HHS Public Access

Author manuscript

Arch Environ Occup Health. Author manuscript; available in PMC 2023 February 09.

Published in final edited form as:

Arch Environ Occup Health. 2023 ; 78(2): 118–126. doi:10.1080/19338244.2022.2105287.

Hazardous dusts from the fabrication of countertop: a review

W. Kyle Mandler[a], **Chaolong Qi**[b], **Yong Qian**[a]

[a]Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Morgantown, WV, USA

[b]Division of Field Studies and Engineering, National Institute for Occupational Safety and Health, Cincinnati, OH, USA

Abstract

Artificial countertop materials, including solid surface composites (SSC) and engineered stone (ES) may pose significant pulmonary health risks for workers who manipulate them. These materials have rapidly become popular in the multibillion-dollar countertop industry, rivaling that of natural materials such as granite and marble due to their variety of desirable esthetic qualities and reduced costs. Both SSC and ES consist of a mineral substrate bound together in a polymer matrix. For SSC the mineral is about 70% aluminum trihydrate (ATH) while ES contains up to 95% crystalline silica by weight. Both materials emit airborne dusts when being manipulated with power tools during the fabrication process. Several deaths and dozens of cases of silicosis have been identified worldwide in workers who fabricate ES, while a single case of fatal pulmonary fibrosis has been associated with SCC dust exposure. This review examines the current state of knowledge for both SSC and ES regarding the composition, particle emission characteristics, workplace exposure data, particle constituent toxicity, and possible methods for reducing worker exposure.

Keywords

Aluminum trihydrate; airborne dust; engineered stone; pulmonary toxicology; respirable crystalline silica; solid surface composites

Background

Workers in the countertop industry, including those who manufacture, fabricate, and install commercial and residential countertops and bathroom fixtures, may not at first glance, appear to be at risk for serious dust-related pulmonary occupational hazards. Indeed, while their work may not elicit images of dust-laden air associated with underground miners, sandblasters and concrete cutters, countertop workers have been increasingly reported to be at risk due to hazards associated with the composition of some emerging popular materials they fabricate.[1] While workers in this industry may install countertops made from a variety

CONTACT W. Kyle Mandler, oex1@cdc.gov, National Institute for Occupational Safety and Health, Health Effects Laboratory Division, 1095 Willowdale Road, Morgantown, WV 26505, USA.

Disclosure statement

The authors declare that they have no competing interests.

of natural materials such as granite, marble, slate, and soapstone, this review focuses on the most popular manmade materials—solid surface composite (SSC)—and engineered stone (ES) and addresses the questions that have been raised in the last decade about the occupational safety of working with both ES and SSC.

SSC material is composed of a proprietary mixture of approximately 70% powdered alumina trihydrate ($Al(OH)_3$, ATH) bound together in an acrylic polymer matrix, along with other binders, pigments, and trace metals[2] SSC is usually extruded as slabs of varying thickness by the manufacturer, and then transported to local fabrication shops. Workers in the shops cut, grind, drill, sand, and polish the materials to fit the desired application. Fabricating SSC generates respirable airborne particulates of mixed composition, as well as small amounts of volatile organic chemicals (VOCs).[3]

ES is a manmade material with increasing popularity in the countertop industry around the world. Composed of an aggregate of crystalline silica (mostly quartz and occasionally cristobalite) and various polymer binders, ES materials can contain greater than 90% of crystalline silica by mass and are manufactured in large slabs.[4] Following manufacturing, the slabs are transported to local fabrication shops where they are shaped to meet the needs of each customer. Working with ES generates respirable crystalline silica (RCS), in fabrication shops and installation sites. Grinding of stone countertop using a handheld grinder is a fabrication task associated with the highest RCS exposure.[5,6] Overexposure to RCS leads to progressive, irreversible, and potentially disabling pulmonary silicosis and is associated with increased risk of lung, autoimmune diseases, tuberculosis, cancer, emphysema and kidney disease.[7] The Occupational Health and Safety Administration (OSHA) estimated that 12,085 workers in the greater stone cutting industry were exposed to RCS in the workplaces in 2013. Of those, 7,441 experienced exposures greater than the previous permissible exposure limit (PEL) of 0.1 mg m^{-3}.8 Recognizing that RCS remains a serious occupational hazard in the United States, in 2016, OSHA reduced its PEL from 0.1 to 0.05 mg m^{-3} as an eight-hour time-weighted-average.[9] In February of 2020, a new National Emphasis Program for RCS was instituted, specifically targeting the countertop industry for increased scrutiny for worker RCS exposures (Table 1).[10]

Main text

Components of SSC are associated with lung toxicity

There have not yet been any scientific investigations of lung disease in workers exposed to airborne emissions while working with SSC materials. However, exposure to principal components of the SSC emissions has been associated with lung disease in humans and animal models. Shaver and Riddell reported rapidly fatal pneumoconiosis in approximately 20% of observed alumina abrasives manufacturing workers.[11] In addition, manufacturing plastic-containing acrylate polymers releases ultrafine particles and VOCs.[12] About 5–13 months of pulmonary exposure to polyacrylate nanoparticles induced shortness of breath and pleural effusions in a group of 7 female workers (aged 18 – 47 y); lung pathology revealed nonspecific inflammation, fibrosis and granulomas, and transmission electron microscopy revealed acrylate nanoparticles deposited in the cytoplasm of pulmonary epithelial and mesothelial cells.[13] Animal studies showed that intratracheal instillation ATH-produced

numerous confluent nodules and firm fibrotic masses at 60- day post-exposure, and by day 210 the majority of the lungs had developed into a mass of confluent collagenous tissue.[14] While there are no studies reporting data on workplace exposure to SSC emissions, these findings suggest that SSC emissions constituents have the potential to induce lung injury in humans.

