Appendix

NSPS Scientific and Evidence Report - Silicosis in Australia

Silicosis is an entirely preventable, fibrotic occupational respiratory disease, which is specifically caused by inhalational exposure to silica dust. In recent years, there has been a major re-emergence of silicosis in Australia, particularly associated with the engineered (artificial) stone benchtop industry.

This report covers:

1.1 What is silica?

Silica or silicon dioxide (SiO2) is one of the most abundant minerals on earth, accounting for 60% of the mass of the earth's crust. Silica is the main constituent of more than 95% of the known rocks. Silica exists in both crystalline and amorphous forms. Inhalation of the crystalline form (e.g. quartz, cristobalite, and tridymite) is associated with a spectrum of occupational diseases.

Quartz is the most common type of crystalline silica and is a major component of rocks, including granite, slate and sandstone. Granite contains about 30% free silica, slate about 40% and sandstone is almost pure silica[1]. "Free" crystalline silica is unbound to other minerals. "Combined" forms of silica, called silicates, are compounds in which silica is bound to other minerals. Examples of silicates used in industry include asbestos, talc and kaolinite, a major component of kaolin (China clay).

Crystalline silica is highly toxic to the lungs, however particles must be small enough to penetrate deep into the lungs where they can do damage. These particles need to be less than 10µm in diameter, and this size is defined as "respirable". This form of dust is referred to as "respirable crystalline silica" or RCS.

1.2 Which workers are at risk of exposure to silica dust?

Occupational exposure to silica dust is one of the oldest known causes of lung disease[2, 3] and remains a risk in many Australian industries and workplaces[4, 5]. Recently, due to the identification of numerous workers contracting silicosis in the stone benchtop fabrication and installation industry, the very high risk of exposure to silica dust from engineered stone has become apparent[2, 3, 6-10].

Generally, workers are at risk of silica exposure if a dust is created from any material containing silica (such as mining and quarry work), or when silica is used in industrial and manufacturing processes.

Silica is used in an ever-expanding range of industrial processes and materials due to its inherent chemical properties but also its abundance and low cost. Examples of industrial uses include concrete, tiles, foundry casting, dental castings, and water filtration. Silica flour is an extremely fine, highly purified form of crystalline silica, which is still actively produced in Australia through a process of sand milling. Silica flour is used as an abrasive additive in soaps, skin care products, toothpastes and paints, and as a filler in paints, plastics and rubber, and a number of pharmaceuticals. The risk associated with silica flour production is notable and has been well established for many decades[11-13]. A paper published in 1981 described 23 / 86 workers at two southern Illinois silica mining and milling operations (producing silica flour) to have radiological features of silicosis, (26%) with simple silicosis and 7 (11%) with complicated silicosis (Progressive Massive Fibrosis - PMF)[12].

Due to the content of silica in the earth's crust historically, workers at risk of silicosis have been employed in occupations where silica is naturally encountered, such as construction, tunnelling, mining and quarry work. Workers are also at risk in industries where relatively low amounts of crystalline silica are present. One example is workers in the demolition sector who are exposed to relatively low silica containing dust (an average of 7%), and yet those exposures are routinely above the Workplace Exposure Standard. It is probable that workers in the demolition sector are exposed to silica at levels that would cause silica-related diseases but many of those workers are not captured in health monitoring programs at present[14-18].

Force is required to produce respirable sized crystalline silica particles. Activities where silica containing sand, ceramics, stone or rock is moved, crushed, milled, processed, drilled, polished, broken or cut have the potential to produce silica dust.

Between the 1970s and mid-2010s, industries most frequently associated with silicosis in Australia were foundries, brickworks/furnace construction, mining/quarries, and excavation/tunnelling[19, 20]. During that period, exposure of miners and construction workers to high levels of silica dust typically occurred when performing tasks with concrete and cement or working near crushers[3, 5]. Between 2000 and 2019, accepted silicosis compensation claims recorded by Safe Work Australia were predominantly in the manufacturing and construction industries (41% each), followed by mining (8%), and electricity, gas, water and waste services (5%), with other industries accounting for the remainder[21].

The Australian Work Exposures Study, a 2012 cross-sectional survey of the Australian working population aged 18-65 years, estimated that 6.6% of Australian workers were exposed to silica dust and 3.7% were exposed to high levels when carrying out tasks at work[5]. Occupations with the highest exposure, in order of mean exposure, were miners, construction workers, engineers, plumbers, handypersons, heavy vehicle drivers, farmers, machine operators, animal and horticultural workers, scientists, metal workers, and electrical workers. Those with the highest proportion of workers exposed to high levels of silica dust were miners, construction workers, plumbers, handypersons, engineers[5].

A complete, up-to-date understanding of which Australian industries currently expose workers to harmful levels of silica dust is lacking.

Table 1: Example occupations and tasks at risk of silica dust exposure	
Underground coal mining	Excavation
Surface coal mining	Earth moving
Hard rock mining	Drilling
Tunnelling	Clay and stone processing
Hydraulic fracturing of gas and oil wells	Cutting and laying pavers or tiles
Quarrying and stone cutting	Mineral ore-treating processes
Foundry work	Fabrication and installation of stone benchtops
Stonemasonry	Demolition
Blast furnaces	Abrasive blasting Agricultural earthworks
Steelworks	Hydraulic fracturing of gas and oil wells
Rolling and finishing mills	Pottery
Road construction and repair	Rock crushing
Building	Cement and concrete production
Construction	Brick manufacturing Paint manufacturing
Production or use of silica flour	Glass manufacturing
Dental prosthesis production	Ceramics production
Jewellery polishing	Teachers (ceramics)

1.3 The engineered stone benchtop industry

Silicosis has rapidly emerged as a major occupational disease among workers cutting and grinding stone for domestic kitchen, bathroom and laundry benchtops in Australia and worldwide[2, 3, 6-10, 22].

Engineered stone (also referred to as artificial, agglomerated or reconstituted stone and quartz conglomerate) is a very high silica containing material primarily used for the fabrication of household benchtops. Slabs of stone are manufactured by mixing finely crushed rock with a polymeric resin and pigments, then molded into shape and heat-cured[23]. The silica content of engineered stone slabs is typically over 90%, which is far higher than the natural stones which have traditionally been used to produce benchtops (marble 3% and granite 30% on average).

Engineered stone materials were introduced into Australia in the early 2000s and since then have become an extremely popular choice for benchtops. In comparison to marble, engineered stone is generally cheaper, is stain resistant and is available in a wide range of colours. By 2010, engineered stone accounted for 32% of all new benchtops in Australia, increasing to 45% in 2016[24].

Most Australian stone benchtop companies import slabs of engineered stone from overseas manufacturers. The benchtop fabricators will then process the slab of stone based on client specifications. Workers cut the slabs to the required size, create cut-outs for sinks and tap wear, and polish the final product. Commonly workers will use high powered hand tools such as grinders. Cutting and grinding engineered stone without water dust suppression (dry processing) has been noted to be an extremely common practice in the industry[22].

Since the increase in cases of silicosis from the engineered stone industry, studies have investigated the levels of silica dust produced when fabricating benchtops from engineered stone. A study performed by

the US National Institute of Occupational Safety and Health (NIOSH) noted that the dust generated during grinding of engineered stone had a mean silica content of 62%[25]. A study of personal exposure to silica dust to stone fabrication workers found exposures exceeded the Workplace Exposure Standard by orders of magnitude when using dry cutting methods, and exceeded the standard when using wet cutting methods also[26]. A small 2015 study noted that dry cutting of engineered stone generated levels of silica dust 150 times above the recommended limit of exposure for a 30 minute period[27]. Although there were limitations to this study, it also suggested that even with the use of water and local exhaust ventilation during cutting, the silica level was still double the recommended limit.

Other studies have indicated that engineered stone dust has particular properties which are likely to influence its toxicity. Both experimental and field studies have demonstrated generation of nanosized particles when working with engineered stone[28, 29]. A recent experimental study reported that in addition to the high concentrations of very fine particles (<1 um) emitted during cutting, that dust emissions from engineered stones had both larger surface areas and higher surface charge in comparison to natural stone[30]. The presence of very fine particles (<1 um) and nanoparticles is an important finding because it influences the effectiveness of control measures and is potentially associated with effects beyond the respiratory system such as autoimmune disease. The increased surface area and charge are also important factors relating to toxicity[30]. The types of tools used during fabrication and the composition of water used for dust suppression also influences the composition of the dust, such as the presence of a range of metals including iron, zirconium, titanium and aluminium[31].

Engineered stone also typically contains epoxy resins used as a binding agent. These resins may form a protective coating over freshly produced silica particles increasing their toxicity, and have also been described in association with other occupational lung diseases including asthma and hypersensitivity pneumonitis[31].

The surge in cases has highlighted inadequate dust control measures, ineffective health monitoring and insufficient enforcement of existing occupational health and safety laws in the engineered stone benchtop industry[3, 21].

1.4 What is silicosis and what are other diseases associated with silica exposure?

Silicosis refers to a range of fibrotic (scarring) lung diseases caused by the inhalation and deposition of RCS particles in the lungs. Although there are likely to be individual levels of susceptibility due to genetic factors, the cumulative dose of silica exposure (the level of silica dust exposure and exposure duration) is the most important factor in the development of silicosis[32].

