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Silica, silicosis and tuberculosis

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SUMMARY

Exposure to crystalline silica dust causes multiple diseases, but silicosis and silica dust-associated tuberculosis (TB), in particular, are the two diseases that remain high on the list of occupational health priorities in low-income countries and that still occur in some high-income countries. The prevalence of silica-related TB is exacerbated by the human immunodeficiency virus (HIV) epidemic in low-income countries. This review describes the morphology of silica and the variable potency of the different forms. Sources of crystalline silica are discussed, with emphasis on less commonly recognised sources, such as small-scale mining operations and agriculture. Trends in the prevalence of silicosis are also presented. Although efforts have been made for many years in most countries to reduce silica dust levels, silicosis continues to occur even in young people. The clinical and pathological features and diagnosis of silicosis, with emphasis on chest radiography, are described. The high risk of mycobacterial infection in silica-exposed individuals is given particular attention, with emphasis on control. Treatment for latent TB is recommended. The management of silicosis and silica-associated TB, including monitoring for early detection of disease and surveillance to identify disease-causing workplaces, are discussed in detail. Prevention of disease, in the form of dust control, remains the focus of the World Health Organization and International Labour Office Global Elimination of Silicosis Campaign. However, clinicians must be aware that silica-associated diseases will be around for many years to come.

KEY WORDS: silica; silicosis; tuberculosis

SILICA-ASSOCIATED DISEASES remain an important public health concern in the twenty-first century because crystalline silica is one of a handful of toxins that causes multiple serious diseases and increased mortality.[1] Exposure contributes to serious epidemics of tuberculosis (TB) in southern Africa and other low-income regions of the world, and silica exposure is still common in both low- and high-income countries. At least 1.7 million workers in the United States are potentially exposed,[2] and an estimated 119 000 are overexposed in some industries.[3] Many millions are exposed in low- and middle-income countries such as India (with about 3 million exposed workers[4]), China and Brazil. Workers in mining countries such as South Africa are particularly affected by silica-associated diseases.

Diseases associated with silica exposure include silicosis, a fibrotic nodular disease of the lung parenchyma, pulmonary TB, lung cancer, chronic obstructive pulmonary disease (COPD) and autoimmune and renal diseases. It is not surprising that the International Labour Organization (ILO) and the World Health Organization (WHO) have embarked upon a Global Elimination of Silicosis Campaign.[5]

This review focuses on sources of exposure to silica, silicosis and silica-associated TB, which is of particular concern in low-income countries, and aspects of management and prevention. Although the focus of this article is silicosis and silica-associated TB, silica is linked to a range of pulmonary and extra-pulmonary diseases (Table 1) which have recently been reviewed by the National Institute of Safety and Health (NIOSH)[2] and the American Thoracic Society (ATS),[7] and are com-

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Table 1 Diseases associated with respirable crystalline silica

Pneumoconiosis
Chronic silicosis
Accelerated silicosis
Alveolar lipoproteinosis
Progressive massive fibrosis (PMF)
Chronic bronchitis
Emphysema
Mineral dust airways disease (MDAD)
Mycobacterial disease
Pulmonary and extra-pulmonary tuberculosis
Non-tuberculous mycobacterial (NTM) disease
Lung cancer
Autoimmune diseases
Scleroderma
Systemic lupus erythromatosis
Rheumatoid arthritis
Renal disease

Adapted from *Murray and Nadel's Textbook of Respiratory Medicine* (Table 61.2), with kind permission from Elsevier Saunders.[6]

prehensively described in Parkes.[8] Silica-associated lung cancer is of concern in many countries but is not discussed here, as it has been reviewed recently.[9]

CRYSTALLINE SILICA

In the health context, silica usually refers to respirable crystalline silica dust, respirable dust being the fine particles, usually invisible to the naked eye and mostly smaller than 7 microns in diameter. Silica comprises an atom of silicon and two atoms of oxygen (SiO_2) uncombined with other elements, hence sometimes called 'free silica', i.e., free of other elements. Because silicon and oxygen are so abundant in the earth's crust, many minerals contain SiO_2 usually combined with other elements, often cations. These minerals are known as silicates, rather than silica, and are ubiquitous; even the varieties of asbestos are silicates.

There are six natural forms or polymorphs of free crystalline silica, most quite rare and produced by extreme heat and pressure.[8] By far the most common polymorph is quartz, a constituent of many sands, stones and rocks. Because of this, quartz is sometimes confusingly used as a synonym for crystalline silica. Besides quartz, tridymite and cristobalite are hazardous polymorphs of silica that may be present when high temperatures are used in industrial processes.[8]

Free silica in a non-crystalline structure is amorphous silica, a form less hazardous than crystalline silica; but heat and pressure can change amorphous silica to crystalline forms. For example, diatomaceous earth, used commercially as a filtering and insulating material, is amorphous silica formed by skeletons of diatoms which is converted to cristobalite on heating and milling.