SSC particle characteristics

The physicochemical characteristics of airborne particles from some types of SSC have been determined in the previous studies. Qi et al.[3] used an automated tool-testing system in a custom-built chamber to generate and measure the emissions from sawing SSC in a controlled environment.[3] They found that the respirable (diameter < 5 μm) particles in the emissions were 82% ATH by mass, and the emitted particles have a mode at 1.05 μm (the number based geometric mean diameter) and another mode for ultrafine particles at 11.8 nm. In the follow-up studies, Kang et al. sought to determine the content of ATH across a range of particle sizes from both sawing and sanding SSC, as well as to characterize any emitted VOCs.[15,16] Although the particle size distribution is affected by the fabrication process (eg, sawing vs. sanding), the aluminum content in the particles is consistent between fabrication processes and sandpaper materials, suggesting that the SSC material is the main source of the aluminum. Methyl methacrylate (MMA) was the most abundant VOC from both sawing and sanding SSC, but its generation rate was much lower from sawing and does not vary much with different sandpaper materials. Considering the generation rates of respirable particles and MMA during sanding and sawing SSC, the exposure to respirable particles not otherwise regulated (PNOR) could reach its PEL much faster than the exposure to individual VOCs (including MMA) could reach their PELs. To date, there have been no published data regarding workplace measurements of particle exposures during the SSC fabrication process, but these laboratory findings suggest that working with SSC materials using power tools can generate respirable particles at hazardous concentrations.

Exposure to SSC emissions has been associated with lung disease

Thus far, a single case of idiopathic pulmonary fibrosis associated with exposure to SSC emissions has been reported—that of a man, aged 64, who had worked with SSC materials in a home workshop for 16 years.[17] Raghu et al. reported that the subject machined, ground, drilled, and sanded SSC. It is unclear what types of dust-mitigation techniques were used, if any, and photos of the workspace depict piles of accumulated SSC dust. Radiography of the patient's lungs indicated interstitial pneumonia, while polarized light microscopy of lung biopsy sections revealed birefringent particles were localized in areas of fibrosis. Electron and dispersive X-ray spectroscopy of these particles indicated that they were composed primarily of aluminum and oxygen. Further Raman spectroscopy analysis indicated the presence of ATH and aluminum oxide. A single-case study is insufficient to causally link the SSC emission to pulmonary fibrosis; however, the presence of ATH deposits in the lung and no other known patient history of fibrogenic exposures suggest that the accumulation of SSC particles in the lungs contributed to the observed outcome.

In vivo and in vitro investigations of the toxicity of SSC emissions

Our group has investigated the potential for toxicity of the emissions from sawing SSC.[18,19] To characterize the acute toxicity and particle clearance, male mice were exposed to either phosphate buffer saline (PBS, negative control), 62.5, 125, 250, 500, or 1000 μg of SSC particles or 1000 μg crystalline silica (Min-U-Sil 5; US Silica Company, Berkeley Springs, West Virginia, positive control) via oropharyngeal aspiration. Body weights were measured for the duration of the study. Bronchoalveolar lavage fluid (BALF) and tissues were collected for analysis at 1- and 14-days post-exposure. Enhanced-darkfield and histopathologic analysis was performed to assess particle distribution and inflammatory responses. BALF cells and inflammatory cytokines were measured. The geometric mean diameter of the particles from sawing SSC following suspension in PBS was 1.25 μm. BALF analysis indicated that lactate dehydrogenase (LDH) activity, inflammatory cells, and pro-inflammatory cytokines were significantly elevated in the 500 and 1000 μg SSC particles exposure groups at days 1 and 14, suggesting that exposure to these concentrations of SSC particles induced inflammatory responses. Histopathology indicated the presence of acute alveolitis at all doses at day 1, which was largely resolved by day 14. Alveolar particle deposition and granulomatous mass formation were observed in all exposure groups at day 14. The SSC particles were poorly cleared, with 81% remaining at the end of the observation period.