Silicosis is one of the oldest known occupational diseases. The term silicosis was first used by Achille Visconti in 1871, however the association of silica containing dust and lung disease dates back to Hippocrates (460-375 BCE)[33]. In 1690 Lohneiss recorded that "... the dust and stone fall upon the lungs, the men have lung disease, breathe with difficulty and at last have consumption." In 1713 Rammazini described silicotic nodules in post mortems of stone cutters presenting with respiratory symptoms[33].

Some of the most tragic and wanton examples of occupational disease have been due to silicosis, such as the Hawks Nest, Gauley Bridge disaster in West Virginia (1931)[34]. This is considered to be one of the worst industrial disasters in American history. Congressional hearings placed the death toll at 476 for 1930-35, but some estimate 1000 deaths[34]. The tragedy brought recognition of acute silicosis as occupational lung disease and compensation legislation to protect workers. Australia is not absent of these tragic examples, with numerous men succumbing to silicosis from constructing our nations earliest sewer tunnels in the early 1900s[35].

There are three described forms of silicosis primarily related to the characteristics of occupational exposure and radiological characteristics:

1. Acute silicosis (also known as silicoproteinosis), associated with very high intensity exposure and may present within a range of weeks to five years from the time of initial exposure[36]
2. Accelerated silicosis, develops within ten years of moderate to high level exposure
3. Chronic silicosis, occurs after ten or more years typically at a low to moderate exposure dose.
   a) Chronic simple silicosis (also known as nodular silicosis): characterised by multiple discrete lung nodules 1-10 mm in size
   b) Chronic complicated silicosis (also known as PMF): coalescence of lung nodules causing conglomerated masses 10 mm or more in size.

Table 2: Forms of silicosis

Form	Duration of RCS exposure	Radiological features
Acute silicosis (silicoproteinosis)	Weeks to 5 years	• Numerous bilateral, multi-focal nodular ground-glass opacities • Areas of consolidation
Accelerated silicosis	Less than 10 years	• Variable features • May include combination of features of acute silicosis, simple silicosis and complicated silicosis
Chronic simple silicosis	Over 10 years	• Multiple discrete lung nodules 1-10mm • Predominance in upper lobes • Enlarged mediastinal/hilar lymph nodes, sometimes with calcification
Chronic complicated silicosis	Over 10 years	• Conglomerated masses (coalescence of nodules) over 10mm (PMF) • Upper lobe predominance • Distortion of surrounding lung parenchyma • Cavitation of masses • Emphysema • Pleural thickening • Enlarged mediastinal/hilar lymph nodes, sometimes with calcification

Acute silicosis (also known as silicoproteinosis) may develop within a few weeks to less than five years of high intensity silica exposure and presents with shortness of breath which may progress rapidly to respiratory failure and death[32]. The dose of exposure required to develop acute silicosis is poorly studied but thought to be in the order of 1-10 mg/m^3/year[37]. Acute silicosis has been described most commonly in sandblasters, but also in tunnel workers and silica flour processing[38-40]. Acute silicosis is a progressive condition with non-specific symptoms including dyspnoea, cough, fatigue, weight loss, fever, and pleuritic chest pain[33, 41]. Disease progression may be rapid and there is high associated mortality[33, 42].

Radiological features are limited to small case series and reports. Chest computed tomography (CT) findings include numerous bilateral centrilobular nodular opacities, focal ground glass opacities, and patchy areas of consolidation[43, 44].

Accelerated silicosis has historically been described as a more rapidly progressive disease compared to chronic silicosis. The presenting features however depend on the stage at which disease is identified. Initially there may be a similar pattern to simple silicosis, followed by more rapid development of coalescent masses, parenchymal distortion and fibrosis[32]. Silica exposure is usually of moderate to high intensity, over 2-10 years. Currently there is a gap in knowledge regarding the dose of exposure associated with accelerated silicosis. There may be an overlap with acute and accelerated silicosis, and both are associated with risk of developing PMF[33], even without ongoing exposure to RCS[45].

The radiological features of accelerated silicosis vary depending on the stage when the condition is identified and have not yet been well described. In the early stages of accelerated silicosis, plain chest x-ray may not detect diagnostic features. A study of Chinese miners noted 26.9% had a negative result on chest x-ray screening despite findings for pneumoconiosis on high-resolution computed tomography (HRCT)[46].

Chronic silicosis is the most common form of silicosis and develops following 10 years or more from the time of initial silica exposure. It has two forms: simple (or nodular) silicosis and complicated silicosis (or PMF). Chronic silicosis has typically been associated with long latency, often over 20 years, therefore may only become apparent after workers leave employment[36].

Large retrospective cohort studies demonstrate that the progression from simple silicosis to PMF occurs in 18-37% of workers over an average of five years[47, 48]. Ongoing silica exposure and smoking are significant factors that increase the risk of radiological progression from simple to complicated silicosis, as well as progressive loss of lung function[49]. Mortality is increased for those diagnosed at a younger age, the presence of conglomerate disease, history of smoking and co-existent tuberculosis[50].

Chronic simple silicosis is characterised by discrete nodules (up to 1cm in size), usually in an upper lobe predominant distribution on chest radiology. Patients may be asymptomatic although up to 70% of patients in some case series report exertional dyspnoea, chronic cough and sputum production[51].

Chronic complicated silicosis where silicotic nodules may coalesce to form conglomerate masses (>1cm in size) which is characteristic of PMF. Central cavitation may occur, leading to increased risk of mycobacterial infections[33]. Enlarged hilar or mediastinal lymphadenopathy may be seen in up to 75% of silicosis patients[52]. Significant distortion of surrounding lung parenchyma and peribronchial vessels occurs in PMF and increases the risk of spontaneous pneumothoraces. Pleural thickening is often present[53].

It may be difficult to differentiate radiological features from lung cancer, tuberculosis and sarcoidosis. Lymph node enlargement is common. Calcification may also develop within the conglomerated masses and in lymph nodes. Destruction of lung tissue results in impaired lung function and elevated pulmonary vascular resistance.

Clinically, patients may experience progressively worsening shortness of breath, low blood oxygen levels, respiratory failure and death. Following development of pulmonary silicosis, the disease may progress even after cessation of RCS exposure. Risk of progression has been associated with cumulative RCS exposure, younger age, lower lung function and larger size and extent of lung opacities on chest radiology at time of initial visit and follow-up duration[45, 54, 55].

Other conditions associated with silica exposure

In addition to silicosis, occupational silica exposure is associated with a range of other respiratory and non-respiratory conditions including autoimmune disease, lung cancer and pulmonary infections, as detailed in Table 3.

Internationally, one of the primary causes of morbidity and mortality in silica exposed workers is the development of Mycobacterium tuberculosis infection[56]. The risk of a patient with silicosis developing tuberculosis (TB) is higher (2.8 to 39 times, depending on the severity of the silicosis) than for healthy controls[56]. TB rates are extremely high in silica-exposed workers from regions with high background rates of TB and HIV. South African gold miners have pulmonary TB rates of 3000 per 100,000[57]. TB infection is associated with increased risk of silicosis progression and disease severity[57]. The effects of TB are also aggravated by HIV infection and smoking which commonly coexist, especially in developing countries, such as South Africa[32, 57]. Silica exposure increases the risk of TB, even without silicosis, and continues after exposure has ceased[57].

Clinical management can be difficult as the radiological features of silicosis and TB frequently overlap[58]. International guidelines recommend TB screening for silica exposed workers (especially over 10 years of exposure) and chemoprophylaxis for latent TB in the setting of silicosis[57].

RCS (a-quartz and cristobalite) has been classified as a human carcinogen by the International Agency for Research on Cancer (IARC) since 1997 and since that time there has been increasing evidence of a dose-response relationship[59-61]. An analysis of 65,980 workers from pooled cohort studies found

cumulative exposure, with a 15-year lag, was a strong predictor of lung cancer[61]. The estimated excess risk (through age 75) of lung cancer for a worker exposed from age 20 to 65 at 0.1 mg/m^3 RCS was 1.1-1.7%, above background risks of 3-6%[61]. There is a significant additive, and close to multiplicative, joint effect of silica exposure and cigarette smoking with lung cancer[40, 60, 62]. There is also increasing evidence that the risk of lung cancer can increase in those working with RCS in the absence of radiological features of silicosis[40, 60]. It has been suggested through animal models that initial inflammation induced by silica exposure is followed by the development of an immunosuppressive microenvironment that supports the growth of lung tumours[63].

Silica is associated with several autoimmune diseases such as systemic sclerosis (SSc), rheumatoid arthritis (RA), systemic lupus erythematosus and antineutrophil cytoplasmic antibody (ANCA)-related vasculitis[64]. Postulated mechanisms include increased levels of autoantibody production, immune complexes and excess production of immunoglobulin. A meta-analysis of 242 silica exposed RA cases indicated that disease risk for exposed individuals was more than three times that of unexposed individuals (3.43, 95% CI 2.25-5.22), and the risk appeared to be higher in males[65]. Although SSc is less common than RA, several studies have demonstrated an increased risk of SSc in association with silica exposure. A meta-analysis of 16 studies examined the association between SSc and occupational exposure to silica, noting a RR of 3.02 (95% CI 1.24-7.35) for males[66]. The risks in the three cohort studies included were extremely high, with over 15-fold higher risks[66]. Patients with silica-associated SSc had greater prevalence of anti-DNA topoisomerase 1 autoantibodies compared to those with idiopathic disease[67, 68].

