

September 30th, 2026

California Division of Occupational Safety and Health (Cal/OSHA)
Department of Industrial Relations
State of California

Re: Public Comment on Emergency Rulemaking to Prohibit the Fabrication of Engineered Stone Countertops Containing More than One Percent Crystalline Silica

Dear Petition Coordinator:

On behalf of Dr. Dennis Paustenbach, Dr. Aryatara Shakya, Mr. Michael Stevens, Mr. Quincy Perry, and Dr. David Weill, we respectfully submit the attached materials in response to Cal/OSHA's request for public comment on the Prohibition of Fabrication and Manufacture of Artificial Stone with more than 1.0% Crystalline Silica, under Petition File No. 609.

This submission consists of a draft scientific review that provides technical analysis and supporting evidence relevant to Cal/OSHA's ongoing evaluation of engineered stone fabrication, occupational RCS exposures, silicosis risk, and appropriate occupational risk-management measures. The attached document is titled:

Comparative Assessment of Respirable Crystalline Silica in Engineered and Natural Stone Fabrication Dust and Human Health Implications

The review examines the scientific literature on RCS exposures and toxicity associated with engineered-stone fabrication and evaluates several prominent scientific and regulatory issues.

- It briefly summarizes the available information on the crystalline silica content of the three most popular stone countertop materials (quartzite [80-100% CS], granite [20-60% CS], and engineered stone).
- It evaluates engineered stone and natural stone exposure data that directly compare NS and ES materials under identical controls and conditions. **It is noteworthy that natural stone materials release more total dust and therefore more RCS proportional to their bulk crystalline silica content under the same fabrication techniques.** This is crucial in evaluating whether a product-composition threshold appropriately reflects the determinants of worker exposure and risk.
- It addresses various suspected physiochemical differences in engineered stone dust compared to natural stone dust and their potential to alter the dose-response for

silicosis. **Ultimately, the weight of scientific evidence does not support the hypothesis that the silicosis dose-response for RCS from engineered stone is materially greater than that from natural stone or other occupational sources.**

- It critically evaluates the use of the term AS-Silicosis. **The current literature does NOT establish that AS-silicosis is a separately classified disease with unique diagnostic, radiologic, or pathologic criteria.** The proposed distinguishing characteristics i.e. short latency, rapid progression, poor outcome, ground-glass opacity, nodular disease, progressive massive fibrosis, cavitation, and infection, substantially overlap with established manifestations and complications of acute, accelerated, and chronic silicosis

Taken together, the evidence reviewed in the attached document is directly relevant to Cal/OSHA's consideration of the proposed prohibition on the fabrication and manufacture of artificial stone containing more than 1.0% crystalline silica. We respectfully encourage Cal/OSHA to consider this analysis, together with the evidence concerning engineering controls, work practices, exposure monitoring, respiratory protection, and enforcement of existing occupational RCS requirements.

The views expressed in this review are solely those of the authors and do not necessarily reflect the views, positions, or policies of their respective employers.

We appreciate Cal/OSHA's consideration of this scientific information and its continuing efforts to prevent occupational silicosis and reduce harmful RCS exposures among workers in the stone-fabrication industry.

Sincerely,

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Dr. Aryatara Shakya

Mr. Michael Stevens

Mr. Quincy Perry

Dr. David Weill

Comparative Assessment of Respirable Crystalline Silica in Engineered and Natural Stone Fabrication Dust and Human Health Implications

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The recent occurrence of silicosis among workers, particularly in the stone fabrication industry, has raised questions about whether airborne respirable crystalline silica (RCS) from different material sources, specifically engineered stone (ES) compared to natural stones (NS), such as granite and quartzite, should be treated as a distinct toxicological hazard. The weight of evidence ultimately supports the conclusion that surface reactivity, particularly the presence and configuration of silanol groups, ability to deposit in the deep lung, biopersistence and, most importantly, dose (inhaled airborne concentration x duration) are the principal drivers of the adverse biological effects of RCS (regardless of the source). Several physiochemical characteristics have been evaluated in the literature for their potential role in enhancing or reducing toxicity, including particle size, morphology, resins, metals, and silica polymorph. An evaluation of these collective characteristics does not suggest that, on an equal RCS basis, engineered stone dust should be treated as a unique hazard in comparison to other common occupational sources of RCS (e.g., natural stone or concrete). Instead, the recent reports of acute, accelerated, and chronic silicosis among stone countertop workers are attributable to inadequate control of RCS exposures. There is overlapping and substantial variation in composition across both natural and engineered stones, but any stone across this range of crystalline silica content can be fabricated safely using proper controls. However, bulk crystalline silica content is not synonymous with exposure, as resin-based materials, under the same conditions, have been shown to release substantially less total dust than solid rock. Consistent with mechanistic and epidemiological evidence, the development of silicosis appears to exhibit a threshold-like behavior, wherein disease occurs only after normal pulmonary clearance and defense mechanisms are repeatedly overwhelmed. Estimates of risk based on epidemiology indicate that disease is not anticipated below the OSHA permissible exposure limit (PEL) for RCS (0.05 mg/m³) as an 8 or 10-hour time-weighted average.

