

The educational framework should be more frequently used to develop and test preventive strategies and solutions, for example, by motivating students to select such themes their theses.

The issues of quality of courses and professional competence are also crucial and have to be properly addressed.

Lack of multidisciplinary approaches and intersectoral collaboration

Efficient and sustainable hazard prevention can only be ensured through a multidisciplinary approach, involving occupational health professionals and stakeholders, such as managers, production personnel, and workers.

Intersectoral collaboration, at national and local levels, is also of great consequence and, in most places, needs to be improved, emphasizing the joint planning and action required to avoid duplication and to make the best use of available resources.

Too much emphasis on quantitative evaluations

It often happens that more attention is given to exposure assessment than to hazard prevention and control. “Occupational health programmes and services should give due importance to primary prevention in relation to exposure assessment and monitoring which, although essential components of occupational hygiene practice, can only disclose or confirm but never prevent exposure. There often is more interest in identifying and evaluating occupational exposures and their consequences, than in actually preventing them” (5). This issue is interlinked with legislation since it is the legal framework that often requires numerical values to characterize exposure.

It may happen that quantitative exposure assessment is unfeasible, but this should never constitute a blockage to required preventive interventions. In fact, even if feasible, it may not be necessary to quantitatively evaluate in order to establish an obvious need to control.

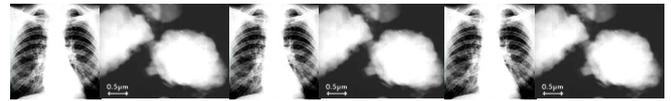
In this context, pragmatic approaches based on qualitative and semi-quantitative assessment methodologies were developed and can be very useful in many cases. If well validated and properly used, these may constitute good tools to assess certain risk situations, establish priorities for action, and guide the decision of “what to do next” in terms of control and which control strategy and measures to adopt.

The HSE COSHH (Control of Substances Hazardous to Health) Essentials, successfully used in the UK, relies on such principles (9;10). Other examples of pragmatic approaches include the SOBANE Methodology (11) and the GTZ methodology (12).

Concerning exposure to silica, the HSE has developed the excellent Silica Essentials control guidance sheets (available online at the HSE website; <http://www.hse.gov.uk/pubns/guidance/index.htm>).

Inadequate programmes

Preventive efforts may be hindered if specific control measures are not integrated into multidisciplinary, competently managed, efficient and sustainable programmes. In this respect, it is important to mention the ILO guidelines on occupational safety and health management systems: ILO-OSH 2001 (13).



Silica-Related Disease: It's not just silicosis

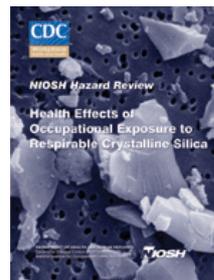


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Introduction

Silicosis is one of the oldest and best-known occupational diseases. Recognized since ancient times, cases of this incurable, but preventable, fibrotic lung disease have been identified in many countries and in many occupational settings and cases continue to be found in developed and less-developed countries. However, research studies published in the last century pointed to other diseases in workers exposed to respirable crystalline silica dust. The U.S. National Institute for Occupational Safety and Health (NIOSH) conducted a review of the large body of international health-related silica literature and published the results in 2002 (1). NIOSH found that occupational exposure to respirable crystalline silica is associated with the development of several diseases including silicosis, and may be related to the development of others. The results of the NIOSH review were incorporated into the WHO Concise International Chemical Assessment Document (CICAD) on quartz (2).

Lung cancer and other respiratory diseases



Debate about whether crystalline silica could be an occupational lung carcinogen heightened in the 1980s after publication of several key works on the topic (3-6). In 1996, the International Agency for Research on Cancer (IARC) 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”

(7). In the same year the American Thoracic Society (ATS) adopted an official statement that described the adverse health effects of exposure to crystalline silica, including lung cancer (8). The ATS found that:

- The available data support the conclusion that silicosis produces increased risk for bronchogenic carcinoma.
- However, less information is available for lung cancer risk among silicotics who never smoked and workers who were exposed to silica but did not have silicosis.
- Whether silica exposure is associated with lung cancer in the absence of silicosis is less clear.

NIOSH concurred with the conclusions of the IARC working group and the ATS (1). The United States National Toxicology Program (NTP) concluded that respirable crystalline silica, “primarily quartz dusts in industrial and occupational settings” is a known human carcinogen (9).

