MINISTRY OF BUSINESS, INNOVATION & EMPLOYMENT
HĪKINA WHAKATUTUKI

COVERSHEET

Proactive release of 'Review of scientific evidence relating to the risks of working with engineered stone'

Minister	Minister Van Velden	**Portfolio**	Workplace Relations and Safety
Cabinet paper	N/A	**Date to be published**	7 March 2024

List of documents that have been proactively released		
Date	**Title**	**Author**
11 December 2024	*Review of scientific evidence relating to the risks of working with engineered stone*	Centre for Public Health Research, Massey University, Wellington

Information redacted **YES / NO** *[select one]*

Any information redacted in this document is redacted in accordance with MBIE's and MFAT's policy on Proactive Release and is labelled with the reason for redaction. This may include information that would be redacted if this information was requested under Official Information Act 1982. Where this is the case, the reasons for withholding information are listed below. Where information has been withheld, no public interest has been identified that would outweigh the reasons for withholding it.

Caveats

This is not a full review of the evidence of health effects associated with respirable crystalline silica exposure, but instead provides brief answers to 9 specific questions posed by MBIE related to specific aspects of health effects and exposure-control options related to occupational respirable crystalline silica exposure.

Review of scientific evidence relating to the risks of working with engineered stone

Centre for Public Health Research, Massey University, Wellington

Dr Amanda Eng, Prof Jeroen Douwes, Prof John Potter, Dr Collin Brooks

11 December 2024

Table of Contents

Introduction

The Ministry of Business Innovation & Employment (MBIE) contracted with the Centre for Public Health Research, Massey University, to conduct a literature review of scientific evidence relating to the risks of working with engineered stone. A condensed summary of the evidence for nine specific questions/statements was requested from MBIE in a table format (see Appendix 1). For readability, we have prepared the responses to the nine questions in a report format using the sub-headings from the table; each of the questions has its own references section. For many of the questions, the evidence is sparse or inconclusive, which largely reflects the infancy of the research area and does not imply absence of risk.

Communique statement 1:

When engineered stone is processed, the dust generated has different physical and chemical properties that likely contribute to more rapid and severe disease

1. Toxicity of submicron particles of amorphous silica (emerging evidence)

1a. What is known with certainty (i.e. established evidence)

There is clear evidence of several toxic and pathophysiological consequences (inflammation, oxidative stress, dyspnea, fibrosis, reduced lung function, emphysema, enlarged pulmonary lymphatic tissue) in *in vitro* and animal studies of exposure to amorphous silica and evidence that the smaller the particle size, the greater the deleterious consequences.[1-3] Many of the observed effects of amorphous silica in animal studies were fully or largely reversible upon cessation of exposure.[3-6]

1b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

In two small human studies, there was some evidence of silicosis following occupational exposure to amorphous silica, although contamination with crystalline silica (CS) could not be ruled out.[7,8] It is therefore unclear whether silicosis was caused by amorphous silica. Several other studies have found no silicosis in workers exposed to amorphous silica;[9-12] some lung function impairment was seen in the chronically exposed but could not be clearly distinguished from the effects of smoking.[9] Thus, amorphous silica exposure is largely not associated with silicosis.

Several animal studies have shown that exposure to amorphous silica nanoparticles can cause systemic inflammation and progression of fibrosis.[5,6,13] Two occupational studies following long-term exposure to amorphous silica fumes also showed fibrosis in a proportion of workers, but evidence remains limited.[14,15] These studies did not specifically focus on submicron particles.

A large study in a population with long-term occupational exposure to amorphous silica (funded by synthetic amorphous silica producers) showed statistically significant associations with impaired lung function and COPD, with strongest associations observed for respirable (<4µm) silica exposures when compared to inhalable (<100 µm) silica exposure (both exposures were based on job task/title and work histories rather than direct individual exposure measurements).[16-18] These findings add to the evidence that smaller particles are more likely to cause harmful effect: although the focus was not specifically on submicron particles, a proportion of respirable silica falls typically in the submicron range and may therefore have played a role.

