Settling the Dust
Silica Past, Present & Future
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Crystalline silica has been one of the most widely studied substances in the history of occupational disease and industrial hygiene. It is the most toxic form of silica, and estimates suggest that approximately 1.7 million workers are exposed to respirable crystalline silica in the U.S. Free silica is a term commonly used to describe quartz, a form of silica that is most prevalent in the environment and, therefore, in the workplace. Less common forms, or polymorphs, include cristobalite and tridymite, which differ from quartz only in structure (Madl, Donovan, Gaffney, et al., 2008; OSHA, 2013).

High exposures to crystalline silica can occur during construction activities such as abrasive blasting, jackhammering and tuck-pointing, as well as in other industries such as mining, foundry work, concrete product, paint and coating manufacturing (OSHA, 2013). In addition, NIOSH has found high exposures to respirable crystalline silica in hydraulic fracturing operations (OSHA & NIOSH, 2012). NIOSH (2005) estimates that more than 15,000 silicosis deaths occurred over the past 3 decades. Between 1995 and 2004, the number of U.S. deaths from silicosis decreased slightly, with NIOSH (2008) reporting approximately 150 to 250 deaths annually. However, the number of workers exposed to crystalline silica could rise with the increasing prevalence of hydraulic fracturing (fracking), in the U.S.

Despite the large number of workers exposed to crystalline silica, OSHA currently has no standard that requires employers to assess employees’ silica exposure, monitor potential health effects or provide necessary worker training. Instead, silica exposure is regulated solely through an OSHA permissible exposure limit (PEL) adopted more than 40 years ago (OSHA, 2003). The current PEL for general industry is dependent on the amount of respirable quartz in the collected sample and is calculated using a formula proposed by American Conference of Governmental Industrial Hygienists (ACGIH) in 1968 (OSHA, 2010):

$$PEL = \frac{10 \text{mg/m}^3}{\%SiO_2 + 2}$$

To assess respirable crystalline silica exposure, dust is collected using filter-based sampling in conjunction with a respirable cyclone. In the U.S., X-ray diffraction is the most popular analytical method to identify percentage of silica, although infrared (IR) spectroscopy is also commonly used (Madl, et al., 2008; Madsen, Rose & Cee, 1995; OSHA, 1999).

The current PEL formula for construction and shipyards (and an alternative PEL for general industry) for respirable crystalline silica requires outdated and obsolece particle counting technology (OSHA, 2013). According to OSHA (2013), the methodology has been “long rendered obsolete by gravimetric respirable mass sampling... Since the current construction and shipyard PELs are expressed only in terms of mppcf [million particles per cubic foot], the results of the gravimetric sampling must be converted to an equivalent mppcf value.”

Furthermore, OSHA (2010) states that workers remain at significant risk of developing silicosis at exposures below the current PELs. Several studies have reported that a high prevalence of silicosis has been observed even at the current PEL and argue that it is not sufficiently protective (Hinizdo & Slu...
is-Cremer, 1993; Kreiss & Zhen, 1996; Rosenman, Reilly, Rice, et al., 1996; Steenland & Brown, 1995). In contrast, other studies have reported that maintaining occupational exposures to crystalline silica have reduced the prevalence of silicosis to a low level (Graham, Ashikaga, Hemenway, et al., 1991; Graham, Vacek, Morgan, et al., 2001). In September 2013, OSHA proposed a rule for public comment that would lower the PEL for respirable crystalline silica as part of a comprehensive standard.

Silica in the 20th Century (1900 to 1989)

Human Health Studies

Lung diseases resulting from dust exposure have been recognized by health professionals since the 16th and 17th centuries. Dusty work environments were prevalent during the industrial revolution in the U.S. due to the increasingly widespread mechanization and the use of pneumatic tools in industry and mining operations (Lanza, 1938). These dust exposures were largely uncontrolled, as dust suppression measures were not often utilized and workers did not typically wear respiratory protection. As a result, disease and mortality rates resulting from silica exposures significantly increased during this period within the dusty trades, as compared to other occupations at this time (Air Hygiene Foundation of America, 1937).