Autoantibodies (blood markers associated with autoimmune diseases) have been noted to occur at higher frequency in silica exposed individuals without autoimmune disease compared to the general population[67]. Autoimmune disease can also occur with silica exposure, but in the absence of silicosis, suggesting that the development of fibrosis and nodular lesions may not be required for development of autoimmunity[67-69].

Table 3: Diseases apart from silicosis associated with RCS exposure[40]	
Disease	**Comment**
Other pulmonary conditions	
Lymphadenopathy	• With or without co-existent parenchymal silicosis • Calcification may be present
Chronic Obstructive Pulmonary Disease (COPD)	• Association with chronic bronchitis, emphysema and airflow obstruction • COPD association independent of smoking and silicosis[32, 70]
Pulmonary fibrosis	Case control studies have demonstrated association between silica exposure and pulmonary fibrosis[71, 72]
Sarcoidosis	May be difficult to differentiate from complicated silicosis
Lung cancer	• Close to multiplicative increased risk if co-existent smoking • May develop in absence of silicosis
Caplan's syndrome	Combination of rheumatoid arthritis or elevated rheumatoid factor with pneumoconiosis
Mycobacterial disease	
Pulmonary tuberculosis	2.8-39 times higher risk of TB, depending on silicosis severity
Autoimmune disease	
Rheumatoid arthritis	3 times increased risk compared to non-RCS exposed, especially males.
Systemic lupus erythematosus	Dose-response associations with increasing intensity or duration[73]
Scleroderma (Erasmus syndrome)	• Development of systemic sclerosis with or without silicosis • Risk Ratio (RR) 3.02 for males with RCS exposure • Greater prevalence of anti-DNA topoisomerase 1 autoantibodies
ANCA-associated vasculitis	Exposure to RCS associated with Odds Ratio (OR) 2.56 (95% CI 1.51-4.36)[74]
Renal disease	
Chronic renal disease	• Histopathology varies from focal to crescentic and necrotizing glomerulonephritis with aneurysm formation[72, 75] • May occur without pulmonary disease[76] • Suggested to be a result of a direct nephrotoxic effect by the microcrystalline silica particles and immune-mediated processes in the context of autoimmune disease[77, 78]

1.5 Engineered stone silicosis

The first cases of silicosis associated with engineered stone were reported in 2010 in three male Spanish workers in their 30s[79]. Silicosis in young workers following short exposure periods have since been reported numerous times subsequently[80]. In 2020, León-Jiménez and colleagues published details of 106 male workers with engineered stone silicosis. They reported a mean age of 36 years and an average of 12 years duration of engineered stone associated work. 28 workers had exposure of less than 10 years, consistent with accelerated silicosis[81].

The sentinel Australian case was reported by Yates and colleagues in 2015, and was a 54 year old Vietnamese refugee who developed complicated silicosis due to extremely poor working conditions[82]. Subsequently a case series described 7 male patients from various regions of Australia, with an age range as young as 26

(median 44 years)[8]. All were involved in dry cutting and ventilation typically relied on ceiling extraction fans or passive airflow through open doors. Only 3 were provided any respiratory protection, which was usually in the form of a disposable mask. None had previously been screened for lung disease through a health surveillance program. The findings demonstrate an exceedingly poor understanding of the risk and regulations associated with working with a high silica containing material since its introduction into Australia.

The Queensland and Victorian Governments have operated large scale screening programs open to stonemasons exposed to crystalline silica dust from engineered stone[22]. The Queensland program provided the first credible insight into the prevalence of disease in this industry. As of March 2022, the program has completed assessments of 1053 workers and identified 238 with silicosis, including 35 with PMF. This suggests a 23% prevalence of silicosis in this industry.

These enhanced assessments have demonstrated poor chest radiography and spirometry sensitivity for screening silica exposed stone benchtop workers. In Victoria, initial results indicated 23 of 65 (35%) workers with simple silicosis had "normal" chest radiographs (ILO category 0) but had consistent CT chest features[83]. Mean forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) percentage predicted values were noted to be over 80% for both simple and complicated silicosis[83].

Emerging evidence indicates engineered stone silicosis differs from silicosis acquired from working with natural stone, such as mining or quarry work, in particular a higher risk of disease progression. A small case series from Australia noted a rapid decline in lung function[8]. Over a median follow up of 16 months a decline in FEV1 of 386 mL/year (SD 204 mL) and FVC of 448 mL/year (SD 312 mL) was observed[8]. The largest cohort with engineered stone silicosis (106 patients) and the longest duration of follow up was recently reported from Spain[81]. Over a median follow up of 4 years there was an average yearly decrease in lung function of 83.4 mL FEV1 and 86.8 ml FVC. However, 25% of patients had an annual decrease of > 133 mL in FEV1 and > 157 mL in FVC[81]. After 4 years, 56% of patients had progressed two or more International Labour Office chest x-ray subcategories, and the number with PMF had increased from 7 to 40. Multivariable analysis showed that lower FVC at diagnosis and longer duration of exposure to silica were associated with progression to PMF[81].

In a recent study of 18 engineered stone workers with silicosis from China followed over 6-12 months, a subgroup of workers experienced similar rapid deterioration in lung function, with median declines in FEV1 of 625 mL/year and FVC 587 mL/year[84]. This study also noted a much shorter duration of exposure at diagnosis compared to patients with silicosis associated with natural stone work (6.4 vs 29.3 years, $P < 0.01$)[84].

1.6 What is the epidemiology of silicosis in Australia?

Although in many countries there has been a decline in silicosis-related mortality due to improved dust control measures over the last 50 - 100 years, silicosis remains a major public health issue internationally and outbreaks of severe silicosis continue to occur[85]. In 2017, the Global Burden of Disease (GBD) study identified 23,695 incident cases of silicosis (age-standardized incidence rate (ASIR) = 0.30 per 100,000), which represents 39% of the 60,055 incident cases of pneumoconiosis[86].

Internationally, silicosis is most prevalent in countries of low and middle income, where the burden is often under-reported because of poor surveillance. China has the most patients with silicosis, with more than 500,000 cases recorded between 1991 and 1995, and 6000 new cases and more than 24,000 deaths reported annually. The problem is particularly acute for workers in small-scale mines, who often have an accelerated form of disease. In the Brazilian gold-mining area in Minas Gerais alone, more than 4500 workers were reported to have had silicosis between 1978 and 1998. Of gold miners in South Africa dying from external causes (e.g., injuries, burns, poisoning, and drowning), proportions with silicosis identified at autopsy increased from 3% to 32% for black miners and from 18% to 22% for white miners between 1975 and 2007.

Accurate Australian data regarding the prevalence and incidence of silicosis is not available. Most data are sourced from workers' compensation claim statistics and cause-of-death statistics[3, 87]. As such, the full scale and impact of silicosis in Australia is unknown. However, available data indicate that numbers are increasing.

A 2003 review of 1467 compensated silicosis cases in New South Wales indicated a significant decline in incidence during the 20th century[20]. While 63% of the cases were compensated before the end of the 1960s, only 9% were compensated between 1979 and 2000[20]. Retrospective analysis of national

mortality data between 1979 and 2002 noted the crude mortality rates for silicosis showed a sustained decline, from 1.8 per million in 1982-1984 to 0.5 per million in 1997-1999[19]. An accurate understanding of the epidemiology of silicosis in Australia has however been limited, due to reliance on worker's compensation and mortality statistics.

In recent years there has been a major increase in the number of workers diagnosed with silicosis[8, 82, 83, 88]. In New South Wales for example, the annual number of certified silicosis cases increased from 9 in 2015-2016, to 107 in 2019-2020[89]. This surge has primarily been driven by the stone benchtop industry and handling of engineered stone, however almost half of the cases of silicosis reported to June 30 2021 were from industries outside of stone benchtop fabrication therefore highlighting the wide-spread nature of this issue[90, 91].

Following recognition of the initial cases of engineered stone silicosis, some Australian governments have offered enhanced screening for workers. As of March 2022, the Queensland program has completed assessments of 1053 workers and identified 238 (23%) with silicosis, including 35 with PMF. Similar results have been noted in Victoria with 108 workers confirmed to have silicosis during the first year of screening[92]. All Victorian workers diagnosed were male with a mean age of 42 years, 62% had been born in a country other than Australia[83] and 26% had worked in the stone benchtop industry for less than 10 years, consistent with the accelerated form of silicosis[83].

The National Occupational Respiratory Disease Registry (NORDR)

As part of the Australian Government's response to the re-emergence of silicosis, a National Occupational Respiratory Disease Registry (NORDR) is in development[90]. Occupational disease registries are well established overseas and provide valuable insights into causes and trends in occupational respiratory diseases, and are a means of early identification of new exposures or industries of concern. It is planned that the NORDR will require mandatory notification of all cases of silicosis by clinicians and encourage the voluntary notification of all other occupational respiratory diseases, such as work-related asthma. For an occupational respiratory disease registry to be effective it will require ongoing close engagement with stakeholders including physicians, workers, unions, occupational hygienists and government agencies. To assist in targeting and monitoring the effectiveness of interventions and prevention strategies, the registry will need to capture and report data on the numbers of new cases, causative exposures, industries of exposure, occupations and job tasks where the exposure occurred, and determine incidence trends[90].