Variability in respirable silica potency

Recent publications have considered factors that may affect the disease-causing potential of silica from different sources.[10,11] Differences in three cohorts of Chinese tin, tungsten and pottery workers suggest that silica dust characteristics may affect the risk of silicosis.[11] The United Kingdom's Health and Safety Executive (HSE) reviewed potency factors with regard to their ability to cause silicosis and summarised their conclusions in a respirable crystalline silica potency matrix.[10] The HSE concluded that extremely small particle size enhances potency, while wetting, ageing and aluminium-containing clay coatings reduce potency. For the practitioner, this means being on the alert for exposure settings producing very fine, freshly fractured, dry silica (high speed dry drilling of rock and sandblasting are good examples).

SOURCES OF EXPOSURE TO RESPIRABLE SILICA

Free crystalline silica (referred to as silica from now on) is so common in the earth's crust that whenever the surface of the earth is disturbed—for example during mining or when sand, stones or rocks are drilled, moved, crushed or processed—potentially hazardous exposure may occur, particularly when dust suppression measures are inadequate.

Due to its crystal structure, quartz is resistant to being broken into smaller and smaller sizes, and thus the percentages of silica in bulk samples of materials are often higher than in the fine respirable dust. The percentage of silica in the unworked raw material may therefore be a misleading measure of risk; the respirable levels of silica need to be determined. In general, though, the greater the mechanical forces applied, the more likely is the generation of respirable dust.

A surprisingly large number of industries generate respirable silica dust, as shown in Table 2. A comprehensive description of exposures is available from several sources,[2,8] but the most common exposures occur in mining and mining-related occupations, such as milling ores, quarrying, tunnelling and excavation. Country rock, i.e., the rock in which the mined mineral is located, often determines the silica risk. Hence, coal mining, for example, is a silica risk in some regions. Industries with well known silica risks include ceramics, construction and foundries. Occupations associated with furnace masonry, stone-working or cutting (e.g., monumental masonry and working tombstones), cutting and polishing gem stones and those in which fine silica materials are used (e.g., in fillers and abrasives) have a long history of producing silica-associated disease. Abrasive blasting with sand, which has widespread applications in engineering, shipbuilding and in the metal and automotive repair industries, is particularly dangerous, and recommendations have been made to ban sand blasting in some countries.[12]

Small workplaces and those with less well known uses of silica remain of concern because, even in high-income countries, there may be a relative lack of awareness of the hazards of silica outside the occupations traditionally associated with silicosis. This contention is supported by data from the USA showing that recent

Table 2 Sources of exposure to respirable crystalline silica

Sources of exposure	Comment
General	
Moving, drilling, working, processing, crushing or mining sand, stones or rocks	Free silica content in respirable fraction of dust determines risk
Mining and related activities	
Mining and milling	Country rock* an important determinant of risk. Gold, coal, tin, copper, mica, uranium, crocidolite, iron, important in some regions
Small-scale mining	Under-researched, but exposure may be high
Mining related	Quarrying, tunnelling, excavating, digging wells and boreholes. Country rock and mineral determines risk. Quarrying granite, sandstone, flint, quartzite, shale and slate may produce high levels of quartz. Potency of silica may be reduced in some clays
Major industrial sources	
Foundry	
Ceramics	Pottery, tiles, brick and refractory articles
Glass manufacture	
Furnace masonry	Cutting, grinding, etc., refractory articles
Construction	Cutting, grinding, etc., concrete, tiles or bricks. Digging foundations
Stoneworking and monumental masonry	Making, cutting, abrasive polishing, etc., of tombstones, billiard tables, slate pencils, cladding and surfaces, including granite counter tops
Abrasive blasting with sand (sandblasting) or siliceous material	Very high exposures common. Usually cleaning or preparation for coating of metal pieces, but also unusual applications, e.g., sandblasting jeans
Minor industrial sources	
Fillers and scourers	Fine silica may be used for fillers in paints, coatings, plastics, rubber, explosives, dental supplies, etc., or in scouring materials (such as cleaning agents and those used for polishing flour) or grinding materials
Jewellery	Cutting, buffing, etc., semi-precious gems
Diatomaceous earth	Calcined material contains cristobalite
Craft work	Stone carvers, sculpture, pottery. Cases unusual unless frequent exposure, e.g., most working days
Less well established sources	
Agriculture	High levels of quartz exposure possible in farming sandy soils but silica-associated disease rare
Non-occupational	Frequent exposure to sand storms

* Country rock = rock hosting the mineral or being mined. Silica content varies from location to location, even within a mine.

silica-associated deaths in younger subjects have occurred after exposure in the construction and manufacturing sectors, with none from mining.[13]

Agriculture

Although listed in some publications as an activity associated with silica exposure,[2,14] agriculture is not widely recognised as a significant risk for silica-associated diseases. Although good studies show that farming activities in sandy soils can produce respirable silica levels well above generally accepted safe levels[15] and that sandy soils are commonplace in drier regions of many countries, silica-associated diseases are rarely reported in agricultural workers. The paucity of cases may be because silica potency is reduced by other soil components such as iron oxides and clay silicates. In addition, silica in soils is likely to be aged, with most farming activities producing little freshly fractured material. Under-diagnosis is also a possible explanation. Cases of silicosis have been reported, however,[16] and silica exposure in farming deserves further research.