The International Agency for Research on Cancer (IARC) does not suggest that amorphous silica causes lung (or any other) cancer in humans.[19]

To date, there are too few data on humans to clearly establish the risk of adverse health effects of airborne amorphous silica exposure. Although there are data from *in vitro* and animal studies, human studies that have specifically focused on submicron amorphous silica particles are largely absent and specific evidence of the health effects of submicron particles of amorphous silica is therefore inconclusive.

1c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

It may be that (sub-micron) amorphous silica nanoparticles are associated with an elevated risk of subsequent lung damage and other organ damage (due to translocation of sub-micron particles via the bloodstream) but this will only be clearly established with sufficiently large and well designed studies – in occupationally exposed populations – that characterise exposure to fractions with specified particle size, including sub-micron particles. We are not aware of any such studies in progress or planned.

1d. How does this compare with natural stone or other masonry products

Engineered stone (ES) with amorphous silica and natural stone are two different products, both associated with health consequences upon occupational exposure (with different levels of evidence for each type of stone) but there are no data that provide direct comparisons between the two.

1e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

Given that wet-cutting and other dust control measures reduce overall dust exposure (see section 5e.), it is very likely that it will also reduce the exposure to submicron amorphous silica particles when cutting/grinding ES manufactured from amorphous silica, but this has not been studied. Of note, resuspension of submicron particles once water has evaporated may create secondary exposure, particularly if sludge is not removed from the workplace in a timely and appropriate fashion.

References

1. Miao C, Jia P, Luo C, et al. The size-dependent in vivo toxicity of amorphous silica nanoparticles: A systematic review. *Ecotoxicology and Environmental Safety* 2024;271:115910.
2. Shin SW, Song IH, Um SH. Role of Physicochemical Properties in Nanoparticle Toxicity. *Nanomaterials* 2015; 5(3).
3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Silica. US Department of Health and Human Services, Centers for Disease Control and Prevention (CDC). 2019.
4. Merget R, Bauer T, Kupper HU, et al. Health hazards due to the inhalation of amorphous silica. *Arch Toxicol* 2002;75(11-12):625-34.
5. Murugadoss S, Lison D, Godderis L, et al. Toxicology of silica nanoparticles: an update. *Arch Toxicol* 2017;91(9):2967-3010.
6. Napierska D, Thomassen LC, Lison D, et al. The nanosilica hazard: another variable entity. *Part Fibre Toxicol* 2010;7(1):39. doi: 10.1186/1743-8977-7-39
7. Mohrmann W, Kann J. [Amorphous silica can cause silicosis] In German. *Gentner Verlag, Stuttgart* 1985;587
8. Philippou S, Teschler H, Morgenroth K. [Pulmonary fibrosis after inhalation of amorphous silicic acid] In German. *Zentralbl Pathol* 1992;138(1):41-46.
9. Choudat D, Frisch C, Barrat G, et al. Occupational exposure to amorphous silica dust and pulmonary function. *Br J Ind Med* 1990;47(11):763-6.
10. Plunkett ER, Dewitt BJ. Occupational exposure to Hi-Sil and Silene. Report of an 18-year study. *Arch Environ Health* 1962;5(5):469-72.
11. Volk H. The Health of Workers in a Plant Making Highly Dispersed Silica. *Archives of Environmental Health: An International Journal* 1960;1(2):125-28.
12. Wilson RK, Stevens PM, Lovejoy HB, et al. Effects of chronic amorphous silica exposure on sequential pulmonary function. *J Occup Med* 1979;21(6):399-402.
13. Maser E, Schulz M, Sauer UG, et al. In vitro and in vivo genotoxicity investigations of differently sized amorphous SiO2 nanomaterials. *Mutat Res Genet Toxicol Environ Mutagen* 2015;794:57-74.
14. Robalo-Cordeiro AJA, Baganha MF, Azevedo-Bernarda R, et al. Biological Effects of Fume Silica (Amorphous Type). In: Beck EG, Bignon J, eds. In Vitro Effects of Mineral Dusts. Berlin, Heidelberg: Springer Berlin Heidelberg 1985:489-96.
15. Vitums VC, Edwards MJ, Niles NR, et al. Pulmonary fibrosis from amorphous silica dust, a product of silica vapor. *Arch Environ Health* 1977;32(2):62-8.
16. Antoniou EE, Nolde J, Torensma B, et al. Nine human epidemiological studies on synthetic amorphous silica and respiratory health. *Toxicol Lett* 2024;399 Suppl 1:12-17.
17. Taeger D, McCunney R, Bailer U, et al. Cross-Sectional Study on Nonmalignant Respiratory Morbidity due to Exposure to Synthetic Amorphous Silica. *J Occup Environ Med* 2016;58(4):376-84.
18. Yong M, Morfeld P, McCunney R. Extended Investigation of Exposure to Respirable Synthetic Amorphous Silica Dust and Its Potential Impact on Non-malignant Respiratory Morbidity. Front Public Health 2022;10: Article 801619
19. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. A review of human carcinogens. Part C: Arsenic, metals, fibres, and dusts. Lyon: IARC 2012.