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1. Introduction

The use of engineered stone (ES) in countertops has greatly increased over the past decade and currently represents more than a third of slab materials for new or upgraded countertops in the United States (Houzz 2026). The overall increased demand for countertops, including crystalline silica-containing natural stone (NS), coincides with the US housing boom (Tanner et al. 2026). It was recently estimated that quartzite and granite account for 10-11% and 15-20% of new countertop materials, respectively, over the last few years (Houzz 2026). In combination, these two natural stones make up roughly another third of the marketplace. As such, ES, granite, and quartzite account for the vast majority of modern countertop materials in the US.

In recent years, clusters of silicosis cases reported among workers engaged in the fabrication and installation of countertops, such as those in California, USA and Queensland, Australia (Barnes et al. 2019), have generated considerable attention within the occupational health community. The California Department of Public Health (CDPH) has recently identified 632 confirmed cases of silicosis, 457 of which occurred in Los Angeles or Orange County (California Department of Public Health (CDPH) 2025). Despite these often being reported as ES-associated silicosis incidents, it is important to note that these workers practically always fabricated a combination of ES and NS slabs during their career, rather than working exclusively with either material. To put it in perspective, the CDPH case definition is two-fold: (1) meets the NIOSH definition of silicosis and (2) has silica exposures associated with countertop fabrication (California Department of Public Health (CDPH) 2026). It is important to understand that the NIOSH definition of silicosis is not distinct to a particular material, where the CDPH instead stretches the definition to ES-silicosis to if a stone worker with silicosis fabricated at a minimum one ES in their career (California Department of Public Health (CDPH) 2026). Thus, incidence- or case-report suggestions that this occupational health issue is unique to ES are not well supported.

The absence of systematic disease surveillance in the countertop industry prior to the last few years (2019 in California) limits the ability to establish historical incidence trends (California Department of Public Health (CDPH) 2025). In addition, changes in fabrication practices, dust-control measures, and the increasing use of immigrant labor further complicate interpretation of the available data. Collectively, these factors further limit the conclusions that can be drawn from existing case reports and incidence studies regarding the extent to which the observed disease burden is attributable specifically to engineered stone. In our review of the available toxicological information, there is no reason to suspect that recent incidence of silicosis in this industry is not fully explained as a predictable outcome of excessive RCS exposure and time.

Nonetheless, Australia recently banned the import, manufacture, processing, and installation of engineered stone benchtops, panels, and slabs containing $\geq 1\%$ crystalline silica (Safe Work Australia 2025). Although coinciding regulatory efforts have been made to limit exposures across this industry, this material ban does not apply to any natural stone equivalents (i.e., natural stones containing $\geq 1\%$ crystalline silica). A similar proposal is being considered in California (State of California Department of Industrial Relations 2026). At this time, the evidence that engineered stone is more hazardous than natural stone of the same bulk crystalline silica-content is not convincing and, indeed, may be entirely incorrect. As such, a weight of evidence comparison of physiochemical properties and human health implications of RCS from ES compared to NS seems timely.

Silicosis is neither a novel condition nor one confined to the engineered stone industry. It has been recognized for more than a century across diverse occupational environments in which workers were exposed at length to sufficiently high concentrations of RCS containing dust (National Institute for Occupational Safety and Health (NIOSH) 2002). Indeed, silica-related pneumoconiosis is an ancient disease, with paleopathologic evidence reported in Egyptian mummies (Tapp et al. 1975), although explicit recognition of dust exposure as a cause of respiratory disease is more clearly documented in later Greek and early occupational-medicine writings (Zanatta et al. 2026). Although silicosis in the United States has been on the decline over the past several decades (Centers for Disease Control and Prevention (CDC) 2026), recent litigation and attention in the countertop industry have prompted questions regarding whether RCS derived from ES possesses unique toxicological properties that distinguish it from RCS encountered in more traditional industrial settings.