Construction

Building and related work have become prominent considerations for silica exposure. Cement is usually low in silica, but cutting, grinding or drilling building materials containing sand and stone, such as concrete, brick, granite and tiles, will generate respirable quartz, as will digging deep foundations, especially in confined spaces.[17] Many construction jobs carry a risk of excessive silica exposure and silica-associated diseases.[18] The risk is reduced when the percentage of time spent grinding, cutting or drilling concrete or building materials is low.[19]

Small-scale mining

Small-scale mining is on the rise in many low-income countries, employing an estimated 13 million people in 1999,[20] but little is known about silica exposure in this setting. Despite being typically labour-intensive with low levels of mechanisation, silica levels can be high, obviously depending on the country rock, as shown in small-scale mining in Tanzania.[21]

New and unusual applications

Silica-containing materials are plentiful and relatively cheap, and new and unusual applications are to be expected. Consequently, exposure is found in unexpected occupations, as illustrated by recently described cases of silicosis from sandblasting jeans with silica-rich sand,[22] exposure to fine dust from heat-dried mud used in Tatami mat manufacturing in China,[23] and dental supply factory workers using quartz- and cristobalite-containing fillers in the USA.[24]

Non-occupational exposure

Unlike asbestos, there is little evidence that low-level exposure to silica causes adverse health effects, but this does not mean that significant non-occupational exposures do not occur, as dangerous levels of silica can be encountered in the environment and domestically. Silicosis, including advanced disease, has been found in Himalayan villagers exposed to frequent dust storms,[25] in women hand-milling corn in South Africa (hut lung)[26] and in children living among stonecutters in India.[27]

SILICOSIS

Pathology

The pathological features of the different forms of silicosis are well described in a number of texts, such as *Murray and Nadel's Textbook of Respiratory Medicine*[6] and *Pathology of Occupational Lung Disease*, edited by Churg and Green.[28] Chronic (nodular) silicosis, the most common form of the disease, occurs after 10–20 years of exposure to silica dust. Silicotic nodules (Figure 1), which first develop in the hilar lymph nodes,[29] are the distinguishing feature. In the lungs, the nodules initially involve both upper lobes (Figure 2) and measure around 3 mm in diameter. As disease progresses, they may be found in the mid and basal zones. Nodules can also be found in the visceral pleura. Each nodule has a well-demarcated central area of whorled hyalinised collagen fibres, with aggregates of dust-laden macrophages at the periphery; the intervening lung parenchyma is normal. Using light microscopy, birefringent particles can be seen in the nodules.

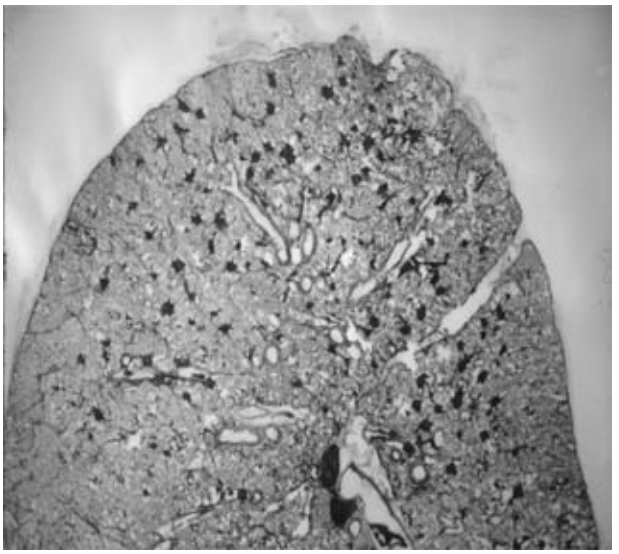

Figure 1 Whole lung section with chronic silicosis. Small circumscribed nodules are seen in the upper zone.

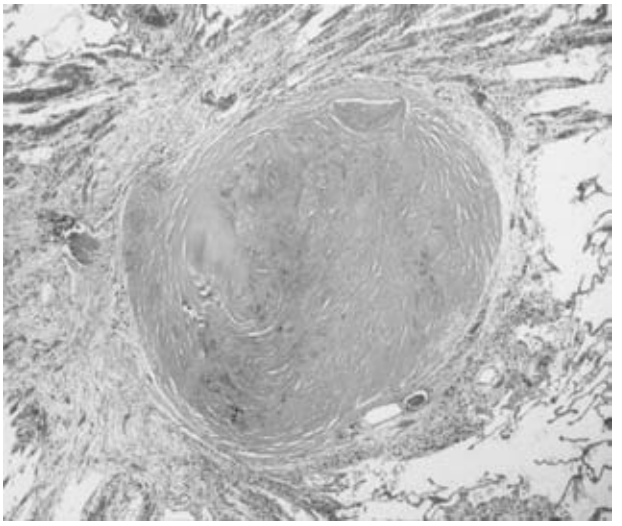

Figure 2 Silicotic nodule characterised by a central zone of hyalinised collagen with a whorled appearance and peripheral dust-containing macrophages.