2. Evidence of difference between ultrafine particles generated with natural stone or engineered stone

2a. What is known with certainty (i.e. established evidence)

Only two studies[1,2] have made direct comparisons between airborne ultrafine particles generated by dry cutting and grinding a small number of samples of engineered stone and natural stone (granite). Although these studies suggested some differences in levels of ultrafine particles generated when grinding/cutting these materials, evidence that ES created more fine dust particles was limited (see section 2b. below).

Therefore, we do not know with certainty that high-silica ES can generate higher levels of ultrafine particles when subjected to dry-grinding/-cutting than natural stone subjected to the same activity.

2b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

One study – involving an experimental design compared ultrafine particles generated by dry-cutting/grinding three ES samples (2 with 91% and 1 with <10% silica) and one granite sample (31% silica) – showed a larger number of ultrafine particles generated for the high-silica-content ES than low-silica-content ES and granite.[2] In a similar experimental study,[1] one of two (~90% and ~50%) CS-containing stones showed a higher concentration of ultrafine particles than granite and a low-silica ES. Another study showed that those working with ES had a higher concentration of ultrafine particles in induced sputum than a non-exposed control group, indicating the potential of exposure to ultrafine particles in workers processing ES.[3]

One larger experimental study involving 12 ES and two granite and one marble sample showed no clear difference in average dust-particle size generated during dry-cutting/grinding with average particle size ranging between 300-700 nm.[4] A specific comparison of ultrafine particles was not made. This study found that cutting/grinding generated significantly higher levels of respirable crystalline silica (RCS) compared to natural stone. It also showed that ES showed more irregular shapes with sharp surface edges and fractures, but provided no specific information on ultrafine particles.

Thus, based on a limited number of studies involving a small number of samples, there is suggestive evidence that high-silica ES can generate higher levels of ultrafine particles when subjected to dry-grinding/cutting compared to natural stone subjected to the same activity.

2c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

The reason that current evidence is inconclusive or suggestive only, is that very few studies have assessed the ultrafine dust fraction generated during ES cutting/grinding and made direct comparisons with natural stone using the same experimental set-up. More studies involving a greater number of samples will provide further insights, thus providing a greater confidence level when determining whether observed differences between ES and natural stone are real.

2d. How does this compare with natural stone or other masonry products

See section 2c. above.

2e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

The experiments conducted to date have used dry-cutting/grinding only and no information is therefore available about how wet cutting would affect the generation of ultrafine particles. However, given that wet-cutting and other dust-reducing measures reduce overall dust exposure (see section 5e.), it is very likely that it will also reduce the exposure to ultrafine particles when cutting/grinding ES. However, resuspension of ultrafine particles once water has evaporated may create further exposure, particularly if sludge is not removed from the workplace in a timely and appropriate fashion.

References

1. Thompson D, Qi C. Characterization of the Emissions and Crystalline Silica Content of Airborne Dust Generated from Grinding Natural and Engineered Stones. *Ann Work Expo Health* 2023;67(2):266-80.
2. Carrieri M, Guzzardo C, Farcas D, et al. Characterization of Silica Exposure during Manufacturing of Artificial Stone Countertops. *International Journal of Environmental Research and Public Health* 2020; 17(12).
3. Ophir N, Bar Shai A, Korenstein R, et al. Functional, inflammatory and interstitial impairment due to artificial stone dust ultrafine particles exposure. *Occup Environ Med* 2019;76(12):875-79.
4. Ramkissoon C, Gaskin S, Thredgold L, et al. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. *Sci Rep* 2022;12(1):4351.

