne crystalline silica (Table 2) (Russell et al., 1929; Kuechle, 1934; Air Hygiene Foundation of America, Inc., 1937b; National Bureau of Standards, 1938). However, no formal scientific review process had been conducted in recommending an OEL until 1946, when the American Conference of Governmental Industrial Hygienists (ACGIH) developed its first maximum allowable concentration (MAC) for silica (ACGIH, 1964). Because it was well recognized that the percentage of crystalline silica in dust dramatically affects the risk of silica-related disease, three MACs were developed based on the content of quartz in airborne dust.
HEALTH HAZARDS OF SILICA AND ABRASIVE BLASTING

Prior to the establishment of the 1946 ACGIH MAC for silica, quartz content in dust was typically determined using a colorimetric assay that measured the amount of silicon. This method was often inconsistent, because the test was based on a timed reaction that could differ significantly between operators. Berkelhamer (1941) reported that x-ray diffraction (XRD) could be used to quantify the amount of various pneumoconiosis-producing dusts. This method relied on the unique scattering of x-ray radiation produced by the crystalline lattice structure of each polymorph of silica and had several advantages over previous methods that used microscopy or colorimetric tests (i.e., analytical speed and sensitivity, minimal sample preparation time, ability to identify the different polymorphs of crystalline silica, capabilities for automation). The disadvantages of this technique were that very small amounts of silica were not detected efficiently, particularly when the sample contained a mixture of crystalline silica polymorphs (Berkelhamer, 1941).

Abrasive Blasting Studies and Developments in Industry

Abrasive Blasting in Foundries and Shipyards  Foundry studies published in the 1940s and 1950s focused on the effectiveness of previously installed dust controls for reducing the incidence of silicosis. These studies were motivated by workers' compensation claims for silicosis that continued to appear through the 1940s and suggested that health risks from airborne dust were still a problem for foundry workers. In 1948, the USPHS and the Illinois State Department of Public Health conducted a 1-yr study of 18 representative ferrous foundries in Illinois to evaluate whether dust-control measures were adequately protecting workers against silicosis (U.S. Public Health Service and Illinois Department of Public Health, 1950). Abrasive blasting operations across the different foundries were performed using a variety of materials, including sand, synthetic abrasive, chilled grit, and shot. The authors concluded that foundry dust levels were generally lower than had been reported over the previous two decades, it was noted that further improvement was still necessary. Concurrent medical examinations of nearly 1937 male employees at 16 foundries revealed pulmonary fibrosis in 9.2% of foundry workers, with the highest incidence observed in workers performing cleaning and finishing operations (U.S. Public Health Service and Illinois Department of Public Health, 1950).

In addition to formal studies conducted by public health agencies, numerous articles published throughout this time period in the National Safety News (a monthly publication of the NSC) addressed issues related to abrasive blasting. One of the overarching themes was the need to ensure that blast rooms, cabinets, and rotating tumbler were sealed and properly maintained to prevent silica-containing dust from escaping into the general work area (Castrop, 1948; National Safety Council, 1945, 1956). It was also emphasized that employees working in the blast rooms should wear air-supplied hoods, and that helmets should be stored in a separate area to prevent
contamination with silica dust. These recommendations were not limited to abrasive blasting using silica-containing sand; it was recognized that the use of steel grit as an abrasive could also generate significant dust levels, and the same safety measures were recommended (Castrop, 1948).

Abrasive blasting was also used widely in the maritime industry for surface preparation of hulls, tanks, and other components, as well as for removing marine growth from the bottom of ships, a process referred to as “sandblast fanning” (U.S. Bureau of Ships, 1955). In contrast to abrasive blasting in foundries, these operations were often conducted in the open air (e.g., hull preparation) or in tightly confined areas on a ship (e.g., surface preparation in a tank) (Figure 8). The engineering controls typically used in foundries were simply not feasible for many shipyard operations, necessitating that shipyards develop alternative means for protecting workers against the hazards associated with abrasive blasting. One common control involved attaching dust-removal apparatuses to blasting hoses to collect dust as it was generated. Some automation was implemented; for example, self-filling hoppers and remote-control systems were sometimes used for abrasive blasting (U.S. Bureau of Ships, 1959). Remote-control systems allowed the blaster to control the flow of compressed air and abrasive material from the handheld nozzle, negating the need for a pot tender. This method was particularly useful for blasting operations in tanks or on scaffolding during preparation of ship hulls where the blaster was not near the hopper.

Investigation of Alternative Abrasives In 1950, the use of silica sand was banned for abrasive blasting in Great Britain (Factories Act), followed by the Netherlands in 1956 and Belgium in 1964 (Council of Europe, 1970). The ban on silica as an abrasive material prompted research related to alternative materials that would provide comparable surface preparation without the toxicity of silica. The majority of this research was performed in Europe. Studies in Norway reported that olivine was satisfactory for cleaning foundry castings (Forbes et al., 1950). Around this same time period, animal studies were also conducted to evaluate the potential toxicity of these alternatives relative to silica sand; intratracheal installation of iron silicate did not produce fibrosis in rats, whereas exposure to silica did

![Figure 8](image-url)
HEALTH HAZARDS OF SILICA AND ABRASIVE BLASTING

(Holmqvist & Swensson, 1963). A 1970 report by the Council of Europe detailed a number of alternative abrasives, including cut steel wires, certain corundum (various aluminum oxides), certain metallic oxides or mixtures thereof, crushed slags from metal smelting or other metal working processes (copper, iron), glass (ground or pelleted), zirconium silicate, and crushed or granulated vegetable products (e.g., cherry pits, almond shells) (Council of Europe, 1970).

The use of alternative abrasives was also considered in the United States as an option to reduce the health risk associated with abrasive blasting operations (Brandt, 1943). The most frequently recommended alternative abrasive around this time was steel shot. However, it was generally recognized that sand had superior qualities as an abrasive and, in many cases, was less expensive than some of the common alternatives. Because of the large quantities of abrasive used in blasting operations, cost was a major factor for most industries. Furthermore, abrasives such as steel shot were known to create metallic dust, which required the same safety measures (supplied-air respirators and abrasive blasting helmets) as silica abrasives (Brandt, 1943). Therefore, because silica sand was viewed as an important industrial resource in the United States, rather than banning the material, the United States sought to control occupational exposures associated with abrasive blasting operations through engineering controls and respiratory protection.

Respiratory Protection Guidelines for Abrasive Blasting

Many of the recommendations for respiratory protection during abrasive blasting operations given by the USPHS, USBM, DOL, and AFA in the 1920s and 1930s were adopted and/or expanded upon by more specialized groups, specifically the maritime industry, in which abrasive blasting was performed frequently. Because of the unique nature of abrasive blasting operations in shipyards, special considerations regarding clean-air sources for airline respirators and falling or entanglement hazards were sometimes needed. In 1943, the U.S. Navy and U.S. Maritime Commission jointly published Minimum Requirements for Safety and Industrial Health in Contract Shipyards. While the requirements for respiratory equipment during abrasive blasting were not specific, they stated that adequate respiratory protective devices should include “abrasive blasting helmets” and “dust respirators” (U.S. Navy and Maritime Commission, 1943). In 1960, the DOL published Safety and Health Regulations for Ship Repairing. In a section devoted to mechanical paint removal, the regulations stated that abrasive blasters were required to be protected by hoods and air-line respirators or helmets with positive-pressure air flow (U.S. Department of Labor, 1960). Employees in the vicinity of blasters, including the pot tenders and recovery men, were to be provided with both eye and respiratory protective equipment. Both the Navy and DOL documents recommended that all respiratory equipment be approved by the USBM (U.S. Department of Labor, 1960; U.S. Navy and Maritime Commission, 1943).

In addition to the maritime industry, independent standard-setting organizations also began publishing safety and health standards related to silica exposure and abrasive blasting. The American Standards Association (now the American National Standards Institute, or ANSI) published the American Standard Safety Code for Heads, Eyes, and Respiratory Protection in 1959 and later published the American National Standard Practices for Respiratory Protection in 1969. These two documents not only recommended respiratory protection for abrasive blasting, but were the first to identify the limitations of different types of respiratory protection (ASA, 1959; ANSI, 1969). It was acknowledged that, in some circumstances, there may be physical limitations to wearing a hose mask or air-line respirator, such as when the operation requires the worker to maneuver or work around obstructions not amenable to the proper use of such a device (ASA, 1959; ANSI, 1969). In these situations, the respirator itself may create additional entanglements or falling hazards. ANSI clarified its position on mechanical filter respirators in a 1968 standard titled “Ventilation and Safe Practices of Abrasive Blasting Operations.” This standard stated that, according to the recommendation of a proper authority having jurisdiction over the work site (i.e., an employer), particulate-filter respirators were acceptable only for occasional or short-term dust exposures when nonsilica abrasives were used, and such respirators were not to be used as a continuous form of protection when silica was used as the abrasive. ANSI further indicated that only USBM-approved abrasive blasting respirators should be worn by workers performing abrasive blasting (ANSI, 1968).
Government and industry-based professional industrial hygiene associations, such as the AIHA and ACGIH, also followed suit in providing recommendations for respiratory protective equipment for abrasive blasting operations. In 1963, the AIHA and ACGIH jointly published the first manual devoted solely to the selection and use of respirators, which became widely used throughout industry. In this manual, abrasive blasting respirators were defined as one of two general designs: (1) a mask or tight-fitting face piece with a connection to a source of supplied air, and including a hood for rebound protection; or (2) a helmet that covers the neck, with a connection to an air supply through a hose line (AIHA-ACGIH, 1963).


Studies of Health Effects

Exposure and Health Surveillance by the Government Following the passage of the Occupational Safety and Health Act by Congress in 1970, the Occupational Safety and Health Administration (OSHA), in the DOL, was formed to enforce occupational safety and health laws while the National Institute for Occupational Safety and Health (NIOSH), now part of the Centers for Disease Control and Prevention in the Department of Health and Human Services, was to be responsible for research and recommendations related to occupational safety and health. NIOSH and OSHA initiated surveys in several different industries to characterize the extent of silica exposure and disease rates among the U.S. workforce. One of the first efforts led by NIOSH was the National Occupational Health Survey (NOHS), in which approximately 5,000 establishments were surveyed and over 9,000 potential occupational hazards were identified (NIOSH, 2006a). Based on this initial survey, NIOSH estimated that over 3 million workers in 238,000 plants were potentially exposed to silica (NIOSH, 1983). In the early 1980s, a second survey, the National Occupational Exposure Survey (NOES), included approximately 4500 establishments (NIOSH, 2006b, 2006d). Both surveys involved site visits and interviews with management to collect information about each facility and occupational health and safety policies. The details of each site visit were entered into a large database, which provided records describing potential workplace exposures by industry, occupation, and chemical. In addition to the industry-wide surveys, NIOSH conducted health hazard evaluations (HHEs) of individual workplaces at the request of employers or employees (NIOSH, 2006a). Over the past 30 yr, there have been more than 200 HHE evaluations pertaining to silica, which are available to the public.