With increasing duration and intensity of exposure, the nodules in the lung become confluent, obliterating normal intervening lung parenchyma and resulting in progressive massive fibrosis (PMF). The lesions may reach many centimetres in diameter and completely efface the upper lobes.

Accelerated silicosis occurs after 3–10 years of exposure. Although the changes are similar to those seen in chronic silicosis, the nodules develop sooner and are more cellular than fibrotic in nature.

Alveolar lipoproteinosis is uncommon and develops after intense short exposures, sometimes only months, to fine dust with a high silica content.[7,28] The alveolar spaces become filled with granular lipoproteinaceous material, comprising alveolar surfactant. If not too small, silica particles may be seen in this lipoproteinaceous material.

Duration and intensity of exposure are not the only determinants of the pathogenicity of silica dust. Host factors influence individual susceptibility, and smaller particles increase the fibrogenicity of the dust. The processes of inflammation and fibrogenesis induced by silica dust are well described by Donaldson.[30]

Silicosis is often progressive even without further exposure,[31,32] and may first present radiologically well after exposure has ceased. In a cohort study of gold miners, 57% of those with silicosis developed the disease after exposure ceased, on average 7.4 years after, but some after more than 20 years.[33]

Clinical features

Chronic silicosis is often a radiological diagnosis, the silicosis itself not producing symptoms. Exertional dyspnoea may occur if silicosis progresses to PMF, or if TB, lung cancer or COPD develops. A productive cough may be present and is usually due to chronic bronchitis; it may, however, be due to lung cancer or TB. Clubbing or systemic symptoms, such as fever and weight loss, should be attributed to lung cancer or TB until proven otherwise. The clinical features of accelerated silicosis are similar to those of chronic silicosis, but develop sooner. Patients with acute silicosis may become disabled within months of exposure, with clinical features similar to alveolar proteinosis of other aetiologies.

Chest imaging

Chronic silicosis is characterised by symmetrically distributed, small (usually 1–3 mm) rounded opacities, initially in the upper zones of the chest radiograph[34] (Figure 3). However, these opacities may occasionally not be visible on a chest radiograph, even in advanced silicosis determined by histology.[35] The ILO has developed standard chest radiographs and guidelines to assist with the reading of the radiographs of pneumoconioses.[34]

PMF is defined by opacities larger than 1 cm (Figure 4); they usually appear bilaterally in the upper zones and can be very large, occupying most of both lung fields. Over time, the fibrotic masses contract towards the mediastinum, leaving hypertranslucent zones at their margins. With the development of PMF, discrete small rounded opacities may disappear, making the diagnosis of silicosis more difficult.

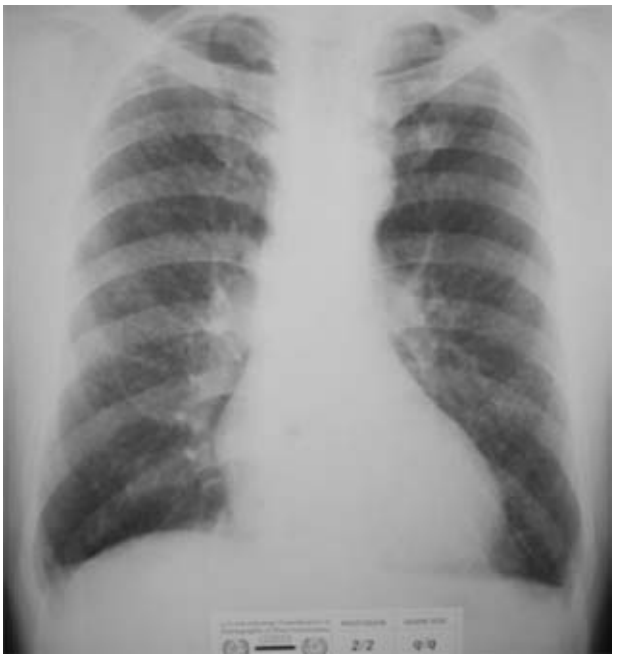

Figure 3 Typical 1.5–3 mm in diameter rounded opacities of chronic silicosis predominantly in the upper zones of the lungs. The radiograph is from the set of International Labour Organization (ILO) standard films. International classification of radiographs. Reproduced with the kind permission of the ILO. Copyright © 2000 International Labour Organization.