3. Presence of resin/VOCs in engineered stone (may influence risks)

3a. What is known with certainty (i.e. established evidence)

Resin makes up approximately 8-20% of (non-sintered) ES,[1] and upon dry-cutting or active machining, resin decomposition can lead to the release of a range of volatile organic compounds (VOCs)/polycyclic aromatic compounds including styrene, pthalic anhydride, benzaldehyde, and toluene.[2-4]

Exposure to VOCs (such as the aforementioned compounds) is associated with a range of respiratory and physiological responses including oxidative stress, inflammation, endothelial dysfunction and smooth muscle constriction; respiratory outcomes in adults include asthma exacerbations and increased morbidity/mortality associated with obstructive lung diseases (reviewed in [5]).

VOCs can coat RCS moieties, potentially affecting their reactivity.[3]

3b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

To date, most work has been experimental and with the exception of one exposure assessment study reported as a conference proceeding[6] there is limited evidence of excessive VOC exposure in ES workers in a real-world setting.

As both can separately affect respiratory physiology, immunity and function, it is plausible that co-exposures to RCS and VOCs increase disease burden for ES workers.

It has been suggested that VOC coating of RCS may potentially alter their reactivity and presentation to the immune system or lung; however, this is speculative and based entirely on *in vitro* work.[3,7]

3c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

There is currently no conclusive evidence that co-exposures to RCS and VOCs may increase disease burden for ES workers.

Mechanisms by which VOCs and RCS may interact on the respiratory system are unclear. It is also unclear whether effects may be additive or synergistic.

Epidemiological studies measuring adverse respiratory outcomes and both RCS and VOCs in workers involved in ES fabrication/processing will provide further insight into whether there are additive or synergistic effects of combined exposures. Similarly, animal and *in vitro* work focused on combined exposures will increase our understanding around this.

3d. How does this compare with natural stone or other masonry products

Natural stone or sintered products do not contain resin, therefore do not release VOCs during cutting; this potential co-exposure is therefore not relevant when handling natural stone.

3e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

Standard methods for dust control and personal protective equipment (PPE) designed to reduce dust exposure are unlikely to control for the risk of exposure to VOCs – particularly gaseous VOCs. In particular, PPE such as disposable dust masks (P1 or P2) are ineffective against VOCs; a combination air purifying respirator protecting against gases, organic vapours and particles would be required.

References

1. Ramkissoon C, Gaskin S, Thredgold L, et al. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. Sci Rep. 2022;12(1):4351.
2. Ramkissoon C, Gaskin S, Hall T, et al. Engineered Stone Fabrication Work Releases Volatile Organic Compounds Classified as Lung Irritants. Ann Work Expo Health. 2023;67(2):288-93.
3. 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.

4. Hall S, Stacey P, Pengelly I, et al. 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):139-49.
5. Lag M, Ovrevik J, Refsnes M, Holme JA. Potential role of polycyclic aromatic hydrocarbons in air pollution-induced non-malignant respiratory diseases. Respir Res. 2020;21(1):299.
6. Madden C, Davidson M, O'Donnell G, Reed S, editors. Characterisation of respiratory hazards during the manufacture and installation of engineered and natural stone products. 37th Annual Conference and Exhibition of the Australian Institute of Occupational Hygienists; 2019 30 November-4 December; Perth, WA, Australia.
7. Di Benedetto F, Giaccherini A, Montegrossi G, et al. Chemical variability of artificial stone powders in relation to their health effects. Sci Rep. 2019;9(1):6531.

4. Potential contribution of metal ions in engineered stone to disease risk (suggestion)

4a. What is known with certainty (i.e. established evidence)

Although variable, metals typically make up <8% of ES content, and include aluminium, iron, sodium, magnesium, potassium and calcium, and trace amounts of cobalt and titanium.[1-4]

Some of these metals (e.g. aluminium) can be found at high levels in biopsies from silicosis patients and those who have had long-term exposure.[4]

Cobalt, iron and aluminium oxide nanoparticles have various cytotoxic and genotoxic effects on human cell lines *in vitro*, via oxidative stress pathways.[5]

One study has found that aluminium and cobalt content in ES emissions may be associated with *in vitro* cell line cytotoxicity.[6]

Inhalation of metallic compounds is associated with a variety of respiratory diseases, depending on metal type/quantity/format, deposition, clearance/persistence, and host factors (reviewed in [7]); however this has not been studied in workers involved in ES fabrication/processing.

