Criteria for Updating the Crystalline free silica PEL

PEL Recommendation:

Change the PEL for both cristobalite and quartz to 0.05 mg/m3. Change the distribution for the respirable fraction to the ISO/CEN/ACGIH standard with the 50 % cut at 4 micrometers. (ISO/CEN is the International Organization for Standardization/European Standardization Committee). The current OR-OSHA PEL for crystalline silica is actually a respirable particulate PEL calculated from the percent crystalline silica in the dust. NIOSH recommends a PEL of 0.05 mg/m3 for both cristobalite and quartz to reduce the risk of developing silicosis, lung cancer, and other adverse health effects.

Background:

Silica: Silica is commonly found in the earth’s crust and refers to the chemical compound silicon dioxide (SiO2). Silica occurs in both crystalline and non-crystalline forms. Crystalline forms are physical states in which the silicon dioxide molecules are arranged in a repetitive pattern that has unique spacing, lattice structure and angular relationship of the atoms. Crystalline silica forms (often referred to as polymorphs) include quartz, cristobalite, tridymite, keatite, coesite, stishovite and moganite [1,2,3]. In nature, quartz is the crystalline form most commonly encountered and is so abundant that the term quartz is often used in place of the general term crystalline silica [4,5]. Quartz is abundant in most rocks and soils and is also present in sand, mortar, concrete, fluxes, abrasives, construction aggregate, porcelain, paints and brick [6]. In addition, quartz containing dust may be generated in any process which involves movement of earth (e.g., mining, farming, construction), disturbance of silica-containing products such as masonry and concrete, or use of sand and other silica containing products (e.g., foundry processes). Consequently, workers are potentially exposed to quartz dust in many occupations and industries. Cristobalite and tridymite can be found in volcanic rocks and soils. These polymorphs can also be produced in some industrial operations. For example, cristobalite transformations occur in foundry processes, calcining of diatomaceous earth, brick and ceramics manufacturing, and silicon carbide production [7,8,9]. Burning of agricultural waste or products such as rice hulls may also cause amorphous silica to become cristobalite [6,10]. The other crystalline polymorphs (i.e., keatite, coesite, stishovite, and moganite) are very rarely or never observed in nature and are formed only under very high pressures [1]. Since these polymorphs are generally formed in small quantities, they are therefore of limited industrial hygiene interest. Non-crystalline, or amorphous, forms of silica exist when the silicon dioxide molecules are randomly arranged. Fly ash, silica fume and silica gel contain amorphous silica. Diatomaceous earth is 88% amorphous silica and is composed of the skeletons of small prehistoric aquatic plants related to algae [11]. In unusual instances of extreme heat and very slow cooling conditions, amorphous silica can be transformed into crystalline silica as mentioned above. Similarly, various crystalline silica forms can transform into different crystalline forms when subjected to high heat or high heat and pressure. [6]
(From the NIOSH Manual of Analytical Methods (NMAM®), 4th ed.DHHS (NIOSH) Publication 94-113 (August, 1994), Chapter – R; Determination of Airborne Crystalline Silica, pages 260 to 280)

Effects Of Exposure To Crystalline Silica:

Silicosis is one of the world's oldest known occupational diseases, dating back to ancient Greece. Since the 1800s, the silicotic health problems associated with crystalline silica dust exposure have been referred to under a variety of common names, including consumption, ganister disease, grinders' asthma, grinders' dust consumption, grinders' rot, masons' disease, miner's asthma, miner's phthisis, potters' rot, sewer disease, stonemason's disease, chalicosis, and shistosis. Silicosis was considered the most serious occupational hazard during the 1930s and was the focus of major federal, state and professional attention.

Silicosis is a disabling and often fatal lung disease caused by breathing dust that has very small pieces of crystalline silica in it. Silicosis, a scarring and hardening of lung tissue, can result when particles of crystalline silica are inhaled and become embedded in the lung. The disease can be progressively debilitating and fatal.