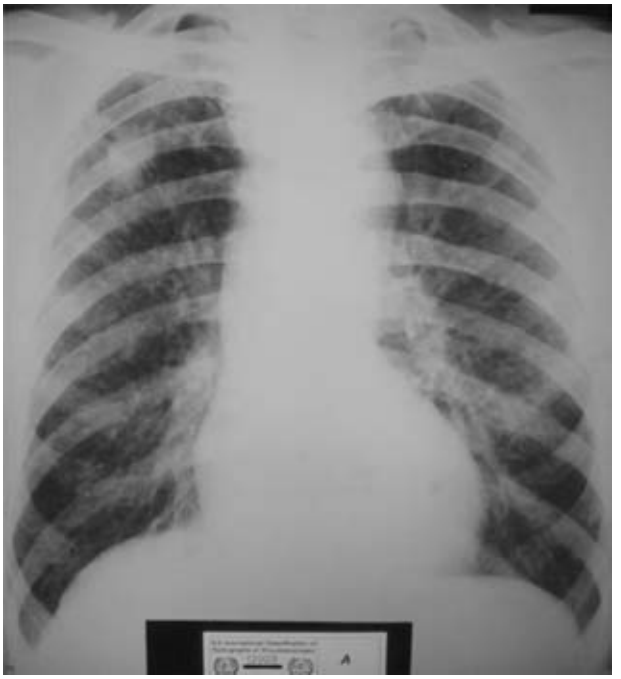

Figure 4 A massive opacity in the right upper zone of the lung. Massive opacities may be much larger and bilateral. The radiograph is from the set of International Labour Organization (ILO) standard films. International classification of radiographs. Reproduced with the kind permission of the ILO. Copyright © 2000 International Labour Organization.

Somewhat surprisingly, high resolution computed tomography (HRCT) has not been shown to be consistently more sensitive than chest radiography in detecting early silicosis,[36] but is useful in detecting lymph nodes, emphysema and accompanying pathology, e.g., of the pleura. HRCT should be reserved for cases with unexplained significant symptoms or atypical radiological features.

Lung function

In chronic silicosis, spirometric tests are often normal in early disease. As disease progresses, airflow limitation may develop,[7,32] but it is likely that much of this is attributable to associated emphysema[37] or TB.[38] Occasionally, even in the absence of radiological silicosis, there may be airflow limitation.

Diagnosis

Silicosis is diagnosed on the basis of a history of exposure, the characteristic radiological features and exclusion of other conditions; the differential most commonly includes TB, sarcoidosis and histoplasmosis. Lung biopsy is rarely necessary, but may be required to distinguish PMF from lung cancer or TB.

A history of silica exposure is necessary to make the diagnosis, and this is usually available because

most often long-term exposure, typically over 20 years, in one of the common dust-generating industries has preceded disease onset. In lower-income countries or uncontrolled environments, exposure may be shorter and disease onset more rapid. However, an exposure history may be difficult to ascertain—exposure may have occurred many years ago, or be forgotten, short or in an unusual occupation. Consequently, a thorough and comprehensive occupational history is necessary in patients with typical radiological features.

For diagnosis and to assess current risk, it may be useful to objectively evaluate silica exposure. Airborne concentrations of agents below which most workers are safe from adverse health effects are generally known as occupational exposure limits (OELs). OELs exist for quartz, cristobalite and trydimite, and are expressed as the weight of respirable material per cubic metre of air (mg/m^3) averaged over a shift or substantial part of it. In theory, then, provided respirable silica dust has been measured, it should be possible to ascertain whether or not workers have been over-exposed, but practitioners need to be alert to analytic and interpretative issues, some of which are listed in Table 3.

Table 3 Considerations in interpreting workplace air concentrations of respirable silica and occupational exposure limits (OELs)

OELs are occupational standards. OELs are mostly 8-hour standards, but environmental exposure may be for much longer and populations often include the young, the elderly and other vulnerable groups.

Occupational exposure is highly variable. Production rates, varying concentrations of silica in raw materials and country rock and variable ventilation and wetting (natural and engineered) greatly affect air levels of respirable dust. 8-hour shift concentrations have been estimated to vary between 3- and 4000-fold.[39]

Free crystalline respirable silica must be measured. Silica (SiO_2) combined with other elements is present in many minerals.

Current concentrations may underestimate past exposure. Dust control improvements over time are not unusual.

Measurement error is to be anticipated. Very small amounts of dust and silica are collected for analysis: respirable quartz OELs are typically at or below 0.1 mg/m^3 and these concentrations are often close to detection limits for most laboratories. Experienced, quality-assured occupational hygienists and laboratories are needed for reliable measurement.

Some OELs have been greatly reduced in recent times. For example, an OEL known as the threshold limit value (TLV)* was 0.1 mg/m^3 for respirable quartz in 1999 and 0.025 mg/m^3 by 2006. Many OELs are not protective against silica-associated diseases.[1]

Visible dustiness may be misleading. Respirable dust is very fine: extremely dusty operations may have little respirable dust and ostensibly clean processes may be 'dusty'.