Silica was also identified as one of five key industrial hazards when OSHA initiated its “Target Health Hazards Program” in 1972. By 1976, more than 5000 inspections were conducted under the auspices of this program. Approximately 60,000 air samples were collected by OSHA, including over 8000 samples of airborne silica. Many of the silica samples were collected at foundries, with 37% of the samples collected in all industries showing concentrations in excess of the OEL (Ryer, 1978). A follow-up analysis of the OSHA inspection data collected from 1972 to 1982 revealed that 20% of samples across all industries evaluated exceeded the TLV by a factor greater than two (NIOSH, 1983). OSHA’s findings were also supported by independent research studies involving foundries (Oudiz, 1986). In total, exposure studies and OSHA sampling data indicated that, despite continued efforts, dust levels were not always sufficiently controlled (NIOSH, 1985, 2006a; Froines et al., 1986; NIOSH, 1976a).

Incidence of Silicosis Declines During the 1980s, exposure data compiled by OSHA and NIOSH were coupled with health statistics to specifically track the incidence of silicosis within various industries across the United States. These efforts involved collaboration between NIOSH and the National Center for Health Statistics (NCHS) to develop the National Occupational Mortality System (NOMS), which facilitated surveillance of cause-specific mortality by the decedent’s industry code or occupation (NIOSH, 2006d). In addition to this program, NIOSH established the National Surveillance System of Pneumoconiosis Mortality (NSSPM) in the late 1980s, which provided annual updates of the total number of deaths in the United States for which pneumoconiosis was listed as the underlying cause (NIOSH, 2006d). Despite evidence that silica exposure levels continued to exceed regulatory
standards in many industries, the overall number of deaths due to silicosis (as an underlying or contributing cause) decreased steadily throughout this era (Figure 9). Age-adjusted mortality rates in the United States, where silicosis was identified as the underlying cause of death, were at least twofold lower by 1990 compared to 1970 (NIOSH, 2003). A greater than fivefold reduction over the same time period was noted for deaths where silicosis was identified as a contributing cause of death.

This favorable trend of silicosis reduction was seen not only in industries involving quartz, but also in those primarily associated with cristobalite exposure. A large-scale reevaluation of diatomaceous earth workers found, for example, that lung changes associated with occupational exposure were observed in 2.8% of workers, compared to 9% in the original 1953–1954 USPHS survey. This reduction was attributed to dust-control practices and use of respirators (Cooper & Cralley, 1958; Cooper & Jacobson, 1977). Similarly, in 1970–1971, periodic chest x-rays taken of Canadian brick and tile workers since 1958 showed no cases of silicosis among 1166 production workers (Rajhans & Budlovsky, 1972).

**Retrospective Analysis of Silica Cohorts** Up until this era, cohort studies typically reported rates of silicosis among the current worker population and did not follow employees who had left the work force. For chronic silicosis, there is at least a 10-yr latency period for the development of disease. Thus, a potentially large number of workers could have been misclassified as nondiseased, because they were evaluated before any clinical symptoms developed. Consequently, epidemiological studies published in the 1970s and 1980s focused on disease rates among those who had remained in the work force long enough to potentially develop silicosis. Many of the studies conducted at this time evaluated large occupational cohorts, some of which included people who had worked in the 1940s. The availability of environmental and medical data for a subset of workers who had been exposed to varying concentrations of airborne silica for decades allowed researchers to provide quantitative estimates of the exposure-response relationship for silica. This type of analysis represented an important advancement from earlier studies in the granite industry, which could

![Figure 9](image_url) **Figure 9.** Number of deaths due to silicosis (underlying or contributing cause) among U.S. residents age 15 yr and over (1968–1999) (NIOSH, 2005).
establish only that workers in areas where airborne silica concentrations were kept below a certain level did not appear to develop disease.

Despite the longer follow-up periods, researchers still faced methodological challenges. A major source of uncertainty was the lack of complete and quantitative historical industrial hygiene data. This lack of information was due, in part, to the fact that air samples were often taken sporadically and under a variety of conditions. Changes in sampling technology also presented a major hurdle in estimating exposure over time, especially when older sampling data were collected using the impinger method and data regarding the silica content of the dust were not available. For example, in the late 1960s and early 1970s, researchers and industrial hygienists began moving away from the impinger method (e.g., particle number) to the gravimetric method to assess airborne silica concentrations in the work environment. Later, size-selective sampling devices were developed that separated out larger dust particles and measured only those that were capable of penetrating the lungs (Ayer et al., 1968; NIOSH, 1974). Gravimetric sampling allowed measurements to be taken over a full shift and also provided enough sample volume to allow for analysis of free-silica content. However, with this shift in air-sampling methodology, it was necessary to determine how the results of one method correlated with the other, because historical measurements had been derived from impinger or particle-count measurements.

To address the discrepancy between impinger and gravimetric data, a factor to convert particle count into particle mass concentrations was developed (Ayer, 1969; Ayer et al., 1973). Specifically, Ayer et al. (1973) simulated 1920 conditions in granite sheds and measured free silica by dust count, using both the Greenburg–Smith impinger and the gravimetric method. A comparison of the two types of measurements suggested that 10 mppcf was roughly equivalent to 0.2 mg/m³. Subsequent studies aimed at correlating the two types of measurements did not always find a relationship, which generated some controversy at the time, because nearly all of the studies conducted through the 1960s had relied on particle counts (NIOSH, 1983; Rice et al., 1984). Despite this controversy, the original conversion factor developed by Ayer (1973) was the one most frequently used in epidemiological studies and by standard-setting groups to convert older particle count values to particle mass measurements.

In addition to the uncertainties associated with interpreting older exposure data, the criteria for identifying and diagnosing silicosis in worker populations varied over time and by industry, making it difficult to retrospectively estimate the true disease rate in a given population. This discrepancy in diagnosing silicosis has hampered direct comparisons of studies. While many studies relied on International Labor Office (ILO) criteria for interpreting chest radiographs for pneumocnosis, the cutoff point (e.g., 1/0 vs. 1/1) used to determine whether a person was classified as “silicotic” often varied. International studies occasionally used criteria other than the ILO classification system for reading chest radiographs for silicosis (Chen et al., 2001, 2005). In addition to variations based on diagnostic criteria, the choice of the comparison population used to generate standard mortality ratios (SMRs) in many studies contributed an additional level of uncertainty. The strength of the association could vary considerably depending on whether local or national death rates were used to generate risk estimates.

**Exposure-Response Studies** Studies of the granite industry conducted in the late 1960s by the Harvard School of Public Health and the Vermont Industrial Hygiene Division were among the first to address the relationship between lifetime exposure to quartz and lung disease (Theriault et al., 1974a, 1974b, 1974c). In these studies, personal lapel samples, collected using the gravimetric method, were used to estimate worker exposures to respirable dust. Lifetime dust exposure was calculated, and a factor of 10 was applied for all exposures prior to 1940, based on the assumption that airborne dust concentrations prior to the installation of dust controls were likely higher than what had been reported in more recent samples. A study by Ayer et al. (1968) reported that the respirable fraction of

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6 Gravimetric sampling provided a solution for many of the problems associated with the particle count method, such as agglomeration of dust particles, which could affect the proportion of particles of respirable size that were actually counted by the method. Particle count results could be highly variable, and because of the short sampling time of the impinger, multiple samples were often required to estimate exposure levels.
the dust in the 1960s contained 7.2% quartz, whereas dust measurements from the first studies of the granite industry reportedly contained between 25 and 35% quartz (Theriault et al., 1974a). Workers were divided into different exposure groups, similar to the four categories evaluated in 1929 by the USPHS (Russell et al., 1929). Again, no respiratory disease, defined by abnormal chest X-rays, was reported in workers in the lowest exposure group (9 mppcf), with average cumulative granite dust exposures of 19 dust-yr (equivalent to an exposure of 0.5 mg/m³ for 19 yr) and thus provided further support for the protectiveness of the OSHA PEL (Theriault et al., 1974c).

The dose-response relationship for silicosis was also evaluated in workers in the dusty trades in North Carolina. These data were collected as part of a formal state program that had been in place since 1935 and included information related to worker pre-employment health status, occupational history, annual physical exams, and air measurements from workplace inspections (Baucom, 1986). The dusty trades included in the program have expanded over time. While early operations around 1900 were limited to mica mining, industries such as hard-rock mining, quarrying, mineral processing, and foundries were incorporated into the program as they developed in the state. Similar to historical surveys of the Vermont granite sheds and quarries, the incidence of silicosis in the North Carolina worker cohort correlated with changes in workplace practices and dust-control measures over time. Because the study period covered a long time span (1935–1980), a cumulative exposure model was selected to evaluate the threshold at which no silicosis was observed (Rice et al., 1986). Cumulative exposure estimates were calculated by adjusting the total dust concentrations by the average quartz content within each industry (estimated from historical settled dust samples). The cumulative exposure at which no increased risk for silicosis was observed was roughly equivalent to a mean exposure of 1 mppcf over a 40-yr working lifetime. Moderately elevated odds ratios (ORs) (ranging from 1.03 to 3.04) were observed among workers with a mean exposure of 2.5 mppcf over a 40-yr working lifetime. Conversion of the 1-mppcf estimate to respirable mass unit yielded a value of approximately 0.1 mg/m³, which was equivalent to the OSHA PEL and consistent with what had been reported in the Vermont studies. The exposure and health experience of the Vermont granite workers and the tradesmen of North Carolina provided additional support that the OSHA PEL was sufficiently protective against silicosis.

Understanding Early Studies of Silica and Lung Cancer

As the incidence of silicosis continued to decrease throughout this era, the focus of many of the health studies shifted to the potential relationship between silica exposure and lung cancer. Several autopsy studies conducted in the 1950s and 1960s suggested that there might be an increased risk of lung cancer in mineral-dust workers, but these were not considered persuasive evidence of a causal association (Gloyne, 1951; Heuper, 1966). By the mid-1970s, the observation of higher rates of lung cancer among metal miners prompted researchers to consider silica as a potentially carcinogenic component of mining dust. Although the presence of other known carcinogens (such as radium ore decay products or amosite asbestos) in the mining industry was well known, several studies reported mixed associations between silica exposure and lung cancer (Gillam et al., 1976; McDonald et al., 1978; Brown et al., 1986; Higgins et al., 1983; Finkelstein et al., 1986). However many of these studies did not control for confounding exposures, and accurate information regarding smoking history was generally unavailable or not properly gathered. As a result of these shortcomings, these investigations were not widely supported within the scientific community.