Considering the importance of alveolar macrophages to the development of pulmonary fibrosis, we exposed a model of human alveolar macrophages (THP-1) to (<5 μm) SSC particles *in vitro.* We also determined relative toxicities of size fractions (0.07, 0.66, 1.58, 5.0, and 13.42 μm diameter) of the airborne particles. Differentiated macrophages were exposed for 24 h to respirable particles from sawing SSC at concentrations of 0, 12.5, 25, 50, or 100 μg/ml PBS. In the size-fraction experiment, one dose (100 μg/ml) was used for each size. Exposure to respirable SSC particles induced THP-1 macrophage toxicity in a dose-dependent manner. Viability was decreased by 15% and 19% after exposure to 50 and 100 μg SSC particles/ml, respectively, which correlated with increased cell culture supernatant LDH activity by 40% and 70% when compared to control. Reactive oxygen species (ROS) production and inflammatory cytokines increased in a dose-dependent manner. For the size-selected particles, we observed a size-dependent relationship to toxicity. SSC particles of 0.07, 0.66, and 1.58 μm diameter killed 36%, 17%, and 22% of cells, respectively, while the larger particles showed no significant effect. These results indicate a potential for cytotoxicity of respirable SSC particles and a relationship between particle size and toxicity, with the smallest fractions appearing to exhibit the greatest toxicity.

Occupational exposure to engineered stone dust (ESD) is associated with lung disease

Epidemiologists have described several clusters of silicosis related to the ES fabrication industry. The earliest cases were detected in Israel and Spain when ES products were relatively new on the market. Kramer et al. studied patients with silicosis associated with ES over a 14-year period. During that time, 25 cases of silicosis with restrictive lung disease were observed, with 10 of those eventually receiving lung transplants, and 3 patients died while waiting for their transplant. The average length of employment was 18.5 years. All

silicosis cases were associated with dry cutting ES material. Compared to International Society for Heart and Lung Transplantation registry data, the incidence of silicosis among these workers was more than 14x higher than average.[20] Another cluster was reported in Spain in 2014 involving 46 cases of silicosis.[21] Among these workers, the median age was 33, with 11 years on average working in the industry. Pérez-Alonso et al. reported that poor industrial hygiene, little or no dust mitigation, and inadequate or missing personal protective equipment were commonplace, even when performing dry cutting tasks. In addition, mandatory annual chest radiographies were not performed to screen for emerging silicosis. In the United States, the first case of ESD-associated silicosis was reported in 2014 in Texas, where a 37-year-old male had been exposed to ESD for 10 years.[1] In subsequent years, 18 cases of silicosis, and 2 deaths, in ES workers have been reported in California, Colorado, Texas, and Washington states.[4] In 2018, the government of Australia undertook a nationwide program of silicosis screening for all at-risk ES workers. Of the 799 workers screened, silicosis was identified in 98 (12%), with 15 suffering from progressive massive fibrosis.[22] These findings suggest that silicosis in the ES industry may be more prevalent than indicated through self-reporting alone. Considering that ES has only become widely popular in this industry in the last two decades, and with latency periods observed for some workers in the range of 10–20 years, many workers with chronic exposure to ESD may only just now be experiencing symptoms of silicosis.

Exposure to ESD is associated with changes in lung function

Functional changes in the lungs are often noted in ESD exposures. Ophir et al. observed significant reductions in diffusion lung carbon monoxide in a single breath, forced expiratory volume, forced vital capacity, total lung capacity, and vital capacity in a group of 68 workers with up to 20 years of occupational exposure to ESD, compared to unexposed control. Furthermore, the authors found a significant association between the severity of these functional deficits and the number of respirable-sized silica particles in sputum collected from subject airways.[23] Hoy et al. noted restrictive and obstructive defects, reduced gas transfer, and progressive declines in forced expiratory volume and ventilatory capacity.[24] Pulmonary function testing showed restrictive defects with reduced diffusion capacity.[4] Wu et al. found that workers with silicosis from exposure to ESD had more severe outcomes with fewer years of exposure than workers with natural stone dust-induced silicosis.[25]

Acute, chronic, and accelerated silicosis

There appear to be differences between ESD-associated silicosis and silicosis from other occupational sources.[26] The principal trend reported globally is the short time of exposure to ESD to the onset of symptoms, which appears to be approximately 10 years. Some cases are occurring after only a few years of exposure.[4,24] Along with rapid onset and progression of symptoms, another hallmark of ESD-silicosis is the severity of symptoms and poor prognosis for affected workers. Incidence of disabling loss of lung function, lung transplants, and death appear to be higher in workers exposed to ESD than comparable occupations with RCS exposure.[20] Taking these trends into account, it may be useful to understand the etiology of silicosis and its various categories.

Silicosis can be broadly categorized via pathophysiology into two ways: acute or chronic. So-called acute silicosis differs from chronic types in several ways. For acute silicosis, the histology reveals proteinaceous fluid filling the alveolar space. The source of this proteinaceous material appears to be hypertrophic type ii pneumocytes.[27] Interstitial fibrosis and pneumonitis, with small silicosis nodules may also be presented.[28] Acute silicosis is also differentiated from chronic silicosis in the speed of progression of symptoms. Alveolar proteinosis may progress rapidly, with onset of symptoms usually occurring in less than 4 years of exposure.[29] The prognosis for patients with acute silicosis is poor; a bilateral lung transplant is often required. Typically, cases of acute silicosis are associated with extreme occupational overexposures to very fine crystalline silica dust of very high purity, such as used in sandblasting, abrasives manufacture, or drilling and blasting through quartz rock.[30]

In contrast to the acute form, chronic silicosis typically develops only after prolonged exposure to RCS, with onset occurring after 20 years or more. Histopathologic analysis of lung tissue typically reveals well-developed silicotic nodules. These nodules eventually mature into dense, largely acellular bundles of deposited collagen. Polarized light microscopy can detect birefringent polyhedral silica particles of 1 and 2 μm in diameter interspersed among the nodules. Radiography shows rounded opacities. Functionally, chronic silicosis results in restrictive lung disease, causing impaired gas exchange and dyspnea.