It is anticipated the NORDR will initially focus on the medical specialties that are most likely to diagnose occupational respiratory diseases: respiratory and occupational physicians. It is expected that the NORDR will be operational by late 2022.

1.7 What are the health, social and economic impacts of silicosis?

Silicosis is irreversible and generally has a poor prognosis. The National Dust Disease Taskforce has described the impact of a diagnosis of an occupational respiratory dust disease 'on all aspects of people's lives' as 'terrible'[21].

Health impacts

Qualitative research undertaken for the National Dust Disease Taskforce among Australians with silicosis or other dust diseases, and their families, reported psychologically debilitating fluctuations in health from day to day[93]. People described experiencing breathlessness and weakness, chest pains, flu-like symptoms, and coughing, and significant concerns about their increased susceptibility to other conditions such as influenza or COVID-19[93]. Family members reported that, in the later stages of the disease, supplemental oxygen and mobility aids such as scooters and handles in the home were needed[93].

The report described significant adverse effects on mental health for the person with silicosis and their family[93]. Workers with silicosis reported fear and anxiety about their condition, their future, and their family, feelings of guilt about being unable to work, feelings of hopelessness about having a terminal illness and the lack of control over the prognosis, anger, sadness, boredom and frustration at having to rely on others for help[93].

Social impacts

Australians with silicosis and other dust diseases have described their uncertainty and inability to plan for the future as deeply debilitating[93]. Many reported damage to relationships due to their anger, frustration, and physical limitations such as being unable to play with their children or grandchildren[93].

Economic impacts

Australians with silicosis and other dust diseases participating in research for the National Dust Disease Taskforce have reported immediate termination from their job role, with few offered a replacement role by their employer[93]. The period of compensation was limited, as was assistance in finding other employment[93].

Those able to work are often unable to find alternative work[93]. National workers' compensation data show that respiratory system diseases were responsible for a sharp increase in lost working weeks between 2017-18 and 2018-19[94].

Safe Work Australia reported that, among workplace injury other than nerve and spinal cord injury, respiratory system diseases were responsible for the greatest cost increase (473%) from 2000-01 to 2018-19[94].

1.8 What measures are there to prevent silicosis?

Silicosis is an entirely preventable lung disease. Silicosis is caused by failure to protect workers from exposure to RCS dust.

In the stone benchtop sector, there have been numerous failures of control documented associated with the surge in cases. Some of these failures include the use of a high silica content material and the failure to identify the hazard of crystalline silica, failure to manage the risk of exposure, failure to provide training in the use, maintenance and storage of personal protective equipment, failure to provide the correct respiratory protective equipment, failure to conduct personal air monitoring and a failure to provide health monitoring for workers[95].

There are four key steps involved in managing risks to health and safety of workers. They are to a) identify the hazard, b) to assess the risks if necessary to understand the nature of the harm that could be caused by the hazard, how serious it could be and the likelihood of it happening, c) to control the risks, and d) to review the control measures to ensure they are working as planned[96].

Identifying the hazard for products that are procured by businesses, such as engineered stone, is performed through obtaining the Safety Data Sheet (SDS) as a tool to provide important information pertaining to composition, health risks and control measures. In the case of a complex mixture such as engineered stone, SDSs may be problematic in terms of specific information on overall health risks. A recent study compared empirically-determined mineral, metallic and organic resin content of 25 individual engineered products across six suppliers, with the corresponding SDS information[97]. Although the resin content for all engineered stone samples was within the SDS reported ranges for most suppliers, there was considerable variability in the crystalline silica content when comparing with supplier's SDS. Potentially toxicologically relevant metallic and mineral constituents were not reported. Some supplier SDSs were found to provide more information than others. Only one of the six suppliers provided crystalline mineral content other than silica, and only two suppliers provided any information about metals.

It has been reported that SDSs are not always available in the engineered stone sector, which presents a significant challenge[98]. Identifying the hazard for silica-containing products, whether they be procured or in-situ, without an SDS is significantly more difficult as it relies on the knowledge of the concentrations of silica in the various products used as part of the work.

Challenges also exist with risk assessment. There is no national requirement for persons to have obtained a specific competency in order to undertake a risk assessment for RCS. Therefore, it is possible for these to be conducted by persons with limited knowledge of the degree of risk. The flow on effect is that if the risk of exceeding the Workplace Exposure Standard is not appropriately assessed, then the requirements for exposure monitoring or health monitoring for crystalline silica as required under most work health and safety legislation would not routinely occur. Further, there is currently no standardised training required for workers to enable an understanding of the linkage between exposure to silica dust and their common sources, and the development of occupational illness and disease. The importance of education and

training to prevent silicosis, particularly around control measures, has been highlighted by findings that most early cases of engineered stone-associated silicosis occurred in small businesses with deficiencies in workplace control measures, including inadequate ventilation, lack of provision of appropriate respiratory protection, and 'dry-processing' of engineered stone[10]. Some workers had never heard of silicosis before their diagnosis[93].

A recent editorial noted that, 'Of the few reports on silicosis … most circle back to education as a tool in the fight against the disease.'[99].

The prevention strategies needed all have the same principles based on the hierarchy of control measures as a layered approach. There are some differences in the specific control measures used across different industries which take into consideration the differences in the source, the type of work environment and the type of work performed. Notwithstanding, relying on one control measure such as water suppression or personal protective equipment alone, does not adequately protect workers from the risk of RCS[6].

Hierarchy of controls

Aside from elimination of the source, a single combination of controls has not been shown to guarantee exposure below the current Workplace Exposure Standard when working with silica-containing materials. The ways of controlling risks are ranked from the highest level of protection and reliability to the lowest. This ranking is known as the hierarchy of risk control. Table 4 provides some common examples of measures as per this hierarchy[96].

Table 4: Hierarchy of controls in order of effectiveness for silicosis prevention

Elimination
• An import ban is under consideration for materials with very high silica content • Sometimes unfeasible, such as presence of silica in the mining, construction, tunnelling, and demolition environment due to it being naturally occurring
Substitution
• Substitute silica-containing materials with low or non-silica-containing materials • Use silica-containing materials that do not need to be cut, ground or polished • Design buildings with pre-built recesses so there is no/less need to cut or drill masonry • Use materials for production of benchtops with no or less silica
Isolation
• Enclosed room with restricted access for dust-generating activities (fitted with appropriate ventilation systems) • Enclosures around transfer points (crushers, conveyor systems, etc) • Enclosures around drop chutes and waste bins • Enclosed cabins fitted with filtration systems for heavy plant and machinery operators • Physical barriers between different workers • Shielding around automated processes • Designating a separate area for changing and amenities
Engineering controls
• Automated cutting, grinding and drilling • Wet-cutting methods • Dust suppression systems • Ventilation and dust capture systems

- Local exhaust ventilation to remove silica dust close to the source before it reaches the breathing zone of a worker
- On-tool dust extraction and filtration
- H-class industrial vacuum cleaners for cleaning

To note: Wet-cutting and exhaust ventilation may not sufficiently control silica dust levels to below the Workplace Exposure Standard alone

Administrative controls

- Obtaining the Safety Data Sheet for all materials used and informing and consulting with workers of the hazards and necessary controls to be applied
- Written rules and policies for handling silica-containing materials and disposing of waste
- Planning to minimise the number of cuts performed by workers that are necessary
- Shift rotation policies to avoid extended exposure
- Restricted area access policies
- Policies for wetting down dusty areas
- Regular housekeeping, including a prohibition of dry sweeping or dry cleaning methods
- Inspection and testing programs (e.g. regular inspection and testing of ventilation systems)
- Corrective action programs in circumstances where non-compliant monitoring results are reported
- Training in the hazards and risks of RCS, the health effects of exposure, the control measures needed to eliminate or otherwise minimise exposure, methods applied to monitor the effectiveness of control measures
- Supervision of workers
- Frequent cleaning and changing of uniforms to avoid contaminating non-work areas, providing washing and showering facilities
- Respiratory protection programs which include respiratory fit-testing for close-face fitting respiratory protection
- Clean shaven policies mandated for workers who are required to wear close-face fitting respiratory protection
- Exposure monitoring program
- Health monitoring program

Personal protective equipment

- Includes use of a well-fitting, appropriately selected respirator
- Close face-fitting respiratory protection with an effective face seal, correctly fitted and fit-tested

Sources: References[4, 6, 10, 21]

Monitoring exposure

Measuring workers' exposure to silica dust via air monitoring is necessary to establish compliance with the Workplace Exposure Standard and to determine if control measures are working as planned. Exposure monitoring to determine a worker's exposure involves measuring the level of silica dust in the breathing zone of workers using a personal sampler during their usual shift activities (including routine breaks) and is performed by an occupational hygienist[4].

If exposure monitoring demonstrates that a worker has been exposed to silica dust above the Workplace Exposure Standard there are existing legal requirements for that exceedance to be reported and acted upon in the resources sector[100]. However, work health and safety legislation, which covers most at-risk workers listed in Table 1 contains no such provisions.

Preventing worker exposure to silica dust (8-hour TWA of 0.05 mg/m^3)[6, 101] is difficult to achieve in some workplaces where workers handle materials with a very high silica content, if only one control measure is

used[10]. A layered approach to controlling exposure is needed which includes implementing all reasonably practicable controls in the above-mentioned hierarchy of controls.