* TLV is an exposure guideline of the American Conference of Governmental Industrial Hygienists.[40]

Trends in the prevalence of silicosis

As sources of silica are so common, disease potential is high. Nevertheless, the prevalence of silicosis has been decreasing in some high-income countries such as the USA,[41] and some, such as Sweden, have virtually eliminated significant exposure.[42] In contrast, lower-income countries have large at-risk populations, poor capacity to control dust and to enforce legislation and standards, and many competing public health needs, traumatic injury and infectious diseases among them. Consequently, silicosis is still common. During the period 1991–1995, China recorded more than 500 000 cases of silicosis, with around 6000 new cases and more than 24 000 deaths occurring each year.[43] Up to 20% of older, in-service gold miners in South Africa have silicosis,[44] and higher prevalences are found in ex-miners.[45,46] High prevalences have also been reported in Chinese pottery workers and tin and tungsten miners,[11] as well as in certain Brazilian workforces—about 54% of stone carvers.[47] Serious forms of the disease are common in high-exposed groups[48] and occur even in young workers with short exposure.[22] Cross-sectional studies will underestimate eventual disease burdens, well shown in southern African gold miners (Table 4), and former workers need to be monitored even if exit medicals are negative.

SILICA-ASSOCIATED TUBERCULOSIS

The association between silicosis and TB has been known for a long time, but more recent findings show that exposure to silica, without silicosis, may also predispose individuals to TB.[49–51] The increased risk of both pulmonary and extra-pulmonary TB is lifelong even if exposure ceases.[50] TB rates in subjects with advanced simple silicosis in high background TB settings can be very high, up to three-fold higher than those in the same workforce without silicosis,[49] and

Table 4 Proportion of gold miners in South Africa with silicosis, by study design and vital status

Authors	Study design	Study population	Diagnostic tool	Rate of silicosis %
Churchyard et al., 2004[44]	Cross-sectional	Employed miners (>37 years)	Chest X-rays	18.3–19.9*
Steen et al., 1997[45]	Cross-sectional	Living ex-miners	Chest X-rays	26.6–31.0*
Trapido et al., 1998[46]	Cross-sectional	Living ex-miners	Chest X-rays	22.0–36.0*
Murray and Hnizdo, 2005 (personal communication)	Cohort study	Deceased gold miners	Autopsy	51.6

* Radiological reader-dependent.

the risk increases with severity of silicosis. The risk is considerably higher in patients with acute and accelerated silicosis.[7] The combined risks of silicosis and HIV infection are multiplicative, with the result that TB remains as much a silica-related occupational disease in non-HIV-infected as in infected miners.[52] Pulmonary TB rates can be extremely high in silica-exposed groups with high background population TB and HIV rates, as is the case in South African gold miners with pulmonary TB rates of 3000 per 100 000.[53]

Non-tuberculous mycobacteria (NTM) may account for a large proportion of the mycobacterial disease in some populations,[7,54,55] *Mycobacterium kansasii* being the most common type. The clinical features and treatment of NTM lung disease are discussed in a recent review.[56]

An integral component of the management of silicosis is the control of mycobacterial disease. New onset radiological findings or the presence of typical features of TB such as persistent cough, haemoptysis, weight loss and fever should be pursued to exclude the infection. In general, the diagnosis can be made on sputum smear and culture, but active disease may be more difficult to detect in the silicotic than in the non-silicotic subject. In many instances, it is the chest radiograph rather than clinical features that gives the first indication of TB in the presence of silicosis. Periodic radiographic screening has been shown to be as effective as, if not more than, sputum examination for the early detection of TB, but the radiological features may be subtle.[57] Therefore, detection is enhanced by comparing sequential films in good reading conditions.

Individuals with active pulmonary TB respond well to directly observed rifampicin-based short-course chemotherapy even in the presence of HIV infection.[58] There is evidence of an increased case-fatality rate,[59] but silicosis and silica dust exposure are not risk factors for recurrence, relapse or reinfection and drug susceptibility.

Treatment for latent TB infection in people with silicosis (especially those with HIV infection) is recommended[7] and may be warranted even in those without silicosis, given the high risk associated with long periods of silica exposure even in the absence of radiological evidence of silicosis.[49–51] A recent study of HIV-infected gold miners with a high prevalence of silicosis reported a reduction in TB incidence of 38% overall, after routine isoniazid (INH) preventive therapy.[60] Screening for latent TB can be problematic. In countries with low background rates of TB, tuberculin skin tests are used to diagnose latent TB, after which a 9-month course of INH is recommended.[61] However, in countries with high background rates of TB, tuberculin skin tests do not accurately and reliably diagnose latent TB infection. Interferon-gamma assays are promising alternatives to these tests.[62] In these cases, it is advisable to offer treatment to everyone with silicosis and/or long silica dust exposure, but care must be taken to exclude individuals with active TB. Treatment uptake and adherence need to be considered before instituting treatment for latent TB.

Although active case-finding other than household contact screening is not indicated for TB control in most settings, this is not the case for silica-exposed populations.