Silicosis is the result of the body's response to the presence of silica dust in the lung(s). The respirable fraction of the dust (particles generally considered to be smaller than five-millionth of a meter) can penetrate to the innermost regions of the respiratory systems. These are the alveoli (airsacs) where the exchange of oxygen and carbon dioxide occurs. When workers inhale crystalline silica, they land on the alveoli, and white blood cells (macrophages) try to remove them. However, the particles of free crystalline silica cause the macrophages to break open. The lung tissues react by developing fibrotic nodules and scarring around the trapped silica particles.

Formation of large numbers of "scars" following prolonged exposure causes the alveolar surface to become less elastic. This is noticed as shortness of breath following exertion. Symptoms may not appear in the early stages of chronic silicosis. In fact, chronic silicosis may go undetected for 15 to 20 years after exposure.

Workers may develop any of three types of silicosis, depending on the concentration of airborne silica:

- Chronic silicosis, which usually occurs after ten or more years of exposure to crystalline silica at relatively low concentrations.
- Accelerated silicosis which results from exposure to high concentrations of crystalline silica and develops five to ten years after the initial exposure.
- Acute silicosis, which occurs where exposure concentrations are the highest and can cause symptoms to develop within a few weeks to four or five years after the initial exposure.

Symptoms and Effects of Silicosis

Early stages of the disease may go unnoticed. Continued exposure may result in a shortness of breath on exercising, possible fever and occasionally bluish skin at the ear lobes or lips.

Silicosis makes a person more susceptible to infectious diseases of the lungs, such as tuberculosis. Progression of silicosis leads to fatigue, extreme shortness of breath, loss of appetite, pains in the chest, and respiratory failure, which may cause death.

Medical evaluations of silicosis victims usually show the lungs to be filled with silica crystals and a protein material. Pulmonary fibrosis (fibrous tissue in the lung) may or may not develop in acute cases of silicosis depending on the time between the exposure and the onset of symptoms. Furthermore, evidence indicates that crystalline silica is a potential occupational carcinogen.
(From the Federal OSHA website at http://www.osha.gov/SLTC/silicacrystalline/mineoja/demolition.html)

Rationale:

Occupational exposures to respirable crystalline silica occur in a variety of industries and occupations because of its extremely common natural occurrence and the wide uses of materials and products that contain it. At least 1.7 million U.S. workers are potentially exposed to respirable crystalline silica [12], and many are exposed to concentrations that exceed limits defined by current regulations and standards. Silicosis, usually a nodular pulmonary fibrosis, is the disease most associated with exposure to respirable crystalline silica. Although the reported mortality associated with silicosis has declined over the past several decades, many silicosis-associated deaths still occur (nearly 300 deaths were reported each year during the period 1992–1995) [13; 14]. In addition, the number of silicosis associated deaths among persons aged 15 to 44 has not declined substantially [15, 16]. An unknown number of workers also continue to die from silica-related diseases such as pulmonary tuberculosis (TB), lung cancer, and scleroderma. The number of cases of silicosis and silica-related diseases in the United States today is unknown. Symptoms of acute silicosis, another form of silicosis, may develop shortly after exposure to high concentrations of respirable crystalline silica. Epidemiologic studies focus on chronic silicosis, which develops years after exposure to relatively low concentrations of respirable crystalline silica. Epidemiologic studies have found that chronic silicosis may develop or progress even after occupational exposure has ceased [17; 18; 19; 20; 21; 22]. Over a 40- or 45-year working lifetime, workers have a significant chance (at least 1 in 100) of developing radiographic silicosis when exposed to respirable crystalline silica at the Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL), the Mine Safety and Health Administration (MSHA) PEL, or the National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL). Silicosis may be complicated by severe mycobacterial or fungal infections. About half of these are caused by *Mycobacterium tuberculosis* and result in TB. Epidemiologic studies have firmly established that silicosis is a risk factor for developing TB.