Silicosis was not recognized as a distinct disease until the early 1900s. Some of the earliest accounts linking silica exposure to severe lung disease came from studies of miners in the U.K. and Australia, as well as published reports by the Miner’s Phthisis Prevention Committee (1916) and the South African Institute for Medical Research (Lanza, 1917). Although chest X-rays and other detection methods were being frequently used in the 1930s, early detection of silicosis was difficult.

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of silicosis. The study’s findings were ultimately used as the basis for the first occupational exposure limit (OEL) for silica. The granite studies confirmed that dose, duration and percent silica content of the inhaled dust were significant risk factors associated with the incidence of lung disease (Madl et al., 2008). In addition, other studies confirmed the prevalence of silicosis among foundry workers associated with welding, cleaning, finishing and abrasive blasting operations (McLaughlin, Goodman, Garrad, et al., 1950; Sander, 1938).

By the 1970s and with passage of the OSH Act, silica was identified as one of five key industrial exposure hazards in the U.S. Epidemiologic studies conducted during the 1970s and 1980s began to examine disease rates and latency with silica-exposed cohorts (Rice & Stayner, 1995; Theriault, Burgess, DiBerardinis, et al., 1974; Theriault, Peters & Johnson, 1974). According to these studies, the OSHA PEL was protective for silicosis.

Toxicology Studies

In the 1930s, animal studies involving silica exposure were conducted mainly at the Saranac Lake Laboratory, and results were shared during the Saranac Lake Symposia (Kuechle, 1934). These studies and others demonstrated that silica depressed the function of leukocytes and that silica was directly toxic to macrophages, the primary cells involved in immune responses in the lung, thus resulting in increased susceptibility to bacterial diseases such as tuberculosis (Gardner, 1938; Mavrogordato, 1929).

In the late 1930s, it was first reported that the extent of tissue damage was inversely proportional to particle size. Particles greater than 10 µm in diameter had no appreciable effect on lung cells (Gardner, 1938). The dose response relationship between silica exposure and lung disease, as well as duration of exposure, were confirmed via animal inhalation studies.

In 1953, King, Mohanty, Harrison, et al. (1953), found that tridymite produced the most severe and rapid response in rat lungs. These studies formed the basis for development of OELs for the various forms of silica. Numerous researchers also showed that silica particles ranging from 0.5 to 8.0 µm produced fibrosis in the rat lungs (Goldstein & Webster, 1966; King, et al., 1953).

The primary focus of animal studies conducted during the 1980s was the carcinogenic potential of crystalline silica; however, it was discovered that silica’s ability to cause lung tumors was limited to rats (IARC, 1996).

Regulatory Actions/Guidelines

The first OEL for silica was recommended by the U.S. Public Health Service based on studies of granite, gold and anthracite miners (Russell, et al., 1929). The recommendation in 1929 was for 9 to 20 million particles per cubic foot (mppcf), less than or equal to 10 µm (Madl, et al., 2008).

The recommended levels decreased as more research was conducted. By 1946, ACGIH had recommended an OEL that included the percent of free silica within the threshold limit value (TLV) calculation (ACGIH, 1946). By 1962, when the potencies of the various forms of silica were established, ACGIH’s previously formulated maximum allowable concentrations were changed to TLVs for crystalline and noncrystalline silica.

Workplace Controls

Beginning in the 1920s, engineering controls associated with abrasive blasting in the automotive industry were evaluated. Engineering controls such as enclosed units, positive-pressure air-supplied helmets, wetting methods, ventilation controls and good housekeeping afforded the best protection of worker exposure (Bloomfield & Greenberg, 1933; National Silicosis Conference, 1938; Winslow Greenburg & Reeves, 1920). In the 1950s and 1960s, several associations and other groups, including AIHA and ACGIH, published recommendations regarding respiratory protection use during abrasive blasting.