Workplace Exposure Standard

The current national Workplace Exposure Standard for quartz (respirable dust) (in force from 1 July 2020 in most jurisdictions)[102] is a time-weighted average (TWA) of 0.05 mg/m^3[101], reduced from the previous standard of 0.1 mg/m^3. The TWA is based on exposure occurring over an 8-hour working day followed by 16 hours of no exposure, over a 5-day working week[6, 103]. A Workplace Exposure Standard is an airborne concentration that must not be exceeded.

However, the standard itself is contested, given evidence that a lower standard of 0.02 mg/m^3 has been reported to be optimal for health[21, 104]. Adoption of this lower exposure standard is supported by Cancer Council Australia[105] and by some jurisdictions. For example, WorkSafe Victoria recommends that employers take a precautionary approach and reduce workers' exposure to an 8-hour TWA average of below 0.02 mg/m^3 to prevent silicosis[106]. In contrast, the Australian Institute of Occupational Hygienists (AIOH) together with the National Association of Testing Authorities (NATA) have stated that there are known challenges in measuring silica dust levels at this low concentration consistently and accurately using currently available technology[107].

Despite exposure monitoring being the key way that businesses determine compliance with the exposure standard, very few businesses in the stone benchtop industry have undertaken the necessary monitoring[95].

1.9 How can workers be screened for silicosis?

In recent years, several jurisdictions in Australia have provided health screening programs for workers in the stone benchtop industry. These programs have been extremely important, in particular as a means to identify workers with silicosis (who are often asymptomatic) and provide them with appropriate management. The programs have also provided further knowledge about the benchtop industry in Australia and engineered stone silicosis. As an indication of the extent of silicosis in the benchtop industry, in March 2022 the Queensland program had completed assessments of 1053 workers and identified 238 (23%) with silicosis, including 35 with PMF.

The standard means of health monitoring of RCS exposed workers includes a standardised respiratory questionnaire, spirometry (FEV1, FVC and FEV1/FVC) and a chest x-ray (full PA view). Results from the first year of screening benchtop workers in Victoria however indicated mean spirometric parameters were well above 80% for workers identified to have silicosis, and 36% with simple silicosis were noted to have a normal (ILO category 0) chest x-ray[83]. These findings call into question the sensitivity of standard screening techniques for this industry. In January 2021, legislation was passed in Western Australia for low dose CT scan to replace chest x-ray as the radiological screening test for occupational exposure to silica.

The purpose of occupational health monitoring (or surveillance) is to detect an adverse effect from a workplace's exposure at an early, pre-clinical stage which allows interventions to protect the health of the worker. The identification of any worker with an adverse health effect is also an indicator of failure to control the exposure at the workplace, thereby putting other workers at risk and requires thorough review of prevention measures at the workplace.

A 'person conducting a business or undertaking' (PCBU), usually the employer, has a legal obligation under Work Health and Safety Regulations to monitor the health of workers where there is 'significant risk' to health because of exposure. RCS is identified under Schedule 14 of the WHS Regulations to be a hazardous substance requiring provision of health monitoring[108]. The PCBU has a duty to determine if significant risk is present and to decide if health monitoring is necessary. Significant risk decisions are made taking into consideration the likelihood of exposure to a hazardous chemical in conjunction with the known health effects of the chemicals.

Despite the prevalence of occupational RCS exposure in Australia and internationally, the evidence base supporting standard screening methods is limited, and outside of the stone benchtop industry, there has been little research of the role of modern lung function testing and radiological techniques, such as CT imaging.

Multidisciplinary meetings

Multidisciplinary meeting (MDM) discussion has been internationally recommended for the diagnosis and management of interstitial lung diseases (ILDs)[109, 110]. A review of the evidence showed MDMs improve inter-observer agreement, increases the proportion of high confidence diagnoses and reduces the frequency of unclassifiable diagnoses[110]. The typical constitution of a MDM for ILDs include respiratory physicians, radiologists and pathologists. In the setting of occupational respiratory diseases, such as silicosis, an occupational physician would have valuable input into an MDM discussion such as the suitability of return-to-work options. Some patients with silicosis may have associated health issues requiring involvement of other specialities such as rheumatologists and mental health professionals[111]. For patients with severe and progressive disease, the MDM discussion can assist with determining appropriateness of other interventions such as anti-fibrotic drugs, whole lung lavage and lung transplantation.

1.10 What are the treatment options for silicosis?

There is no currently available treatment to reverse silicosis. General management measures are similar to those for other chronic lung diseases, and include:

- Smoking cessation
- Vaccinations, including COVID-19, influenza and pneumococcal
- Screening for tuberculosis (TB) and consideration of treatment of latent TB
- Disease education
- Exercise programs
- Psychological support
- Consider enrolling in clinical trials, if available
- Have a plan to manage flare-ups of the disease
- Oxygen support or other ways to manage chronic respiratory failure, may be needed
- Regular review with general practitioner and respiratory physician
- Palliative care
- Lung transplantation: For those with near fatal respiratory failure, lung transplantation remains the only option. Currently approximately 200 lung transplantations are performed in Australia annually and the reported survival of bilateral lung transplant recipients at 1, 3 and 5 years is 90%, 74% and 68%, respectively[112]. Limited data suggests that survival in silicosis patients following lung transplantation was not reduced compared to idiopathic pulmonary fibrosis[113].

It is quite likely that after a diagnosis of silicosis, a patient will not be able to return to their pre-injury occupation, due to risk of further silica exposure. Following a diagnosis of silicosis, it is important to avoid any further silica exposure, which is likely to contribute to worsening of disease over time. This can be a very stressful experience for a patient and their family, and can be associated with significant disruption to income. Usually, a patient will submit a workers compensation insurance claim, that will be assessed by the relevant insurance agency.

The benefits of work to physical and mental health are well established. If a patient is physically and mentally fit enough, then the insurance agency or work, health and safety regulator may engage the worker in a return-to-work program. The treating doctor will play an important role in determining if a patient is fit enough to return to work and if any identified work options are suitable.

Other treatment options for silicosis that have undergone limited studies include:

- Whole lung lavage
- Immunosuppression
- Anti-fibrotic drug therapy.

Whole lung lavage

Given the pathological similarities with pulmonary alveolar proteinosis, whole lung lavage has been trialled for acute silicosis, and more recently for engineered stone silicosis[114, 115]. Whole lung lavage has been

demonstrated to decrease the presence of dust particles, macrophages, and cytokines on subsequent bronchoalveolar lavage (BAL) analysis, and may slow lung function decline in the short term[114]. The impact on long term outcomes or mortality is unclear[114]. There is however recent emerging evidence regarding the role of whole lung lavage specifically for engineered stone associated silicosis, which has indicated it to be a well-tolerated and potentially useful treatment for this condition[115].

Immunosuppression

Immunosuppression has also been trialled in chronic silicosis, with little success. Older studies[116] report a transient improvement in lung function with prednisolone but with no apparent sustained benefit or reduction in mortality. There is limited evidence that prednisolone reduces dyspnoea and cough, although benefits appeared to be associated with longer periods of exposure[116]. In a case series of patients with accelerated silicosis undergoing lung transplantation, it was observed that almost all were previously treated with prednisolone, which did not change the clinical course[117]. Infliximab reduced the histological inflammatory and fibrotic response in silicosis-induced rats[118], but this is yet to be translated into human studies.

Antifibrotic drug therapy

Given the relative success of the use of anti-fibrotic therapy in idiopathic pulmonary fibrosis (IPF), the use of nintedanib has been tested in experimental silicosis animal models[119, 120]. In March 2022, nintedanib was added to the Australian Pharmaceutical Benefits Scheme for people with progressive fibrosing interstitial lung disease, in addition to the existing listing for IPF. Other antifibrotic agents including tadalafil and relaxin are also being trialled in animal models[121, 122].

Cell-based therapy including bone-marrow derived mononuclear cell[123] and mesenchymal cell transplantation[124] have both demonstrated some benefit in animal models and are currently undergoing trials in humans[125].

1.11 What are the gaps in knowledge related to silicosis, and its prevention and management?

There are major gaps in the epidemiological understanding of silicosis, especially in Australia.

Reasons for these gaps include:

- Poor awareness of occupational lung diseases and complacency with regard to these diseases
- Lack of an operational National Occupational Respiratory Disease Registry (NORDR) that links with State-based registers
- Lack of an Australian air monitoring exposure registry
- Reliance on workers compensation and death certification data which has been repeatedly demonstrated to severely under report cases
- Delayed recognition of occupational respiratory diseases by medical professionals
- Delayed case identification due to deficiencies in occupational health monitoring programs and the use of low sensitivity methods.