2. Background

According to the International Union of Geological Sciences (IUGS), granite, the most popular natural stone countertop material, may contain 20-60% quartz by volume (same by weight) (Le Bas and Streckeisen 1991). A more quartz-rich rock would fall under a different definition (e.g., quartzite, which is also commonly used in countertops). Quartzite is often reported as 80 or 90-100% quartz by volume (same by weight) (Le Bas and Streckeisen 1991; Occupational Safety and Health Administration (OSHA) and National Institute for Occupational Safety & Health (NIOSH) 2015; Badouna et al. 2020; HASpod 2022; Tanner et al. 2026). Marble, representing 3-4% of the countertop market (Houzz 2026), typically contains a much lower percentage of silica, generally $\leq 5\%$ by volume (same by weight) (Badouna et al. 2020). However, some veins may be substantially higher.

The distinction between respirable dust (RD) and RCS should be made clear whenever discussing the physiochemical properties of airborne particulates. At times, this has not been done in existing scientific literature. RD refers to the fraction of airborne

particulate matter sufficiently small ($<10\text{ }\mu\text{m}$) which can penetrate to the alveolar region of the lung, independent of its chemical composition (Cherrie 2022; Queen Mary University of London n.d.). RCS represents a subset of the RD fraction, that is a crystalline silica polymorph (e.g. quartz, cristobalite, tridymite). When discussing a disease like silicosis, the specific agent, in this case RCS, is the primary focus.

It is important to recognize that the hazards of airborne concentrations of RCS are governed by a combination of factors that extend well beyond the nominal crystalline silica content of a material. These include the physical properties of the substrate (e.g., hardness and fracture behavior can alter the release of dust), the nature of the task (cutting, grinding, polishing, layout, installation, housekeeping), the tools and energy applied, and the presence and effectiveness of engineering controls such as water suppression and local exhaust ventilation. Accordingly, the generation of respirable dust, and the fraction thereof that is crystalline silica, can vary substantially across occupational environments. Stone fabrication, like many occupational scenarios, represents a situation in which several of these determinants may converge to produce elevated airborne concentrations of RCS when controls are insufficient.

Thompson and Qi (2023), in a comparative experimental study, evaluated grinding emissions from various stone countertops. Two were engineered stones composed of silica in a polyester resin matrix (Stone A and Stone B) and one was natural granite. Stone A contained the highest bulk crystalline silica content (90% by weight), followed by Stone B (50% by weight), which was near the upper range of silica content typically found in granite (20-60% by weight). The granite in this study was not measured for its bulk crystalline silica content. After grinding under the same conditions, granite (30 mm thick slab) released significantly greater total dust in comparison to the resin-based engineered stones (30 mm and 32 mm thick for Stone A and B, respectively). Stone A exhibited the highest RCS generation rate (15.6 mg/cm^3), followed by granite (10.8 mg/cm^3), and Stone B (6.31 mg/cm^3) (Thompson and Qi 2023). Despite similar crystalline silica compositions and thicknesses, granite released 171% more airborne RCS in comparison to Stone B, highlighting the inaccuracies in comparing countertop materials solely based on crystalline silica content.

A later study by Martinez-Gonzalez et al. (2024) performed controlled dry cutting, dry polishing, and wet cutting on granite (42% quartz) and silestone (an 80.8% quartz ES). Under dry cutting, dry polishing, and wet cutting conditions, peak alpha-quartz exposures for granite were 16.79 mg/m^3 , 5.44 mg/m^3 , and 1.48 mg/m^3 , respectively. For silestone, corresponding values were 22.58 mg/m^3 , 8.52 mg/m^3 , and 2.01 mg/m^3 , respectively (Martinez-Gonzalez et al. 2024). By comparison, the wet cutting of marble ($<2\%$ crystalline silica) had a peak exposure of 0.34 mg/m^3 . Despite the fact that silestone reportedly

contained nearly double the quartz by weight compared to the granite, the resulting difference in airborne RCS quartz exposures were proportionally much smaller (34%, 57%, and 36% greater for the respective tasks compared to the 100% greater one might expect). Ultimately, the data from Martinez-Gonzalez et al. (2024) showed that exposure magnitude is not proportional to bulk crystalline silica content and similar to Thompson and Qi (2023), indicated that resin-based materials release significantly less dust than harder natural stones.

Based on the data reported in Thompson and Qi (2023) and Martinez-Gonzalez et al. (2024), it is reasonable to estimate that a 40-60% crystalline silica (by weight) NS may produce roughly the same airborne RCS exposure as a 70-90% crystalline silica (by weight) ES under identical fabrication conditions. In discussion with several fabricators, it has been noted that ES products often require fewer fabrication steps, particularly polishing, to achieve an acceptable finish. NS slabs may require additional finishing because of naturally occurring microfissures, mineralogical heterogeneity, and other material inconsistencies.

