REVIEW   **Open Access**

A review of silicosis and other silica-related diseases in the engineered stone countertop processing industry

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Abstract

Background Engineered stone (ES), a material that has become widespread for its use in kitchen and bathroom countertops since the 1980s, is composed of over 90% crystalline silica by weight, significantly exceeding the silica content of natural stones such as granite (40–50%) and marble (< 10%). Workers fabricating ES are exposed to dangerously high levels of respirable crystalline silica (RCS) and other toxic chemicals, which increases the risk of developing silicosis and other lung and systemic diseases. The purpose of this review is to explore the epidemiology, occupational risks, regulatory gaps, diagnostic evaluation, and clinical challenges associated with ES dust exposure.

Main body ES silicosis was first described in the early 2010s among ES countertop workers in Spain, Italy, and Israel. Since then, hundreds of cases have emerged worldwide, namely in China, Australia, the United States, the United Kingdom, and Belgium. Silicosis from ES dust is accelerated and diagnosed after 7–19 years of exposure, often affecting young individuals (median age 33–55 years) from marginalized or immigrant communities. Morbidity and mortality are poor, with high rates of lung transplantation and death. Industrial hygiene air sample monitoring data shows that despite engineering controls such as wet saws and exhaust ventilation, exposure to respirable crystalline silica when cutting ES frequently exceeds safe exposure levels. Diagnostic evaluation and treatment are clinically challenging due to delayed medical screening, misdiagnosis, and lack of treatment options.

Conclusions This review underscores the urgent need for enhanced occupational safety regulations, active screening, and healthcare support to address the rising burden of ES silicosis among vulnerable worker populations globally.

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Background/introduction

Silicosis is the most prevalent pneumoconiosis worldwide. It is a fibrotic lung disease that usually occurs after the chronic inhalation of respirable crystalline silica (silicon dioxide, SiO_2) by workers in mining, quarrying, or sandblasting. Although documented as early as ancient Egypt, the 1930 international Johannesburg Conference on Silicosis produced the first general scientific consensus opinion regarding the disease [1]. This occurred concurrently with international outbreaks noted in UK scouring powder manufacturers, South African gold miners, and the US Hawk's Nest Tunnel disaster [2]. These events prompted many of the silicosis prevention efforts of the 20th century, particularly in the US, where it incited the Secretary of Labor's Stop Silicosis campaign, the formation of the Occupational Safety and Health Administration, and the implementation of occupational permissible exposure limits for silica dust [3]. Similarly, identifying new and accelerated silicosis affecting a relatively young population of engineered stone countertop workers has prompted a resurgence of new research into an entity known as engineered stone silicosis.

Engineered stone (ES; also called artificial or agglomerate stone) is relatively novel and has become an increasingly ubiquitous material for kitchen and bathroom countertops since its debut in the 1990s (colloquially referred to as quartz countertops). Although it is similar in appearance to natural stones such as marble or granite, it contains significantly higher levels of silica – typically greater than 90% silica by weight, compared to granite (40–50% at the most) and marble (< 10%) [4, 5]. Workers who cut, polish, and finish countertops made from ES can, therefore, be exposed to much higher levels of respirable crystalline silica (RCS), increasing their risk of developing silicosis.

ES Silicosis is diagnosed on average after a relatively short work tenure of 7–19 years and at a median age between 33 and 55 years [6–8]. This contrasts the historically longer latency periods of chronic silicosis seen among underground miners that more often affect older workers [9, 10]. However, the relatively short latency period of ES silicosis draws similarities to the epidemic of silicosis among denim sandblasters in Turkey from the mid to late 2000s who developed the disease at similarly young ages and short work tenures [11–13]. The relatively short latency period of ES silicosis has prompted biomedical science to reconsider ES silicosis as a novel entity and explore its unique mechanisms [14]. There is continued need for further studies to understand the pathophysiologic implications of materials found within engineered stone countertops in addition to silica, such as resins and metals (e.g., Cobalt and Aluminum). Additionally, there is early evidence that particle size may modify chemical interactions and therefore disease risk [15]. The current state of knowledge of the pathogenic mechanisms of ES silicosis is detailed at length in a recent review by Ramkissoon et al. and is beyond the scope of this review [16].

This narrative review aims to provide a multidisciplinary discussion of the epidemiology, occupational processes, regulation and enforcement, and the diagnosis, treatment, and prognosis of ES silicosis and other associated diseases. To explore these aspects of ES silicosis, we conducted a comprehensive search on the Web of Science and PubMed using the keywords "Silicosis" AND "engineered stone" OR "Artificial stone" OR "Quartz conglomerate." We included articles in all languages to ensure a broad scope of data collection. Additionally, we reviewed the bibliographies of the included studies to identify any pertinent articles that may not have surfaced in our initial search. This approach allowed us to capture a wide range of relevant literature and insights on the subject.

Epidemiology

ES silicosis was first reported in 2010 and 2011 in Spain with 20 total identified cases [17–19], and again in 2012 among 7 of 29 Italian countertop workers [20]. Following this, 25 cases were identified at a lung transplant referral center in Israel, diagnosed between 1997 and 2010, corresponding to the opening of an ES manufacturing company nearby 10 years prior (see Fig. 1) [21, 22]. In 2014, 46 cases were identified in southern Spain [23]. In the ensuing decade, reports of cases continued to emerge globally, including in Spain, China, Australia, the United States, the United Kingdom, and Belgium [6, 7, 24–30].

Best estimates of silicosis prevalence among stone countertop workers are derived from an active government screening program in Queensland, Australia, which began in 2018, and as of August 2024, has found that 224 of 1,054 workers screened (21%) have silicosis, 36 (3.6%) with progressive massive fibrosis [31]. In the United States, the only estimates of prevalence come from active surveillance of workers from two workplaces in California using spirometry and chest radiographs, which found that 5 out of 43 (12%) had silicosis [32], likely an underestimate given the limited sensitivity of chest radiograph (35–48%) compared to high resolution computed tomography (HRCT) [33–35].

In addition to affecting predominantly young workers with shorter work tenures (accelerated silicosis), many case series note the disproportionate impact on marginalized and migrant communities. In the United States, more than 95% of all affected workers are Spanish-speaking immigrants from Mexico or Central America [5, 8]. The majority are likely to be uninsured or underinsured secondary to low wages and undocumented immigrant status [8]. Similarly, in Australia, more than half of those with ES silicosis had been born outside of the country [36], and in China, migrant interior construction workers
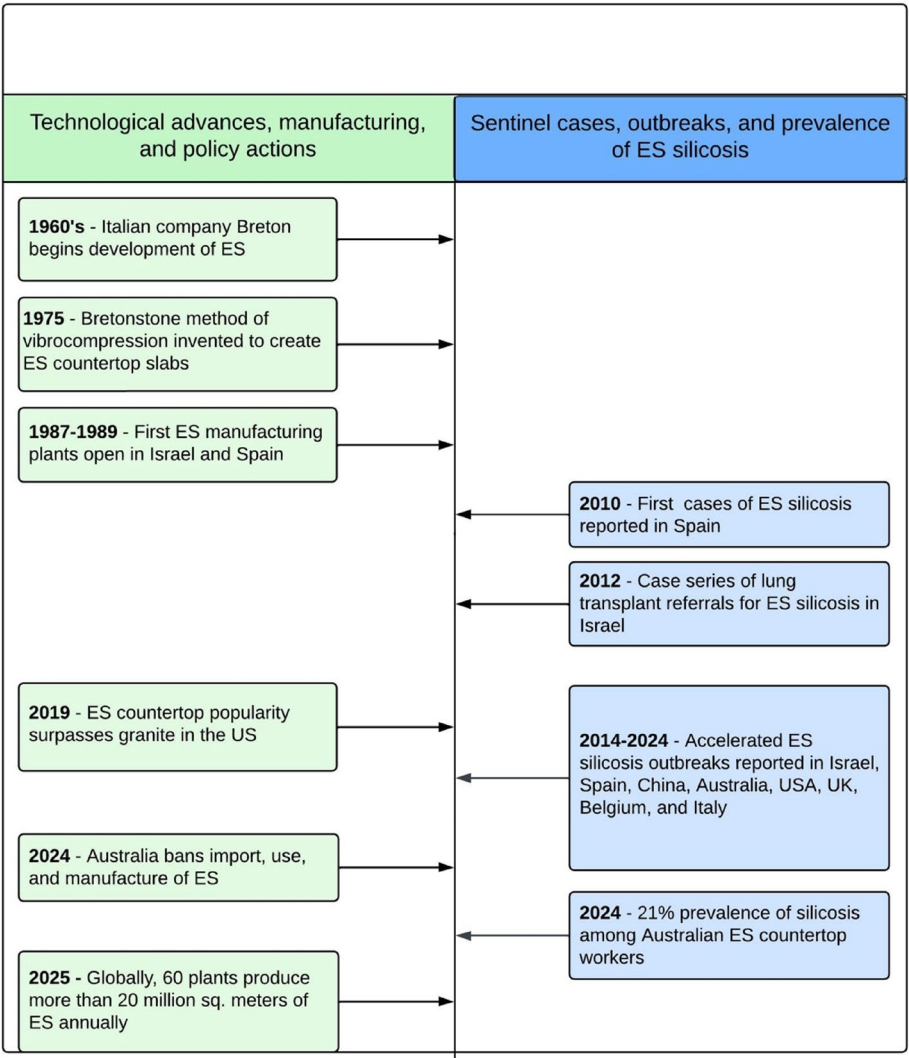
Fig. 1 Timeline of selected major events in technological advances, manufacturing, policy actions, and epidemiology of ES silicosis [40]. Definitions: ES = engineered stone, USA = United States of America, UK = United Kingdom