The carcinogenicity of crystalline silica in humans has been strongly debated in the scientific community. In 1996, the International Agency for Research on Cancer (IARC) reviewed the published experimental and epidemiologic studies of cancer in animals and workers exposed to respirable crystalline silica and concluded that there was “sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources” [23]. In the same year, directors of the American Thoracic Society (ATS) adopted an official statement that described the adverse health effects of exposure to crystalline silica, including lung cancer [24]. The ATS found that “the available data support the conclusion that silicosis produces increased risk for bronchogenic carcinoma.” However, the ATS noted that less information was available for lung cancer risks among silicotics who had never smoked and for silica-exposed workers who did not have silicosis. They also stated that it was “less clear” whether silica exposure was associated with lung cancer in the absence of silicosis. NIOSH has reviewed the studies considered by IARC and ATS, and NIOSH concurs with the conclusions of IARC [23] and the ATS [24]. These conclusions agree with NIOSH testimony to OSHA, in which NIOSH recommended that crystalline silica be considered a potential occupational carcinogen [25]. Statistically significant excesses of mortality from stomach or gastric cancer have been reported in various occupational groups exposed to crystalline silica. Occupational exposure to respirable crystalline silica is associated with chronic obstructive pulmonary disease, including bronchitis and emphysema. Significant increases in mortality from nonmalignant respiratory disease (a broad category that can include silicosis and other pneumoconioses, chronic bronchitis, emphysema, asthma, and other related respiratory conditions) have been reported for silica-exposed workers [26, 27; 28; 29; 30; 31; 32; 33; 34; 35; 36; 37] and silicotics [38; 39; 40]. Many case reports have been published about autoimmune diseases or autoimmune-related diseases in workers exposed to crystalline silica or workers with silicosis. In addition, several recent epidemiologic studies reported statistically significant numbers of excess cases or deaths from known autoimmune diseases or immunologic disorders (scleroderma, systemic lupus erythematosus, rheumatoid arthritis, sarcoidosis), chronic renal disease, and subclinical renal changes. Until improved sampling and analytical methods are developed for respirable crystalline silica, NIOSH will continue to recommend an exposure limit of 0.05 mg/m^3 to reduce the risk of developing silicosis, lung cancer, and other adverse health effects. NIOSH also recommends minimizing the risk of illness that remains for workers exposed at the REL by substituting less hazardous materials for crystalline silica when feasible, by using appropriate respiratory protection when source controls cannot keep exposures below the NIOSH REL, and by making medical examinations available to exposed workers.

(NIOSH HAZARD REVIEW, Health Effects of Occupational, Exposure to Respirable Crystalline Silica, DEPARTMENT OF HEALTH AND HUMAN SERVICES, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, April 2002; DHHS (NIOSH) Publication No. 2002–129)

The following tables are a summary of current Occupational Exposure Limits for Crystalline Free Silica.

Table 1

	PEL
Current OR-OSHA PEL:	10 mg/m3 / (% quartz + 2 x % cristobalite + 2) for respirable dust
OSHA:	10 mg/m3 / (% quartz + 2 x % cristobalite + 2) for respirable dust
1989 OSHA PEL:	Respirable quartz: 0.1 mg/m3, Respirable cristobalite and tridymite: 0.05 mg/m3
NIOSH REL:	Respirable cristobalite, quartz and tridymite: 0.05 mg/m3
2003 ACGIH TLV:	Respirable cristobalite, quartz and tridymite: 0.05 mg/m3
MSHA PEL	10 mg/m3 / (% quartz + 2) for respirable dust

Table 2
Other States:

California PEL	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3
Michigan PEL	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3
N. Carolina PEL	10 mg/m3 / (% quartz + 2 x % cristobalite + 2) for respirable dust
Washington PEL	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3
Hawaii	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3
Minnesota	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3
Vermont	Respirable Quartz: 0.1 mg/m3, Respirable Cristobalite: 0.05 mg/m3

Respirable Fraction

The OR-OSHA and OSHA PELs are actually calculations that determine the respirable dust PEL from the percent quartz and cristobalite found in the dust. For both these standards the respirable particulate fraction is based on the fraction of the particles in the air that conform to the size distribution outlined in the following table. The Aerodynamic Diameter is in micrometers.

Table 3

Aerodynamic Diameter (Unit Density Sphere)	Percent Passing Selector
2	90
2.5	75
3.5	50
5.0	25
10	0

This is typically referred to as a 50 % cut of 3.5 micrometers.