Several recent field studies have corroborated the controlled study findings of Thompson and Qi (2023) and Martinez-Gonzalez (2024), reporting the following:

“... exposures to respirable quartz from engineered stone were not generally higher than exposures to respirable quartz from granite.” (Phillips et al. 2013, pg. 371)

“There was no statistically significant difference for stone type on short term task-based exposure to respirable crystalline silica. This is also consistent with the study by Phillips et al. [2013].” (Qi and Lo 2016)

“... stone type was not a significant factor for [airborne] RCS concentration” (Mcgowan 2025, pg. 33)

The deposition and clearance of all RD particles are governed largely by particle size and aerodynamic behavior, although the biological activity of RCS is dependent on a number of physiochemical properties following deposition in the lung. For example, freshly fractured particles tend to exhibit greater reactivity, which diminishes over time as the surface undergoes aging and interaction with environmental constituents (Dutta Gupta et al. 2024; Marrocco et al. 2024). Other physicochemical attributes, morphology, the presence of organic matrices such as resins, co-exposure to metals, and crystalline polymorph (i.e., cristobalite or quartz), have been evaluated for their potential to modify this reactivity, and more importantly, silicosis outcomes.

In light of these considerations, the present analysis was undertaken to examine whether the physicochemical characteristics of RCS derived from ES pose a greater human health hazard relative to RCS from sources such as NS or concrete. Particular emphasis was

placed on disentangling process driven characteristics from material driven factors and evaluating the extent to which the combination of these factors materially alters the dose-response curve for RCS. One objective is to understand why the recent cases of silicosis occurred, how they could have been prevented, and whether existing exposure limits are appropriate.

3. Methods

A scoping review of the scientific literature was conducted to evaluate whether respirable crystalline silica (RCS) generated from engineered stone (ES) differs toxicologically from RCS generated from natural stone (NS) or other occupational sources of crystalline silica in a manner that would be expected to result in a meaningfully different human health risk at an equivalent RCS exposure. A scoping-review approach was selected because the relevant evidence spans multiple disciplines, including particle physics, mineralogy, industrial hygiene, exposure science, toxicology, epidemiology, regulatory guidance, and occupational medicine.

The review was organized around predetermined scientific questions identified by the authors, based on prior knowledge of the RCS literature, as the principal issues relevant to evaluating whether RCS source materially modifies toxicological potency or health risk. These questions were grouped into six areas: (1) particle size; (2) particle surface area and surface chemistry; (3) resins and organic constituents of ES; (4) silica polymorph, particularly quartz and cristobalite; (5) metals; and (6) RCS exposure-response relationships, including the potential for a biological threshold or tolerable dose.

Relevant literature was identified through searches of PubMed and Google Scholar through June 18, 2026. Searches combined terms related to RCS, silicosis, engineered and natural stone, and countertop fabrication with topic-specific terms related to particle size and deposition, surface area and chemistry, silanols, freshly fractured silica, zeta potential, resins, volatile organic compounds (VOCs), metals, silica polymorphs, dose-response, cumulative exposure, and thresholds. These terms were combined, as appropriate, with topic-specific terms to identify each question. Searches were not restricted to ES because studies involving other occupational or experimental sources of RCS may provide information relevant to the intrinsic properties and biological activity of crystalline silica. Reference lists of relevant articles and reviews were also examined to identify additional studies, including older mechanistic and toxicological literature. The authors additionally included relevant literature and regulatory documents based on their prior knowledge of the topic that may not have been identified through the search strategy.

Studies were considered relevant, at the authors' discretion, if they informed one or more of the predetermined research questions. The weight of evidence was evaluated based on consistency across study types, biological plausibility, dose-response information, comparability of exposure conditions, and potential alternative explanations for reported differences. Particular emphasis was placed on studies directly or indirectly comparing ES with NS or other RCS-containing materials and studies evaluating materials under comparable experimental or fabrication conditions.

4. Results

4.1. Particle Size

- i. How does aerodynamic equivalent diameter influence respiratory tract deposition, and why do respirable fraction metrics (e.g., ISO conventions) fail to precisely predict alveolar deposition?