When several European countries banned silica in abrasive blasting in the 1950s, researchers began to investigate alternatives and their toxicity (Holmqvist & Swensson, 1963). Throughout the 1970s, NIOSH actively investigated abrasive blasting practices and the results highlighted the respiratory protection measures needed to adequately protect workers against silica exposures. These specifications included the use of a separate air supply (either as a supplied-air respirator or an air-supplied hood) (NIOSH, 1974).

Modern Years (1990 to 2013)

Current Health Hazard State-of-Knowledge

A major focus of health studies during the late 20th century and beginning of the 21st century was to further characterize the dose-response relationship between crystalline silica and silicosis. Improvements in exposure assessment and dose reconstruction methods, as well as follow-up with the established occupational cohorts, allowed scientific research to generate quantitative estimates of disease risk at different levels of silica exposure over a working lifetime (Madl, et al., 2008). These new studies suggested that the OSHA PEL for silica did not provide sufficient protection against the development of disease (Madl, et al., 2008). 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) at which illness would not be expected (Madl, et al., 2008; Rice & Stayner, 1995).

Human Health

After IARC designated silica as a group 2A carcinogen (probably carcinogenic to humans) in 1986, various epidemiological studies were conducted to address the relationship between exposure to silica and the development of silicosis and lung cancer (Madl, et al., 2008). These studies reported increased lung cancer risk; however, in many cases, these associations were not statistically significant.
or statistically significant findings were limited to those workers clinically diagnosed with silicosis (Madl, et al., 2008). In fact, the issue as to whether silicosis is a necessary step in the development of lung cancer through exposure to silica has been a controversial issue (Checkoway & Franzblau, 2000).

In 1996, IARC reevaluated the carcinogenicity of crystalline silica, with the committee relying on the least confounded epidemiological studies (those with the least bias arising from the co-occurrence or mixing of effects of extraneous factors). IARC (1996) concluded that “the epidemiological findings support increased lung cancer risk from inhaled crystalline silica (quartz and crystabolite) resulting from occupational exposure” that could not be explained by known confounders or any other biases. Therefore, IARC designated crystalline silica a group 1 carcinogen (carcinogenic to humans). However, IARC (1996) also noted that “carcinogenicity in humans was not detected in all industrial circumstances studied.”

In 2002, NIOSH published a health hazard review for silica. The review focused on the same epidemiologic cohorts as the 1996 IARC assessment, with some exclusions based on confounding exposures (NIOSH, 2002). Overall, the review supported IARC’s conclusion that an association exists between lung cancer risk and silicosis.

Toxicology

The mechanisms underlying silica-induced carcinogenesis in animals are still not fully understood. During the 1990s, researchers focused on characterizing the mechanisms by which silicosis occurs and its apparent role in lung cancer development (Madl, et al., 2008). As Madl, et al. (2008), report, many toxicological studies at the time focused on how particle surface chemistry, intercellular signaling pathways and oxidant stress may induce inflammation and stimulate the immune system leading to tissue fibrosis. Some studies also led to the search for potential biomarkers of exposure, effect and susceptibility for silicosis (Gulumi-an, Borm, Vallyathan, et al., 2006). The formation of lung tumors in rats exposed to silica may also be consistent with a nonspecific response to persistent inflammation and increased cell proliferation (Mossman, Jimenez, BeruBe, et al., 1995).

Regulatory Actions/Guidelines

The ACGIH TLV for quartz remained at 0.1 mg/m³ throughout the 1990s. In 2000, it was decreased 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 the established acceptable risk level of 1 in 1,000. In addition, ACGIH designated quartz as a group A2 carcinogen (suspected human carcinogen).

In 2006, ACGIH combined the TLVs for quartz and crystabolite, and set the combined TLV at 0.025 mg/m³. The basis for the change was epidemiologic studies in the diatomaceous earth industry (exposure to crystabolite); conducted during the late 1990s and early 2000s, these studies reported that the exposure-response risk for silicosis was similar to that for quartz (ACGIH, 2006).