Silica dust is sampled using techniques to collect the fraction of dust that penetrates into the deep lung. The ISO/CEN/ACGIH definition for this respirable particulate mass (RPM) is slightly different than the current OR-OSHA and OSHA definitions.

It is defined as:

$$\text{Collection efficiency} = 0.5\left(1 + e^{-0.06\, d_{ae}}\right)\left(1 - F(x)\right)$$

d_{ae} = aerodynamic diameter of the particle in micrometers

Γ = 4.25 micrometers

Σ = 1.5

F(X) = the cumulative probability function of the standardized normal variable x and

$$X = \frac{\ln\left(d_{ae}/\Gamma\right)}{\ln(\Sigma)}$$

With this particle size distribution 50 % of the 4 micrometer particles pass the size selector. This distribution lis displayed in the following table.

Table 4

Aerodynamic Diameter(μm)	Percent Passing Size Selector
1	97
2	91
3	74
4	50
5	30
6	17
7	9
8	5
9	3
10	1

Exposures

Nationwide at least 1.7 million workers are exposed to crystalline free silica according to 1991 NIOSH statistics. Morbidity and Mortality Weekly Report (April 29, 2005/54(16); 401-405) states that "Crystalline silica exposure and silicosis have been associated with work in mining, quarrying, tunneling, sandblasting, masonry, foundry work, glass manufacture, ceramic and pottery production, cement and concrete production, and work with certain materials in dental laboratories."

NIOSH's Work Related Lung Disease Surveillance Program (Pub 2003-111) states that there were 2405 deaths in the US from 1990 to 1999 caused by silicosis. 23 of these deaths were in Oregon. The most frequently recorded industries were (in order) construction; metal mining; coal mining; blast furnace, steel works, rolling and finishing mills; nonmetallic mining and quarrying; iron and steel foundries; miscellaneous nonmetallic mineral and stone products; not specified manufacturing industries; machinery, except electrical and structural clay products. The most frequently recorded occupations were mining machine operators; laborers, except construction; managers and administrators; supervisors, production occupations; janitors and cleaners; machine operators, not specified; construction laborers; molding and casting machine operators; supervisors and proprietors, sales occupations and truck drivers.

MSHA data for Oregon from 1979 to 1999 for metal and nonmetal mining shows 98 overexposures to the PEL and 110 overexposures to the REL

OR-OSHA Silica exposure data from 1989 to 2003 in Oregon has 398 overexposures to the old PEL and would have had 430 overexposures to PELs of 0.05 mg/m3 quartz and 0.05 mg/m3 cristobalite. SIC codes where overexposures were found are 0161, 0723, 0760, 1411, 1541, 1542, 1611, 1622, 1721, 1741, 1742, 1743, 1751, 1761, 1771, 1799, 2063, 2411, 2851, 2952, 3083, 3252, 3272, 3281, 3295, 3312, 3321, 3325, 3364, 3441, 3444, 3555, 3949, 4959, 5032, 5082, 5148, 5211, 5719, 5941, 5945, 8093. These include many phases of agriculture, construction and manufacturing. Also included are logging, and wholesale and retail businesses.

Comparison of current OR-OSHA PEL with proposed PEL

Table 5 contains comparisons of the current OR-OSHA PEL and the proposed PEL. The first column in Table 5 is percent quartz present in a sample. The second column is the allowable PEL for the respirable dust PEL calculated from the percent quartz in column 1 and the formula for the current OR-OSHA PEL (see Table 1). The third column is the amount of quartz present in the air (in mg/m3) at the calculated respirable dust PEL and the percent quartz in the dust. Column four is the NIOSH REL or the ACGIH TLV. This is the proposed PEL. The fifth column is a ratio of the quartz exposure represented by the current PEL to the proposed PEL (the REL or TLV). Figure 1 is a graphic representation of this data.

For the current OR-OSHA PEL the allowable quartz exposure is always less than 0.1 mg/m3. This can be seen in Table 5 where the third column is always less than 0.1 or Figure 1 where the PEL line is always less than 0.1 mg/m3. The allowable quartz exposure with the proposed PEL of 0.05 mg/m3 is less than the current PEL when quartz is greater than 2 %. This can be seen in Table 5 where the values in column 5 are less than or equal to one, quartz is less than or equal to 2 percent.