are exposed to unsafe levels of respirable dust with inadequate respiratory protection [37]. Additionally, migrant/immigrant or minority workers are found to be at higher risk of occupation-related illness or injury and were also disparately affected by severe forms of silicosis in the West Virginia Hawk's nest tunnel disaster [2, 38]. Immigrants are likely to delay or forego medical care due to cost, difficulty navigating the healthcare system, or language barriers, and therefore, are likely to present in late stages of disease utilizing emergency services [39]. In fact, nearly half of ES countertop patients with silicosis in California initially presented to the emergency department instead of through medical surveillance or primary care [8].

Occupational processes
Manufacturing of ES slabs

The process for manufacturing ES was invented in Italy in the early 1970s by Marcello Toncelli's company, Bretonstone [41]. The process, which is still generally used today, is known as vibro-compression under vacuum. Typically, manufacturers combine quartz and resin binder under high heat, vacuum, and compression to create a countertop slab (Fig. 2). After vibro-compression into a slab, manufacturers distribute the ES slabs to smaller workshops or subcontractors who cut, sand, and polish the material and install it as a kitchen or bathroom countertop. To the best of our knowledge, there have been no peer-reviewed studies that present air sampling exposure monitoring results for RCS exposure during the manufacturing process or detailed descriptions of the hazards associated with this process. This absence of
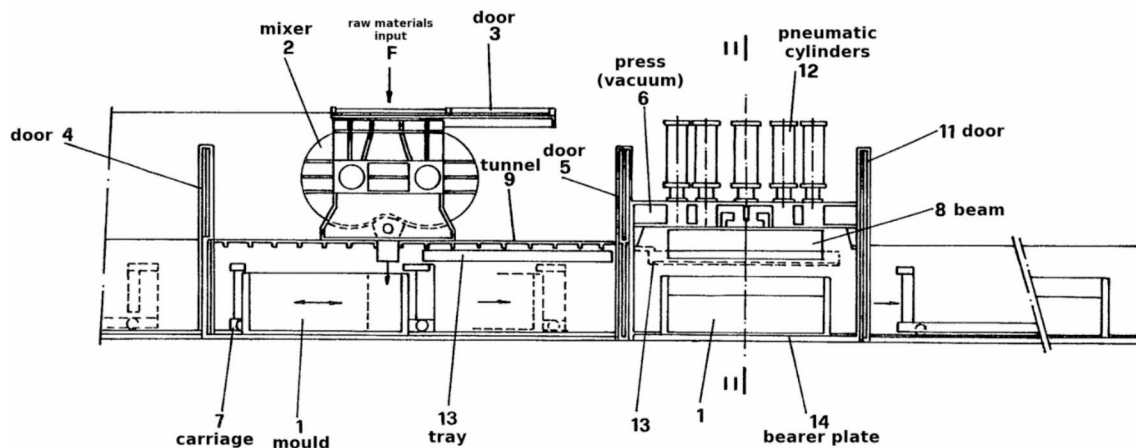
Fig. 2 Schematic from US Patent October 6, 1987, of the Bretonstone press used to make ES slabs [41].

data presents a gap in the literature and underscores how manufacturing companies either withhold this information or have not collected or conducted these industrial hygiene assessments. A criminal court in Spain convicted manufacturer Cosentino for not providing adequate information to companies performing further processing of the slabs, and journalists have uncovered compensation agreements with manufacturing companies with confidentiality clauses [42]. The primary understanding of worker exposures when working with ES slabs is limited to the further processing of the stone slabs, which is described in detail in the next section, referred to as "fabrication."

Further processing of ES Countertops/ Slabs (Commonly referred to by industry in the United States as Fabrication)
The process commonly referred to as fabricating by owners of stone countertop companies and their employees includes the further processing and transformation of an ES slab into a countertop by performing polishing, beveling, and cutting using a variety of tools which vary depending on the business sophistication and types of equipment. This variation ranges from fabricators at small shops primarily using hand tools for the entire process to larger shops who invest in robotic and computer numerical control (CNC) machinery (saws, water laser jets, and polishing) that allow workers to create distance

from the sources of air contaminants. First, ES slabs are cut to specified measurements. Then, interior sink holes are cut out of the slab, followed by polishing the slab's surfaces. When adding edging to a countertop, two slabs must be cemented together using an adhesive to create a larger countertop surface, in a process called lamination. Finally, finished countertops are transported to the site and installed [43–46].

Fabrication shops typically process a wide range of countertop slabs, including, but not limited to, porcelain, granite, marble, sandstone, quartzite, and ES slabs. ES slabs are comprised of generally greater than 90% crystalline silica, resin, pigments and metals for color, and other additives to assist with the curing of the resin. Depending on the type of resin used in the slab, volatile organic chemicals [(VOCs), such as styrene, methyl methacrylate, and phthalic anhydride] may be produced during the fabrication of the slabs [47, 48]. The dust generated from cutting and grinding ES slabs comprises a high concentration of very fine (< 1 μm) particles [49].

In general, workers at fabrication shops will be assigned tasks in one or more of the following four similar exposure groups (SEG) categories: automated tool operators, small tool operators, lamination, and support workers [35, 50, 51]. First, automated tool operators are typically responsible for programming and operating CNC machinery (routers, water laser jets, and bridge saws),

robotic arm saws, miter saws, and automatic polishers. The advantages of these tools for the worker are that most are outfitted with water delivery systems to reduce the generation of airborne particulates and that operators can remain out of the vicinity of the machinery during the process, reducing their potential exposure. While the water used in their tools reduces the dust particulates in the air, dust may be generated, as shown in Fig. 3(A), or dust particles may agglomerate onto water droplets. Second, small tool operators typically use a variety of sanders, angle grinders, polishers, and cutting saws that may or may not have integrated water delivery systems [43–46]. For the smallest fabrication shops, these tools will be the entry point for beginning a stone fabrication business, given their relatively low cost compared to automated tools. Figure 3(B) shows an employee using a small hand tool to polish ES dry, while Fig. 3(C) shows this same process being performed wet. Even for shops with a wide range of automated tools, much of the final polishing and finishing work is done by hand.

A third category of tasks for employees at a stone fabrication shop is lamination. Lamination involves using an adhesive to cement two slabs together or to glue an edge to a countertop [43]. The employees performing this task potentially have the greatest exposure to silica dust and other air contaminants due to work being conducted dry to prevent interfering with the lamination adhesive from curing, as shown in Fig. 3(D). Because the adhesive must be dry to cure, some employees use compressed air or heat guns to dry the surfaces, which may produce airborne dust [52]. The final category of workers is those either performing support tasks within the fabrication shop or performing the installation. Support employees perform various tasks, including moving slabs, as shown in Fig. 3(E), performing maintenance on equipment, packaging slabs for shipping or installation, or general housekeeping. The exposure to silica and other air contaminants for these tasks will depend on the engineering controls, shop ventilation, methods used for fabrication, and housekeeping of the shop, as settled dust can be kicked up and suspended in the air. The exposure to silica and other air contaminants for installers will depend on the need for additional cuts or adjustments, ventilation, and availability of control methods at the installation site [52].

Exposure and controls
Overview of exposure to silica and other air contaminants when fabricating engineered stone
Elevated exposure to respirable crystalline silica during ES countertop fabrication was first documented in 2012 with a report that showed dry finishing of countertop slabs to produce dust concentrations ranging from 215 μg/m^3 to 519 μg/m^3, significantly above most occupational exposure limits (OELs; 50 μg/m^3 in the US and Australia, 100 μg/m^3 in UK and EU) [27, 53, 54]. This was followed by investigations that additionally examined the efficacy of engineering controls, such as using wet methods and exhaust ventilation.