Occupational exposure to respirable crystalline silica is associated with chronic obstructive pulmonary disease (COPD), including bronchitis and emphysema (1). In addition, significant increases in mortality from non-malignant respiratory disease (a broad category that could include silicosis and other pneumoconioses, chronic bronchitis, emphysema, asthma, and other related respiratory conditions) were reported in several studies of silica-exposed workers and also in studies of silicotics (1). A

review published in 2003 by researchers at NIOSH reviewed epidemiologic and pathologic studies of COPD and concluded that the evidence “suggests that chronic lower levels of silica exposure may lead to the development of emphysema, chronic bronchitis, and/or mineral dust airways disease (MDAD) that can lead to airflow obstruction, even in the absence of radiological signs of silicosis.” (10)

Pulmonary tuberculosis and other infections

Silica dust exposure and silicosis increase the risk of tuberculosis (11). “Silicotuberculosis” is a common problem in many developing countries and in communities where active tuberculosis is common (12). A study published in 2006 reported that pulmonary tuberculosis is “currently epidemic” in South African goldmines and is associated with both silicosis and HIV infection (13). Silicosis can also be complicated by infections with non-tuberculous mycobacteria (NTM) such as *Mycobacterium kansasii* and *Mycobacterium avium-intracellulare* (1)(14). Other infections in workers with silicosis may be caused by *Nocardia asteroides* and *Cryptococcus* (1)(15)(16).

Autoimmune Diseases and autoimmune-related diseases

In the last century, many case reports were published about various autoimmune disorders in workers or patients occupationally exposed to crystalline silica. The majority of these reports described scleroderma (systemic sclerosis), systemic lupus erythematosus (lupus), rheumatoid arthritis, autoimmune hemolytic anaemia, and dermatomyositis or dermatopolymyositis (1). Additionally, NIOSH and WHO cited several epidemiologic studies that reported statistically significant numbers of excess deaths or cases of immunologic disorders and autoimmune diseases in silica-exposed workers, including scleroderma (17)(18), rheumatoid arthritis (19)(20), and systemic lupus erythematosus (17). Further research is needed to determine the cellular mechanism for development of autoimmune responses and diseases in workers exposed to crystalline silica (1)(21).

Chronic renal diseases, sub-clinical renal changes

The NIOSH (1) and WHO (2) reviews noted that some recent epidemiologic studies conducted in several countries reported statistically significant associations of crystalline silica exposure with renal disease incidence (22) or mortality (23)(17), Wegener’s granulomatosis (24), and sub-clinical renal changes (25)(26)(27). Four epidemiologic studies, published after the content included in those reviews, evaluated an exposure-response relationship for renal disease and silica exposure (28)(29)(30)(31). The studied silica-exposed cohorts were: a) 4,626 industrial sand workers in the U.S. (28), b) 2,670 male employees of the North American sand industry (29), c) a combined (i.e., pooled) U.S. cohort of the aforementioned 4,626 industrial sand workers, 3,348 gold miners, and 5,408 granite workers (30), and d) 4,839,231 U.S. deaths from various causes that occurred over a 14-year period in 27 states (31). Of the sand worker studies, one found a “pronounced” monotonic trend of increased incidence of end-stage renal disease (18 cases) with increasing cumulative silica exposure (28), while the other study investigated the relationship of cumulative exposure with mortality from nephritis/nephrosis or kidney cancer but found no increasing trend (29). Both studies analysed relatively small numbers of renal disease deaths or cases compared with the three-cohort pooled analysis of 204 deaths with renal disease listed on the death certificate as an underlying or contributing cause. That pooled study found an excess of renal disease mortality and a statistically significant monotonic trend of increased renal disease mortality with increasing cumulative silica exposure (30). The large U.S. case-control analysis of various

causes of death assessed crystalline silica exposure qualitatively and did not find a significant and increasing exposure-response trend with any renal outcome investigated (31). A mechanism for silica-related renal disease has not been well-established.

Other adverse health effects

The NIOSH (1) and WHO (2) reviews found a number of adverse health effects noted in the published literature including non-lung cancers of various sites; an association between these non-pulmonary cancers and occupational silica exposure has not been confirmed.

Conclusion

Occupational exposure to respirable crystalline silica is associated with silicosis, lung cancer, pulmonary tuberculosis, and airways diseases. In addition, it may be related to development of autoimmune disorders, chronic renal disease, and other adverse health effects (1) (see Table 1). Exposure-response analyses predicted that the excess or absolute risk of death or disease from lung cancer, silicosis, and kidney disease in crystalline silica-exposed workers varies, but exceeds one per 1,000 after 45 years of exposure to silica concentrations near or lower than the U.S. (OSHA) standard (32). However, the good news is that occurrence of these diseases in silica-exposed workers is preventable; WHO admonishes that “**Action should be taken before exposure happens**” (33).