4b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

It is believed that metals in ES originate from pigments and resins.[6]

As they have both been shown separately to cause respiratory disease, it is plausible that co-exposures to RCS and metals can increase disease burden for ES workers, although, again, this has not been studied in workers involved in ES fabrication/processing.

4c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

It is unclear if the excess metals found in biopsies of silicosis patients plays a role in disease or is simply a bystander.

There is currently insufficient evidence that co-exposures to RCS and metals/metal ions increase disease burden for ES workers.

Epidemiological studies measuring adverse respiratory outcomes and both RCS and metals in workers involved in ES fabrication/processing will provide further insight into whether there are additive or synergistic effects of combined exposures. Similarly, animal and *in vitro* work focused on combined exposures will increase our understanding in this area.

4d. How does this compare with natural stone or other masonry products

Natural stone (e.g. granite, marble) often contains more metal (30-40% by weight; mainly aluminium, calcium and magnesium) than ES, although there is considerable variation between different ES, natural stone, and other building materials.[1]

There is currently insufficient evidence to determine whether metals in natural stone affect the risk of RCS on respiratory outcomes.

4e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

There is currently insufficient evidence – however, metal and metal oxide particulate exposure would plausibly be managed using similar controls as with RCS.

References

1. Ramkissoon C, Gaskin S, Thredgold L, et al. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. Sci Rep. 2022;12(1):4351.
2. 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.
3. Di Benedetto F, Giaccherini A, Montegrossi G, et al. Chemical variability of artificial stone powders in relation to their health effects. Sci Rep. 2019;9(1):6531.
4. Leon-Jimenez A, Manuel JM, Garcia-Rojo M, et al. Compositional and structural analysis of engineered stones and inorganic particles in silicotic nodules of exposed workers. Part Fibre Toxicol. 2021;18(1):41.
5. Rajiv S, Jerobin J, Saranya V, et al. Comparative cytotoxicity and genotoxicity of cobalt (II, III) oxide, iron (III) oxide, silicon dioxide, and aluminum oxide nanoparticles on human lymphocytes in vitro. Hum Exp Toxicol. 2016;35(2):170-83.
6. Ramkissoon C, Song Y, Yen S, et al. Understanding the pathogenesis of engineered stone-associated silicosis: The effect of particle chemistry on the lung cell response. Respirology. 2024;29(3):217-27.
7. Nemery B. Metals and the respiratory tract. In: Nordberg GF, Costa M (Eds). Handbook on the toxicology of metals. 5th edition: Elsevier; 2022.

Communique statement 2:

There is no scientific evidence for a safe threshold of crystalline silica content in engineered stone, or that lower silica content engineered stone is safer to work with.

5. Crystalline silica risks stem from exposure not directly from the level of content in materials. Epidemiology is therefore based on exposure, and not content as such

5a. What is known with certainty (i.e. established evidence)

Cumulative RCS *exposure* is conclusively linked to adverse health effects in a wide range of occupational settings. As with other occupational risk factors, health effects are dependent on exposure and dose, which, among other factors, such as dust suppression controls, specific tasks, and process-related variables, are themselves dependent on the CS content in the crude product being processed. This also applies to ES as indicated by simulation studies showing that the higher the CS content, the higher the RCS levels produced during processing.[1,2] Therefore, it is clear that lower CS content in ES reduces exposure, but that the magnitude of the reduction remains difficult to predict as this is, at least in part, dependent on the other factors described above.

5b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

Very few studies of silicosis in ES workers have included measurement of personal RCS levels and none have assessed the combined effects of CS content and RCS exposure. Therefore, although suspected, there is no evidence that ES silica content is proportionally related to risk. A recent study collected personal measurements from ES workers and found that the geometric mean of the RCS/respirable dust (RD) ratio for 8 primary tasks ranged from 0.31-0.56,[3] suggesting that job task affects RCS content in RD, which may be independent of CS content of the crude product, thus indicating an additional factor that may ultimately determine exposure/dose.

