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## NIOSH HAZARD REVIEW

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# 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

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## NIOSH HAZARD REVIEW

### Health Effects of Occupational Exposure to Respirable Crystalline Silica

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## 1 Introduction

### 1.1 Definition of Crystalline Silica

Silica refers to the chemical compound silicon dioxide ( $\text{SiO}_2$ ), which occurs in a crystalline or noncrystalline (amorphous) form. Crystalline silica may be found in more than one form (polymorphism). The polymorphic forms of crystalline silica are alpha quartz, beta quartz, tridymite, cristobalite, keatite, coesite, stishovite, and moganite [Ampian and Virta 1992; Heaney 1994; Guthrie and Heaney 1995]. Each polymorph is unique in its spacing, lattice structure, and angular relationship of the atoms. In nature, the alpha (or low) form of quartz is the most common [Virta 1993]. This form is so abundant that the term *quartz* is often used in place of the general term *crystalline silica* [BOM 1992; Virta 1993]. Quartz is a common component of soil and rocks; consequently, workers are potentially exposed to quartz dust in many occupations and industries (see [Section 2.3](#)). Cristobalite and tridymite are found in rocks and soil and are produced in some industrial operations when alpha quartz or amorphous silica is heated (such as foundry processes, calcining of diatomaceous earth, brick and ceramics manufacturing, and silicon carbide production) [NIOSH 1974; Weill et al. 1994; Virta 1993; Altieri et al. 1984]. Burning of agricultural waste or products such as rice hulls may also cause amorphous silica to become cristobalite (a crystalline form) [Rabovsky 1995; IARC 1997]. The other polymorphs (i.e., keatite, coesite, stishovite, and moganite) are rarely or never observed in nature [Ampian and Virta 1992].

### 1.2 Current Health Issues

Occupational exposure to respirable crystalline silica is a serious but preventable health hazard. Since 1968, reported mortality associated with silicosis has declined; however, 200 to 300 such deaths were reported each year during the period 1992-1995 [NIOSH 1996a; Althouse 1998]. Furthermore, the number of silicosis-related deaths among persons aged 15 to 44 did not decline substantially during 1968-1994, accounting for 207 of the 14,824 silicosis-related deaths during this period [CDC 1998a,b]. In addition, an unknown number of unreported or undiagnosed worker deaths occur each year from silicosis and other silica-related diseases such as pulmonary tuberculosis (TB), lung cancer, and scleroderma. The number of current cases of silicosis and silica-related disease in the United States is also unknown.

Prevention and elimination of silicosis and silica-related disease in the United States are priorities of the National Institute for Occupational Safety and Health (NIOSH), the Occupational Safety and Health Administration (OSHA), the Mine Safety and Health Administration (MSHA), and the American Lung Association [DOL 1996]. International health agencies have also expressed

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concern about the continuing occurrence of silicosis and silica-related diseases. The International Agency for Research on Cancer (IARC) recently reviewed the results of post-1986 epidemiologic studies of lung cancer and occupational exposure to crystalline silica. They concluded that there is "sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources" (i.e., IARC category "Group 1" carcinogen) [IARC 1997]. In 1991, the International Labour Office published a document describing methods for preventing and controlling occupational lung diseases, including silicosis [ILO 1991]. And in 1993, the Office of Occupational Health of the World Health Organization (WHO) called for increased medical surveillance of mineral-dust-exposed workers to prevent pneumoconioses such as silicosis and asbestosis [WHO 1993]. Epidemiologic studies published after the IARC review [IARC 1997] provide additional evidence for an exposure-response relationship of respirable crystalline silica with lung cancer mortality or morbidity (see [Section 3.4.2.1](#)).

Several recent epidemiologic studies indicate that current occupational standards are not sufficiently protective to prevent the occurrence of chronic silicosis. Epidemiologic studies of workers in the United States [Kreiss and Zhen 1996; Steenland and Brown 1995a; Rosenman et al. 1996; Hughes et al. 1998], Canada [Muir et al. 1989a,b; Muir 1991], Hong Kong [Ng and Chan 1994], and South Africa [Hnizdo and Sluis Cremer 1993] have reported significant risks of silicosis over a working lifetime at concentrations of quartz or respirable dust containing quartz that are below the current NIOSH recommended exposure limit (REL) [NIOSH 1974], OSHA permissible exposure limit (PEL) [29 CFR\*1910.1000], and the MSHA PEL [30 CFR 56, 57, 70, 71] (see [Appendix](#) and [Table 12](#) in Chapter 3).