It was not until the early 1980s that more robust studies of lung cancer in silicotics appeared in the scientific literature (Figure 10). Analyses of data from the Swedish pneumoconiosis register (which contains all silicosis cases, compensated or uncompensated, after 1931) indicated that workers employed in the mining and iron and steel industries had higher rates of lung cancer (Westerholm, 1980). The authors noted, however, because of the presence of co-carcinogens in these industries

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7Initially, dust samples were analyzed for silica content using chemical and petrographic methods. Samples were first treated with a series of acids to remove soluble compounds and break down silicates (Drinker, 1954). The petrographic analysis involved the evaluation of certain physical properties, including specific gravity, hardness, color, crystal structure, and others, using the same light-field method of counting impinger samples (Drinker & Hatch, 1954). Based on this type of analysis, it was estimated that Vermont granite dust contained between 25% and 35% quartz (Russell et al., 1929). The colorimetric method, using hydrofluoric acid, was later used to analyze quartz, but it could not distinguish between the polymorphs.
FIGURE 10. Timeline of key studies and events that led to the recognition of the hazards of silica (1970–1989).
and the lack of smoking information, this association did not necessarily indicate a causal relationship. Similarly, Ontario gold miners receiving compensation for silicosis were found to have elevated rates of cancer, as well as mortality from all causes (Finkelstein et al., 1986). Although these early mortality studies reported increased cancer rates (as measured by proportionate mortality ratio [PMR] and SMR, respectively) among silicotics, a major limitation of these studies was that they did not account for smoking history.

In the early 1980s, Goldsmith et al. (1982) brought the issue to the attention of the scientific community (Figure 10). In their study, Goldsmith et al. (1982) indicated that excess mortality rates of respiratory cancer had been reported in North American and European studies in iron and steel foundry workers, steel casting workers, abrasive blasters, metal molders, miners (nonuranium), and ceramic workers, all of which had historically experienced excessive exposures to silica. The reports from the Swedish Pneumoconiosis Register and the Ontario Ministry of Labor were also cited by Goldsmith as evidence that silica exposure was associated with an increased risk of lung cancer (Finkelstein et al., 1982; Westerholm, 1980). Although nearly 20 studies were considered by Goldsmith et al. (1982), the results did not allow for easy inference of a direct causal relationship between silica and lung cancer. This difference was largely due to the fact that the observed associations were as likely to be due to other factors, such as smoking or concurrent exposure to other known occupational carcinogens (i.e., polycyclic aromatic hydrocarbons [PAHs] among foundry workers, radioactive radon daughters and asbestos among mine workers) (Ng, 1994). Other limitations included selection and information bias, as well as a lack of dose-response patterns. Nonetheless, several hypotheses regarding the carcinogenic effects of silica were proposed by Goldsmith et al. (1982): (1) Silica directly produces lung cancer; (2) silicosis may serve as an intermediate pathologic state leading to lung cancer; and (3) silica, linked with PAHs either from smoking or from the ambient working environment, impairs lung clearance and increases the effective dose and/or duration of exposure, inducing neoplasia in the adjacent pulmonary tissue.

**Experimental Animal Studies** The primary focus of animal studies conducted in the 1980s was to evaluate the carcinogenic potential of crystalline silica (Figure 4). The incidence of lung tumors was measured in rats, hamsters, and mice following exposure by multiple routes (e.g., inhalation, intratracheal instillation, intrapleural, intrathoracic, and intraperitoneal injection, and intravenous administration) (Wagner, 1976; Wagner et al., 1968, 1980; IARC Working Group, 1987; Wagner & Wagner, 1972). Positive results were limited to rats, in which lung tumors were observed following inhalation and intratracheal instillation, and thoracic malignant histiocytomas developed following intrapleural and intrathoracic administration (Holland et al., 1983, 1986; Dagle et al., 1986; IARC Working Group, 1987). Although rat inhalation studies reported tumor incidence ranging from 20 to 30% (Dagle et al., 1986; Muhle et al., 1995; Holland et al., 1986), a consistent dose-response pattern was not documented. In addition, tumors were not observed in hamsters, mice, or guinea pigs (Holland et al., 1983; Holland, 1995; Stenback et al., 1986; Niemeier et al., 1986). Based on the animal studies, in 1986, International Agency for Research on Cancer (IARC) classified crystalline silica as a group 2A carcinogen, a designation indicating that there was sufficient evidence available to classify silica as a carcinogen to animals, but only “limited evidence” that silica was carcinogenic in humans (IARC Working Group, 1987).

The fact that evidence of carcinogenicity was limited to the rat has raised several questions about whether silica (alone) is a lung carcinogen. It has been suggested that chronic inflammation, which is not observed in mice or hamsters, may play a role in the carcinogenesis of silica in the rat (Holland, 1995). It has also been proposed that lung fibrosis produced by silicotic lesions in rats may have contributed to the development of lung tumors. These questions have stemmed from (1) observations of the inconsistent latency of tumor incidence (e.g., tumors observed after acute and chronic exposures), (2) co-occurrence of fibrosis and tumors in the rats (i.e., fibrosis is required for the development of carcinogenic tumors), (3) lack of dose-response patterns, and (4) inconsistencies among studies (Holland, 1995; NIOSH, 2002a). Given that malignant tumors were not observed in other animal species (even in a mouse strain with a high background incidence of lung adenoma), it was suggested that rats may have some specific, unique susceptibility to the adverse effects of silica (ILSI, 2000). In addition, silica was not mutagenic in *Salmonella typhimurium* or *Escherichia coli* assays, and in vitro studies in mammalian cells were positive only for micronuclei.
induction (IARC Working Group, 1987). In short, typical tests for mutagenicity have not indicated that silica is likely to be an initiator for cancer.

Occupational Exposure Limits for Silica

The introduction of the respirable mass method as a sampling technique and its validation in the granite worker cohorts paved the way for proposing a new TLV for quartz in 1971 (Table 2). Like the previous standards, the formula used based the TLV on % of crystalline silica. The new TLV did not replace the previously existing particle-count approaches, but provided industrial hygienists with an option to use either method (i.e., both methods or formulas were considered equivalent). In addition to the new respirable-mass TLV for quartz, ACGIH recommended that TLVs for cristobalite and tridymite be limited to one-half the value calculated for quartz (this approach was applied to both respirable mass and particle-count formulas). The ACGIH subsequently decided to drop the mppcf formula entirely from future TLVs (ACGIH, 1966, 1971).

In 1971, OSHA adopted the ACGIH TLVs as a PEL, which was a federally enforceable limit. Although OELs for silica had been in place for nearly 40 yr, this was the first time in history that employers across the nation were required by federal law to maintain airborne contaminants in the workplace below airborne concentrations specified by the government. The PEL was also based on the gravimetric approach, as well as the option for the particle-count approach. In 1983, the ACGIH opted to simplify the guideline by replacing the formulas for calculating TLVs with recommended limits for respirable and total dust for specific concentrations: 0.1 mg/m³ for respirable dust and 0.3 mg/m³ for total dust (ACGIH, 1983). Similarly, the TLVs for cristobalite and tridymite were changed to an explicit and single value of 0.05 mg/m³ (ACGIH, 1983). Because measurements of respirable dust were easily obtained and were considered the most relevant in terms of health risk, the TLVs for total dust were eliminated by ACGIH in 1986.

In 1974, NIOSH published its “Recommended Standard for Occupational Exposure to Crystalline Silica,” in which it proposed a respirable quartz dust limit of 0.05 mg/m³ in the workplace environment as a time-weighted average, in combination with a program of work practices and medical examinations. This recommendation was based, in part, on the studies in the granite industry by Theriault et al. (1974a, 1974b, 1974c), in which pulmonary function tests and chest x-rays were correlated with dust measurements using the new size-selective respirable mass sampling and gravimetric method. NIOSH acknowledged that these studies did not identify a “safe” level of exposure (that is, one that presents no risk but, rather, tolerable risk) and noted that the exposure-response model used in the study predicted a significant incidence of silicosis when zero dust exposure was used as the input. Nonetheless, NIOSH recommended that the limit of exposure to quartz, cristobalite, and tridymite in workplace environments should be no greater than 0.05 mg/m³ (Theriault et al., 1974a, 1974b, 1974c; NIOSH, 1974).

The 1974 NIOSH document was fairly controversial at the time. Not only did NIOSH equate all three crystalline forms of silica (despite the decades of research showing that the hazards of quartz, cristobalite, and tridymite were not the same), but their findings were based on Theriault et al. (1974a, 1974b, 1974c), which NIOSH had acknowledged was not a source suitable for deriving an OEL. OSHA and ACGIH did not adopt NIOSH’s recommendation, due in part, to a study performed by Graham et al. (1981), which evaluated the same group of workers as Theriault et al. and found an increase, rather than a decrease, in pulmonary function (OSHA, 1989). When OSHA updated its standard in 1989, the PEL was changed to 0.1 mg/m³ for respirable dust containing quartz (regardless of percentage) and to 0.05 mg/m³ for cristobalite and tridymite, although these values were later rescinded by the U.S. Court of Appeals based on administrative procedural grounds (OSHA, 1989). A respirable quartz OEL of 0.1 mg/m³ was also recommended by the American Society for Testing and Materials (ASTM) in 1986 (ASTM, 1986). ASTM commented that the Theriault et al. (1974a, 1974b, 1974c) studies did not demonstrate that an OEL higher than 0.05 mg/m³ would be unsafe and criticized the extrapolation of particle-count standards to particle mass concentrations based on concerns that the granite-shed studies (10 mppcf, or approximately 0.1 mg/m³) may not be representative for all types of quartz-containing dusts.
Abrasive Blasting Studies and Developments in Industry

The hazards of abrasive blasting were well known when OSHA was promulgated in 1970. Nonetheless, cases of acute silicosis among abrasive blasters continued to be reported in the literature (Suratt et al., 1977; Owens et al., 1988; Giles et al., 1978). In general, these reports documented silica exposures far in excess of the TLV, with rapid onset of disease (usually within a few years). In addition, exposure studies during abrasive blasting operations were conducted in the Louisiana Gulf area throughout the 1970s (Samimi et al., 1974, 1975, 1978a; Ziskind et al., 1976). With no mining or quarrying industry, silicosis was a relatively rare disease in Louisiana until around 1950, when the offshore drilling, shipbuilding, and ship repair industries developed. Abrasive blasting was commonly used for surface preparation in all of these industries (Hughes, 1982).

In 1972, a longitudinal study of silicotics was initiated in the Louisiana Gulf area. Study participants were identified through personal consultations and local hospitals; by 1982, 83 men with silicosis were enrolled (Hughes et al., 1982). Disease progression was monitored by x-ray examinations and lung function tests. Follow-up ranged from 1 to 7 yr. Air samples for various job titles obtained from Samimi et al. (1978b) were combined with work histories to estimate cumulative silica dust exposures; impairment of lung function was greater among men in the higher exposure categories. Most of the study participants had worked as abrasive blasters in the 1950s and 1960s, and the average length of exposure to abrasive blasting operations was 11.3 yr. These observations were consistent with what had been published previously regarding acute silicosis in abrasive blasters (Merewether, 1936; Jones et al., 1975).

Exposure studies that were also conducted during this period focused specifically on abrasive blasting in Louisiana steel fabrication yards during slow (up to 2.5 h per day), moderate (2.5–5 h), and busy (5 h and above) conditions (Samimi et al., 1974, 1975, 1978a). Air samples were collected for a variety of job types, including the abrasive blaster, pot tender, helper, and other nearby workers (Figure 11). Samples were also collected from inside and outside abrasive blasting hoods (both air-fed and non-air-fed). It was noted by Samimi et al. (1974) that, with the exception of the abrasive blaster, workers rarely wore respiratory protection during abrasive blasting. Airborne

**FIGURE 11.** Respirable silica exposures for different abrasive blasting jobs preformed with and without appropriate respiratory protection (Samimi et al., 1974, 1975).