Studies have shown that despite using predominantly wet methods, ES fabrication often exposes workers to silica concentrations above the recommended exposure levels, as exposure levels during entirely automated wet processes may range from 20 μg/m^3 to 680 μg/m^3 [51]. However, when workshops incorporate dry tasks such as the use of dry hand tools, exposures increased up to 3880 μg/m^3 [51]. The US NIOSH conducted a series of studies and a Health Hazard Evaluation at stone fabrication shops in Minnesota and Texas following the identification of silicosis cases. The results of these assessments (which included a series of short-term and full-shift samples) consistently showed that even when wet methods were used, exposures were consistently above occupational exposure limits, with the highest levels among workers using pneumatic wet grinders with diamond cup wheels [44, 45, 55]. Qi and Echt conducted another site assessment in 2021 and examined the use of several different engineering control methods to control dust during the use of hand tools for grinding and polishing. They found that integrated water systems (water spray and center-feed) failed to adequately reduce the airborne concentrations of silica, with the short-term exposures ranging from 51.5 to 568.5 μg/m^3 [46]. Only with the addition of sheet-flow wetting (where an additional flow of water is added to the surface) were exposures reduced to levels below occupational exposure limits [56].

Dry cutting with wall exhaust ventilation is not sufficient to reduce potential over-exposure. A study by Guarnieri et al. assessed the use of wall exhaust ventilation during dry cutting and polishing at large (greater than 200 employees) factories in Italy from 2016 to 2019. The study measured exposure to silica for an 8-hour time-weighted average (TWA), which ranged from 260 μg/m^3 to 744 μg/m^3 when dry cutting and polishing [57]. Another Italian study of four facilities, conducted from 2018 to 2019, found exposures below the European Directive exposure limit of 100 μg/m^3 for silica but above 50 μg/m^3 [35].

Seneviratne et al. conducted an exposure assessment at six workplaces and measured the full-shift silica exposure for thirty-four workers performing a variety of wet cutting, dry finishing, and wet finishing. The results of this study further demonstrated that 85% of workers performing dry finishing and 71% of workers using water-fed hand tools were over the Australian Workplace Exposure Standard (WES) of 50 μg/m^3, with one worker using a high-pressure water-fed pneumatic grinder being exposed to almost 20 times the WES [50]. The exposures

Fig. 3 Examples of jobs and tasks conducted by countertop fabricators. **A** Automated Tool Operator using a Bridge Saw with Water Delivery. **B** An employee using dry polishing on an ES slab generating respirable crystalline silica. **C** Employee wet polishing. **D** Fabrication of edge following lamination of countertop edge. **E** Support employee positioning the hoist to lift a slab. Picture Credit: **A**, **B**, and **D** by Jenny Houlroyd, Picture **C** and **E** by Mixed Bag Media

measured by Seneviratne et al. were much higher than those reported by Weller et al., where results for the 121 workers sampled ranged from 9 to 120 μg/m^3 [52].

Finally, the California Division of Occupational Safety and Health (Cal/OSHA) reported the results of targeted inspections from January 2019 to February 2020 at 47 workplaces where they monitored 152 employees for exposure to respirable crystalline silica, where 25% of the workers sampled had exposures above 50 μg/m^3 [58]. However, this may underestimate exposure since regulatory TWAs for OSHA are required to calculate any time of the workday not sampled as zero exposure, thus diluting the exposure monitoring results.

Control methods and respiratory protection

In 2014, NIOSH conducted a Health Hazard Evaluation to investigate employee exposures to respirable crystalline silica when fabricating ES and natural stone slabs. The results of this survey included a series of recommendations for how employers must follow the hierarchy of controls by implementing engineering controls, administrative controls, and personal protective equipment to protect workers [55]. Since the publication of this report,

there has been a growing consensus that no one method employed will successfully and consistently reduce exposures below the occupational exposure limits [43]. As a result, as outlined below, all aspects of the NIOSH Hierarchy of Controls (Fig. 4) should be considered as methods to protect workers from silicosis [59].

Elimination or substitution

In response to the growing epidemic of ES silicosis cases, the Australian Government applied a precautionary approach in banning the use and import of ES [60]. Although lower silica alternatives may be thought to be less hazardous, the Australian scientific and regulatory expertise conducted between 2019 and 2023 affirmed that there is no guarantee that exposure to very low levels of silica (materials with lower silica content, intensity of exposure below threshold limited value-TWA) is harmless.

Engineering controls

In the places or countries where ES continues to be fabricated, companies should shift to using automated tools, such as the CNC machine or robotic arm saw, to
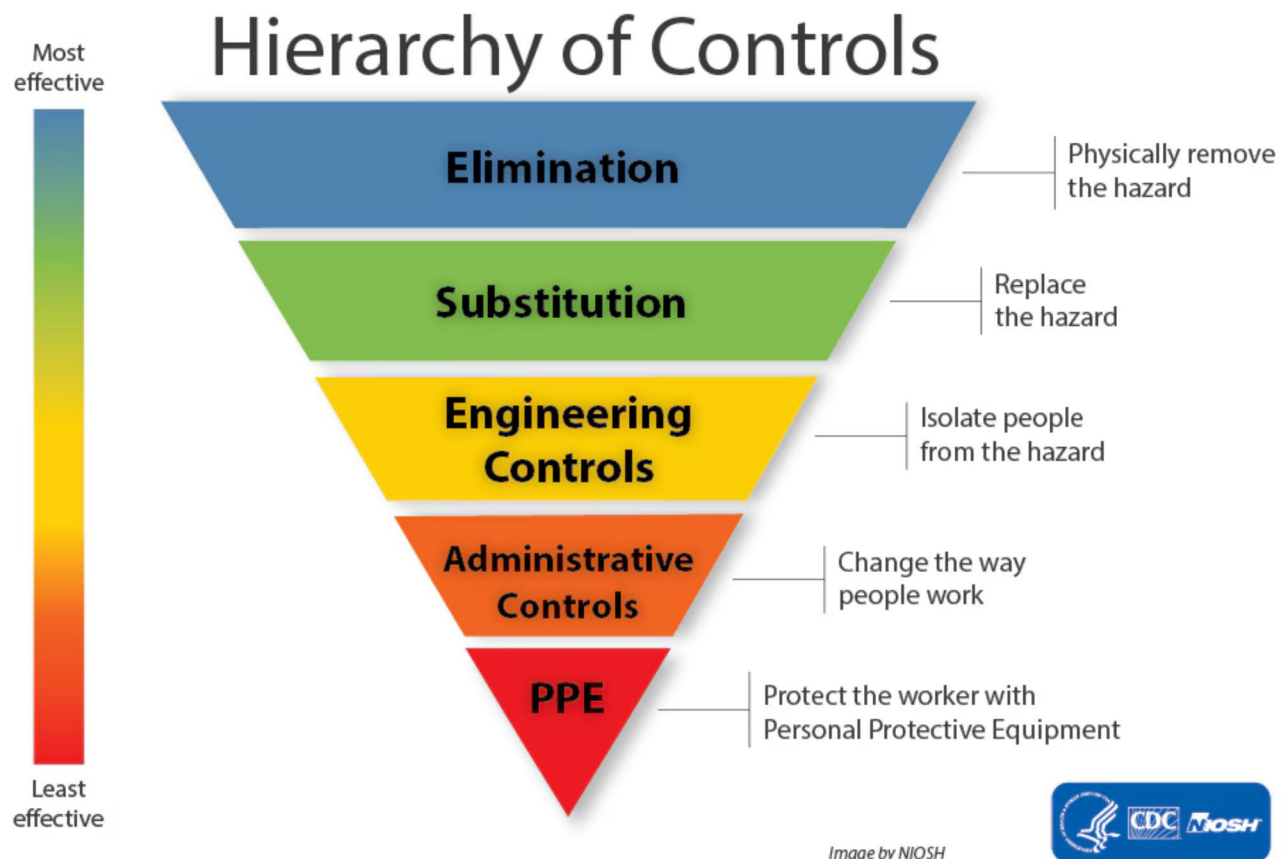

Fig. 4 The Hierarchy of Controls developed by The National Institute of Occupational Safety and Health (NIOSH) is a method for identifying and ranking methods of protecting workers from hazards

eliminate the need for employees to stand near where cutting, grinding, or polishing is being conducted. As a rule, all fabricators conducting tasks with ES should be, at a minimum, done wet to reduce the levels of airborne concentrations of silica. However, wet methods alone do not effectively reduce employee exposure to silica [35, 45, 46, 51, 52, 55, 57, 61]. Additional methods such as sheet wetting, where the slab's surface is flooded with water, have been shown to reduce silica dust further [56], but many shops may find this cost prohibitive and infeasible due to workshop layout and engineering.