Note to readers: Further information about the adverse health effects of occupational exposure to respirable crystalline silica is available from the cited references, the NIOSH Silica topic page <http://www.cdc.gov/niosh/topics/silica/default.html>, and the Publications section of the NIOSH en español website <http://www.cdc.gov/spanish/niosh/pubs-sp.html>.

Disclaimer: “The findings and conclusions in this report are those of the author and do not necessarily represent the views of the National Institute for Occupational Safety and Health.”

Table 1. Conclusions of NIOSH review of adverse health effects of occupational exposure to respirable crystalline silica (1).

Adverse Health Effect	Associated with occupational exposure	May be associated with occupational exposure
Silicosis	U	
Lung Cancer	U	
Pulmonary Tuberculosis	U	
Airways Diseases	U	
Autoimmune Disorders		U
Chronic Renal Disease, sub-clinical renal changes		U



Chronic obstructive bronchitis and emphysema⁽⁴⁾ in hard coal miners



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Long-term exposure to crystalline silica-containing dust in hard coal mines is not only associated with

⁴ Emphysema is characterized by loss of elasticity of the lung tissue and destruction of its structures

The Global Occupational Health Network

ISSUE No. 12 - 2007



World Health Organization

GOHNET

GOHNET NEWSLETTER

ELIMINATION OF SILICOSIS



Silicosis is a form of *pneumoconiosis* caused by inhalation of crystalline *silica* dust, and is marked by *inflammation* and scarring in forms of nodular lesions in the upper lobes of the

lungs. Silicosis (especially the acute form) is characterized by shortness of breath, fever, and cyanosis (bluish skin). It may often be misdiagnosed as *pulmonary edema* (fluid in the lungs), *pneumonia*, or *tuberculosis*. The best way to prevent silicosis is to identify workplace activities that produce crystalline silica dust and then to eliminate or control the dust.

What you may not know is that the full name of this disease is a 45 letter word and the *longest word* in the *English language*: *pneumonoultramicroscopicsilicovolcanoconiosis*.

Newsletters and references for articles are posted on the Occupational Health website at: www.who.int/occupational_health/publications/newsletter/en/index.html.

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The ILO/WHO Global Programme for the Elimination of Silicosis (GPES)



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In the field of occupational health there are few risk factors and thus few health outcomes that can be completely eliminated at a global level. Silicosis is a positive exception. In countries like the US and in Europe where appropriate measures have been taken,

the incidence of silicosis has decreased dramatically. It is clear that to eliminate silicosis, the main focus has to be on prevention.

Silicosis is a well-known fibrogenic lung disease which is probably the most ancient occupational illness. Its prevention has a long history in the ILO and WHO. The First International Conference on Silicosis was convened by the ILO 75 years ago in Johannesburg, South Africa, to discuss prevention of silicosis that was highly prevalent in miners. The silicosis conferences organized by ILO during the last eight decades have greatly contributed to the advance of respiratory medicine around the world. They have always focused on important current issues, as reflected by the expanding conference themes and titles. In 1930, it was the International Conference on Silicosis; in 1950, it was the International Pneumoconiosis Conference. By 1992, it became the International Conference on Occupational Lung Diseases and by 1997, the International Conference on Occupational Respiratory Diseases (ICORD). The recent 10th ICORD (April 2005, China) has provided an excellent forum for deliberations on best practices for prevention and control of occupational respiratory hazards in the 21 century.

Despite all efforts to prevent it, silicosis still persists worldwide. This incurable disease affects tens of millions of workers engaged in hazardous dusty occupations in many countries. In 1997, the International Agency for Research on Cancer (IARC) classified

IN THIS ISSUE

The ILO/WHO Global Programme for the Elimination of Silicosis (GPES)	1
Elimination of Silicosis: The importance of preventing occupational exposure to dust	3
Silica-Related Disease: It's not just silicosis	6
Chronic obstructive bronchitis and emphysema in hard coal miners	7
Silicosis prevention program in Mutual de Seguridad, Chile	9
Elimination of Silicosis in the Americas	10
Launch of Silica Essentials	12
Silicosis and its control in small scale silica mills in India	12
National Program for the Elimination of Silicosis, Brazil (NPES-B)	15
GOHNET Newsletter - Contributors' Information	17
How to join GOHNET	19