*workers did not wear respiratory protection.

**workers wore air-supplied hood.
concentrations, reported as the ratio of respirable dust to the TLV at that time, were noted to be 0.3 (inside air-supplied hoods) and 14.1 (outside the hood) during abrasive blasting. Ratios for other types of workers (e.g., helpers, pot tenders, painters, etc.) ranged from 0.7 to 7.4. A 1975 publication of the same data indicated that the respirable dust concentrations were taken “continuously” during the workday and represented the potential time-weighted average concentration during blasting and nonblasting intervals (Samimi et al., 1975). As such, they included time when the hood was off the worker and did not represent exposures when the hood was properly used. For blasters who did not continuously wear air-supplied hoods during abrasive blasting operations, the ratio of respirable silica dust to the contemporaneous TLV ranged from 2.7 to 33.7 (Samimi et al., 1974). Samples collected inside the air-fed hood only during blasting averaged respirable silica concentrations 1/3 the TLV value at that time, indicating that when worn continuously, continuously air-fed hoods could significantly reduce exposures to the abrasive blaster.

The studies conducted by Samimi et al. (1974, 1975) consistently demonstrated that concentrations of respirable dust during abrasive blasting were considerably above the TLV, despite the fact that many of these activities occurred outside. In the late 1970s, OSHA also conducted surveys during abrasive blasting in foundries. Data compiled from inspections indicated a median value of 13% silica in the dust, and nearly 50% of air samples of silica exceeded a level of 1.2 times the PEL (Oudiz, 1986). In addition, some of the health hazard evaluations and walk-through surveys conducted by NIOSH documented misuse of respiratory equipment or other problems with work practices or control equipment.

In response, NIOSH conducted a large-scale survey to determine the number of workers that performed abrasive blasting and characterized practices across different industries (Blair, 1974). Six target locations were chosen, and selection was focused to give a large representation to shipyards (five of the six target locations contained seaports). Other industries surveyed (172 companies in total) included the monument industry, auto body repair shops, painting and construction, and foundries. Questionnaire responses indicated that a high proportion of the abrasive blasting work was performed in unconfined areas (outdoors and in large work areas) or in enclosed areas (i.e., tank with the worker on the outside). Silica sand was used in 44.7% of abrasive blasting operations. Other abrasives that were reportedly used included steel shot (16.7%), steel grit (9.7%), alumina (9.3%), flint/garnet (7%), glass beads (4.6%), carbides (3.5%), slag (3.1%), and organics such as cobs or pecan shells (1.1%) (Blair, 1974). With respect to typical abrasive blasting practices, the majority of industries surveyed used hand-held hoses for the dry-blast processes. The authors noted that the only other process reported in significant numbers was the centrifugal or “airless” process; workers generally did not use respirators when performing airless blasting. In addition, some job sites reported that workers did not wear respiratory protection during outdoor abrasive blasting or other abrasive blasting operations on stone.

In 1974, NIOSH published its “Industrial Health and Safety Criteria for Abrasive Blast Cleaning Operations,” which described typical practices based on a survey of 92 manufacturers known to perform abrasive blasting (Goodier et al., 1974). Airborne dust concentrations at participating plants were measured, and it was reported that when silica sand was used as an abrasive, airborne silica measurements usually exceeded OSHA and ACGIH OELs, which was not observed with metallic shot or slag abrasives (Goodier et al., 1974). Soon thereafter, in 1976, NIOSH published several documents pertaining to safe abrasive blasting operations. These generally focused on engineering controls and work practices and also provided an overview of the various types of blasting equipment and dust control methods, as well as recommended ventilation guidelines for abrasive blasting operations (NIOSH, 1976a, 1976b). In addition, it was also suggested that blasters use some of the newer abrasives available at the time, such as wet-bottom boiler slag (“Black Beauty”) or copper slag, both of which contained less than 1% silica (NIOSH, 1976a).

**Respiratory Protection Guidelines for Abrasive Blasting**

The creation of OSHA and the institution of federal regulations in the early 1970s also affected guidelines for the use of respiratory protective equipment. In 1971, OSHA published its first respiratory protection standard based on the 1969 ANSI standard on respiratory protection...
and ANSI’s standard on identification of gas mask canisters. The OSHA standard required that employers provide proper respiratory protection to their employees when necessary to protect their health and refers the reader to ANSI Z88.2 for guidance as to how to choose the proper respiratory protection. It should be noted that OSHA did not allow that respiratory protection be a primary means of control, rather substitution, isolation, and ventilation should be the principle means of controlling exposure (OSHA, 1971a).

Although the OSHA respiratory protection standard did not specifically address abrasive blasting, the Department of Labor adopted the Bureau of Labor Standards, Safety and Health Regulations for Ship Repairing outlined requirements for abrasive blasting hoses, nozzle couplings, nozzles, “dead man control,” and personal protective equipment (PPE), which included protective clothing and either (1) hoods and air-fed respirators, or (2) air-fed helmets with positive-pressure air flow. Abrasive blasters working in open spaces were also required to wear air-fed respirators or helmets, unless the abrasive contained less than 1% free silica. For low-silica (<1%) situations, a Bureau of Mines USBM-approved filter respirator appropriate for lead dusts was permitted. Eye and respiratory protection, in addition to protection against impact, were also required for workers in proximity to abrasive blasters (e.g., pot tenders, recovery men) (U.S. Department of Labor, 1964).

NIOSH became involved in respiratory protection when it began approving respirators in 1972. NIOSH jointly approved respirators with USBM until 1973, when the Mining Enforcement Safety Administration (MESA) was created from the regulatory division of the USBM and assumed safety and health enforcement functions. In 1974, MESA assumed the former USBM role of jointly approving respirators with NIOSH, until 1977, when the Federal Mine Safety and Health Act was passed. The passage of this act created the Mine Safety and Health Administration (MSHA) by moving MESA from the Department of the Interior to DOL (U.S. Department of Labor, 1977; OSHA, 1998b). NIOSH and MSHA jointly approved respirators until 1995, when NIOSH became the sole approval agency for all respiratory protection equipment, with the exception of selected mining-specific respirators (NIOSH, 1995; OSHA, 1998b).

In the 1970s, NIOSH contracted the Los Alamos Scientific Laboratory (LASL) to provide advice on how to improve the respirator approval process. In 1976, LASL acknowledged the need for the respirator certification program to include fit testing. This recommendation was based on human studies that evaluated how respirators fit on differently sized and proportioned faces (Douglas et al., 1976; Hack et al., 1974). One of the major outcomes was the development of a “fit test panel,” which incorporated dimensions of the face and lips of the various participants in the studies. This information was used during respirator testing to ensure that each respirator would provide adequate protection to a heterogeneous worker population (Douglas et al., 1976).

Quantitative respirator fit testing was incorporated into the ANSI 1980 update of the American National Standard Practices for Respiratory Protection (Z88.2). This document also introduced a method for assigning respirator establishing protection factors based on the respirator, work area, and wearer, which provided a more conservative approach to the selection and classification of respirators (ANSI, 1980).

As described previously, NIOSH was actively involved in investigations of abrasive blasting practices throughout the 1970s. The resulting reports often discussed what respiratory protection measures were necessary to adequately protect workers against silica exposures. The use of a separate air supply (either as a supplied-air respirator or an air-supplied hood) was always recommended, which was consistent with the scientific literature and previously published guidelines in existence at the time (Figure 6) (U.S. Department of Labor, 1964). Specifically, NIOSH recommended a Type C continuous-flow, supplied-air respirator (in combination with a hood or helmet) during abrasive blasting using silica sand (NIOSH, 1974). These recommendations were adopted by OSHA in a 1978 directive, which served as an internal document for OSHA to evaluate compliance with the standard and determine whether there were grounds for citing an employer (OSHA, 1978). Furthermore, a 1976 NIOSH report on engineering controls and work practices for abrasive blasting operations recommended wearing an abrasive blasting respirator that was MESA/NIOSH approved (NIOSH, 1976a).
Studies of Health Effects

Silicosis Exposure-Response Models A major focus of health studies in the 1990s was to further characterize the exposure-response relationship between crystalline silica and silicosis. Improvements in exposure assessment and dose reconstruction methods, as well as continued follow-up of established occupational cohorts, allowed researchers to generate quantitative estimates of disease risk at different levels of silica exposure over a working lifetime. These new studies, which attempted to account for well-established confounders (e.g., diagnostic criteria, length of follow-up), suggested that the OSHA PEL for silica did not provide sufficient protection against the development of disease. Most of these studies offered a quantitative estimate of risk for silicosis mortality, and at least one developed a model to estimate a “no-observed-adverse-effect level” (NOAEL), which would define a threshold dose below which illness would not be expected (Rice & Stayner, 1995).

A cohort morbidity study of Ontario hardrock miners was among the first to develop quantitative risk estimates for silicosis associated with different respirable silica exposures for a working lifetime (Muir et al., 1989a, 1989b; Verma et al., 1989). This study was a joint effort between the management of the gold mines, Canadian government, and Workers’ Compensation board, and involved collecting of data from 2,109 miners at two gold and uranium mines. The highest risks were observed among workers employed in dusty areas, rather than those who had worked the longest in the industry. Although workers were not followed after leaving the mines, there were a total of 32 (1.5%) cases of silicosis identified by a panel of B-readers in this cohort. The authors estimated a cumulative risk estimate (%) of silicosis incidence of 1.2 (0.7–2.1) based on 40 yr of exposure at a mean respirable concentration of the current OSHA PEL of 0.1 mg/m³ (Muir et al., 1989a, 1989b; Verma et al., 1989).

While the analysis of the hardrock miners was one of the more thorough exposure-response studies conducted during this time period, the small number of silicosis cases and the lack of follow-up of workers after they left the work force were considered significant shortcomings, in that the true prevalence of silicosis could have been substantially underestimated. Later cohort studies conducted in the early 1990s included longer follow-up periods and attempted to determine the health status of workers who had left the workplace. In a cohort study by Hnizdo and Sluis-Cremer (1993), the health status of white South African gold miners who had worked at least 10 years between 1940 and the early 1970s was followed through 1991. Of these miners, 14% were silicotic, and of these silicotics, 57% displayed radiological signs of silicosis an average of 7.4 yr after leaving the mines (Hnizdo & Sluis-Cremer, 1993). The reported silicosis rates were considerably higher than in previous studies, which the authors attributed to the longer follow-up period. It was also suggested at the time, however, that the average dust exposures (which had been based on samples collected from 20 gold mines in the 1960s) might have been underestimated (Gibbs & Du Toit, 2002), or that South African quartz produced more toxicity than Canadian quartz. Based on these data, the cumulative risk of silicosis incidence at 9 mg/m³-yr (roughly equivalent to 28 yr at the current PEL) of respirable dust was estimated to be 25%.