Current evidence gaps regarding measures to prevent silicosis include:

- Level of compliance with health monitoring being undertaken by employers across all at-risk sectors, and the sensitivity of currently recommended health monitoring requirements, including effectiveness of screening
- Level of non-compliance with the current Workplace Exposure Standard for RCS, and industries where non-compliance is occurring most frequently
- Verification of the Workplace Exposure Standard for RCS of less than 0.05mg/m^3, and its evidence-based impact on workers' risk exposure
- Understanding of the differences in regulatory compliance activities in different Australian jurisdictions

- Understanding the level of employer and PCBU awareness and methods of implementation of WHS regulations specific to RCS exposure
- Understanding how employers and PCBUs determine if "significant risk" of employee exposure to RCS is present and decide if exposure monitoring and health monitoring is provided
- The adequacy of low levels of exposure monitoring techniques and technology, including their long-term viability
- Impact of the concentration of exposure versus intensity, e.g. the effect of ambient exposure, impact of exposure during longer shifts, impact of specific tasks, etc
- Understanding the frequency that education related to the risk associated with RCS exposure and appropriate control measures is provided to employees and technical college students
- Generation of behavioural insights to build evidence of the best ways to prevent or reduce behaviours that increase risk of workplace RCS exposure
- Research to determine how the generation of dust particles which are in the nanoparticle size range influences effectiveness of control measures (e.g. ventilation, use of water, respirators)
- Research into development of alternative low-silica content materials to substitute those currently in use, including materials currently available and new products under development
- Hazard of materials with different levels of silica, resin components, and other composites of engineered stone
- Research to determine the effectiveness of control measures when processing silica containing materials (e.g. ventilation, use of water, respirators)
- Further research to understand the toxic properties of dust generated from engineered stone, in addition to the presence of silica
- Additional strategies for improving dust control, uptake of control measures, safe work practices and WHS duties.

Current evidence gaps regarding the management of silicosis include:

- Research into optimal, modern health monitoring techniques (such as the role of CT imaging) to aid early detection of disease
- Development and validation of a protocol for Occupational Respiratory Disease specific multidisciplinary meetings
- Research into the longitudinal risk of disease development following hazardous RCS exposure, in particular workers from the stone benchtop industry formerly exposed to dry processing of engineered stone
- Incidence and prevalence of lung disease related to silica exposure, and level of impairment
- Factors which influence return-to-work, including review of return-to-work support services available to workers and how workers interact with these services
- The impact of non-respiratory adverse health outcomes associated with RCS exposure and silicosis including mental health and rheumatological disease
- Research into potential disease modifying therapies for silicosis, such as anti-fibrotic drugs
- Lived experience of affected workers and their families, including health, social and economic impacts.

Version: 1.6
Author: Dr Ryan Hoy
Co-Contributor: Kate Cole OAM
Approved by: Australian Government Department of Health and Aged Care, 23 August 2022

References

1. Lapp NL. Lung disease secondary to inhalation of nonfibrous minerals. Clinics in chest medicine. 1981;2(2):219-33.
2. Hoy RF, Chambers DC. Silica-related diseases in the modern world. Allergy. 2020;75(11):2805-17.
3. Hoy RF, Jeebhay MF, Cavalin C, Chen W, Cohen RA, Fireman E, et al. Current global perspectives on silicosis – convergence of old and newly emergent hazards. Respirology. 2022.
4. Safe Work Australia. Working with silica and silica containing products. Guidance material (February 2022): Safe Work Australia; 2022. Available from: https://www.safeworkaustralia.gov.au/doc/working-silica-and-silica-containing-products.
5. Si S, Carey RN, Reid A, Driscoll T, Glass DC, Peters S, et al. The Australian Work Exposures Study: prevalence of occupational exposure to respirable crystalline silica. Ann Occup Hyg. 2016;60(5):631-7.
6. Safe Work Australia. Managing the risks of respirable crystalline silica from engineered stone in the workplace. Code of Practice (October 2021): Safe Work Australia; 2021. Available from: https://www.safeworkaustralia.gov.au/doc/working-silica-and-silica-containing-products.
7. Barnes H, Goh NSL, Leong TL, Hoy R. Silica-associated lung disease: An old-world exposure in modern industries. Respirology. 2019;24(12):1165-75.
8. Hoy RF, Baird T, Hammerschlag G, Hart D, Johnson AR, King P, et al. Artificial stone-associated silicosis: a rapidly emerging occupational lung disease. Occup Environ Med. 2018;75(1):3-5.
9. Perret JL, Miles S, Brims F, Newbigin K, Davidson M, Jersmann H, et al. Respiratory surveillance for coal mine dust and artificial stone exposed workers in Australia and New Zealand: A position statement from the Thoracic Society of Australia and New Zealand. Respirology. 2020;25(11):1193-202.
10. Hoy RF. Artificial stone silicosis. Curr Opin Allergy Clin Immunol. 2021;21(2):114-20.
11. Johnson WM, Busnardo MS. Silicosis following employment in the manufacture of silica flour and industrial sand. J Occup Med. 1993;35(7):716-9.
12. Banks DE, Morring KL, Boehlecke BA, Althouse RB, Merchant JA. Silicosis in silica flour workers. Am Rev Respir Dis. 1981;124(4):445-50.
13. Nelson HM, Rajhans GS, Morton S, Brown JR. Silica flour exposures in Ontario. Am Ind Hyg Assoc J. 1978;39(4):261-9.
14. Cole, K, editor Controlling exposures to Respirable Crystalline Silica (RCS) in Sydney demolition workers. A Client-led intervention. Australian Institute of Occupational Hygienists Inc 37th Annual Conference and Exhibition Nov 30th - Dec 4th; 2019; Crown Convention Centre, Perth.
15. Flanagan ME, Seixas N, Majar M, Camp J, Morgan M. Silica dust exposures during selected construction activities. AIHA J (Fairfax, Va). 2003;64(3):319-28.
16. Kirkeskov L, Hanskov DJ, Brauer C. Total and respirable dust exposures among carpenters and demolition workers during indoor work in Denmark. J Occup Med Toxicol. 2016;11:45.
17. Normohammadi M, Kakooei H, Omidi L, Yari S, Alimi R. Risk Assessment of Exposure to Silica Dust in Building Demolition Sites. Saf Health Work. 2016;7(3):251-5.
18. U B, Böckler M, W C, D D, S G, H G, et al. Exposure to quartz at the workplace (BGIA-Report 8/2006e). Berlin: Institute for Occupational Safety and Health of the German Social Accident Insurance; 2008.
19. Smith DR, Leggat PA. 24 years of pneumoconiosis mortality surveillance in Australia. Journal of occupational health. 2006;48(5):309-13.
20. Berry G, Rogers A, Yeung P. Silicosis and lung cancer: a mortality study of compensated men with silicosis in New South Wales, Australia. Occup Med (Lond). 2004;54(6):387-94.
21. Australian Government Department of Health National Dust Diseases Taskforce. Health National Dust Diseases Taskforce. Final report to Minister for Health and Aged Care. Canberra: Australian Government Department of Health; 2021. Available from: https://www1.health.gov.au/internet/main/publishing.nsf/Content/ohp-nat-dust-disease-taskforce.htm.
22. Hoy RF, Glass DC, Dimitriadis C, Hansen J, Hore-Lacy F, Sim MR. Identification of early-stage silicosis through health screening of stone benchtop industry workers in Victoria, Australia. Occup Environ Med. 2021;78(4):296-302.

23. Caesarstone®. About Caesarstone® Quartz Surfaces Australia 2019 [Available from: http://www.caesarstone.com.au/about-us.
24. Caesarstone®. Caesarstone Company Overview. 2018 September, 2018.
25. Qi CL, L. Engineering Control of Silica Dust from Stone Countertop Fabrication and Installation. National Institute for Occupational Safety and Health; 2016.
26. Phillips ML, Johnson DL, Johnson AC. Determinants of respirable silica exposure in stone countertop fabrication: a preliminary study. J Occup Environ Hyg. 2013;10(7):368-73.
27. Cooper JH, Johnson DL, Phillips ML. Respirable silica dust suppression during artificial stone countertop cutting. Ann Occup Hyg. 2015;59(1):122-6.
28. Carrieri M, Guzzardo C, Farcas D, Cena LG. Characterization of Silica Exposure during Manufacturing of Artificial Stone Countertops. Int J Environ Res Public Health. 2020;17(12).
29. Ophir N, Bar Shai A, Korenstein R, Kramer MR, Fireman E. Functional, inflammatory and interstitial impairment due to artificial stone dust ultrafine particles exposure. Occup Environ Med. 2019.
30. Ramkissoon C, Gaskin S, Thredgold L, Hall T, Rowett S, Gun R. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. Sci Rep. 2022;12(1):4351.
31. Di Benedetto F, Giaccherini A, Montegrossi G, Pardi LA, Zoleo A, Capolupo F, et al. Chemical variability of artificial stone powders in relation to their health effects. Scientific Reports. 2019;9(1):6531.
32. Leung CC, Yu IT, Chen W. Silicosis. Lancet (London, England). 2012;379(9830):2008-18.
33. Greenberg MI, Waksman J, Curtis J. Silicosis: a review. Disease-a-month : DM. 2007;53(8):394-416.
34. Sherson D. Silicosis in the twenty first century. Occup Environ Med. 2002;59(11):721-2.
35. Report of Sewerage Works Ventilation, (1902).
36. Health effects of occupational exposure to respirable crystalline silica.: National Institute for Occupational Safety and Health Cincinnati, OH, Services DoHaH; 2002. Contract No.: 2002-129.
37. Seaton A, Cherrie JW. Quartz exposures and severe silicosis: a role for the hilar nodes. Occup Environ Med. 1998;55(6):383-6.
38. Koksal N, Kahraman H. Acute silicosis in teflon-coated pan manufacturing due to metal sandblasting. International journal of occupational and environmental health. 2011;17(3):210-3.
39. Suratt PM, Winn WC, Jr., Brody AR, Bolton WK, Giles RD. Acute silicosis in tombstone sandblasters. Am Rev Respir Dis. 1977;115(3):521-9.
40. Rees DM, J. Silica. In: Newman Taylor A CP, Blanc P, Pickering A., editor. Parkes' Occupational Lung Disorders. Fourth ed. Boca Raton, FL.: CRC Press; 2017. p. 187-205.
41. Souza CA, Marchiori E, Goncalves LP, Meirelles GS, Zanetti G, Escuissato DL, et al. Comparative study of clinical, pathological and HRCT findings of primary alveolar proteinosis and silicoproteinosis. European journal of radiology. 2012;81(2):371-8.
42. Ding M, Chen F, Shi X, Yucesoy B, Mossman B, Vallyathan V. Diseases caused by silica: mechanisms of injury and disease development. International immunopharmacology. 2002;2(2-3):173-82.
43. Marchiori E, Ferreira A, Muller NL. Silicoproteinosis: high-resolution CT and histologic findings. Journal of thoracic imaging. 2001;16(2):127-9.
44. Marchiori E, Souza CA, Barbassa TG, Escuissato DL, Gasparetto EL, Souza AS, Jr. Silicoproteinosis: high-resolution CT findings in 13 patients. AJR American journal of roentgenology. 2007;189(6):1402-6.
45. Akgun M, Araz O, Ucar EY, Karaman A, Alper F, Gorguner M, et al. Silicosis Appears Inevitable Among Former Denim Sandblasters: A 4-Year Follow-up Study. Chest. 2015;148(3):647-54.
46. Xing J, Huang X, Yang L, Liu Y, Zhang H, Chen W. Comparison of high-resolution computerized tomography with film-screen radiography for the evaluation of opacity and the recognition of coal workers' pneumoconiosis. Journal of occupational health. 2014;56(4):301-8.
47. Yang H, Yang L, Zhang J, Chen J. Natural course of silicosis in dust-exposed workers. Journal of Huazhong University of Science and Technology Medical sciences = Hua zhong ke ji da xue xue bao Yi xue Ying De wen ban = Huazhong keji daxue xuebao Yixue Yingdewen ban. 2006;26(2):257-60.