Table 5

% Quartz	Respirable dust PEL (mg/m3)	Amount of Quartz Present at PEL (mg/m3)	REL or TLV	Current PEL / REL(or TLV)
1	3.33	0.0333	0.05	0.67
2	2.5	0.05	0.05	1.00
3	2	0.06	0.05	1.20
4	1.67	0.0667	0.05	1.33
5	1.43	0.0714	0.05	1.43
6	1.25	0.075	0.05	1.50
7	1.11	0.0778	0.05	1.56
8	1	0.08	0.05	1.60
9	0.909	0.0818	0.05	1.64
10	0.833	0.0833	0.05	1.67
11	0.769	0.0846	0.05	1.69
12	0.714	0.0857	0.05	1.71
13	0.667	0.0867	0.05	1.73
14	0.625	0.0875	0.05	1.75
15	0.588	0.088	0.05	1.76
16	0.556	0.0889	0.05	1.78
17	0.526	0.0894	0.05	1.79
18	0.5	0.09	0.05	1.80
19	0.476	0.0905	0.05	1.81
20	0.455	0.0909	0.05	1.82
25	0.370	0.0926	0.05	1.85
30	0.313	0.0938	0.05	1.88
35	0.270	0.0946	0.05	1.89
40	0.238	0.0952	0.05	1.90
45	0.212	0.0957	0.05	1.91
50	0.192	0.0962	0.05	1.92
55	0.175	0.0965	0.05	1.93
60	0.161	0.0968	0.05	1.94
65	0.149	0.0970	0.05	1.94
70	0.139	0.0972	0.05	1.94
75	0.130	0.0974	0.05	1.95
80	0.122	0.0976	0.05	1.95
85	0.115	0.0977	0.05	1.95
90	0.109	0.0978	0.05	1.96
95	0.103	0.0979	0.05	1.96
100	0.0980	0.0980	0.05	1.96