Early identification and treatment of active TB not only limits disability in the individual, but also protects silica-affected individuals and assists in controlling spread to co-workers and the community (see Management, below).

MANAGEMENT

As silicosis is incurable, the management goals are to detect early cases of silicosis and TB through monitoring of both currently and formerly exposed workers; to establish surveillance programmes; to slow progression; to prevent TB; and to reduce disability.

The interaction between silica exposure and smoking in the development of TB, lung cancer and COPD[37] makes it particularly important to implement smoking cessation programmes in the workplace.

Monitoring

Baseline, periodic and exit questionnaires, physical examination, chest radiography and spirometry are the usual monitoring tools.

Removal from any further occupational exposure to silica is generally recommended after the diagnosis of silicosis, but in many poorer countries this would result in job loss and prolonged or permanent unemployment, often without unemployment benefits. In these cases a more flexible approach is necessary. A considered approach may be based on current dust levels—it is unclear that low levels of exposure increase disease progression significantly;[31] the feasibility of improving dust control in the current job, even if some time is required; the possibilities of job modification (the most dusty tasks could be re-allocated); and the proximity of retirement, including early retirement (continued exposure for a few years at lower levels may be less harmful than job loss). If immediate relocation to a low-exposure job is not feasible, affected workers can be retained in service but prioritised for relocation as positions may become available over time. Of course, decisions should only be made in consultation with a fully informed patient and recorded in patient notes.

Apart from reporting cases, health care workers should monitor disease rates and maintain record keeping for many years, given the latency of the diseases.

Surveillance

Surveillance systems able to identify disease-causing workplaces for targeted intervention are important aspects of prevention. Public health officials need to

respond to newly identified cases of silica-associated disease at the enterprise level, ideally by assessing current dust control measures in the source workplace and by medical evaluation of current and former co-workers, as appropriate. Surveillance can take many forms, from voluntary reporting of new cases by doctors to a national register or follow-up of workers' compensation claims, but, at least in parts of the US, hospital discharge records identify the greatest number of cases and problem worksites and are the most cost-effective approach.[63] Usually, cooperation by diagnosing doctors is necessary for a successful system.

Wagner comprehensively reviewed screening and surveillance of workers exposed to mineral dust,[64] and the American College of Occupational and Environmental Medicine (ACOEM) more recently published an evidence-based statement.[65]

Key questions in designing surveillance programmes are whom to include in surveillance and how often to perform the periodic evaluations. There are no universal answers, as the primary determinants will often be the risk of TB and the past and current concentrations of silica: higher risks and exposure prompt increased frequency of testing, up to 6-monthly in some settings.

If TB rates are high, periodic active case finding should be complemented by passive case finding: worker and management education on symptoms and benefits of early diagnosis; job protection on diagnosis; easy access to caring workplace services; and ambulatory treatment are all important. If silicosis is the major concern and respirable silica levels are below 0.05 mg/m^3, a baseline evaluation, a 1-year follow-up, then 3-yearly for the first 10 years and 2-yearly thereafter, is recommended by the ACOEM.[65] The WHO recommendation for routine periodic evaluations is less precise, every 2–5 years, but includes the comment: 'Ideally, health surveillance, particularly for workers exposed to silica dust, should be life-long'.[64]

In high HIV infection regions, the prevention of TB is especially important; workers leaving employment need to be re-informed of their life-long increased risk of silica-associated conditions even in the absence of extant diseases, and the need to inform attending health care professionals about their past exposure.

Compensation

Workers' compensation benefits in poorer countries may be critical in providing financial support for families and covering medical costs. Even in the absence of respiratory impairment, workers with silicosis may be excluded from many jobs due to an abnormal chest radiograph, so compensation benefits are generally provided.

The value of active case finding in former workers has been demonstrated in southern Africa.[45,46] Surveys of retired gold miners who had returned to rural labour-sending areas identified many undiagnosed and uncompensated individuals.

PRIMARY PREVENTION—DUST CONTROL

There is no cure for silicosis or other silica-associated diseases apart from TB. Under the guidance of the ILO/WHO global elimination campaign, a number of countries have established national silicosis elimination programmes, including some classed as low- or middle-income; South Africa and Thailand are examples. A comprehensive approach is required to prevent silica-associated diseases. Elements of these programmes include awareness, education, strengthening enforcement of standards, targeting priority industries for dust control, improved case finding and compensation.