Castranova et al. postulated a four-step process for the development and progression of chronic silicosis.[31] First, inhaled RCS reaches the deep lung and causes direct cytotoxicity. Surface silanol groups may act as H-donors and damage biological membranes.[32] $Si^{\cdot}$ and $SiO^{\cdot}$ radicals formed on freshly fractured cleavage planes can generate ROS, causing lipid peroxidation and cell damage.[33,34] Second, alveolar macrophages are recruited to phagocytize the RCS, resulting in further oxidant production.[35] Third, macrophages, recruited polymorphonuclear leukocytes, and alveolar type II epithelial cells release proinflammatory cytokines and chemokines.[36] Finally, alveolar pneumocytes secrete fibrogenic factors, including platelet-derived growth factor (PDGF) and fibronectin.

A third classification of silicosis exists that shares features of both acute and chronic forms and has been termed “accelerated” silicosis. Similar to acute silicosis, accelerated is associated with shorter, intense exposures, typically in the range of 2–10 years.[37–40] Histologically, in a pattern more resembling early chronic silicosis, the accelerated form is also associated with fibrotic masses composed of collagen and reticulin enveloping silica particles. The alveolar septa undergo type II epithelial cell hypertrophic and hyperplastic thickening.[37,41]

Exposure to ESD is associated with autoimmunity

Silicosis has been associated with systemic inflammation and resultant autoimmune disorders for more than a century as cases of diffuse scleroderma were first described in a group of stone masons.[42–44] Research has linked diseases such as systemic lupus erythematosus,[45–47] systemic sclerosis,[48] and rheumatoid arthritis.[42,49,50] The biologic mechanism underlying silica-induced autoimmune disorders is not clear. The presence of elevated antinuclear antibodies (ANA) is often reported in a subset of silicosis cases.[51] In a

group of 68 patients with silicosis of varying progression, lung antibodies were detected in 47. Of that antibody positive group, all expressed IgG lung antibodies, while 32 presented IgA, and IgM were detected in 15.[52]

Autoimmune disorders have also been described in patients with ESD-associated silicosis. Shtraichman et al. found that in 40 cases of silicosis in Israeli countertop fabricators, 9 (23%) presented autoimmune symptoms.[53] Of these nine subjects, six reported symptoms of arthritis, four Raynaud's disease, two dysphagia, one myalgia, and one xerostomia. Six out of nine subjects were smokers, a factor which has been found to exacerbate silicosis and autoimmunity.[54] Turner et al. recently identified a group of three Australian workers with an average exposure duration of 19.3 years. In addition to functional deficits, these workers presented clinical features of autoimmunity including arthropathy, sicca, Raynaud's phenomenon, and positive autoantibody titers. A common theme among these workers was dry cutting techniques generating very high exposures, coupled with minimal or no respiratory PPE.[55] In a survey of 18 cases of ES workers with silicosis across several states, Rose et al. identified five workers with symptoms of autoimmune disease. This subset of patients was found to suffer from either rheumatoid arthritis or scleroderma. Crucially, in some workers who presented with autoimmune symptoms, silicosis was not initially suspected. Only after lung biopsy was performed on these subjects was their silicosis discovered.[4]

ESD characterization and exposure control

Efforts have recently been made to characterize emissions from manipulating ES materials with various powered tools. Johnson et al. measured respirable particle emissions and the fraction of RCS in a controlled, simulated workspace environment.[56] The emissions generated by different tasks included cup grinding, wheel grinding, polishing, blade cutting, and core drilling. Each task was performed with and without local exhaust ventilation (LEV), as well as with or without wetting techniques. The ES material used in this case contained >85% quartz. They found that, when operated dry with no LEV, silicon carbide wheel grinding resulted in the highest RCS exposure, followed by sawing and cup-grinding. The percentage of crystalline silica in the collected particulate mass averaged 53% overall (range: 9.3–76.2%).

Following the hierarchy of controls,[57] eliminating or substituting crystalline silica in the ES is the most preferred method of reducing worker exposure to RCS. Using a strategy of short-term task-based sampling, Qi et al. reported that working with ES alone led to significantly higher RCS exposures than working with mixed stone types (ES and granite, which is a natural stone) with $P = 0.03$ for grinding and $P \ll 0.01$ for polishing.[8] Carrieri et al. determined the physicochemical characteristics of particles generated by grinding three types of ES and compared them to the particles from grinding granite.[58] Overall, grinding the granite produced the most respirable particles. However, the types of ES with high (90%) silica content, produced 3 and 4-fold greater RCS than granite (30% silica), even though the overall respirable particles concentration was lower with ES. The type of ES with low (9%) silica content, unsurprisingly, produced the least RCS, approximately the third of the amount as granite, and far less than the high silica ES materials.