5c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

There is a limited number of studies assessing RCS levels in ES workers, including studies of processing lower/zero CS content ES products. Also, there are no comparative health studies assessing risk in workers processing lower/zero CS content ES products and workers processing high CS content ES. It therefore remains unclear whether low CS content ES will substantially reduce the risk of silicosis and other associated health outcomes.

5d. How does this compare with natural stone or other masonry products

Whilst the silica content of natural stone can vary widely, simulation studies that have examined natural stone have found that RCS levels generally reflect the lower CS content of natural stone compared to ES, but it is unclear how this affects risk of adverse health effects. In particular, although a small number of studies have found an increased risk of silicosis in

granite processing workers,[4] very few studies have specifically compared workers processing low- versus high CS granite, or other natural stone.

5e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

The limited number of studies of interventions/controls have focused on RD and RCS. Evidence from both simulation studies and field studies indicates that substantial reductions can be achieved with wet methods and automation.[5,6] Several studies have also indicated that specific wet methods (e.g. sheet wetting) are more effective than others (e.g. water spray only);[7,8] however, these studies specifically assessed cutting/grinding, which is now largely automated.

Although there has been a widespread transition to automation, certain tasks are carried out by hand or are semi-automated, particularly hand polishing. Several field studies, including one conducted recently during 2022-2023, found that high levels (above occupational exposure limits (OELs)) remain for manual tasks such as hand polishing, even with the adoption of wet methods.[3] For some manual tasks, simulation studies have indicated that wet methods combined with local exhaust ventilation (LEV) are more effective,[5,7] whereas one study found the combination to be less effective than individual methods alone.[9] A recent review[10] concluded that there does not appear to be consensus on best practice for wet methods and LEV to control dust at source.

There are no or limited studies on PPE performance and housekeeping practices in ES workers but the detection of high RCS levels despite the use of wet suppression methods suggests that the use of respirators (and regular fit-testing) and sludge disposal management are important.

References

1. Ramkissoon C, Gaskin S, Thredgold L, et al. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. Sci Rep. 2022;12(1):4351.
2. Hall S, Stacey P, Pengelly I, et al. 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):139-49.
3. Weller M, Clemence D, Lau A, et al. An assessment of worker exposure to respirable dust and crystalline silica in workshops fabricating engineered stone. Ann Work Expo Health. 2024;68(2):170-9.
4. Ding MZ, Hou YF, Tian T, et al. Investigation on the current situation of occupational disease hazards in stone processing enterprises in Weihai City. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2024;42(9):712-5.
5. Cooper JH, Johnson DL, Phillips ML. Respirable silica dust suppression during artificial stone countertop cutting. Ann Occup Hyg. 2015;59(1):122-6.
6. Salamon F, Martinelli A, Vianello L, et al. Occupational exposure to crystalline silica in artificial stone processing. J Occup Environ Hyg. 2021;18(12):547-54.
7. Gaskin S, Thredgold L, Ramkissoon C. Evaluation of on-tool dust control measures for processing of engineered stone. Australian Institute of Occupational Hygienists Inc Conference Proceedings. 2022.
8. Qi C, Echt A. Engineering Control of Silica Dust from Stone Countertop Fabrication and Installation – Evaluation of Wetting Methods for Grinding. National Institute for Occupational Safety and Health,. 2021;EPHB Report No. 2021-DFSE-710.

9. Johnson DL, Phillips ML, Qi C, et al. 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):711-23.
10. Ramkissoon C, Gaskin S, Song Y, et al. From Engineered Stone Slab to Silicosis: A Synthesis of Exposure Science and Medical Evidence. Int J Environ Res Public Health. 2024;21(6).