In addition to water, implementing negative pressure workstations or a waterfall curtain ventilation booth that uses both circulation of air and a stream of water to pull dust particles out of the air may provide added exposure control [43]. Companies should also ensure that all ventilation systems are properly maintained, filter change-out schedules are conducted regularly, and, when necessary, local exhaust ventilation is installed to remove dust further [55].

Administrative controls

Administrative control includes developing a housekeeping schedule that removes any settled silica dust from the work environment. This may include wet mopping, high efficiency particulate air (HEPA) vacuums to clean vertical surfaces, and floor cleaning machines. In the US, the OSHA Final Silica Rule requires employers to train their workers on the hazards of exposure to silica, post adequate hazard signage, and supervise work practices to ensure proper implementation of engineering controls [54]. Additionally, employers are required to enroll all employees exposed to silica above the Action Level (AL) of 25 μg/m^3 in medical surveillance, which should be conducted initially and every three years to screen employees for silicosis [54]. Employers are required to keep up-to-date documentation of their employees' medical exams. Finally, supervision of work practices is necessary to ensure that all engineering controls are fully and properly implemented. Similarly, other countries, such as the UK, Brazil, and Australia, have mandated medical surveillance. In the UK, for example, workers exposed to silica above 100 μg/m^3 are mandated to receive a medical surveillance exam after 15 years of exposure [62].

Personal protective equipment (PPE)

Respiratory protection is the most important personal protective equipment to protect workers from exposure to respirable crystalline silica and other inhalation hazards associated with ES. Employers should select the necessary level of respiratory protection based on air sampling results based on the silica standard for general industry. Weller et al. reported that based on the air sampling results conducted at Australian countertop fabrication shops, a Class P2 or an N95 filtering facepiece respirator with an Assigned Protection Factor (APF) of 10 would adequately protect most workers based on Australian Standards for silica [52]. The analysis by Weller et al. did not break down respiratory protection recommendations based on the task and assumed all work would be conducted with wet methods. An N95 filtering facepiece respirator is designed to only protect from particulates and not the VOCs produced when fabricating ES slabs.

A systematic review of ES silicosis cases from 2019 summarized individual protective measures that ES countertop workers reported [6]. They found that respiratory protection was not worn or only worn by a minority. Even in cases where workers use respiratory protection, they are often not fit-tested, trained in its proper use and maintenance, or lack consistency in wearing it [6, 21]. Similar practices were reported in later studies where even when employees were provided with respirators, there continued to be barriers to their effectiveness given a lack of fit-testing, training, requirements for employees to be clean-shaven, and general support to select and wear the correct respirator [35, 50, 52, 58]. Additionally, workers who wore tight-fitting full-face respirators, positive air purifying respirators (PAPRs) with tight-fitting masks, or PAPRs with loose-fitting hoods, although providing greater protection than half-face elastomeric respirators or N95s, reported reduced visibility due to water accumulation on their masks surface [52]. This finding highlights the practical challenge of keeping workers consistently and effectively protected from overexposure. The California/OSHA updated permanent standard now requires tight-fitting, full-face PAPRs for all trigger tasks that produce exposure to respirable crystalline silica, which would include all similar exposure groups previously listed [63].

Regulation and enforcement

Globally, occupational exposure standards for silica vary, and most countries, aside from a few European countries, Australia and Mexico, do not have as stringent of a standard as the current US standard, which mandates a permissible exposure level (PEL) of 50 μg/m^3 or less as an 8-hour-time-weighted average. Most low and middle-income countries have no workplace regulations on silica at all [64].

United States

The Hawk's Nest Disaster of 1930 is the deadliest example of silicosis in modern US history, where over 700 workers died of acute or accelerated silicosis while mining a three-mile-long tunnel through Gauley mountain in West Virginia to build a hydroelectric plant [65]. Congress investigated this disaster in 1936. However, silica regulation was not imposed until the creation of a

national Occupational Safety and Health Administration (OSHA) in 1970, which issued its first silica standard in 1971, codifying a PEL of 250 μg/m^3 as an 8-hour TWA [3]. Despite the recommendation of the National Institute of Occupational Safety and Health (NIOSH), also created in 1970, to reduce the permissible exposure level to 50 μg/m^3 in 1974, OSHA did not make this change until the Final Silica Rule was issued in 2016 [54]. The 2016 Final Silica Rule was established after renewed concern about the health effects of silica exposure in the 1980s and 1990s. For instance, the International Agency for Research on Cancer (IARC) recognized silica as a probable carcinogen (group 2) in 1987 and as a carcinogen for humans (group 1) in 1997 (updated and confirmed in 2012). Silica-exposed workers were also found to be at significant risk of other respiratory diseases, as well as kidney and autoimmune diseases [54]. OSHA instituted a Special Emphasis Program in 1996 to increase enforcement of existing standards and educate and train employers. It also partnered with multiple other agencies, such as the NIOSH, to launch prevention efforts. During this time, OSHA found high rates of noncompliance with the existing standard. The 2016 Final Rule added additional provisions for exposure assessment, methods for controlling exposure, respiratory protection, medical surveillance, hazard communication, and recordkeeping [54].

The first cases of ES silicosis in the US were described in a single case report in Texas in 2014 [66], followed by 18 cases in four different US states described in a 2019 Centers for Disease Control Morbidity and Mortality Weekly Report [5]. Three years later, public health officials and pulmonary physicians in California published the largest US case series of ES silicosis, with 52 cases [8]. As of 2024, public health officials in California have confirmed over 230 ES silicosis cases [67]. In response to California's first reported outbreak of ES silicosis in 2019, which included two fatalities of workers in their 30s, Cal/OSHA initiated a Special Emphasis Program (SEP). The SEP aimed to reduce worker exposure to RCS by enforcing compliance with the silica and respiratory protection standards [68]. The SEP found overexposures in over half and citations in over two-thirds of workplaces, most commonly for failures to perform exposure assessment, communicate RCS hazards to employees, and perform medical surveillance [58]. SEP results in addition to increasing case counts, prompted the Western Occupational and Environmental Medical Association to petition Cal/OSHA to develop a Silica Emergency Temporary Standard (ETS), which was adopted in December 2023 [69]. The ETS imposed more stringent regulations, such as the prohibition of dry cutting or use of compressed air or sweeping to clean workstations, as well as requiring enhanced respiratory protection with full-face, tight-fitting PAPR [63]. During one year of the ETS, Cal/OSHA inspected 85 workshops, shutting down 26% due to immediate safety risks and citing 95% for violations [63]. As of December 2024, the Cal/OSHA standards board adopted an updated permanent standard similar to the ETS [63].

Throughout 2022, Federal OSHA found that about one-quarter of the air sample monitoring in stone countertop fabrication facilities exceeded the PEL, and about two-thirds of the employers with overexposures did not conduct medical surveillance [70]. More recently, federal OSHA launched an initiative to enhance enforcement and provide compliance assistance to workers in stone fabrication and installation nationwide. Initial results from September 2023 to July 2024 identified 68 overexposures among 332 air samples and issued 1,059 citations [71].

Australia
The most comprehensive regulatory and enforcement action globally in ES silicosis has been in Australia, whose experience may serve as a case study for other countries addressing this emerging epidemic. In response to the epidemic of ES silicosis in Australia, a National Dust Disease Taskforce was created in 2019 [72]. Early on, the Taskforce recommended the development of a strategic national approach to the disease, including understanding disease scope through research collaborations and data collection [72]. In their final 2022 report to the Minister of Health, the Dust Disease Taskforce recommended multiple interventions, most notably strengthening work health and safety measures, which included conducting regular dust sample and health monitoring, implementing a licensing schema to restrict access ES to businesses working with the product safely, and initiating the pathway for a ban of importation on ES [73]. The last recommendation was based on evidence that preventive measures did not protect workers effectively [73].

Safe Work Australia, an Australian federal agency, formally entertained three policy options: prohibition of use of all ES, prohibition of use of ES greater than 40% silica, or prohibition of ES greater than 40% silica with an additional licensing scheme for businesses working with materials with less than 40% silica [61]. They formally recommended option one, which was ultimately carried out as of July 2024, with the ban on use of all ES [60]. Australia's experience represents an example of an organized national effort to prevent further ES silicosis.

Overview of lung diseases
Silicosis refers to the spectrum of diseases caused by inhaling free crystalline silica, which mainly denotes pulmonary parenchymal silicosis, although lymphadenopathy may precede pulmonary manifestations [74–76]. The most commonly observed pulmonary parenchymal

manifestation is chronic simple silicosis, the oldest described and the most prevalent type of pneumoconiosis worldwide [77]. Additionally, workers exposed to ES dust may develop several manifestations of lung disease outside of the classically described pneumoconiosis, such as diffuse dust fibrosis, chronic obstructive pulmonary disease (COPD), and lung malignancy.