The South African study was among the first to question the protectiveness of the OSHA PEL and to indicate that exposures at 0.1 mg/m³ for a 40- or 45-yr working lifetime might not protect against silicosis (Figure 12) (Hnizdo & Sluis-Cremer, 1993). Since then, a number of other epidemiological studies reported varying estimates of silicosis risk associated with a lifetime of work at the current occupational standard (Table 4) (Rosenman et al., 1996; Steenland & Brown, 1995b; Kreiss & Zhen, 1996; Chen...
FIGURE 12. Timeline of key studies and events that led to the recognition of the hazards of silica (1990–2008).
et al., 2001; ASA, 1959; Churchyard et al., 2004; Park et al., 2002; Checkoway et al., 1997; Mannetje et al., 2002a, 2005; Mannetje & Pearce, 2005). Risk estimates reported in some of the larger studies have ranged from 1 to 80%, but generally were consistent in that the estimated risk of silicosis incidence exceeded OSHA’s acceptable risk level of 1 in 1,000. Few studies provide an estimate of a “safe” exposure level, although in the Rice and Stayner (1995) study, data from 6 epidemiological studies were reviewed, and the NOAEL was estimated to be between 7 and 100 μg/m³. The authors noted that such a wide range indicates a high degree of uncertainty and demonstrates the difficulty in drawing conclusions about safe exposure levels. Possible reasons for the differences in risk include the large variation in follow-up time, lack of information regarding peak exposures, or differences in exposure patterns (Finkelstein, 2000; NIOSH, 2002a). It has also been suggested that the dose-response curve for silicosis is nonlinear, with risk increasing more rapidly at higher exposure levels (Hnizdo & Sluis-Cremer, 1993).

To address the variability among the studies, IARC conducted a pooled exposure-response analysis of six occupational cohorts (Mannetje et al., 2002a, 2002b). Original exposure data were obtained from the principal investigators, converted to a common exposure metric (mg/m³), and organized into job-exposure matrices (Mannetje et al., 2002b). The analysis focused on mortality from

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<td>Mining</td>
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<tr>
<td>Gold and uranium</td>
<td>Muir et al, 1989a</td>
<td>Incidence</td>
<td>0.4 (0.2-1.1)†</td>
</tr>
<tr>
<td>Gold</td>
<td>Hnizdo and Sluis-Cremer, 1993</td>
<td>Incidence</td>
<td>77% ‡</td>
</tr>
<tr>
<td>Gold</td>
<td>Steenland and Brown, 1995b</td>
<td>Incidence</td>
<td>35–47% ‡</td>
</tr>
<tr>
<td>Hardrock</td>
<td>Kreiss and Zhen, 1996</td>
<td>Incidence</td>
<td>68–80%</td>
</tr>
<tr>
<td>Tin</td>
<td>Chen et al, 2001</td>
<td>Incidence</td>
<td>55%</td>
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| Gold              | Churchyard et al, 2004         | Incidence        | Average prevalence, 19.1%; long term workers, 32%\
| Tin               | Chen et al, 2001               | Incidence        | 55%                                           |
| Tungsten          | Chen et al, 2005               | Incidence        | 7%                                             |
| **Diatomite**     | Checkoway et al, 1997          | Mortality        | RR 3.17 (1.25-8.05)‡                           |
| Hughes et al, 1998| Incidence                      | 1.1% ‡           |
| Park et al, 2002  | Mortality                      | 100-140/1000     |
| **Foundry**       | Rosenman et al, 1996           | Incidence        | OR 2.1 (1.56–2.82)‡                           |
| **Pottery workers**| Chen et al, 2005               | Incidence        | 13% ‡                                         |
| **Dusty trades**  | Rice et al, 1986               | Incidence        | Significant risk‡ at 2.5 mppcf ‡               |
| **Pooled analyses**| t Mannetje et al, 2002a        | Mortality        | 13/1000                                        |

*Table 4. Summary of Exposure-Response Studies and Lifetime Estimates of Silicosis Risk for Silica Exposures at the OSHA PEL (0.1 mg/m³)*

†45 year work history unless otherwise noted.
‡Reported in Chen 2001.
§40 year work history.
□4–55 mg/m³ · yr cumulative respirable silica dust (assuming 30% quartz content at a respirable dust concentration of 0.4 mg/m³ for 37 yr).
*calculated using 0.09 mg/m³ · yr PEL.
**87–93% samples <0.1 mg/m³.
††cumulative exposure (2.1–5 mg/m³ · yr); risk of non-malignant respiratory disease.
‡‡2 mg/m³ · yr crystalline silica exposure; risk of opacities.
\*no risk calculation.
\*Pooled analysis included: Checkoway et al. 1997, Koskela et al. 1994, Costello et al. 1988, Steenland and Sanderson 2001, Steenland and Brown 1995a, DoKlerk et al. 1998. These studies were designed to detect lung cancer exposure-response relationships; the data was pooled and re-analyzed to measure silicosis risk.
†Based on Chinese Method for total dust of 4.6 mg/m³ for 45 yr.
‡Based on Chinese Method for total dust of 2.0 mg/m³ for 45 yr.
silicosis, which the authors noted was seldom studied because of the relatively small number of deaths where silicosis was reported on the death certificate as the underlying cause. Quantitative exposure data were derived from studies of U.S. diatomaceous earth workers, Finnish granite workers, U.S. (Vermont) granite workers, U.S. industrial sand workers, U.S. gold mine workers, and Australian gold miners (Checkoway et al., 1997; Koskela et al., 1994; Costello & Graham, 1988; Steenland & Sanderson, 2001; Steenland & Brown, 1995a; de Klerk & Musk, 1998). Together, the pooled cohort contained 18,364 workers, of whom 170 died from silicosis or an unspecified pneumoconiosis. The silicosis mortality rate for the entire cohort was 28.8 per 100,000 person years, while the estimated cumulative risk of death from silicosis was 13/1000 for exposure to 0.1 mg/m³ from age 20 to 65. Although the authors did not provide an estimate of an occupational exposure limit that would result in less than a 1-in-1000 risk of silicosis mortality, it was emphasized that the current OSHA PEL of 0.1 mg/m³ was not sufficiently protective (t Mannetje et al., 2002a).

**Silica, Silicosis, and Lung Cancer** In the two decades following the IARC 1986 designation of silica as a 2A probable carcinogen, numerous epidemiological studies have been conducted to address the relationship between silica exposure, silicosis, and lung cancer (Figure 12). Similar to the exposure-response studies, there was considerable potential for exposure misclassification and other possible biases (Hessel et al., 2000). The extent to which these factors played a role has varied by industry. Some of the earlier studies during this era were conducted in the pottery and stone workers, but many of the positive associations were attributed to other exposures (e.g., cigarette smoke, PAHs), which confounded the exposure response patterns of silica (Costello and Graham, 1988; McDonald, 1995; McLaughlin et al., 1992). Studies in the pottery and ceramic industries at the time showed conflicting results, which were explained, in part, by exposures to other substances (Meijers et al., 1990; Thomas, 1982, 1990), as well as the selection of the referent populations used to calculate mortality ratios. Although the presence of other carcinogens was less of an issue in the granite industry, there were mixed findings of increased lung cancer mortality among granite workers compared to the general population (Davis et al., 1983; Steenland & Beaumont, 1986; Costello & Graham, 1988; Koskela et al., 1987a, 1987b). A more recent follow-up study of the Vermont granite worker cohort found increased lung cancer mortality among silica-exposed workers (SMR=1.17, p < .05), although a dose-response trend was not seen across all the exposure groups (Attfield & Costello, 2004). Excess lung cancer risk was documented among diatomaceous earth and refractory brick workers, who were exposed primarily to cristobalite (Checkoway et al., 1993; Rice et al., 2001).

Increased lung cancer risk has been reported in studies throughout the 1990s and 2000s. In many cases, however, these associations were not statistically significant, or significant findings were limited to workers with silicosis (Figure 13). The issue of whether silicosis is a necessary step in the development of lung cancer has been a controversial issue for many years (Checkoway & Franzblau, 2000). Beginning in the mid-1990s, several meta-analyses were conducted using studies of silicotics. In these studies, the estimates of relative risk for lung cancer mortality ranged from 1.3 (95% CI 1.2–1.4) to 2.2 (95% CI 2.1–2.4) (Smith et al., 1995; Steenland & Stayner, 1997). While the individual studies included in each meta-analysis varied, many of the original studies contained incomplete (or nonexistent) smoking data.

In 1997, IARC reevaluated the carcinogenicity of crystalline silica (IARC Working Group, 1997) relying upon the “least confounded” epidemiological studies, which included those of the South Dakota gold miners (McDonald et al., 1978; Brown et al., 1986; Steenland & Brown, 1995a), Danish stone-industry workers (Guenel et al., 1989), Vermont granite-shed and quarry workers (Costello & Graham, 1988), U.S. crushed-stone-industry workers (Costello et al., 1995), U.S. diatomaceous-earth workers (Checkoway et al., 1993, 1996), Chinese and Italian refractory-brick workers (Dong et al., 1995; Merlo, 1991), and Chinese and United Kingdom pottery workers (McDonald et al., 1995; Chen et al., 1992; McLaughlin et al., 1992) (Table 5). Cohorts of registered silicotics in North Carolina and Finland were also considered (Amandus et al., 1991; Partanen et al., 1994). IARC concluded in its assessment that “the epidemiological findings support increased lung cancer risks from inhaled crystalline silica (quartz and cristobalite) resulting from occupational exposure” that could not be explained by confounding or other biases. IARC also noted, however, that “carcinogenicity in humans was not detected in all industrial circumstances studied” (IARC Working
Although IARC designated crystalline silica as a Group 1 carcinogen (“sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite”), this decision provoked controversy within the scientific community (Hessel et al., 2000; Soutar et al., 2000; Pelucchi et al., 2006; Checkoway & Franzblau, 2000; Wong, 2002; Vallyathan et al., 1998). Some of the primary arguments against the IARC designation were inconsistencies amongst the studies and weakly significant findings. The lack of smoking information in many of the studies and the unreliability of older exposure data were also noted. Hessel et al. (2000) published a response to the IARC working group, in which an alternative analysis was conducted (Table 5). The authors included nearly 20 of the studies upon which IARC relied, as well as some that had been published since the 1997 evaluation. Studies of compensated silicotics were excluded (Hessel et al., 2000). The analysis did not find an association between lung cancer and exposure to silica dust.