Engineering control is the preferred exposure control method after elimination and substitution. In virtually all countertop workshops, practices often involve sawing, grinding, polishing, and other work that generates airborne particles. A variety of engineering controls have been reportedly used to reduce emissions and the workers' exposure to RCS. However, the real-world implementation of sufficient engineering control methods is far from ideal. Phillips et al. found that approximately 74% of countertop shops in three metropolitan areas of Oklahoma reported using predominantly dry methods in at least one step of their work, and four shops (9%) reported using no particle collection or suppression systems at all.[59] Utilizing wet-sawing techniques has been shown to reduce mean respirable particle and RCS exposure more than 10-fold. Local exhaust ventilation (LEV), similar to what has been employed to reduce welding fume exposures for metal fabricators, can reduce exposure by an additional 10-fold for sawing ES, from an original 69–0.6 mg m^{-3}.60 However, the reduced RCS exposure was still significantly higher than the OSHA PEL of 0.05 mg m^{-3}. When grinding stone countertops in a dust control booth with a filtration unit, the short-term RCS exposure was reduced by 78.7%.[61] Utilization of tools modified with water jets, or a sheet-wetting method that wet the entire work surface continuously, in combination with LEV appears to be able to further reduce RCS exposures during ES edge grinding and blade cutting. However, addition of LEV to some tools with spray-wetting attachments may reduce their effectiveness, so a comprehensive understanding of the effects of these measures is required for each tool.[56] Without the interference of LEV, using the wetting method of combining water spray and sheet-wetting, the short-term RCS exposure was found to be reduced to 0.033 ± 0.011 mg m^{-3} for grinding mixed stone types (ES and granite), which was below the OSHA PEL of 0.05 mg m^{-3}56 (Table 1).

In vitro study of ESD toxicity

To date, there has been only one *in vitro* investigation of ESD toxicity.[63] studied the toxicity of several types of ESD when incubated with human bronchial epithelial cells (BEAS-2B). Compared to silica control, the freshly generated ESD samples produced more ROS, although this difference was reduced over the following hours. In assays of hemolysis and LDH release, all ESD samples generated negligible values when compared to silica. After thermal degradation of the polymer coating the particles, cytotoxicity was increased. In contrast to their hemolysis and LDH assay results, markers for epithelial-mesenchymal transition, a critical step in the development of fibrosis, were increased by all ESD samples, while the presence of a polymer coating on the samples had no effect.

Conclusions and the future directions

There are currently gaps in the knowledge regarding the occupational exposure risk and toxicity mechanisms of both ESD and SSC emissions. For ESD, while several ongoing studies are attempting to characterize the particles emitted during various real-world fabrication tasks, an international effort has been made to identify workers with ESD-associated silicosis, changes in lung function, and autoimmunity. In comparison, relatively little is known about occupational exposures to SSC emissions and only one case of lung disease has been associated with this material. It may be the case that SSC emissions are simply less hazardous, or workers are not exposed to quantities sufficient to cause disease. More information regarding the workers' exposure to SSC emissions is required to

make these determinations. Additionally, working with SSC materials releases VOCs, the inhalation of which in conjunction with solid particles has not yet been investigated.

The rapid progression of accelerated silicosis in ESD-exposed workers is of concern. It is unclear if these cases are the result of massive overexposure to the RCS released by working with these materials, or if it is something intrinsic to the material (trace metals, pigments, and organics) that potentiates this effect. More controlled cell culture and animal studies are required to elucidate the molecular mechanisms underpinning this observed toxicity. Considering the numbers of reported ESD-associated silicosis cases, more prevention efforts are needed to protect workers in this occupational sector. Industrial hygiene techniques that may have been sufficient at keeping RCS under regulatory limits when working with natural stone may be inadequate to protect workers fabricating ES, which contain much greater amounts of crystalline silica as well as polymer binders. While it is beyond the scope of this review to proscribe changes to the industry, the hierarchy of controls doctrine would suggest that elimination of the RCS hazard through a change in material composition may be the most effective course of action.

Although the nature of particles and VOCs differ between emissions from ES and SSC, we observed that both may induce pulmonary damage. Considering the size of the countertop fabrication industry and the sometimes-variable emission controls used in these workplaces, it is imperative that we identify which components of these materials are causing toxicity, as well as any systemic implications they may present.

Funding

The author(s) reported there is no funding associated with the work featured in this article.

List of abbreviations:

ATH	aluminum trihydrate
BALF	bronchoalveolar lavage fluid
ES	engineered stone
ESD	engineered stone dust
LDH	lactate dehydrogenase
LEV	local exhaust ventilation
MMA	methyl methacrylate
OSHA	Occupational Health and Safety Administration
PEL	permissible exposure limit
RCS	respirable crystalline silica
SSC	solid surface composite