6. The existence of a "tipping point" for exposure, at which there is heightened risk of disease

6a. What is known with certainty (i.e. established evidence)

Occupational health studies in other (non ES-related) industries have provided clear evidence of dose-response associations for RCS and several lung diseases, and elevated risks of silicosis[1] and lung cancer[2] at the lowest estimated cumulative exposure levels reported. An increased risk of silicosis mortality has been observed for levels under permissible exposure limits.[3]

6b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

There is insufficient evidence of a clear no-effect level or threshold for RCS. One study funded by the Crystaline Silica Panel of the American Chemistry Council, and based on inflammatory, oxidative stress, and other mechanistic pathways observed in animal (rat) models, and using data from epidemiological studies (not involving ES-related industries), suggested a "tipping point" or threshold exposure estimate that may trigger lung diseases, including lung cancer, in humans.[4] However, this was based on assumptions that these models provide a true reflection of risk and involved extrapolation from animal models. A recent systematic review of RCS (in all industries) and lung cancer risk concluded that there is no overall consensus on a RCS exposure limit but limits set by different agencies generally range from 0.025mg/m^3 to 0.1mg/m^3.[5]

6c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

Exposure assessment for RCS in ES workers has been limited and whilst it has been suggested that cumulative exposure is important, a clear dose-response association in workers processing ES has not yet been established. A "tipping point", if there is one, therefore cannot be established based on current evidence. Even with more data, this will be difficult to determine as highlighted by the absence of a clear threshold exposure for RCS in other industries. This is further compounded by the fact that there is a risk of silicosis progression even after cessation of silica dust exposure, thus further complicating the assessment of dose-response relationships.

6d. How does this compare with natural stone or other masonry products

Most evidence discussed above is based on RCS exposure in general i.e. mostly natural stone and/or masonry. There is no clear evidence that, if there was a "tipping point" below which no health effects occurred, this would be different for natural stone and ES; however, this may well

be the case as ES contains additional components not found in natural stone, such as resins (discussed in section 3 above).

6e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc.

As indicated in section 5e., a small number of recent field studies have found RCS levels above OELs despite the adoption of wet methods to suppress dust.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Silica. US Department of Health and Human Services, Centers for Disease Control and Prevention (CDC). 2019.
2. Ge C, Peters S, Olsson A, et al. Respirable Crystalline Silica Exposure, Smoking, and Lung Cancer Subtype Risks. A Pooled Analysis of Case-Control Studies. Am J Respir Crit Care Med. 2020;202(3):412-21.
3. Liu Y, Zhou Y, Hnizdo E, et al. Total and Cause-Specific Mortality Risk Associated With Low-Level Exposure to Crystalline Silica: A 44-Year Cohort Study From China. Am J Epidemiol. 2017;186(4):481-90.
4. Cox, J. L. A. An exposure-response threshold for lung diseases and lung cancer caused by crystalline silica. Risk Anal. 2011; 31, 1543-1560
5. Rey-Brandariz J, Martínez C, Candal-Pedreira C, et al. Occupational exposure to respirable crystalline silica and lung cancer: a systematic review of cut-off points. Environ Health. 2023;22(1):82.

7. The relationship between percentage silica content and presence of other materials

7a. What is known with certainty (i.e. established evidence)

There appears to be no clear relationship between percentage silica and presence of other materials in ES; any relationship would be entirely dependent on the nature and proportion of components used in ES manufacture.

7b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

Based on the literature showing that high exposure to RCS, VOCs or metals separately leads to respiratory disease (see specific sections), it is plausible that a combination of these exposures increases disease burden, but this requires confirmation from well-designed human, animal, and *in vitro* studies.

7c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

Epidemiological studies measuring adverse respiratory outcomes as well as VOCs, metals, and other additives used in ES in workers involved in ES fabrication/processing will provide further insight into whether there are additive or synergistic effects of combined exposures. Similarly, animal and *in vitro* work assessing the effects of combined exposures will increase our understanding around the risks of such co-exposures.

7d. How does this compare with natural stone or other masonry products

The percentage of silica and other materials (i.e. metals) varies across materials, including different types of ES, natural stone, or other building products.[1] However, natural stone does not contain organic compounds/resins.

7e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc.

N/A- see sections specific to silica content, resins, metals.

References

1. Ramkissoon C, Gaskin S, Thredgold L, et al. Characterisation of dust emissions from machined engineered stones to understand the hazard for accelerated silicosis. Sci Rep. 2022;12(1):4351.

8. If the percentage silica content were limited it would not be easily measured in any particular case without destructive testing.