48. Lee HS, Phoon WH, Ng TP. Radiological progression and its predictive risk factors in silicosis. Occup Environ Med. 2001;58(7):467-71.
49. Ng TP, Chan SL, Lam KP. Radiological progression and lung function in silicosis: a ten year follow up study. British medical journal (Clinical research ed). 1987;295(6591):164-8.
50. Ng TP, Chan SL, Lee J. Predictors of mortality in silicosis. Respiratory Medicine. 1992;86(2):115-9.
51. Talini D, Paggiaro PL, Falaschi F, Battolla L, Carrara M, Petrozzino M, et al. Chest radiography and high resolution computed tomography in the evaluation of workers exposed to silica dust: relation with functional findings. Occup Environ Med. 1995;52(4):262-7.
52. Antao VC, Pinheiro GA, Terra-Filho M, Kavakama J, Muller NL. High-resolution CT in silicosis: correlation with radiographic findings and functional impairment. J Comput Assist Tomogr. 2005;29(3):350-6.
53. Cox CW, Rose CS, Lynch DA. State of the art: Imaging of occupational lung disease. Radiology. 2014;270(3):681-96.
54. Karatas M, Gunduzoz M, Buyuksekerci M, Ozakinci OG, Nadir Ozis T. Radiological progression and lung function decrements among silica-exposed ceramic workers: a longitudinal study. Inhalation toxicology. 2019;31(3):119-24.
55. Mohebbi I, Zubeyri T. Radiological progression and mortality among silica flour packers: a longitudinal study. Inhalation toxicology. 2007;19(12):1011-7.
56. Barboza CE, Winter DH, Seiscento M, Santos Ude P, Terra Filho M. Tuberculosis and silicosis: epidemiology, diagnosis and chemoprophylaxis. Jornal brasileiro de pneumologia : publicacao oficial da Sociedade Brasileira de Pneumologia e Tisilogia. 2008;34(11):959-66.
57. Rees D, Murray J. Silica, silicosis and tuberculosis. The international journal of tuberculosis and lung disease : the official journal of the International Union against Tuberculosis and Lung Disease. 2007;11(5):474-84.
58. Skowronski M, Halicka A, Barinow-Wojewodzki A. Pulmonary tuberculosis in a male with silicosis. Advances in respiratory medicine. 2018;86(3).
59. International Agency for Research on Cancer. Silica dust, Crystalline, in the form of quartz or cristobalite. IARC Monograph. 2018;100C-14.
60. Liu Y, Steenland K, Rong Y, Hnizdo E, Huang X, Zhang H, et al. Exposure-response analysis and risk assessment for lung cancer in relationship to silica exposure: a 44-year cohort study of 34,018 workers. American journal of epidemiology. 2013;178(9):1424-33.
61. Steenland K, Mannetje A, Boffetta P, Stayner L, Attfield M, Chen J, et al. Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. Cancer causes & control : CCC. 2001;12(9):773-84.
62. Lai H, Liu Y, Zhou M, Shi T, Zhou Y, Weng S, et al. Combined effect of silica dust exposure and cigarette smoking on total and cause-specific mortality in iron miners: a cohort study. Environmental health : a global access science source. 2018;17(1):46.
63. Sato T, Shimosato T, Klinman DM. Silicosis and lung cancer: current perspectives. Lung Cancer (Auckland, NZ). 2018;9:91-101.
64. Miller FW, Alfredsson L, Costenbader KH, Kamen DL, Nelson LM, Norris JM, et al. Epidemiology of environmental exposures and human autoimmune diseases: findings from a National Institute of Environmental Health Sciences Expert Panel Workshop. Journal of autoimmunity. 2012;39(4):259-71.
65. Khuder SA, Peshimam AZ, Agraharam S. Environmental risk factors for rheumatoid arthritis. Reviews on environmental health. 2002;17(4):307-15.
66. McCormic ZD, Khuder SS, Aryal BK, Ames AL, Khuder SA. Occupational silica exposure as a risk factor for scleroderma: a meta-analysis. International archives of occupational and environmental health. 2010;83(7):763-9.
67. Pollard KM. Silica, Silicosis, and Autoimmunity. Frontiers in immunology. 2016;7:97.
68. Conrad K, Mehlhorn J. Diagnostic and prognostic relevance of autoantibodies in uranium miners. International archives of allergy and immunology. 2000;123(1):77-91.

69. Parks CG, Conrad K, Cooper GS. Occupational exposure to crystalline silica and autoimmune disease. Environmental health perspectives. 1999;107 Suppl 5:793-802.
70. Hoet P, Desvallees L, Lison D. Do current OELs for silica protect from obstructive lung impairment? A critical review of epidemiological data. Critical reviews in toxicology. 2017;47(8):650-77.
71. Baumgartner KB, Samet JM, Coultas DB, Stidley CA, Hunt WC, Colby TV, et al. Occupational and environmental risk factors for idiopathic pulmonary fibrosis: a multicenter case-control study. Collaborating Centers. American journal of epidemiology. 2000;152(4):307-15.
72. Kim SY, Kang DM, Lee HK, Kim KH, Choi J. Occupational and Environmental Risk Factors for Chronic Fibrosing idiopathic Interstitial Pneumonia in South Korea. J Occup Environ Med. 2017;59(11):e221-e6.
73. Parks CG, de Souza Espindola Santos A, Barbhaiya M, Costenbader KH. Understanding the role of environmental factors in the development of systemic lupus erythematosus. Best practice & research Clinical rheumatology. 2017;31(3):306-20.
74. Gomez-Puerta JA, Gedmintas L, Costenbader KH. The association between silica exposure and development of ANCA-associated vasculitis: systematic review and meta-analysis. Autoimmunity reviews. 2013;12(12):1129-35.
75. Mulloy KB. Silica exposure and systemic vasculitis. Environmental health perspectives. 2003;111(16):1933-8.
76. Osorio AM, Thun MJ, Novak RF, Van Cura EJ, Avner ED. Silica and glomerulonephritis: case report and review of the literature. American journal of kidney diseases : the official journal of the National Kidney Foundation. 1987;9(3):224-30.
77. Stratta P, Canavese C, Messuerotti A, Fenoglio I, Fubini B. Silica and renal diseases: no longer a problem in the 21st century? Journal of nephrology. 2001;14(4):228-47.
78. Ghahramani N. Silica nephropathy. The international journal of occupational and environmental medicine. 2010;1(3):108-15.
79. Martinez C, Prieto A, Garcia L, Quero A, Gonzalez S, Casan P. Silicosis: a disease with an active present. Archivos de bronconeumologia. 2010;46(2):97-100.
80. Leso V, Fontana L, Romano R, Gervetti P, Iavicoli I. Artificial Stone Associated Silicosis: A Systematic Review. Int J Environ Res Public Health. 2019;16(4).
81. León-Jiménez A, Hidalgo-Molina A, Conde-Sánchez M, Pérez-Alonso A, Morales-Morales JM, García-Gámez EM, et al. Artificial Stone Silicosis: Rapid Progression Following Exposure Cessation. Chest. 2020.
82. Frankel A, Blake L, Yates D. LATE-BREAKING ABSTRACT: Complicated silicosis in an Australian worker from cutting engineered stone countertops: An embarrassing first for Australia. European Respiratory Journal. 2015;46(suppl 59):PA1144.
83. Hoy RF, Glass DC, Dimitriadis C, Hansen J, Hore-Lacy F, Sim MR. Identification of early-stage silicosis through health screening of stone benchtop industry workers in Victoria, Australia. Occup Environ Med. 2020.
84. Wu N, Xue C, Yu S, Ye Q. Artificial stone-associated silicosis in China: A prospective comparison with natural stone-associated silicosis. Respirology. 2020;25(5):518-24.
85. Hoy RF, Jeebhay MF, Cavalin C, Chen W, Cohen RA, Fireman E, et al. Current global perspectives on silicosis-Convergence of old and newly emergent hazards. Respirology. 2022.
86. Shi P, Xing X, Xi S, Jing H, Yuan J, Fu Z, et al. Trends in global, regional and national incidence of pneumoconiosis caused by different aetiologies: an analysis from the Global Burden of Disease Study 2017. Occup Environ Med. 2020;77(6):407-14.
87. Alif SM, Glass DC, Abramson M, Hoy R, M.R S. Occupational Lung Diseases in Australia 2006 - 2019. Monash University; 2020.
88. Matar E, Frankel A, Blake LKM, Silverstone EJ, Johnson AR, Yates DH. Complicated silicosis resulting from occupational exposure to engineered stone products. The Medical journal of Australia. 2017;206(9):385-6.