VOC	volatile organic compounds

References

1. Friedman GK, Harrison R, Bojes H, Worthington K, Filios M. Centers for Disease C, et al. Notes from the field: silicosis in a countertop fabricator - Texas. MMWR Morb Mortal Wkly Rep. 2014;64(5):129–130.
2. DuPont. Corian Solid Surface Product Overview. Wilmington, DE: DuPont; 2018.
3. Qi C, Echt A, Murata TK. Characterizing dust from cutting Corian(R), a solid-surface composite material, in a laboratory testing system. Ann Occup Hyg. 2016;60(5):638–642. doi:10.1093/annhyg/mew005. [PubMed: 26872962]
4. Rose C, Heinzerling A, Patel K, et al. Severe silicosis in engineered stone fabrication workers – California, Colorado, Texas, and Washington, 2017–2019. MMWR Morb Mortal Wkly Rep. 2019;68(38):813–818. doi:10.15585/mmwr.mm6838a1. [PubMed: 31557149]
5. Zwack LM, Brueck SE VK, Qi C. Evaluation of Crystalline Silica Exposure during Fabrication of Natural and Engineered Stone Countertops. HHE Report, 2016 Contract No.: 2014–02153250. Atlanta, GA: U.S. Department of Health and Human Services Centers for Disease Control and Prevention; 2016.
6. Qi C, Ea Lo L. editor Exposure to Respirable Crystalline Silica during Stone Countertop Grinding/Polishing using Handheld Tools. The 36th Annual Conference of Amercian Association for Aerosol Research 2017. October 16–20, 2017; Raleigh, NC.
7. National Institute for Occupational Safety and Health. Health Effects of Occupational Exposure to Respirable Crystalling Silica (Publication no. 2002–129). Atlanta, GA: Department of Health and Human Servies, Centers for Disease Control and Prevention; 2002.
8. Deparment of Labor, Occupational Safety and Health Administration. Occupational Exposure to Respirable Crystalline Silica; Proposed Rule. Washington, DC: Deparment of Labor, Occupational Safety and Health Administration; 2013.
9. Occupational Exposure to Respirable Crystalline Silica, Final Rule. Washington, DC: Deparment of Labor, Occupational Safety and Health Administration; 2016.
10. Occupational Safety Health Administration. National Emphasis Program—Crystalline Silica. Directive Number: CPL 03–00-023; 2020.
11. Shaver CG, Riddell AR. Lung changes associated with the manufacture of alumina abrasives. J Ind Hyg Toxicol. 1947;29(3):145–157. [PubMed: 20296238]
12. Unwin J, Coldwell MR, Keen C, McAlinden JJ. Airborne Emissions of Carcinogens and Respiratory Sensitizers during Thermal Processing of Plastics. Ann Occup Hyg. 2013;57(3):399–406. [PubMed: 23091110]
13. Song Y, Li X, Du X. Exposure to nanoparticles is related to pleural effusion, pulmonary fibrosis and granuloma. Eur Respir J. 2009;34(3):559–567. doi:10.1183/09031936.00178308. [PubMed: 19696157]
14. Stacy B, King E, Harrison C, Nagel-schmidt G, Nelson S. Tissue changes in rats' lungs caused by hydroxides, oxides and phosphates of aluminium and iron. J Pathol Bacteriol. 1959;77(2):417–426. doi:10.1002/path.1700770212. [PubMed: 13642189]
15. Kang S, Liang H, Qian Y, Qi C. The composition of emissions from Sawing Corian®, a solid surface composite material. Ann Work Expo Health. 2019;63(4):480–483. doi:10.1093/annweh/wxz009. [PubMed: 30852605]
16. Kang S, Liang H, Qian Y, Qi C. The composition of emissions from Sanding Corian® with different sandpapers. Aerosol Air Qual Res. 2021;21(2):200377. doi:10.4209/aaqr.2020.07.0377. [PubMed: 35371190]
17. Raghu G, Collins BF, Xia D, Schmidt R, Abraham JL. Pulmonary fibrosis associated with aluminum trihydrate (Corian) dust. N Engl J Med. 2014;370(22):2154–2156.
18. Mandler WK, Qi C, Orandle MS, et al. Mouse pulmonary response to dust from sawing Corian®, a solid-surface composite material. J Toxicol Environ Health A. 2019;82(11):645–663. doi:10.1080/15287394.2019.1640816. [PubMed: 31290376]