OSHA needs to modernize its occupational exposure limits for silica for all industries. The current OSHA PEL, which is based on the 1968 ACGIH TLV of [10 mg/m³ (% quartz +2)] for respirable dust and [30 mg/m³ (% quartz +2)] for total dust has not changed since 1971. Furthermore, OSHA’s PEL for construction and shipyards (derived from the 1970 ACGIH TLV) is based on an obsolete particle counting technology. NIOSH and ACGIH recommend 50 µg/m³ and 25 µg/m³ exposure limits, respectively, for respirable crystalline silica (OSHA, 2010). Both industry and worker groups have recognized that a comprehensive OSHA standard for crystalline silica is needed to provide for exposure monitoring, medical surveillance and worker training (Iafolla, 2013).

Current Activities

In 2002, OSHA began developing a comprehensive standard for occupational exposure to crystalline silica in earnest. The agency completed the Small Business Regulatory Enforcement Fairness Act (SBREFA) report for the proposed rule in December 2003, and finalized the peer review of silica health effects and risk assessment in January 2010 (OSHA, 2010). The proposal then was stalled for more than 2 years in the White House Office of Management and Budget’s (OMB) Office of Information and Regulatory Affairs (OIRAJ) (Iafolla, 2013). OSHA finally published the proposed rule in September 2013, with public hearings to begin in March 2014 (as this issue of Professional Safety went to press).

In 2011, several organizations voiced concerns regarding the delays in promulgating the rule. For example, ASSE wrote a letter to an OIRA administrator in September 2011 encouraging that agency to complete its review of the proposed rule. In its letter, ASSE noted that the “silica issue first appeared
on OSHA’s Unified Agenda in 1997 [and] OSHA completed the SBREFA Report at the end of 2003” (ASSE, 2011). The group also stated that inadequate resources for standards development, potential future litigation and political motivations have contributed to OSHA’s difficulty in setting timely standards.

In addition, AIHA sent comments to the Secretary of Labor and the OMB director in November 2011, encouraging them to avoid further delays so that the public review of the rulemaking could begin as soon as possible (AIHA, 2011). AIHA stated that it recognized the merit of OIRA review with regard to costs/benefits and quality assessment, but that an additional delay was “unacceptable.” The group also noted the lack of transparency in OMB’s review process, and stated that the rule needed to be released for publication so that industry, labor and the SH&E community could provide meaningful input in a public forum. The group also encouraged OMB to defer to “OSHA’s scientific judgment as much as possible.”

OSHA is proposing a respirable crystalline silica PEL of 50 µg/m³ as an 8-hour TWA. The proposed standard also includes an action level that would trigger additional provisions related to topics such as health screening and training (OSHA, 2013).

Conclusion

Knowledge regarding health hazards from crystalline silica exposure and how to best safeguard exposed workers has evolved drastically over the past century. However, regulatory uncertainties and the fact that many workers are still overexposed to silica dust in the U.S. and abroad have led to calls for a comprehensive OSHA standard. Some labor groups and occupational safety advocates have even called for a ban on all types of abrasive blasting, including methods that do not use sand or silica, as a means to safeguard workers.

Promulgating a standard that contains requirements for engineering controls, respiratory protection, exposure monitoring, training and medical surveillance might create an environment in which such a ban would be unnecessary. In addition, such a standard would likely serve to protect workers on a global scale, since many countries would presumably adopt the standard.

References


Challenges to the Proposed Silica Standard

Several organizations, such as Construction Industry Safety Coalition, American Chemistry Council (ACC), and members of the U.S. Chamber of Commerce, have challenged the proposed silica rule and its supporting analyses (BLR, 2013; ForConstructionPros.com, 2013; Lee & Iafolla, 2013). Concerns include the high cost of compliance as well as questions regarding OSHA’s ability to effectively enforce the current PEL, let alone the various provisions of the proposed standard. In addition, ACC has posited that the PEL is already “appropriate” to prevent silica-related disease, noting that current silicosis cases are a result of noncompliance with the existing PEL (ACC, 2013).

ACGIH. (1946, April). Proceedings of the eighth annual meeting of the American Conferences of Governmental Industrial Hygienists, Chicago, IL.


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