Silicosis (Pneumoconiosis from silica dust exposure)
Silicosis is typically diagnosed in individuals with a history of exposure to respirable silica dust with consistent radiographic or pathological findings [78]. Silica exposure can result in one of three distinct disease patterns of silicosis: (1) chronic silicosis, typically developing after more than 10 years of exposure to respirable dust containing less than 30% quartz; (2) subacute or accelerated silicosis, which arises after shorter periods of heavier exposure, usually between 2 and 10 years; and (3) acute silico-proteinosis, often occurring after a few months of intense exposure to fine dust with high silica content.

Chronic silicosis develops after 10 years of exposure. There are two common phenotypic presentations: (1) simple silicosis and (2) complicated silicosis, also known as progressive massive fibrosis (PMF). Simple silicosis is characterized by the presence of numerous, small, predominantly rounded nodular opacities (measuring less than 10 mm in diameter) with a centrilobular and peripheral (perilymphatic) distribution and predilection for the posterior upper zones of the lung due to less efficient clearance (Fig. 5). Histologically, the silicotic nodule in simple silicosis has a central acellular zone of "whorled" collagen fibers, often with dust-laden macrophages in the periphery. Silica particles are weakly birefringent under polarized light and can be found within macrophages, lymph nodes, or silicotic nodules on biopsy [79].

In some patients, these small nodules may enlarge and coalesce into mass-like fibrotic opacities in the upper zones of the lungs measuring larger than 10 mm, a pattern known as PMF or complicated silicosis (Fig. 6). However, PMF can occur in some patients without radiographic simple silicosis preceding it [80]. Though often considered a rare entity, ES fabricators have higher rates of PMF than other occupational groups with silica exposure [12, 81], with 30–40% developing PMF [8, 24, 25]. Those with PMF radiographically are more likely to present with a mixed restrictive-obstructive pattern on spirometry, a reduced total lung capacity (TLC), and reduced diffusion capacity (DLCO) [82]. PMF has a poor prognosis with rapid progression to cardiopulmonary failure in the absence of lung transplantation [21, 25, 83].

Acute silicoproteinosis (also referred to as "acute silicosis") is the acute form of silicosis. It is caused by very high levels of silica dust exposure over a short period (weeks to a few years) [84, 85]. Reflective of the high levels of silica exposure, multiple case series of artificial stone fabricators have demonstrated the pathologic findings of silicoproteinosis on lung biopsy or transplant explant (7–9%) [8, 86]. However, this is likely an undercount as the diagnosis of silicosis is often made without pathology. Pathologically, acute silicoproteinosis has a similar appearance to pulmonary alveolar proteinosis (PAP) and is characterized by the accumulation of an acellular lipoproteinaceous material in the alveoli [87, 88], which is periodic acid-Schiff (PAS) stain positive on histologic evaluation, and can contain silica crystals seen as birefringent in polarized light microscopy [89]. This accumulation of lipoproteinaceous material is due to the impaired ability of alveolar macrophages to clear lipoproteins from the alveolar space. This pathophysiology similarly occurs in idiopathic PAP but is due to
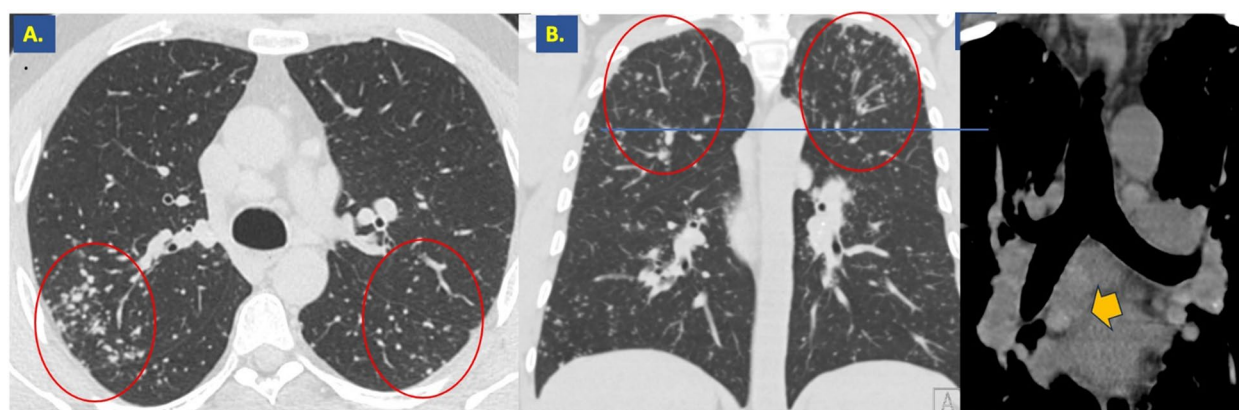

Fig. 5 Simple Chronic Silicosis. A 35-year-old male who worked as an engineered stone cutter for 16 years presented with incidental imaging abnormalities at an outside hospital but no pulmonary symptoms. CT Chest (**A**, axial; **B**, coronal) shows posterior, upper-lung predominant, well-defined, sub-5-mm but variably-sized, perilymphatic nodularity (red circle) without ground-glass or consolidative opacities. Multi-compartmental mediastinal lymphadenopathy (**C**, yellow arrow) is present predominantly in the bilateral hilar, subcarinal and paratracheal compartments with intrinsically hyperdense appearance and occasional stippled calcifications. Transbronchial biopsy showed material with refraction of polarized light, consistent with silicosis
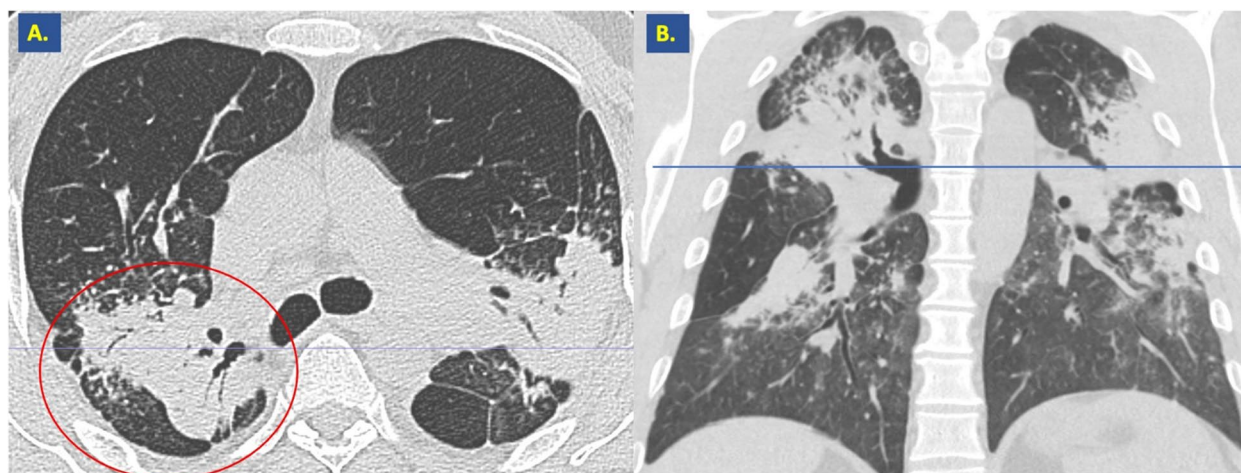
Fig. 6 Complicated Silicosis/Progressive Massive Fibrosis. A 44-year-old man who worked in the engineered stone countertop manufacturing industry, presented with dyspnea. CT Chest (**A**, axial; **B**, coronal) revealed large bilateral posterior-upper lung predominant masses with partially-sharp margins, hilar and pulmonary distortion, bronchiectasis and mild para-cicatricial emphysema (red circle), consistent with progressive massive fibrosis. Given the typical imaging findings in the setting of the occupational history, a biopsy was not performed
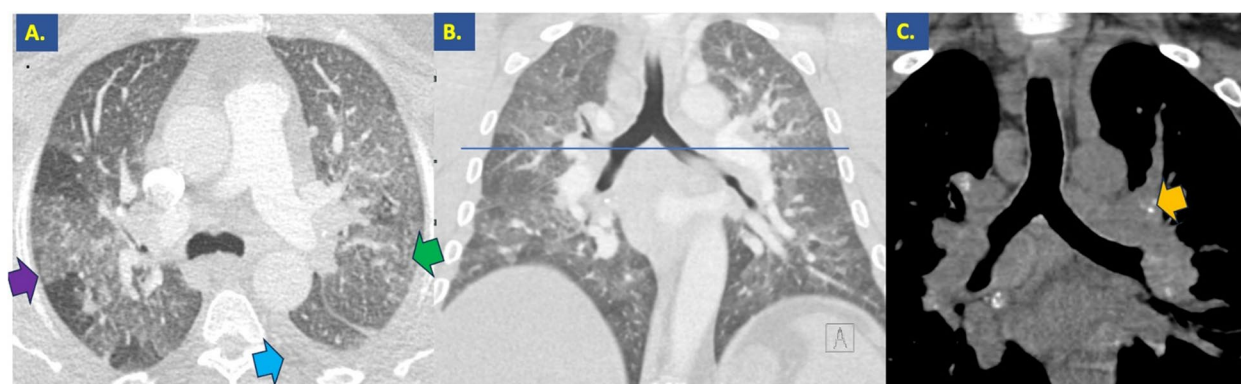