19. Mandler WK, Kang S, Farcas M, Qi C, Friend SA, Qian Y. In vitro toxicity assessment of respirable solid surface composite sawing particles. Toxicol Ind Health. 2020;36(4):250–262. doi:10.1177/0748233720921683. [PubMed: 32379541]
20. Kramer MR, Blanc PD, Fireman E, et al. Artificial stone silicosis [corrected]: disease resurgence among artificial stone workers. Chest. 2012;142(2):419–424. doi:10.1378/chest.11-1321. [PubMed: 22383661]
21. Pérez-Alonso A, Córdoba-Doña JA, Millares-Lorenzo JL, Figueroa-Murillo E, García-Vadillo C, Romero-Morillos J. Outbreak of silicosis in Spanish quartz conglomerate workers. Int J Occup Environ Health. 2014;20(1):26–32. doi:10.1179/2049396713Y.0000000049. [PubMed: 24804337]
22. Kirby T Australia reports on audit of silicosis for stonecutters. Lancet. 2019;393(10174):861. doi:10.1016/S0140-6736(19)30478-7. [PubMed: 30837133]
23. 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;76(12):875–879. doi:10.1136/oemed-2019-105711. [PubMed: 31562234]
24. Hoy RF, Baird T, Hammerschlag G, et al. Artificial stone-associated silicosis: a rapidly emerging occupational lung disease. Occup Environ Med. 2018;75(1):3–5. doi:10.1136/oemed-2017-104428. [PubMed: 28882991]
25. 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):518–524. doi:10.1111/resp.13744. [PubMed: 31828940]
26. Leung CC, Yu IT, Chen W. Silicosis. Lancet. 2012;379(9830):2008–2018. doi:10.1016/S0140-6736(12)60235-9. [PubMed: 22534002]
27. Hoffmann EO, Lamberty J, Pizzolato P, Coover J. The ultrastructure of acute silicosis. Arch Pathol. 1973;96(2):104–107. [PubMed: 4352308]
28. Buechner HA, Ansari A. Acute silico-proteinosis: a new pathologic variant of acute silicosis in sandblasters, characterized by histologic features resembling alveolar proteinosis. Dis Chest. 1969;55(4):274–284. doi:10.1378/chest.55.4.274. [PubMed: 5775743]
29. Chapman EM. Acute silicosis. JAMA. 1932;98(17):1439–1441. doi:10.1001/jama.1932.02730430015006.
30. Gardner LU. Pathology of so-called acute silicosis. Am J Public Health Nations Health. 1933;23(12):1240–1249. doi:10.2105/ajph.23.12.1240. [PubMed: 18013881]
31. Castranova V From coal mine dust to quartz: mechanisms of pulmonary pathogenicity. Inhalation Toxicol. 2000;12(sup3):7–14. doi:10.1080/08958378.2000.11463226.
32. Karlsson HL, Toprak MS, Fadeel B. Chapter 4 - toxicity of metal and metal oxide nanoparticles. In: Nordberg GF, Fowler BA, Nordberg M, editors. Handbook on the Toxicology of Metals. 4th ed. San Diego: Academic Press; 2015. p. 75–112.
33. Vallyathan V, Shi X, Dalal N, Irr W, Castranova V. Generation of free radicals from freshly fractured silica dust. Am Rev Respir Dis. 1988;138(5):1213–1219. doi:10.1164/ajrccm/138.5.1213. [PubMed: 2849348]
34. Castranova V, Pailes WH, Dalai NS, et al. Enhanced pulmonary response to the inhalation of freshly fractured silica as compared with aged dust exposure. Applied Occupational and Environmental Hygiene. 1996;11(7):937–941. doi:10.1080/1047322X.1996.10389993.
35. Vallyathan V, Mega JF, Shi X, Dalal NS. Enhanced generation of free radicals from phagocytes induced by mineral dusts. Am J Respir Cell Mol Biol. 1992;6(4):404–413. doi:10.1165/ajrcmb/6.4.404. [PubMed: 1312851]
36. Driscoll KE, Maurer JK. Cytokine and growth factor release by alveolar macrophages: potential biomarkers of pulmonary toxicity. Toxicol Pathol. 1991;19(4 Pt 1):398–405. doi:10.1177/0192623391019004-108. [PubMed: 1667554]
37. Nakladalova M, Stepanek L, Kolek V, Zurkova M, Tichy T. A case of accelerated silicosis. Occup Med (Lond). 2018;68(7):482–484. doi:10.1093/occmed/kqy106. [PubMed: 30053129]
38. Baird T, Putt M, Dettrick A. A stonemason with accelerated silicosis in the setting of tumour necrosis factor alpha inhibitor therapy. Respirol Case Rep. 2016;4(5):e00171. [PubMed: 27516887]