Primary prevention of silicosis, the control of dust to concentrations at which disease will not occur—more comprehensively covered in industrial hygiene publications—is the optimum form of prevention and should be the overriding goal of national and workplace occupational health programmes. It may be cost-effective in both high- and low-income countries ($106–$109 saved per healthy year or disability adjusted life year).[66] Even if silica concentrations cannot be lowered to levels that will prevent all silica disease, it is important, for at least two reasons, to reduce concentrations to the lowest level that can be achieved. First, to prevent serious disease, e.g., rapid radiological progression and PMF have been associated with higher cumulative exposures and lung dust burdens, and acute silicosis is a consequence of intense exposure.[8,31] Second, even relatively short excursions over control limits, particularly if well above these limits, have been found to increase the risk of silicosis dramatically.[67]

Widely used current standards of 0.05–0.1 mg/m^3 have been shown not to be protective.[1] Greaves calculated that the risk of silicosis following a lifetime of exposure at 0.05 mg/m^3 is likely to be 20–40%.[68] The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended a threshold limit value (TLV) of 0.025 mg/m^3.[40] Attaining legislated levels is challenging, especially in low-income countries and in small enterprises, and even these low levels may not be protective against some silica-associated diseases such as TB.[1]

CONCLUSION

Silica-associated diseases can be prevented, as has been shown in Sweden and other high-income countries. However, even with a focus on primary prevention, silica-associated diseases with long latency will occur well into the future due to contemporary exposure. In low-income countries, new cases of silicosis and associated lung cancer, COPD and TB are likely for decades because reduction to very low concentrations of silica, necessary to prevent disease, will take time to

achieve, and protective standards have not even been established for some silica-associated diseases such as TB.

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RÉSUMÉ

L'exposition à la poussière de silice cristalline provoque de nombreuses maladies, mais la silicose ainsi que la tuberculose (TB) associée aux poussières de silice en particulier sont les deux affections qui restent fréquentes sur la liste des priorités de santé du travail dans les pays en développement et qui existent toujours dans certains pays développés. La prévalence des TB liées à la silice est exacerbée par l'épidémie de virus immunodéficience humaine (VIH) dans les pays en développement. Cette revue décrit la morphologie de la silice ainsi que la puissance variable de ses différentes formes. On y discute les sources de la silice cristalline en insistant sur les sources moins généralement connues telles que les opérations de mine à petite échelle ainsi que l'agriculture. Les tendances dans la prévalence de la silicose sont également mentionnées. Quoique depuis de nombreuses années on ait fait des efforts dans la plupart des pays pour réduire les niveaux de poussière de silice, la silicose continue à se développer, même chez les sujets jeunes. Les caractéristiques cliniques et anatomo-pathologiques ainsi que le diagnostic de la silicose sont décrits en insistant sur le cliché thoracique. Le risque élevé d'infection mycobactérienne chez les individus exposés à la silice fait l'objet d'une attention particulière avec insistance sur le contrôle de la maladie. On recommande le traitement de la TB latente. La prise en charge de la silicose et de la TB associée à la silice, y compris le suivi pour détection précoce de la maladie et la surveillance pour identifier les lieux de travail générateurs de maladies, font l'objet d'une discussion détaillée. La prévention de la maladie, sous forme de contrôle des poussières, reste l'objectif de la Campagne Mondiale d'Elimination de la Silicose conduite par l'Organisation Mondiale de la Santé et le Bureau International du Travail. Toutefois, les cliniciens doivent être conscients du fait que les maladies associées à la silice resteront encore présentes pendant de nombreuses années.

RESUMEN

La exposición a los polvos de sílice cristalina causa múltiples enfermedades, pero la silicosis y la tuberculosis (TB) asociada con silicosis en particular, son dos enfermedades que siguen ocupando un puesto de primer orden en las prioridades de salud laboral en los países en desarrollo y se presentan aún en algunos países desarrollados. La epidemia de infección por el virus de la inmunodeficiencia humana (VIH) ha exacerbado la prevalencia de TB asociada con silicosis en los países en desarrollo. En esta revisión se describe la morfología de la sílice y la potencia variable de sus diferentes formas. Se analizan las fuentes de sílice cristalina, con énfasis en aquellas menos conocidas, como las operaciones de minería en pequeña escala y las actividades agropecuarias. Asimismo, se presentan las tendencias de la prevalencia de silicosis. Si bien en la mayoría de los países se han realizado esfuerzos durante muchos años, con el propósito de disminuir las concentraciones de polvo de sílice, la silicosis sigue presente, incluso en personas jóvenes. Se describen las características clínicas y patológicas y el diagnóstico de la silicosis, haciendo hincapié en la radiografía de tórax. Se concede particular atención al alto riesgo de enfermedades por micobacterias en las personas expuestas a la sílice y sobre todo a su control. Se recomienda el tratamiento de la infección tuberculosa latente. Se expone en forma pormenorizada el tratamiento de la silicosis y de la TB asociada con silicosis, incluidos el seguimiento a fin de detectar en forma temprana la enfermedad y la vigilancia con el propósito de reconocer los ambientes de trabajo que generan enfermedad. La prevención de la enfermedad, mediante el control de la concentración del polvo, sigue siendo el principal objetivo del Programa Mundial de Eliminación de la Silicosis de la Organización Mundial de la Salud y de la Organización Internacional del Trabajo. Sin embargo, los médicos deben tener presente que las enfermedades causadas por la silicosis seguirán presentes durante muchos años.