8a. What is known with certainty (i.e. established evidence)

Currently available methods to assess CS content require destructive testing. No methods are available that allow CS content to be measured without destructively manipulating engineered (or other) stone.

8b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

There are no non-destructive testing methods that allow even an indication of what the CS contents may be in engineered (or other) stone.

8c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

We are not aware of a method development that will allow CS content in engineered (or other) stone to be measured in real-time and not involve destructive testing. If such a method were to be developed, it would probably not be available in the near future.

8d. How does this compare with natural stone or other masonry products

The same applies to natural stone.

8e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

N/A

9. The silica content of individual slabs of the same product varies.

9a. What is known with certainty (i.e. established evidence)

If the definition of "same product" includes all individual slabs from the same supplier and is covered by the same safety data sheet (SDS) irrespective of the specific "model", then silica content will vary as it varies from model to model due to different proportion of other added materials including other minerals and resins. This is reflected in the SDS, which typically provides a range of silica content (e.g. 70-95% or >88%) rather than a fixed weight-based proportion (e.g. 30%). This has been shown in one study[1] that examined the silica content (and other minerals and resins) of multiple samples obtained from the same suppliers. However, silica was expressed as a % of total crystalline content (including non-silica crystalline minerals) rather than as a weight percentage of the total sample, making it difficult to provide a quantitative estimate of differences across individual samples. Nonetheless, it is clear from this that silica contents vary from model to model even if slabs are from the same supplier and covered by the same SDS.

If the definition of "same product" includes individual slabs from the same model and supplier only, then the variance is expected to be considerably smaller, but this has not been tested empirically.

9b. What is suspected /inconclusive (i.e. there is some evidence or it is suspected)

As noted above, if the definition of "same product" includes individual slabs from the same model and supplier only, then the variance is expected to be considerably smaller, possibly negligible, but this has not been tested empirically.

9c. What is not known now but can be expected to be better understood in the future (ie there is no, or limited, evidence currently, but it can realistically be gathered)

This information is relatively easy to obtain through a well-designed experimental study.

9d. How does this compare with natural stone or other masonry products

Silica content may naturally vary within the same type of stone e.g. the silica content for granite may vary from 0-45%.[2]

9e. What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)

N/A

References

1. Kumarasamy C, Pisaniello D, Gaskin S, Hall T. What Do Safety Data Sheets for Artificial Stone Products Tell Us About Composition? A Comparative Analysis with Physicochemical Data. Ann Work Expo Health. 2022;66(7):937-945.
2. Simcox NJ, Lofgren D, Leons J, Camp J. Silica exposure during granite countertop fabrication. Appl Occup Environ Hyg. 1999;14:577-82.

Appendix 1: Output table requested by MBIE

Communique statement	***Adelaide University advice***	***What is known with certainty*** *(I.e. established evidence)*	***What is suspected /inconclusive*** *(ie there is some evidence or it is suspected)*	***What is not known now but can be expected to be better understood in the future*** *(ie there is no, or limited, evidence currently, but it can realistically be gathered)*	***How does this compare with natural stone or other masonry products***	***What is known about the ability to control for the risk, eg with wet cutting, extraction, PPE etc. (ie since 2019)***
When engineered stone is processed, the dust generated has different physical and chemical properties that likely contribute to more rapid and severe disease.	Toxicity of submicron particles of amorphous silica (emerging evidence)					
	Evidence of difference between ultrafine particles generated with natural stone or engineered stone					
	Presence of resin/VOCs in engineered stone					

	(may influence risks)					
	Potential contribution of metal ions in engineered stone to disease risk (suggestion)					
There is no scientific evidence for a safe threshold of crystalline silica content in engineered stone, or that lower silica content engineered stone is safer to work with.	Crystalline silica risks stem from exposure not directly from the level of content in materials. Epidemiology is therefore based on exposure, and not content as such.					
	The existence of a “tipping point” for exposure, at which there is heightened risk of disease					
	The relationship between the percentage silica content and presence of other					

	materials ~~and risks is not known~~					
	If the percentage silica content were limited it would not be easily measured in any particular case without destructive testing.					
	The silica content of individual slabs of the same product varies.					