\*Code of Federal Regulations. See CFR in references.

TB is an infectious disease that poses a threat to the health of silica-exposed workers and the public. A survey of U.S. mortality data for 1979 to 1991 reported that TB comortality was at least several times higher in decedents with silicosis than in decedents with asbestosis, with coal workers' pneumoconiosis (CWP), or without silicosis, asbestosis, or CWP [Althouse et al. 1995]. The U.S. Centers for Disease Control and Prevention (CDC), WHO, and the American Thoracic Society (ATS) have recently published information about risk factors for TB, including occupational exposure to respirable crystalline silica [CDC 1995; WHO 1996; ATS 1997]. The U.S. Environmental Protection Agency (EPA) suggested "further investigation" of the health effects of ambient crystalline silica exposures in potentially sensitive subgroups, including infants and persons with a respiratory infection or disease such as TB or pneumonia [EPA 1996].

Recent epidemiologic studies of occupational exposure to crystalline silica dust have also reported increased incidence of—or mortality from—extrapulmonary diseases such as scleroderma, rheumatoid arthritis, other autoimmune disorders, and renal disease [ATS 1997].

Experimental research has shown that crystalline silica is not an inert dust. The toxicity of crystalline silica particles is related to reactive sites on the surfaces of silica particles. Further discussion of in vitro studies of the biologic activity and factors that modify toxicity are found in [Section 3.2.1](#) and [Section 4](#).

### 1.3 History of NIOSH Activity

In 1974, NIOSH reviewed the available health effects data on occupational exposure to respirable crystalline silica and determined that the principal adverse health effect was silicosis [NIOSH 1974]. At that time, NIOSH recommended that occupational exposure to respirable crystalline silica dust be controlled so that workers would not be exposed to the airborne particulate at a time-weighted average (TWA) concentration greater than 50

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micrograms per cubic meter of air ( $50 \mu\text{g}/\text{m}^3$  —or  $0.05 \text{mg}/\text{m}^3$ ), determined during a full-shift sample for up to a 10-hr workday during a 40-hr workweek. A later NIOSH report (*Review of the Literature on Crystalline Silica*) concluded that additional toxicologic and epidemiologic studies were needed to determine

1. the relationship between respirable crystalline silica dose and the risk of developing silicosis and lung cancer and
2. the adverse effects of crystalline silica on the kidney [NIOSH 1983a].

Since then, additional studies reported an increased incidence of malignant tumors in the lungs of rats exposed to either inhalation or intratracheal administration of various forms and preparations of respirable crystalline silica [Holland et al. 1986; Dagle et al. 1986; Groth et al. 1986; Muhle et al. 1989; Spiethoff et al. 1992]. On the basis of the evidence from the animal studies published by 1986, IARC concluded that "sufficient evidence" existed for the carcinogenicity of respirable crystalline silica in experimental animals but only "limited evidence" existed for carcinogenicity in humans [IARC 1987]. During the 1988 OSHA rulemaking activity on air contaminants, NIOSH recommended an exposure limit of  $0.05 \text{mg}/\text{m}^3$  "as respirable free silica for all crystalline forms of silica" to protect workers from silicosis and cancer [54 Fed. Reg.\* 2521 (1989)]. In addition, NIOSH testimony referred to the IARC [1987] review and recommended that OSHA label crystalline silica a potential occupational carcinogen [54 Fed. Reg. 2521 (1989)].

\*Federal Register. See Fed. Reg. in references.

#### 1.4 Purpose and Scope

The numerous health effects of occupational exposure to respirable crystalline silica are reviewed in the chapters of several recent books [Graham 1998; Davis 1996; Green and Vallyathan 1996; McDonald 1996; Seaton 1995; Morgan and Reger 1995; Elmes 1994; Goldsmith 1994a,b; Weill et al. 1994; Wagner 1994]. This NIOSH Hazard Review summarizes the health effects of occupational exposure to respirable crystalline silica reported in literature published through March 1999. The review emphasizes recent important epidemiologic studies of occupational exposure to respirable crystalline silica with regard to

1. the quantitative risk of chronic silicosis,
2. lung cancer,
3. autoimmune disease,
4. chronic renal disease, and
5. chronic obstructive pulmonary disease. In addition, the review describes limitations of the current sampling and analytical methods for quantifying occupational exposures to silica.

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