In 2002, NIOSH published a health hazard review for silica, in which the lung cancer studies were considered (NIOSH, 2002a). The review focused on the same industries as the 1997 IARC

<table>
<thead>
<tr>
<th>Year</th>
<th>Risk Estimate</th>
<th>Study</th>
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<tr>
<td>1988</td>
<td></td>
<td>Costello &amp; Graham, 1988 - Quarry workers</td>
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<td>Costello &amp; Graham, 1988 - Total shed workers</td>
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<td>Costello &amp; Graham, 1988 - Shed workers (started before 1940)</td>
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<td>Costello &amp; Graham, 1988 - Shed workers (started after 1940)</td>
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<td>1989</td>
<td></td>
<td>Guenel et al, 1989 - Lung cancer cases in the stone industry</td>
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<td>1992</td>
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<td>Amandus et al, 1992 - Silicotics</td>
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<td></td>
<td>Amandus et al, 1992 - Nonsilicotics</td>
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<td>McLoughlin et al, 1992 - Low (0.1-8.69 μg/m³-yr) silica exposure</td>
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<td>McLoughlin et al, 1992 - Med (8.7-26.2 μg/m³-yr) silica exposure</td>
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<td>McLoughlin et al, 1992 - High (≥ 26.3 μg/m³-yr) silica exposure</td>
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<td>1993</td>
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<td>Checkoway et al, 1993 - Diatomite workers</td>
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<td>Partanen et al, 1994 - &lt;2yr follow-up since silicosis diagnosis</td>
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<td>Partanen et al, 1994 - &gt;2, &lt;10yr follow-up since silicosis diagnosis</td>
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<td>Partanen et al, 1994 - &gt;10yr follow-up since silicosis diagnosis</td>
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<td>1995</td>
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<td>Cherry et al, 1995 - Pottery workers</td>
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<td>Costello et al, 1995 - Granite workers</td>
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<td>Dong et al, 1995 - Silicotics</td>
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<td>McDonald, 1995 - Lung cancer cases</td>
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<td></td>
<td></td>
<td>Steenland &amp; Brown, 1995 - Gold miners</td>
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<tr>
<td>1996</td>
<td></td>
<td>Checkoway et al, 1996 - White diatomite workers</td>
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<td>Starzynski et al, 1996 - Coal miners</td>
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<td>Starzynski et al, 1996 - Foundries</td>
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<td>Starzynski et al, 1996 - Quarries</td>
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<td></td>
<td>Starzynski et al, 1996 - Underground workers</td>
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<td>Checkoway et al, 1996 - Nonwhite</td>
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<td>Checkoway et al, 1996 - White</td>
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<td>DeStefani et al, 1996 - Silica exposure</td>
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<td>1997</td>
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<td>Burgess et al, 1997 - Cumulative exposure ≥ 4000 μg/m³-yr</td>
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<td>Burgess et al, 1997 - Duration of employment ≥ 20yr</td>
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<td>Burgess et al, 1997 - Mean silica exposure intensity ≥ 200 μg/m³</td>
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<td>Burgess et al, 1997 - Max silica exposure ≥ 400 μg/m³</td>
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<td>Brown et al, 1997 - Denmark</td>
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<td>Brown et al, 1997 - Sweden</td>
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<td>Checkoway et al, 1997 - Lung cancer cases in diatomite workers</td>
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<td>Chiaze et al, 1997 - White male fiberglass workers</td>
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<td>Rafnsson et al, 1997 - Lung cancer incidence</td>
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<td>Hnizdo et al, 1997 - Gold miners</td>
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assessment, with the exception of studies involving silicotics found in local or national registries or those that were considered to be confounded due to inadequate exposure assessment, selection and/or confounding bias, inadequate controlling for smoking or exposure to other carcinogens, lack of evidence of an exposure-response relationship, or the inability to distinguish differences in the fibrogenic and carcinogenic potencies of the various silica polymorphs (Table 5). NIOSH also discussed several studies that had been published subsequent to the IARC assessment and noted that, overall, their review supported IARC’s conclusion that there was an association between lung cancer risk and silicosis. A review of the literature by ACGIH (2006) noted that, while a number of U.S. and international agencies agreed that a positive association exists between silica exposures and
<table>
<thead>
<tr>
<th>Cohorts</th>
<th>IARC 1997</th>
<th>Hessel, 2000</th>
<th>NIOSH 2002</th>
<th>2002 IARC*</th>
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<td>U.S. gold</td>
<td>Steenland and Brown, 1995a</td>
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<td>Hnizdo and Sluis-Cremer, 1991</td>
<td>Reid and Sluis-Cremer, 1996</td>
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<td>Australian gold</td>
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<td>de Klerk and Musk, 1998</td>
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<td>Finnish granite</td>
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<td>Steenland et al., 2001</td>
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<td>German stone, quarrying, ceramics industry</td>
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<td>Steenland et al., 2001</td>
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<td>U.S. industrial sand</td>
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<td><strong>Refractory brick workers</strong></td>
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<td>Puntoni et al, 1988</td>
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<td><strong>Pottery workers</strong></td>
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<td>Winter et al, 1990</td>
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<td>Kaperna et al, 1986</td>
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<tr>
<td><strong>Result/Conclusion</strong></td>
<td>Overall, “carcinogenic to humans,” support for carcinogenicity of silica varies by industry</td>
<td>Lack of causal relationship between silica and lung cancer</td>
<td>“NIOSH concurs with the conclusions of the IARC (1997) and the ATS”</td>
<td>Estimated excess lifetime risk was 1.7% (95% CI 0.2% – 3.6%)</td>
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</table>

*Steenland et al., 2001.
To address some of the issues posed by the scientific community, Steenland et al. (2001) conducted a pooled analysis using the cohorts that were developed for the exposure-response studies described previously (Steenland et al., 2001). This pooled analysis included 65,000 workers in 10 cohorts and over 1000 lung cancer deaths. Pooling the number of lung cancer deaths across multiple occupational cohorts allowed researchers to increase the statistical confidence in their comparisons on other studies that individually may not have had the statistical power to detect a statistical difference. Data from foundries were excluded to prevent confounding from co-carcinogens, such as PAHs. Results from studies of coal miners were also excluded because of the relatively low silica content of the dust. Smoking information was available for 4 of the 10 cohorts. In this analysis, odds ratios for lung cancer increased by quintiles of cumulative exposure, and similar results were observed when average exposure was considered. The Steenland study reported that the log of cumulative exposure, with a 15-yr lag, was predictive of lung cancer ($p = .0001$), with consistency across studies (test for heterogeneity, $p = .34$). They reported that the results were consistent between underground mines and other facilities (Steenland et al., 2001).

The role of silicosis in the development of lung cancer remains an unresolved matter today. In a review published by Checkoway and Franzblau (2000), 10 studies that presented lung cancer risk estimates separately for silicotics and nonsilicotics were assembled and analyzed (Checkoway & Franzblau, 2000). While the authors noted that epidemiologic and experimental evidence supports the IARC 1997 classification of crystalline silica as a human lung carcinogen, they also concluded that there remains uncertainty about whether excessive lung cancer occurs exclusively among workers with silicosis. A more recent review of studies conducted between 1996 and 2005 (excluding those previously reviewed in the IARC monograph) used random effects models to estimate pooled relative risks (RRs) for lung cancer separately among studies of silicotics, nonsilicotics, and those in which the status of silicosis was unknown (Pelucchi et al., 2006). In total, 45 studies were considered. The pooled RRs for the cohort studies ($n = 28$) were 1.69 (95% CI 1.32–2.16) for silicotics, 1.25 (95% CI 1.18–1.33) when silicosis status was unknown, and 1.19 (95% CI 0.87–1.57) for nonsilicotics. For case-control studies ($n = 15$), pooled RRs were 3.27 (95% CI 1.32–8.2) for silicotics, 1.41 (95% CI 1.18–1.7) when silicosis status was unknown, and 0.97 (95% CI 0.68–1.38) for nonsilicotics. The pooled RR for the two PMR studies considered was 1.24 (95% CI 1.05–1.47).

Based on the current literature, it is uncertain whether the increased risk of lung cancer among silicotics is actually due to the fibrotic nature of the lung or if silicosis is merely a marker for high silica exposures. Only a handful of studies specifically addressed lung cancer risk in the absence of silicosis; these have shown conflicting results (Ulm et al., 1999; Checkoway & Franzblau, 2000). The quality of data regarding smoking, which is known to produce pulmonary fibrosis and lung cancer, must also be considered when evaluating the literature related to this topic. Estimates of an association between silica exposure and lung cancer are weaker than many, if not most, other carcinogens (Steenland et al., 2001). Taken together, the true relationship between silica exposure, silicosis, and lung cancer may never be entirely clear.

**Relationship Between Silica Exposure and Nonpulmonary Disease**

Over the last 10–15 yr, a number of studies addressing the potential association between silica exposure and autoimmune and kidney diseases have appeared in the scientific literature. While case reports in silica-exposed workers were published in the early 1900s, it was not until the mid 1980s that epidemiological studies were conducted to evaluate the relationship between silica and autoimmune diseases such as scleroderma, rheumatoid arthritis (RA), and systemic lupus erythematosus (SLE) (Bramwell, 1914; Calvert et al., 2003; Rosenman et al., 1999; Sluis-Cremer et al., 1985; Rafnsson et al., 1998; Burns et al., 1996). It has been difficult to characterize the risk of autoimmune disease among workers exposed to silica because environmental and genetic factors (e.g., gender, family history) can greatly influence the development and progression of these illnesses, yet they cannot be readily controlled for in occupational studies (Arnett, 2000; Silman & Newman, 1996; Steen, 1999). In addition, women are more susceptible to many autoimmune diseases, but occupational cohorts are predominantly male;
thus, the relative rarity of these diseases among men makes it difficult to achieve the statistical power necessary for epidemiology studies (Jacobson et al., 1997; Parks et al., 1999).

Scleroderma has been the most commonly studied autoimmune disorder in occupational studies involving silica. Scleroderma, which literally means “hardening of the skin,” is a disorder of the connective tissue characterized by increased collagen production (Industrial Disease Standards Panel, 1992). Prior to 1990, the majority of the literature that connected scleroderma to silica exposure included case reports, prevalence surveys, and ecological studies with few case-control studies (Rodnan et al., 1967; Sluis-Cremer et al., 1986; Steen, 1999). More recent studies have evaluated scleroderma mortality rates among workers exposed to silica. Excess rates were reported in some, but not all, of the cohorts (NIOSH, 2002a). The use of mortality data to study scleroderma in occupational settings has been problematic, because in general, scleroderma is not a fatal disease. These studies also failed to account for family history or possible confounding exposures. While the causes of scleroderma are unknown, a family history of scleroderma and other autoimmune disease, and gender (scleroderma is 3–8 times more common in women than in men) are considered the strongest risk factors (Industrial Disease Standards Panel, 1992; Jacobson et al., 1997; Arnett, 2000). Nonetheless, the American Thoracic Society (ATS) has recently concluded that the evidence relating silica exposure to scleroderma is persuasive where there is an appreciable silicosis risk; although NIOSH did not find sufficient evidence to reach the same conclusion, it referenced the literature that has found statistically significant numbers of excess deaths (American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health, 1997; NIOSH, 2002a).

Similar issues must be considered when evaluating the relationship between silica exposure and the development of rheumatoid arthritis (RA). RA, which is characterized by painful joint inflammation, also has a strong genetic component and has been associated with a number of environmental and lifestyle risk factors (e.g., female hormonal changes, cigarette smoking, diet, and infections) (Oliver & Silman, 2006; Klockars et al., 1987; Caplan, 1953). In recent epidemiological studies, positive associations between silica exposure and RA have been reported (Calvert et al., 2003; Rosenman & Zhu, 1995; Rosenman et al., 1999; Sluis-Cremer et al., 1986). However, these studies either did not adjust for confounders (e.g., family history, cigarette smoking) or did not report a concurrent association with other autoimmune diseases such as scleroderma, systemic lupus erythematosus, or sarcoidosis. In 2000, Turner and Cherry reported that cases of RA among pottery, sandstone, and refractory material workers had silica exposures of significantly shorter duration compared to the referents (Turner & Cherry, 2000). An ATS review committee indicated that the association between RA and silica exposure or silicosis is not clear, particularly when RA, silicosis, and most other pulmonary fibrotic diseases may not produce positive serum tests for antinuclear antibodies and rheumatoid factor (American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health, 1997).