89. Insurance and Care NSW. Annual Report 2019 - 20. Annual report. NSW Government, NSW i; 2020.
90. Australian Goverment. National Dust Disease Taskforce, Final Report. Australian Government, Department of Health; 2021.
91. NSW SW. NSW Dust Disease Register Annual Report 2020-21. NSW; 2021.
92. Silica associated lung disease health screening research project: Phase 1.: Monash University; 2020 November, 2020.
93. Quantum Market Research. Dust Disease Research Update. Final report May 2021. South Yarra, Victoria: Australian Government Department of Health; 2021. Available from: https://www1.health.gov.au/internet/main/publishing.nsf/Content/ohp-nat-dust-disease-taskforce.htm.
94. Safe Work Australia. Australian workers' compensation statistics. Canberra: Safe Work Australia; 2021. Available from: https://www.safeworkaustralia.gov.au/sites/default/files/2021-11/Final%20Australian%20Workers%20%20Compensation%20Statistics%202019-20.pdf.
95. Kreitals N, editor "Industry change in the manufactured stone benchtop industry as a result of proactive compliance activities" Australian Institute of Occupational Hygienists 38th Annual Conference & Exhibition; 2021; Sydney.
96. Australia SW. How to Manage Health and Safety Risks Code of Practice. 2011.
97. Kumarasamy C, Pisaniello D, Gaskin S, Hall T. What Do Safety Data Sheets for Artificial Stone Products Tell Us About Composition? A Comparative Analysis with Physicochemical Data. Ann Work Expo Health. 2022.
98. Gaskin S GR, Jersmann H, Pisaniello D. SWSA UoA Engineered Stone Benchtops Final Report for open access. Adelaide: The University of Adelaide; 2020.
99. The Lancet Respiratory M. The world is failing on silicosis. Lancet Respir Med. 2019;7(4):283.
100. Coal Mining Safety and Health Regulation 2017, Chapter 2, Part 12, Section 89A (2017).
101. Safe Work Australia. Workplace exposure standards for airborne contaminants: Safe Work Australia; 2019. Available from: https://www.safeworkaustralia.gov.au/doc/workplace-exposure-standards-airborne-contaminants-2019.
102. Safe Work Australia. Changes to the workplace exposure standard for respirable crystalline silica [web page]. [cited 2022 May]. Available from: https://www.safeworkaustralia.gov.au/safety-topic/hazards/crystalline-silica-and-silicosis/changes-workplace-exposure-standard-respirable-crystalline-silica.
103. Australia SW. Guidance on the Interpretation of Workplace Exposure Standards for Airborne Contaminants 2013.
104. Australian Government. All of goverments' response to the final report of the National Dust Disease Taskforce: Australian Government; 2022. Available from: https://www1.health.gov.au/internet/main/publishing.nsf/Content/45748C2442F5BA74CA2587E4000F8600/$File/All-Govt-Response-Final-Report-Dust-Disease-Taskforce.pdf.
105. Cancer Council Australia, Lung Foundation Australia. Inquiry into the 2021 review of the dust diseases scheme. Submission No 5. 2021. Available from: https://www.parliament.nsw.gov.au/lcdocs/submissions/76636/0005%20Cancer%20Council%20and%20Lung%20Foundation%20Australia.pdf.
106. Worksafe Victoria. Crystalline silica: Safety basics: Victorian Government; 2022 [updated 16 March 2022; cited 2022 May]. Available from: https://www.worksafe.vic.gov.au/crystalline-silica-safety-basics.
107. Australian Institute of Occupational Hygienists. Respirable Crystalline Silica (RCS) Measurement AIOH / NATA Statement of Common Interests 2021.
108. Australia SW. Crystalline silica and silicosis 2018 [Available from: https://www.safeworkaustralia.gov.au/silica.
109. Raghu G, Remy-Jardin M, Myers JL, Richeldi L, Ryerson CJ, Lederer DJ, et al. Diagnosis of Idiopathic Pulmonary Fibrosis. An Official ATS/ERS/JRS/ALAT Clinical Practice Guideline. Am J Respir Crit Care Med. 2018;198(5):e44-e68.

110. Prasad JD, Mahar A, Bleasel J, Ellis SJ, Chambers DC, Lake F, et al. The interstitial lung disease multidisciplinary meeting: A position statement from the Thoracic Society of Australia and New Zealand and the Lung Foundation Australia. Respirology. 2017;22(7):1459-72.
111. England BR, Hershberger D. Management issues in rheumatoid arthritis-associated interstitial lung disease. Curr Opin Rheumatol. 2020;32(3):255-63.
112. Paraskeva MA, Levin KC, Westall GP, Snell GI. Lung transplantation in Australia, 1986-2018: more than 30 years in the making. The Medical journal of Australia. 2018;208(10):445-50.
113. Rosengarten D, Fox BD, Fireman E, Blanc PD, Rusanov V, Fruchter O, et al. Survival following lung transplantation for artificial stone silicosis relative to idiopathic pulmonary fibrosis. American journal of industrial medicine. 2017;60(3):248-54.
114. Zhang YM, Zhang HT, Wang CY, Wang W, Wu J, Wang C. [Long-term therapeutic effects of whole lung lavage in the management of silicosis]. Zhonghua lao dong wei sheng zhi ye bing za zhi = Zhonghua laodong weisheng zhiyebing zazhi = Chinese journal of industrial hygiene and occupational diseases. 2012;30(9):690-3.
115. Chambers DC, Apte SH, Deller D, Masel PJ, Jones CM, Newbigin K, et al. Radiological outcomes of whole lung lavage for artificial stone-associated silicosis. Respirology. 2021;26(5):501-3.
116. Sharma SK, Pande JN, Verma K. Effect of prednisolone treatment in chronic silicosis. Am Rev Respir Dis. 1991;143(4 Pt 1):814-21.
117. Kramer MR, Blanc PD, Fireman E, Amital A, Guber A, Rhahman NA, et al. Artificial stone silicosis [corrected]: disease resurgence among artificial stone workers.[Erratum appears in Chest. 2012 Oct;142(4):1080]. Chest. 2012;142(2):419-24.
118. Zhang H, Sui JN, Gao L, Guo J. Subcutaneous administration of infliximab-attenuated silica-induced lung fibrosis. International journal of occupational medicine and environmental health. 2018;31(4):503-15.
119. Maillet I, Quesniaux V, Wollin L, Ryffel B. Effect of nintedanib on silica-induced lung inflammation and fibrosis in mice. European Respiratory Journal. 2012;40.
120. Wollin L, Distler JHW, Redente EF, Riches DWH, Stowasser S, Schlenker-Herceg R, et al. Potential of nintedanib in treatment of progressive fibrosing interstitial lung diseases. Eur Respir J. 2019;54(3).
121. Abdelaziz RR, Elkashef WF, Said E. Tadalafil reduces airway hyperactivity and protects against lung and respiratory airways dysfunction in a rat model of silicosis. International immunopharmacology. 2016;40:530-41.
122. Li XF, Liao J, Xin ZQ, Lu WQ, Liu AL. Relaxin attenuates silica-induced pulmonary fibrosis by regulating collagen type I and MMP-2. International immunopharmacology. 2013;17(3):537-42.
123. Morales MM, Souza SA, Loivos LP, Lima MA, Szklo A, Vairo L, et al. Pilot safety study of intrabronchial instillation of bone marrow-derived mononuclear cells in patients with silicosis. BMC Pulm Med. 2015;15(1471-2466 (Electronic)):66.
124. Liu WW, Wang HX, Yu W, Bi XY, Chen JY, Chen LZ, et al. Treatment of silicosis with hepatocyte growth factor-modified autologous bone marrow stromal cells: a non-randomized study with follow-up. Genetics and molecular research : GMR. 2015;14(3):10672-81.
125. Lopes-Pacheco M, Bandeira E, Morales MM. Cell-Based Therapy for Silicosis. Stem cells international. 2016;2016:5091838.

Australian Government
Department of Health

Lung Foundation Australia