Fig. 7 Acute Silicosis. A 43-year-old male who worked as a stone-cutter since age 17 years, presented with acute dyspnea, dry cough and chest pain for one week in 2016. The patient did not have these symptoms when evaluated one month prior by a physician. Chest CT (**A**, axial at the carina; **B** and **C**, coronal) showed diffuse heterogeneous mosaic ground-glass attenuation (purple arrow) with indistinct underlying nodularity and areas of subtle "crazy-paving" (green arrow), a small left pleural effusion (blue arrow) and multi-compartmental mediastinal hyperdense lymphadenopathy with stippled calcification (yellow arrow). Differential workup was negative, including for cardiac (heart failure) and infectious (TB, fungal) etiologies. Bronchoalveolar lavage (BAL) showed white, cloudy fluid with pigmented macrophages. Transbronchial pulmonary and mediastinal biopsy showed material with refraction of polarized light. The constellation of findings is consistent with areas of acute silicoproteinosis on early chronic silicosis with lipoproteinaceous inflammatory material

anti-granulocyte-macrophage colony-stimulating factor (GM-CSF) antibodies disrupting the GM-CSF signaling pathway [87, 89]. There is increasing recognition that direct toxicity to alveolar macrophages by either inhaled or systemic toxic processes can create the same clinical presentation by preventing alveolar clearance by the macrophages [90]. Radiographically, silico-proteinosis also appears similar to idiopathic PAP, with areas of peri-hilar ground-glass opacities and septal thickening, often in the upper lobes (Fig. 7) [91]. However, unlike idiopathic PAP, silicoproteinosis is less likely to have a "crazy paving" pattern, though it can be identified [92]. Additionally, lymph node calcification is highly specific for silicosis when differentiating from idiopathic PAP [92].

Accelerated silicosis occurs due to high-level silica exposure and typically develops within 2 to 10 years after the initial exposure. Its imaging characteristics overlap with those of both acute and chronic silicosis, showing ground-glass opacities indicative of silicoproteinosis, along with silicotic nodules and interstitial fibrosis commonly seen in chronic forms of the disease (Fig. 8) [93]. Many workers present with simultaneous findings of fibrosis and areas of acute silicoproteinosis, consistent with their shorter work tenures and higher levels of exposure at the time of diagnosis [8, 93].

Fig. 8 Accelerated Silicosis. The same patient from Figure 7 (A, B, C; CT Chest from 2016) continued to have regular follow-up. The extensive ground-glass opacities and left pleural effusion have resolved, but relatively rapid development of complicated silicosis was noted (red circle). CT Chest from 2017 (**D**) shows upper-lung predominant, subcentimeter, coalescent, perilymphatic nodularity. CT Chest from 2018 (**E**) and (**F**) show progressive massive fibrosis with large consolidative opacities and parenchymal/hilar distortion. The patient was listed for lung transplant

Other silica-related lung diseases

Diffuse dust fibrosis

Workers with a history of silica dust exposure may also present with the *usual interstitial pneumonia* (UIP) pattern, characterized by honeycombing and basilar predominance of abnormalities. Among workers with known silica exposure in all industries, approximately 9–10% have been found to have a UIP pattern radiographically without classical findings of pneumoconiosis/silicosis [94, 95]. Silica exposure is a well-described independent risk factor for interstitial pulmonary fibrosis (IPF) (odds ratio 1.8), and approximately 4% of IPF is attributable to silica dust [96, 97]. Silica particles found on biopsies of patients with IPF were associated with a more rapid decline of pulmonary function [98]. However, case series of ES workers have not described UIP on CT scans [8, 82, 93], though this does not exclude the possibility.

Obstructive lung disease

As noted above, ES, in addition to respirable crystalline silica, also releases vapors of VOCs. Depending on the type of resin used in the slab, chemicals such as styrene, methyl methacrylate, and phthalic anhydride may be produced during the fabrication of the slabs [47, 48]. Many of these have been designated as lung irritants by the American Conference of Governmental Industrial Hygienists (ACGIH) [47, 48], have been linked to obstructive lung diseases such as asthma and constrictive bronchiolitis [99, 100]. Among ES workers with confirmed silicosis,16–33% have demonstrated obstructive or mixed patterns on pulmonary function testing (PFTs) [8, 24, 82]. Additionally, 11% of exposed workers in Australia without confirmed silicosis radiographically had PFTs with an obstructive or mixed pattern concerning for either asthma or chronic obstructive lung disease [24]. An outbreak investigation by Tustin et al. reported an outbreak of work-related asthma (WRA) in a US countertop manufacturing and fabrication facility where among 58 workers, 5 had confirmed WRA and 11 had suspected WRA [101].

Lung cancer

Lung cancer is the most common cause of death from cancer worldwide, causing nearly 1 in 5 (18.4%) cancer deaths. Tobacco is the dominant known cause, however, this is in the setting of widespread under-recognition of occupational determinants of lung cancer [97]. The IARC has identified multiple occupational exposure factors as being carcinogenic to the human lung, including respirable crystalline silica [102]. A meta-analysis by Shahbazi et al. found a significant dose-response relationship between silica exposure and lung cancer [103]. For this reason, there should be a high index of suspicion in patients with asymmetric enlarging nodules, new cavitation, or the development of systemic symptoms such as weight loss or fevers [79].

Sarcoidosis

Exposure to silica dust has been shown to increase the risk of developing sarcoidosis, particularly in certain populations. A Swedish case-control study found that men exposed to respirable silica dust were significantly more likely to develop sarcoidosis than unexposed controls, with the risk highest in younger men (≤35 years) and those with prolonged exposure [104]. Silica dust is thought to act as an environmental trigger in genetically predisposed individuals, initiating immune responses that lead to granuloma formation. This finding aligns with other research suggesting that silica exposure could play a role in the development of inflammatory diseases like sarcoidosis, although the mechanisms remain under investigation [105, 106].

However, misdiagnosis of silicosis as sarcoidosis is a critical challenge, especially in occupational settings involving high silica exposure, such as artificial stone

fabrication. This is not surprising considering the radiographic overlaps as well as the presence of granulomas in both diseases on pathology [27, 107]. Cases have been reported where accelerated silicosis, characterized by multiorgan involvement and rapid disease progression, was initially misdiagnosed as sarcoidosis due to overlapping clinical features like granulomas and elevated ACE levels [107]. In these cases, detailed occupational histories and tissue analyses revealing silica particles were ultimately key to distinguishing silicosis from sarcoidosis. Similarly, misdiagnosis of sarcoidosis has delayed appropriate treatment and removal from further silica exposure in case series of ES workers [8, 27]. These findings underscore the importance of incorporating occupational exposure data into diagnostic practices to avoid misclassification and ensure appropriate treatment. Finally, as sarcoidosis is not (to our knowledge) recognized or compensated as a disease resulting from occupational exposure in any country, the diagnostic confusion between sarcoidosis and silicosis may increase the loss of opportunities for workers to access their social rights.

Silica-associated systemic diseases
Autoimmune diseases
The association between silicosis and autoimmune disease has been apparent since the early to mid-1900s when scleroderma was observed in stone masons [108], systemic sclerosis (SSc) was found among South African gold miners (Erasmus Syndrome) [109], and rheumatoid arthritis (RA) was described in coal miners (Caplan's Syndrome) [110]. Silica exposure has additionally been linked to systemic lupus erythematosus (SLE), dermatomyositis, polymyositis, and antineutrophil cytoplasmic antibody (ANCA) associated vasculitis, among other rheumatological diseases [81, 111, 112]. The association between silicosis and rheumatologic disease has continued to be observed among ES countertop workers with rates of autoimmune disease around 20%, an estimated 7 times higher than the expected prevalence in the general population, most commonly RA, SSc, and SLE, but also ANCA vasculitis and others [25, 86]. Rates of autoimmune and inflammatory marker elevation were also found to increase in prevalence among ES countertop workers based on the severity of disease with anti-nuclear antibody (ANA) and angiotensin-converting enzyme (ACE) prevalence of 24.2% and 10.3% if exposed without silicosis, 34.0% and 34.8% with simple silicosis, and 47.1% and 55.6% with complex silicosis, respectively [113]. Likelihood of positive autoantibodies in silicosis has additionally been associated with increasing age, smoking, and higher exposure to RCS [114]. Among a large cohort of over 1,000 exposed workers with and without silicosis, confirmed autoimmune disease occurred in about 1% [114].