A possible association between silica exposure and systemic lupus erythematosus (SLE) has also been investigated. SLE is an autoimmune disease that affects multiple organ systems and is characterized by immunological abnormalities, such as hyperactive B cells. SLE is generally believed to develop from the combination of a genetic predisposition and an environmental trigger. Family members of autoimmune disease patients are at a greater risk of developing disease, and endogenous hormone levels have been shown to be important in disease development. Environmental triggers that have been identified include numerous pharmaceuticals containing aromatic amines; other suspected triggers include sex hormones, traumatic life events, cigarette smoking, and hair dyes (Bengtsson et al., 2002). Much of the literature related to silica exposure and SLE consists of case reports (Koeger et al., 1995; Costallat et al., 2002; Bolton et al., 1981). More recently, occupational cohort studies have suggested an association between SLE and high occupational exposures to silica, although the results have not always been statistically significant (Calvert et al., 2003; Sanchez-Roman et al., 1993; 5A number of chemicals present in occupational settings, including vinyl chloride, organic solvents such as trichloroethylene, and epoxy resins, have been investigated for a possible association with scleroderma incidence. While a convincing association has not been shown (Steen, 1999), scleroderma has been a compensable occupational disease in South Africa, the former German Democratic Republic, and Ontario, Canada (Industrial Disease Standards Panel, 1992). The majority of claims have been related to silica.
Conrad et al., 1996). NIOSH has concluded that more research is necessary to characterize the relationship between silica exposure and SLE; likewise, the ATS has concluded that a causal association should be suspected only in cases of acute or accelerated silicosis (American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health, 1997; NIOSH, 2002a).

While recent studies have postulated an association between occupational exposure to silica and autoimmune disease, the complex nature of these disorders and the inability to control for important risk factors in epidemiology studies preclude drawing any firm conclusions. In vitro studies have demonstrated that exposing cell cultures to silica can exert an adjuvant effect on antibody production, which could lead to a chronic process of immune stimulation (Parks, 1999). Several mechanisms by which silica could generate an autoimmune response through this pathway have been proposed, but robust animal and experimental studies are lacking. Markers of autoimmune dysfunction have also been documented in abrasive blasters with silicosis, including increased levels of antinuclear autoantibodies, rheumatoid factor, immunoglobins, and immune complexes, although serum levels did not correlate with the severity or progression of lung disease (Doll et al., 1981). Overall, the body of evidence remains unclear, and both NIOSH and ATS have concluded that further research is required before any definitive conclusions can be drawn regarding the association between silica exposure and autoimmune disease (American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health, 1997; NIOSH, 2002a).

In addition, an increased incidence of renal disease has been suggested among silica-exposed workers in a number of published case reports over the last 25 yr (Giles et al., 1978; Haughlustaine et al., 1980; Osorio et al., 1987). However, as was the case with autoimmune disease, it was not until the 1990s that epidemiological studies on this health endpoint were conducted. Small sample sizes and very low incidence have made it difficult to detect associations. There has also been considerable variation in the renal health endpoints included in occupational studies, which has made comparison difficult. Endpoints have ranged from biomarkers of renal function (proteinuria) to specific kidney diseases (glomerulonephritis, hypertensive kidney disease, interstitial kidney disease, Wegener granulomatosis, SLE nephritis, and kidney cancer) to generalized kidney disease (end-stage renal disease, renal disease, and kidney failure) to the physical accumulation of silica in the kidney (Steenland, 2005; Ng et al., 1993; El-Safty et al., 2003a, 2003b; Ng et al., 1992; Boujemaa et al., 1994; Hotz et al., 1995; Nuyts et al., 1995; Steenland et al., 1990; Rapiti et al., 1999; Hogan et al., 2001; Chiazze et al., 1999; Calvert et al., 2003; Attfield & Costello, 2004). The most compelling study, Steenland (2005), found an excess risk of end-stage renal disease of 1.8% (0.8% to 9.7%) in a pooled analysis of 3 cohorts. In their review of the health effects of crystalline silica, NIOSH noted that multiple studies have reported statistically significant relationships between silica exposure and renal disease (NIOSH, 2002a).

Experimental Animal Studies  With a clear understanding of the histopathological and clinical aspects of silicosis, researchers in the 1990s focused their efforts on characterizing the mechanisms by which silicosis occurs and its apparent role in the development of lung cancer. Many of these studies have evaluated how particle surface chemistry, intercellular signaling pathways, and oxidant stress may induce inflammation and stimulate the immune system, ultimately leading to cell proliferation and tissue fibrosis following exposure to crystalline silica (Blackford et al., 1997; Huffman et al., 1998; Kim et al., 1999; Kang et al., 2000; Zeidler et al., 2004; Hamilton et al., 2008). Some of these studies have led to investigation for potential biomarkers of exposure, effect, and susceptibility for silicosis (Gulumian et al., 2006). Overall, the intercellular interactions and cell signaling pathways are complex, numerous cross-talk and feedback mechanisms exist, and species responses and forms of silica differ. Although the use of biomarkers for human populations exposed to silica are far from being validated or feasible, the studies have revealed some of the underlying mechanisms involved in the development of silica-induced lung injury.

The mechanisms underlying silica-induced carcinogenesis in animals, and perhaps in humans, are not well understood. IARC described the experimental evidence for a direct genotoxic action of crystalline silica as “weak” (IARC Working Group, 1997). However, many of the pathways that are believed to be involved in the inflammatory and fibrogenic processes may also be important in the carcinogenic process. For example, the development of lung tumors in the rat appears to be dependent on coexistent chronic inflammation and cell proliferation (ILSI, 2000). ROS produced as a
result of silica exposure can interact with DNA in vitro to form potentially promutagenic lesions (Daniel et al., 1993; Saffiotti et al., 1994; Shi et al., 1994, 1998; Albrecht et al., 2005), and increased cell proliferation can lead to the development of cancer by increasing the likelihood that such lesions will become fixed before they can be repaired by the cell. It has been suggested that the formation of lung tumors in rats exposed to silica appears to be consistent with a nonspecific response to persistent inflammation and increased cell proliferation, and that it may therefore be appropriate to apply a threshold model of carcinogenesis to inhaled silica if silicosis is required for the development of silica-related lung cancer (Mossman et al., 1995).

Occupational Exposure Limits for Silica

The TLV for quartz remained at 0.1 mg/m³ throughout the 1990s. In 1999, ACGIH reevaluated the TLV for quartz and, in 2000, lowered the TLV to 0.05 mg/m³ based on studies suggesting that the risk of silicosis associated with exposure to 0.1 mg/m³ over a working lifetime was well above of OSHA's acceptable risk level of 1 in 1,000 (Table 2) (Muir et al., 1989a; Graham et al., 1991; Hnizdo & Sluis-Cremer, 1993; Steenland & Brown, 1995a; Kreiss & Zhen, 1996; ACGIH, 2000). ACGIH also cited the study by Hnizdo et al. (1993), in which autopsies were conducted to validate previous diagnoses of silicosis based on x-rays, and it was estimated that over half of silicotics would not have been diagnosed as positive using x-ray analysis alone (Hnizdo et al., 1993). ACGIH also designated quartz as a suspected human carcinogen (A2) and noted that, while it did not appear that silica was a direct-acting initiator, there was “compelling evidence that many forms of pulmonary fibrosis constitute major risks for lung cancer” (ACGIH, 2000). Thus, lowering the TLV to a level that was protective against silicosis was considered sufficient to prevent the development of lung cancer. However, ACGIH also acknowledged that cancer studies in rats were lacking in strength, due to the recognition that rats are a poor predictor of human response to the effects of dust (ACGIH, 2000). A significant departure from previous silica TLVs occurred in 2006, when the TLVs for quartz and cristobalite were combined, and lowered to 0.025 mg/m³ (ACGIH, 2006). The primary basis for the combined TLV for quartz and cristobalite was that human studies in the diatomaceous earth industry in the late 1990s/early 2000s indicated that the exposure-response risks for radiologically diagnosed silicosis in this cohort were similar to those for quartz, which was a departure from historical animal studies that showed differential toxicity for the two crystalline forms of silica (ACGIH, 1962, 2006; Hughes et al., 1998; Park et al., 2002).

The current OSHA PEL of 10/(% quartz + 2) mg/m³ for respirable dusts and 30/(% quartz + 2) mg/m³ for total dusts (Table 2) remains unchanged since 1971; however, OSHA has been working toward a revision. A major concern has been the issue of noncompliance with the current standard, and as such, a new standard would have an economic impact on those already in compliance. To address this situation, it has been suggested that OSHA consider improved enforcement and outreach for the existing rule. A timetable for OSHA’s final decision to move forward with rulemaking has not been issued. In addition, MSHA has also given an advanced Notice of Proposed Rulemaking on crystalline silica. Designation of a new occupational health standard for respirable crystalline silica has been listed as a long-term action as recently as April 2006.

Abrasive Blasting Studies and Industry Developments

Because of the very high exposures that can occur during abrasive blasting, NIOSH has continued to evaluate these operations in industries (e.g., shipyards, foundries) long recognized as having excessive

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11The current OSHA PEL is commonly cited as 0.1 mg/m³, which assumes that the percentage of quartz is equal to 100%.
12Under the Small Business Regulatory Enforcement Fairness Act of 1996 (SBREFA), one of the first steps that OSHA is required to take when revising a PEL is to issue a proposal (element of a draft rule) to a panel to demonstrate how a rulemaking might affect small business (U.S. Small Business Administration, 1996). In October 2003, the submitted draft evaluated three PELs under consideration by the Agency: 50, 75, and 100 μg/m³, measured as an 8-hr time-weighted average (TWA) (U.S. Small Business Administration, 2003). In December 2003, OSHA received comments from small-entity representatives (SERs), and the consensus of the majority of SERs was that a new silica rule was not needed.
13According to the Unified Regulatory Agenda, the Peer Review of Health Effects and Risk Assessment for silica, which is also required as a part of the rulemaking, has an undetermined completion date (OSHA, 2007).
silica exposures and a high incidence of silicosis. The development of surveillance programs, such as NIOSH’s Sentinel Event Notification System for Occupational Risks (SENSOR) and OSHA’s Special Emphasis Program (SEP), during this time period have also been important in identifying and investigating high risk industries (NIOSH, 2006d; Galster, 1997). Much of the silicosis and silica exposure experience among abrasive blasters in the 1990s through today were identified and reported through these types of programs (Centers for Disease Control [CDC], 1990, 1998, 2004; Sevinc et al., 2003; Galster, 1997; O’Brien et al., 1990; Irwin, 2003; Jennison & Cocalis, 1995; Short, 1993; Yassin et al., 2005). For silicosis cases, the majority of workers with symptoms and diagnoses reported not using recommended respiratory protection, and workplaces were found to have insufficient engineering controls.