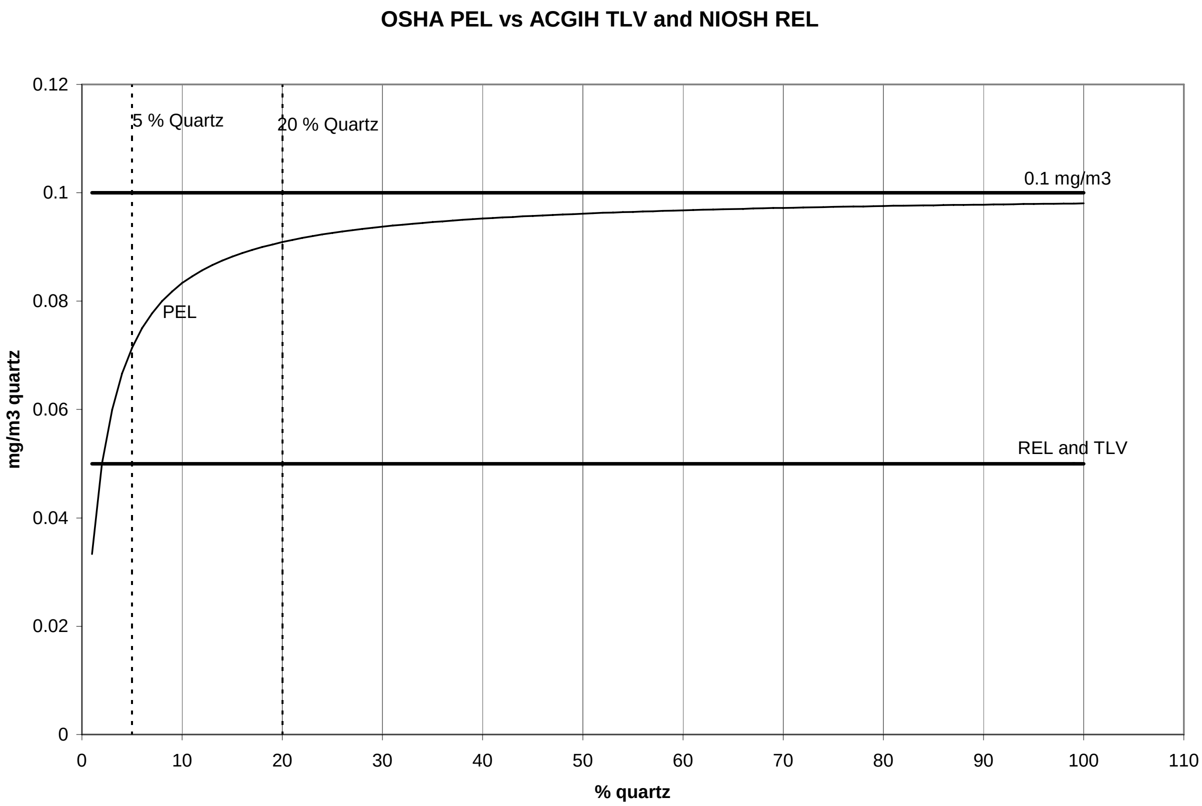

Figure 1

The average of 500 samples analyzed by the OR-OSHA lab from 1989 to 2003 was 13 % for quartz. The median was 8.53 %. About 10 % were less than 2% quartz. The distribution is illustrated in figure 2.

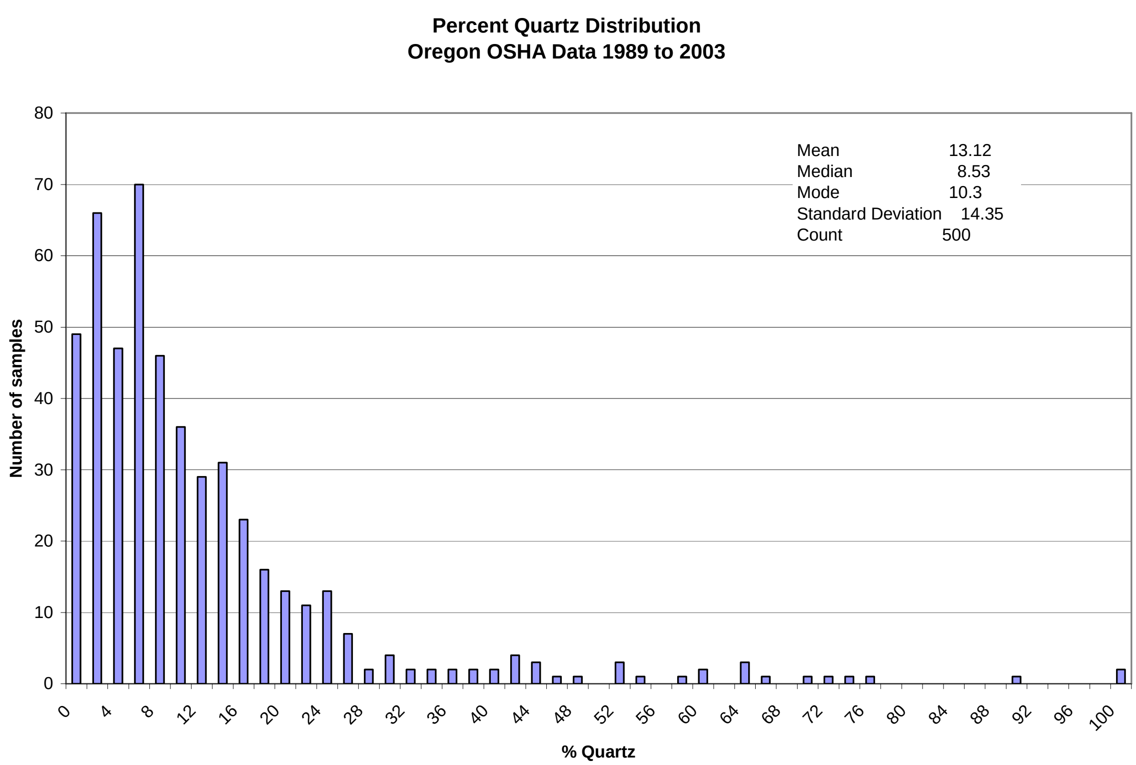

Figure 2

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