The proposed mechanism for silica-induced autoimmunity is that silica-stimulated macrophages in the lung trigger the activation of the inflammasome, which catalyzes the release of pro-inflammatory mediators such as Interleukin 1-Beta (IL-1b), Tumor Necrosis Factor-alpha (TNF-a), and interferons [115]. Because macrophages cannot degrade silica particles, the sustained activity of macrophages leads to nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activation and the production of reactive oxygen species in the mitochondria. Abundant reactive oxygen species result in macrophage death, release of silica particles, and continued inflammation [115].

Mycobacterial infections
An association between tuberculosis and silicosis has also been described, with a recent meta-analysis yielding a pooled relative risk of 4.01 for the development of tuberculosis among patients with silicosis [116]. For instance, in a cohort of 2,758 former South African Miners, the prevalence of silicotuberculosis was 25.7% [117]. Among ES countertop workers in California, 2 in 45 total (4%) patients had confirmed diagnoses of active tuberculosis (TB) [118]. However, because silicosis and tuberculosis have similar symptomatology and imaging findings (micronodular pattern in the upper lung fields), 10 of 45 (22%) were initially misdiagnosed with active pulmonary TB, as opposed to silicosis, which was ultimately ruled out after many had received months of systemic multidrug therapy for active TB [118]. Therefore, although pulmonary tuberculosis remains a relevant comorbid condition in ES silicosis, it may be mistaken as the primary pathology due to its similar miliary micronodular imaging pattern.

Nontuberculous mycobacterial (NTM) infection is associated with underlying lung disease, including in cystic fibrosis and COPD, as well as silicosis. Until the mid-1900s, NTM were considered colonizers, not pathogens. However, in 2007, a consensus definition for pulmonary NTM infection was established and includes symptoms, positive culture for NTM on either two sputum samples or one bronchoalveolar lavage sample, as well as characteristic fibro-cavitary or nodular imaging findings [119]. In ES silicosis, cohorts in Israel and California, found that 9% of patients have NTM disease, the same prevalence as found in silicosis of any cause in a Brazilian cohort from 1999 to 2023 [8, 120, 121]. Predominant imaging findings are cavitary lesions, and the most prominent species include *Mycobacterium* (*M*.) *kansasii*, *M. abscessus*, *M. intracellulare*, *M. fortuitum* and *M. xenopi*. Small case series suggest that comorbid NTM may additionally portend increased mortality and is found more often in the advanced stages of silicosis, i.e., PMF [120, 122, 123]. The geographic prevalence of different mycobacterial

species varies widely. In the United States, the prevalence of NTM is greater than TB, potentially contributing to the higher proportions of NTM than TB in patients with ES silicosis, despite most being immigrants from Latin America. Further study is needed to understand the unique biological and epidemiologic risk factors for NTM disease development in ES silicosis.

The underlying mechanism of association between silicosis and mycobacterial disease is incompletely understood. However, in vitro models have shown that pulmonary macrophage exposure to crystalline silica deregulates cell death pathways, which limits the macrophage's ability to control Mycobacterium tuberculosis growth. This process is associated with increases in pro-inflammatory markers such as TNF-alpha, which ultimately promote necrosis and lead to the progression of tuberculosis [124].

Renal disease
Silica exposure has long been associated with an increased risk of developing renal disease [125–128]. In ES silicosis, the multinational registry by Hua et al. found an estimated prevalence of kidney disease of 10%. However, these were predominantly reported from Israel, where patients were older and had more advanced silicosis [21, 82]. Histologically, many cases show findings consistent with proliferative glomerulonephritis or Immunoglobulin A deposition [129]. The mechanisms underlying silica nephropathy are incompletely understood and likely are caused by a combination of the direct toxic effect of deposited crystalline material in the renal parenchyma or a macrophage-activating autoimmune process caused by silica particles [130, 131].

Evaluation and treatment
The diagnostic work-up of silicosis among ES countertop workers includes a thorough medical and occupational history, radiology, pulmonary function testing, and optional pathology.

Occupational history
A detailed exposure history is crucial in evaluating a stone countertop worker (Table 1). To develop a broader picture of their dust exposure, asking questions regarding work conditions, materials used, and dust control measures is critical. This evaluation should be more in-depth than job title alone, as the level of exposure can vary widely between workplaces depending on job tasks, available engineering controls, work practices, and personal protective equipment. Questions can include overall dustiness, amount of artificial stone (versus natural stone or other materials), types of machinery used, wet or dry cutting/fabricating practices, ventilation, and personal protective equipment. Contributory information can be the presence of disease in coworkers.

Clinical signs and symptoms
Symptoms from chronic silicosis can be insidious, with a prolonged asymptomatic period out of proportion to the severity of imaging. Common respiratory symptoms include shortness of breath, chronic dry cough, chest pain, and back pain. As the disease progresses, weight loss and hypoxia become more common [8]. As noted above, a thorough medical history and physical exam should be taken to evaluate for respiratory disease and other systemic conditions such as opportunistic infections and autoimmune disease.

Pulmonary function testing
Pulmonary function testing demonstrates a range of abnormalities, though restrictive ventilatory pattern is the most common [8, 24, 25]. However, many ES workers with abnormal CT scans of the chest have normal PFTs, especially at the time of diagnosis. In an Italian case series of those with an abnormal CT scan, only 33% had abnormal spirometry, while 50% had abnormal diffusion capacity [107]. Interestingly, even among those with progressive massive fibrosis, only 47.1% demonstrated normal spirometry in one case series in Australia [24]. Therefore, the sensitivity of pulmonary function testing for diagnosis is less than a CT scan of the chest but is a marker for progression and disability.

Imaging findings
Silicosis demonstrates characteristic imaging findings that vary depending on the stage and form of the disease. Simple silicosis is characterized *by* numerous, small, predominantly rounded opacities (measuring less than 10 mm in diameter) with a centrilobular and peripheral (perilymphatic) distribution and predilection for the posterior upper and middle zones of the lung. These nodules increase in profusion as the disease progresses. A hallmark finding is enlarged and hyperdense multi-compartmental mediastinal lymph nodes, often with hilar/subcarinal/paratracheal predominance [132].

As simple silicosis advances, it becomes characterized as PMF, defined as the presence of opacities greater than 1 cm in diameter. These masses are often bilateral and symmetrical, predominantly located in the upper and middle lung zones, and become progressively accompanied by stigmata of fibrosis. These large opacities initially form in the periphery of the lung, migrating centrally over time, either unilaterally or symmetrically. These mass-like opacities often occur superimposed upon a background of small nodules, as seen in simple silicosis. The severe scarring and volume loss can distort mediastinal and parenchymal anatomy with adjacent

Table 1 Critical elements of an occupational history for engineered stone (ES) workers

Occupational History Factors	Details
What do you do for work?	Chronology of jobs - Years of employment in the stone countertop fabrication industry - Year of employment in construction jobs that may include installation or demolition of ES slabs or other exposure to respirable crystalline silica
What are your current and past job tasks?	Examples - Manufacture of ES - Machine operator cutting large slabs (usually with a water-fed CNC machine) - Small tool operator performing polishing, cutting, or grinding on slabs - Fabrication - Lamination - Installation - Helper - Office worker - Manager or Supervisor
Assessing Dust/Workplace Conditions	Types of stone (slabs) used - Proportion of time working with artificial/ES - Years When did the increased use of ES slabs start? Engineering controls - Frequency and trend of dry work - Frequency and trend of wet work - Were tools using water sufficiently keeping dust levels under control - Use of local exhaust ventilation, waterfall ventilation systems, or supplemental water systems to further reduce dust Protected areas - Number of employees working in shared space - Work in an enclosed area (such as a shed or booth) - Frequency around others doing dry work or turning water systems Housekeeping Compressed air used to clean off surfaces or clothing (high exposure risk) - Were drains on the floor removing water slurry out of the shop, or was the slurry remaining on the floor and drying overnight to leave behind dust? - Amount of settled dust observable on the floor and work surfaces
Personal Protective Equipment	Respirator or mask use - Type of mask/respirator was worn (filtering facepiece respirator or N-95, ½ mask elastomeric respirator, full face-piece respirator, Powered Air Purifying Respirator or PAPR, or supplied air respirator) - Respirator loose-fitting hood or helmet or a tight-fitting respirator. - Fit testing for the respirator conducted - Presence of facial hair - Consistency of respirator use over time and in different conditions (heat stress or during specific tasks) - Where respirators were stored and if they were cleaned

paracicatricial emphysematous destruction [80]. Necrosis/cavitation may occur. Due to the carcinogenic nature of RCS, lung cancer is an important differential diagnosis [103], as is superimposed mycobacterial or other infection [116]. For instance, if a lesion is relatively large and unilateral, lung cancer should be considered, and close nodule follow-up or biopsy may be indicated according to the Fleischner Society guidelines for the management of incidental pulmonary nodules [133].