During the 1990s, OSHA remained active in training and educating abrasive blasters and other workers exposed to high levels of silica. In 1996, OSHA started a SEP focused on silicosis, which provided employers and state officials with educational materials about the risks of silica exposure. There was also an inspection component of the program. Of the 332 SEP inspections performed by OSHA, 30% of the air samples were found to be over the OSHA PEL (OSHA, 1996; Linch et al., 1998). This program included recommendations for medical monitoring. In addition, workers, unions, state health departments, and NIOSH all prepared public educational materials about the risks of silicosis and ways to prevent the disease. Most of these publications were aimed at construction and foundry workers, although at least one mock case study was prepared for high school students (NIOSH, 2002b).

**Silicosis Among Construction Workers** During the 1990s, construction workers were increasingly identified as an occupational group at high risk for silica-related disease. Many construction workers perform abrasive blasting as part of their work, and may also be exposed during other activities such as drilling, cutting, and grinding concrete. In a 1996 report titled “Preventing Silicosis and Deaths in Construction Workers,” NIOSH described a number of silicosis case reports among construction workers, including abrasive blasters, in which respiratory protection was either inadequate or completely absent (NIOSH, 1996a). This observation is consistent with reports in other exposure studies of the construction industry (Flanagan et al., 2003; Rappaport et al., 2003).

Flanagan et al. (2006) published a study of silica exposures at construction sites in which three task areas had higher quartz exposures from abrasive blasting. In all, samples from the majority of trade, task and tool categories exceeded the TLV of 0.05 mg/m³. By trade, abrasive blasters had the highest quartz exposure, although exposures classified by task and tool categories showed that silica concentrations during tuck-point grinding and surface grinding exceeded that observed with abrasive blasting (Flanagan et al., 2006). Both engineering and PPE controls were recommended for the workers with the highest exposures; however, it was noted that the nature of construction work makes implementing these controls difficult. The composition of the rock and sand used in cement varies widely, and the portable nature of the work limits the use of engineering controls.

In a recent study of the construction industry, researchers compared the airborne silica concentrations across four different construction trades (i.e., bricklayers, operating engineers, painters, and laborers). Painters, who used abrasive blasting in surface preparation, experienced the highest median silica exposures of 1.28 mg/m³ (Rappaport et al., 2003). The remaining groups of construction workers were shown to have median silica exposures in excess of the TLV at 0.32, 0.08, and 0.35 mg/m³ for bricklayers, operating engineers, and laborers, respectively. Of particular concern were exposures of bricklayers and laborers, who did not wear any respiratory protection in spite of the high exposure levels. Dust suppression was recommended as the most inexpensive way to reduce silica exposures for blasters and other construction workers until the construction industry investigates silica exposures at thousands of sites (Rappaport et al., 2003).

The use of alternative abrasives was often recommended as an additional control method to reduce worker exposures to airborne silica. Abrasive material other than silica sand was already commonly used in many industries, although few exposure and toxicity studies of the dust generated from these abrasive materials had been conducted in the United States. In 1998, NIOSH published a comprehensive study on the effectiveness, operating costs, and airborne concentrations associated with the use of a number of substitute abrasive materials (KTA-Tator, 1998a, 1998b, 1999). The study examined 13 different types of abrasives, including silica sand, and analyzed 30 different chemicals and metals in the airborne abrasive dust for potential health risks. In addition,
the study compared the operating costs of the different abrasive materials. While it was concluded that the alternative abrasives were comparable to silica sand from an economic standpoint and substantially reduced respirable quartz concentrations, it was also found that the alternative abrasives had higher levels of potentially toxic agents, including heavy metals. The study suggested that a broader “vertical health standard” encompassing all health hazards associated with abrasive blasting should be considered (KTA-Tator, 1998a, 1999).

**Respiratory Protection Guidelines for Abrasive Blasting**

Although respirators have never been considered the principle method for controlling exposures in abrasive blasting and the basis for respiratory protection recommendations during abrasive blasting have not changed significantly over the past 15 yr, respirator recommendations during abrasive blasting have become more prescriptive than in the past. For example, in 1992, ANSI again updated the American National Standard Practices for Respiratory Protection (Z88.2) to reflect the current state of the science on qualitative and quantitative fit testing, breathing-air supplies, respirator selection, and assigned protection factors (ANSI, 1992). Specifically, the respirator decision-making process was clarified, oxygen deficiency was redefined, and a requirement for the fit testing of positive-pressure respirators was added. The standard clearly states that respirators specifically approved for abrasive blasting should be used for these operations. Furthermore, the standard mentions that abrasive blasting in confined spaces may generate contaminant levels exceeding the protective capability of any respirator, and such situations require implementation of engineering controls.

In the 1980s, the language regarding recommended respiratory protection for abrasive blasting shifted from air-supplied respirators/hoods/helmets or abrasive blasting respirators to, specifically, Type CE respirators. The first official document to single out Type CE as the only respirator type suitable for abrasive blasting was ASTM 1986 standard, Health Requirements Relating to Occupational Exposure to Quartz Dust. Specifically, it stated that a Type CE supplied-air respirator, including a full face piece, hood, or helmet, operated with positive pressure should be used during abrasive blasting (ASTM, 1986). Later, NIOSH published a pamphlet that stated that abrasive blasting operations involving silica sand demand the highest level of protection available, defined as the Type CE abrasive blasting respirator. The pamphlet states clearly, “If you must sandblast, use type CE positive pressure abrasive blasting respirators” (NIOSH, 1997a). In 1995, NIOSH stated that they had not approved any Type AE or BE respirators, indicating that they had become obsolete (NIOSH, 1995). In the NIOSH Respiratory Protective Devices guidance, which was published in the Federal Register in 1995 (42 CFR 84), the definition of a Type CE respirator is as follows (NIOSH, 1995):

A type “C” supplied-air respirator equipped with additional devices designed to protect the wearer’s head and neck against impact and abrasion from rebounding abrasive material, and with shielding material such as plastic, glass, woven wire, sheet metal, or other suitable material to protect the windows of face pieces, hoods, and helmets which do not unduly interfere with the wearer’s vision and permit easy access to the external surface of such window(s) for cleaning.

NIOSH further clarified this definition in May 1996, when it published a Respirator User Notice to all abrasive blasters. This document clearly defined Type CE abrasive blasting respirators four ways: (1) continuous flow with loose-fitting hood and assigned protection factor (APF) of 25, (2) continuous flow with tight-fitting face piece and APF of 50, (3) positive-pressure respirator with tight-fitting half-mask face piece and APF of 1000, and (4) pressure demand or positive-pressure respirator containing a tight-fitting full face piece and APF of 2000 (NIOSH, 1996b).

The NIOSH User Notice further stated that “air purifying and powered-air purifying respirators are not recommended for abrasive blasting operations, but may be suitable for auxiliary work such as outside clean-up operations.” It also emphasized that silica sand should not be used as an abrasive medium. If silica sand is used, the notice stated that only NIOSH-certified pressure-demand or positive-pressure respirators with a NIOSH-recommended APF of 1000 or 2000 should be used (NIOSH, 1996b).

Similar to respiratory protection recommendations, the respiratory standards developed by OSHA have not changed significantly in recent years. OSHA’s maritime standard still states,
“Abrasive blasters working in enclosed spaces shall be protected by hoods and air line respirators, or by air helmets of a positive pressure type in accordance with the requirements of subpart I of this part” (OSHA, 2004). Subpart I refers to OSHA’s respiratory protection standard updated in 1998, stating that employers must select NIOSH-approved respirators after evaluating the hazard and determining the proper type of respirator to be worn as part of a required respiratory protection program (OSHA, 1998a). The use of NIOSH-approved respirators is also a requirement stated in OSHA’s ventilation standard, which addresses respiratory protection for abrasive blasting (OSHA, 1999). It should be once again noted that under OSHA, it is illegal to rely upon respiratory protection as the principle means of controlling exposure (OSHA, 1998b).

DISCUSSION

Crystalline silica has been one of the most widely studied chemicals in the history of occupational disease, and was among the first industrial chemicals for which exposure standards and work practice guidelines were developed. Our understanding of the health hazards associated with exposure to airborne silica continues to evolve to this day. The silica TLV has been updated several times over the last 5–10 yr and, in 2003, OSHA initiated the process of reevaluating the protectiveness of the PEL for crystalline silica. In all likelihood, the possible lung cancer hazard associated with silica will receive the most attention from scientists and regulators. IARC, ACGIH, ATS, and the World Health Organization (WHO) have designated crystalline silica a human carcinogen; however, many questions remain and continue to be heavily debated by the scientific community. These questions, particularly the exact nature of the relationship between silicosis and lung cancer, will be critical as OSHA considers the scientific basis of the current PEL.

By and large, there has been significant success in reducing silica exposures across the various industries, and the incidence of disease today is a fraction of that observed in prior decades. However, historical and current studies have consistently identified abrasive blasting as one of the more hazardous operations involving silica, and cases of silicosis among abrasive blasters continue to be reported today. Efforts to reduce exposure among this occupational group have included implementing of engineering controls, developing of OELs, and establishing guidelines for the use of respiratory protection. Most of the techniques that formed the basis for the dust-control measures (including respiratory protection) for abrasive blasting operations in the early 20th century are still used today, albeit with a number of improvements. Exposure studies in a variety of industries demonstrated that engineering controls were not always effective at reducing airborne silica concentrations to a level believed to be safe, and consequently, respiratory protection has always been (and continues to be) an important component for protecting abrasive blasters against the respiratory hazards of airborne crystalline silica.

Because abrasive blasting is commonly performed in many industries, the number of people potentially exposed to silica is relatively large. Silica sand is still used as an abrasive in the United States, and abrasive blasting of surfaces such as concrete can also produce airborne silica exposures even when nonsilica abrasives are used. As discussed in this review, exposure surveys conducted over the years have documented failures to maintain airborne concentrations of silica below the PEL, as well as a lack of appropriate respiratory protection programs within many industries. Abrasive blasting operations were often noted to be particularly problematic, and consequently, NIOSH and OSHA have continued to closely monitor abrasive blasters. Guidelines and requirements pertaining to respiratory protection have been updated in recent years to reemphasize the importance of a positive-pressure respirator during abrasive blasting operations. In addition, there has been considerable focus in recent years on the construction industry, where the portable nature of the work makes implementation of traditional engineering controls difficult. The importance of proper respiratory protection has been a consistent theme for abrasive blasters in this industry, and in fact, improper or a complete lack of respiratory protection in the workplace has been noted in many of the silicosis cases identified by NIOSH. As such, it should be recognized that, while the scientific community will undoubtedly continue to debate whether health risks exist at the current PEL or whether silica is a carcinogen, significant attention will undoubtedly be directed at educating employers and enforcing current occupational exposure and respiratory protection standards.
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HEALTH HAZARDS OF SILICA AND ABRASIVE BLASTING