39. Hutyrova B, Smolkova P, Nakladalova M, Tichy T, Kolek V. Case of accelerated silicosis in a sandblaster. Ind Health. 2015;53(2):178–183. doi:10.2486/indhealth.2013-0032. [PubMed: 25567156]
40. Tse LA, Li ZM, Wong TW, Fu ZM, Yu IT. High prevalence of accelerated silicosis among gold miners in Jiangxi, China. Am J Ind Med. 2007;50(12):876–880. doi:10.1002/ajim.20510. [PubMed: 17948247]
41. Castranova V, Vallyathan V. Silicosis and coal workers' pneumoconiosis. Environ Health Perspect. 2000;108 Suppl 4:675–684. doi:10.1289/ehp.00108s4675.
42. Parks CG, Conrad K, Cooper GS. Occupational exposure to crystalline silica and autoimmune disease. Environ Health Perspect. 1999;107 Suppl 5:793–802. doi:10.1289/ehp.99107s5793. [PubMed: 10970168]
43. Pollard KM. Silica, silicosis, and autoimmunity. Front Immunol. 2016;7:97. [PubMed: 27014276]
44. Bramwell B Diffuse sclerodermia: its frequency; its occurrence in stone-masons; its treatment by fibrolysin—Elevations of temperature due to fibrolysin injections. Edinburgh Medical Journal. 1914;12(5):387.
45. Conrad K, Mehlhorn J, Luthke K, Dorner T, Frank KH. Systemic lupus erythematosus after heavy exposure to quartz dust in uranium mines: clinical and serological characteristics. Lupus. 1996;5(1):62–69. doi:10.1177/096120339600500112. [PubMed: 8646229]
46. Mehlhorn J, Gerlach C. Coincidence of silicosis and lupus erythematosis. Z Erkr Atmungsorgane. 1990;175(1):38–41. [PubMed: 2219993]
47. Hrycek A, Olszanecka-Glinianowicz M. Silicosis and systemic lupus erythematosus–case report. Pol Merkur Lekarski. 2008;24(139):18–19.
48. Ferri C, Artoni E, Sighinolfi GL, et al. High serum levels of silica nanoparticles in systemic sclerosis patients with occupational exposure: possible pathogenetic role in disease phenotypes. Semin Arthritis Rheum. 2018;48(3):475–481. doi:10.1016/j.semarthrit.2018.06.009. [PubMed: 30093240]
49. Klockars M, Koskela RS, Jarvinen E, Kolari PJ, Rossi A. Silica exposure and rheumatoid arthritis: a follow up study of granite workers 1940–81. Br Med J (Clin Res Ed). 1987;294(6578):997–1000. doi:10.1136/bmj.294.6578.997.
50. Turner S, Cherry N. Rheumatoid arthritis in workers exposed to silica in the pottery industry. Occup Environ Med. 2000;57(7):443–447. doi:10.1136/oem.57.7.443. [PubMed: 10854495]
51. Doll NJ, Stankus RP, Hughes J, et al. Immune complexes and autoantibodies in silicosis. J Allergy Clin Immunol. 1981;68(4):281–285. doi:10.1016/0091-6749(81)90152-4. [PubMed: 6974746]
52. Cojocaru M, Niculescu T, Spătaru E. Study of lung antibodies in patients with silicosis. Rom J Intern Med. 1995;33(3/4):243–247. [PubMed: 8646197]
53. Shtraichman O, Blanc PD, Ollech JE, et al. Outbreak of autoimmune disease in silicosis linked to artificial stone. Occup Med (Lond). 2015;65(6):444–450. doi:10.1093/occmed/kqv073. [PubMed: 26070814]
54. Stolt P, Yahya A, Bengtsson C, EIRA Study Group, et al. Silica exposure among male current smokers is associated with a high risk of developing ACPA-positive rheumatoid arthritis. Ann Rheum Dis. 2010;69(6):1072–1076. doi:10.1136/ard.2009.114694. [PubMed: 19966090]
55. Turner MT, Samuel SR, Silverstone EJ, Yates DH. Silica exposure and connective tissue disease: an underrecognized association in three Australian artificial stone workers. Am J Respir Crit Care Med. 2020;201(3):378–380. doi:10.1164/rccm.201905-1057LE. [PubMed: 31597045]
56. Johnson DL, Phillips ML, Qi C, Van AT, Hawley DA. Experimental evaluation of respirable dust and crystalline silica controls during simulated performance of stone countertop fabrication tasks with powered hand tools. Ann Work Expo Health. 2017;61(6):711–723. doi:10.1093/annweh/wxx040. [PubMed: 28927166]
57. The National Institute for Occupational Safety and Health (NIOSH). Hierarchy of Controls. Morgantown, WV: Centers for Disease Control and Prevention; 2015.
58. 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):1–15.

59. 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):368–373. doi:10.1080/15459624.2013.789706. [PubMed: 23668829]

60. Cooper JH, Johnson DL, Phillips ML. Respirable silica dust suppression during artificial stone countertop cutting. Ann Occup Hyg. 2015;59(1):122–126. [PubMed: 25326187]

61. Qi C, Echt A. In: CDC/NIOSH/DFSE, ed. Field Evaluation of a Mobile Dust Control Booth for Stone Countertop Grinding. In-depth Survey Report: Houston, TX Field Survey (EPHB Report # 2020-DFSE-165, NIOSHTIC # 20058064)Oxford UK: Oxford University Press; 2019.

62. Hall S, Stacey P, Pengelly I, Stagg S, Saunders J, Hambling S. Characterizing and comparing emissions of dust, respirable crystalline silica, and volatile organic compounds from natural and artificial stones. Ann Work Expo Health. 2022;66(2):139–149. doi:10.1093/annweh/wxab055. [PubMed: 34331440]

63. Pavan C, Polimeni M, Tomatis M, et al. Editor's Highlight: Abrasion of Artificial Stones as a New Cause of an Ancient Disease. Physicochemical Features and Cellular Responses. Toxicol Sci. 2016;153(1):4–17. doi:10.1093/toxsci/kfw101. [PubMed: 27255382]

Table 1.

Composition and particle emission characteristics of SSC and ES.

	Solid surface composite	Engineered stone
Composition	Approximately, 70% powdered alumina trihydrate (ATH); 30% acrylic polymer matrix along with other binders, pigments, and trace metals[3]	Crystalline silica (as high as 90%+); various polymer binders
Particle emission characteristics	From sawing: respirable particles were 82% ATH by mass; and the emitted particles have bimodal size distribution with the number-based geometric mean diameters at 1.05 μm and 11.8 nm for the two modes	From sawing: crystalline silica content in the emitted particles is mostly consistent with that in the bulk material; and the emitted particles have bimodal size distribution with the mass-based geometric mean diameters at 6 μm and 9 μm for the two modes[62]