Traditional surveillance methods, such as chest X-rays, are less sensitive compared to modern imaging modalities like HRCT scans and often fail to detect early-stage disease. This is a critical shortcoming, as individuals diagnosed early can benefit from timely interventions, including removal from further silica exposure, to prevent accelerated disease progression. As a result, there is a growing recognition of the need to update and improve international surveillance strategies, incorporating cost-effective tools and modern diagnostic technologies to enable earlier detection and more comprehensive worker protection.

In simple silicosis, HRCT better defines the nodules, revealing their centrilobular and subpleural distribution in a perilymphatic pattern while demonstrating hilar lymphadenopathy. CT scan of the chest demonstrates an enhanced ability to detect early disease before it becomes visible on conventional radiographs and can better characterize abnormalities by distinguishing between silicotic nodules and other causes of nodular opacities [33]. In complicated silicosis, HRCT better delineates mass borders and internal architecture while showing associated emphysema more clearly, particularly the paracicatricial emphysema around the masses [134, 135]. Additional features may be observed, including various patterns of lymph node calcification, associated emphysema, and

possible complications such as tuberculosis and pleural thickening or plaques.

HRCT provides superior visualization of these findings and is more sensitive in detecting silicosis when compared to chest radiography. Two Australian case series of ES silicosis demonstrated that 35–43% of ES fabricators with silicosis had an abnormal chest CT scan demonstrating silicosis despite having a normal chest radiograph [93, 113]. Another Italian study demonstrated that among 24 workers diagnosed with silicosis via CT scan, only 42% demonstrated an abnormal chest radiograph [57]. For these reasons, Australian and Californian regulators have updated the screening requirements from X-rays to mandate chest CT scans due to their higher sensitivity for early disease [36, 53, 63].

Medical screening and surveillance

Medical surveillance for silicosis plays a crucial role in identifying and protecting at-risk workers, but current approaches face significant limitations. These programs are often overly dependent on air monitoring, which, while essential, is expensive and not feasible in many occupational settings, particularly in low-resource regions. Because of the high risk of over-exposure in the ES industry, areas such as California and Australia have changed screening guidelines for all in the industry rather than relying on air monitoring [36, 63].

In Western European countries, medical surveillance programs for silica-exposed workers are typically systematic and integrated into occupational health frameworks. For example, the French Haute Autorité de Santé (HAS) has developed comprehensive guidelines that provide a structured approach to surveillance beyond imaging alone [136]. These guidelines outline detailed protocols for occupational physicians including exposure assessment methodologies, recommended frequencies of health examinations, standardized questionnaires for respiratory symptoms, pulmonary function testing parameters, and decision algorithms for different exposure scenarios. The HAS guidelines also address post-exposure surveillance requirements, an often-overlooked component of silicosis management. For occupational physicians seeking comprehensive surveillance protocols, these guidelines offer valuable reference.

In the United States, OSHA has established specific medical surveillance requirements under its Respirable Crystalline Silica standard (29 CFR 1910.1053), which mandates baseline and periodic medical examinations for workers exposed above the action level for 30 or more days per year. These examinations must include medical and work histories, physical examinations, chest X-rays interpreted by B-readers, pulmonary function tests, and screening for latent tuberculosis infection [54]. The National Institute for Occupational Safety and Health

(NIOSH) further supplements these requirements with detailed guidance through its B-Reader Program for standardized pneumoconiosis classification [137]. In Australia, responding to the emerging epidemic of accelerated silicosis in engineered stone workers, surveillance guidelines have evolved significantly with the adoption of the Royal Australian and New Zealand College of Radiologists (RANZCR) protocol, which now recommends low-dose CT scans rather than traditional radiography as the primary screening tool for all workers in this industry regardless of exposure duration [138], complemented by comprehensive respiratory function testing and standardized respiratory questionnaires at regular intervals [36, 73].

Furthermore, effective medical surveillance extends beyond diagnostic imaging to incorporate multidisciplinary approaches. This includes standardized respiratory symptom questionnaires, comprehensive pulmonary function testing with diffusion capacity measurement, and consideration of newer biomarkers that may indicate subclinical silica-induced inflammation. Given the varying regulatory frameworks and healthcare systems globally, occupational physicians and public health officials should consider adaptation of these more comprehensive approaches to their specific contexts while maintaining the core elements of early detection, proper exposure assessment, and timely intervention.

Treatment and disease management

There is currently no effective treatment for silicosis. Patients with pneumoconiosis have largely been excluded from pharmacotherapeutic studies for interstitial lung diseases [139]. Interventions are supportive and include using supplemental oxygen for hypoxia, vaccination against respiratory pathogens, and identifying and treating concomitant autoimmune diseases or infections. Scientific investigation and clinical trials are ongoing for immunosuppression, whole lung lavage, antifibrotics, and other novel therapeutics. Thus far, the primary treatment modality for end-stage lung disease due to silicosis, as in other progressive pulmonary diseases, is lung transplantation [21, 82].

Progression and prognosis

Silicosis often progresses regardless of whether exposure to RCS persists after diagnosis. Historically, the proportion of individuals experiencing disease progression is high—generally one-third to two-thirds [140, 141], even up to 88% in one study of South African gold miners from 1988 [142]. Risk factors for silicosis progression include greater radiological severity and younger age at diagnosis, rapid initial increase in the size and number of opacities, and increased cumulative exposure [140]. Considering their high exposure level, ES workers face

an exceptionally high risk of silicosis progression and severity despite shorter work tenures [36]. It is widely accepted that ongoing exposure accelerates silicosis progression. An extensive Swedish longitudinal registry of silicosis cases with diverse exposures and 20-year follow-up published in 1980 found that radiographic disease progression was more likely in individuals with continued exposure than those who ceased exposure [141]. Similarly, ongoing exposure was linked to a heightened risk of progressive massive fibrosis (PMF) among South African and Brazilian gold miners [142, 143]. Although the probability of progression is most influenced by cumulative silica exposure, a study of Iranian quarry workers found that progression occurred in around half of patients 10 years after complete exposure cessation, slightly more so in those already with PMF as opposed to simple silicosis [144]. ES silicosis progression after cessation appears much more rapid, with 56% of patients with simple silicosis showing progression after only four years and 37.7% progressing to PMF in one Spanish study [7]. Figures 7 and 8 demonstrate a progression from acute silicosis to PMF. PMF has a poor prognosis with rapid progression to cardiopulmonary failure in the absence of lung transplantation [21, 83].

Conclusions

ES silicosis represents a growing occupational health crisis driven by the widespread use of high-silica materials and insufficient regulatory oversight in the stone countertop fabrication industry. The global rise in cases underscores the need for proactive interventions, including implementing stricter workplace safety standards, widespread adoption of engineering controls, and personal protective equipment to minimize workers' exposure to respirable crystalline silica. Public health initiatives, such as mandatory screening programs, surveillance systems, and worker education, are critical to identify cases early, mitigate disease progression, and prevent new cases. The example of Australia's ban on ES products and active screening efforts demonstrates the success of concerted, systematic interventions to provide solutions to the epidemic of ES silicosis.

The disproportionate impact on marginalized workers, including immigrant populations, highlights the broader social inequities in occupational health. Many affected workers lack adequate access to healthcare and are underinsured, compounding the challenges of timely diagnosis and treatment. Addressing this crisis requires a multidisciplinary upstream approach integrating public health policies, occupational safety regulations, and targeted outreach to vulnerable communities. Governments, employers, and healthcare systems must collaborate to create safer working environments, provide affordable healthcare access, and prioritize the well-being

of at-risk populations. Without these coordinated efforts, the burden of silicosis among ES workers will continue to rise, perpetuating preventable suffering and economic costs.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not appliable.

Competing interests
The authors declare no competing interests.

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