Particle size, reflected in aerodynamic equivalent diameter (AED), is widely recognized as an important determinant of particle deposition within the respiratory tract, thus it plays a role in estimating the delivered dose to various lung regions (Londahl et al. 2014; Agency for Toxic Substances and Disease Registry (ATSDR) 2019; Lippmann and Leikauf 2020). For RCS, unlike fibers, particles are usually more equant (comparable dimensions in all directions) than elongated, with aspect ratios often near one. For compact, equant particles in the 1–10 μm size range, measured physical diameter and AED may align reasonably well when particle density and dynamic shape factor are near unity (Hinds and Zhu 2022). However, for higher-density minerals such as quartz, AED will exceed physical diameter by approximately the square root of the particle density, assuming near-spherical shape. Because quartz (2.6 g/cm^3) and cristobalite (2.3 g/cm^3) have a density of approximately 2.5 g/cm^3 , the AED of RCS particles is approximately 1.6 times the measured physical diameter when the dynamic shape factor is near one (Stones et al. 2016).

For silicosis, the alveolar (gas-exchange) region is unanimously regarded as the relevant site of fibrotic injury. In this region, RCS particles that evade upper airway defenses will deposit, interact with epithelial cells, and undergo macrophage-mediated processing. It is important to recognize that the respiratory tract functions as an integrated defense system; deposition in the upper respiratory tract (head airways) and tracheobronchial region reduces the fraction of particles reaching the alveoli, such that “... *these rapidly cleared regions serve to protect the more vulnerable region*” (Hinds and Zhu 2022, pg. 209).

Commonly used conventions, such as the ISO 7708 respirable fraction, were developed as practical sampling approximations rather than precise predictors of biological fate (International Organization for Standardization (ISO) 1995; Baron 2016). These

conventions are intended to identify particles able to reach the gas-exchange region, but they do not account for the fact that this is a non-linear relationship and, even when the upper airway and trachea-bronchial (TB) airways are avoided, a substantial proportion of these particles are not retained. As described by Hinds and Zhu (2022), the distinction between particles that reach the alveoli and those that deposit there is consequential; in many cases, especially for smaller particles, a large fraction is subsequently exhaled (described in the next section). Thus, while particle size is clearly relevant to transport, its use as a coarse surrogate for retained dose, particularly in regulatory or risk assessment contexts, should be viewed with appropriate caution.

ii. What mechanisms explain the nonlinear relationship between particle size and deposition efficiency?

The relationship between particle size and deposition efficiency is not linear and reflects the combined influence of several competing physical processes. Within the respirable fraction, relatively large particles (1-10 μm) are efficiently removed in the upper airways due to impaction and sedimentation, whereas smaller particles (0.01-0.2 μm) more efficiently evade these head and TB airways (International Commission on Radiological Protection (ICRP) 1994; Hinds 1999; Kuempel et al. 2015).

Between small and large particles in the respirable region lies a range, often cited as approximately 0.2 to 1 μm , where particles are sufficiently small to evade upper airway deposition but not sufficiently diffusive to deposit efficiently in the deep lung [estimated 5-10% deposition per Hinds and Zhu (2022) and ICRP (1994)]. Essentially, these particles tend to remain entrained in the inhaled airstream because they have insufficient inertia, settling velocity, or diffusivity to deposit in the alveoli. As a result, alveolar deposition efficiency among respirable particles reaches a relative minimum in this size range, even though such particles are frequently classified as “respirable”. As stated in the International Commission on Radiological Protection (ICRP) Publication 66, *Human Respiratory Tract Model For Radiological Protection* (1994):

“Aerosol particles are much less mobile than gas molecules and tend to be carried along with the airflow. There are many paths a particle might follow in its journey through the airways. A particle of minimal intrinsic mobility is least likely to come in contact with an airway surface by inertial impaction or Brownian motion. Particles of about 0.5 μm in diameter are at the minimum of combined inertial (aerodynamic) and diffusional (thermodynamic) motion. Very few of these particles will deposit on the airway walls as they penetrate deep into the respiratory tract during a breath. The fluid mechanics are not completely reversible on exhalation, so particles follow a different path during exhalation; but again, very few particles of minimal intrinsic motion

deposit on airway walls. About 10% of these particles are not exhaled, largely owing to irreversible exchange between tidal air and resident air by actual fluid mixing.” (International Commission on Radiological Protection (ICRP) 1994, pg. 55)

Model-based estimates, such as those derived from the ICRP framework, suggest that alveolar deposition peaks at around 50% retention near $0.02\ \mu\text{m}$ and declines on either side of that value (Figure 1). Alveolar deposition rates are less than 20% when particles are less than $0.004\ \mu\text{m}$ or greater than $0.08\ \mu\text{m}$ (Figure 1).

These findings indicate that respirability should not be conflated with deposition, and that deposition does not indefinitely nor linearly increase as particle sizes get smaller in the respirable range.

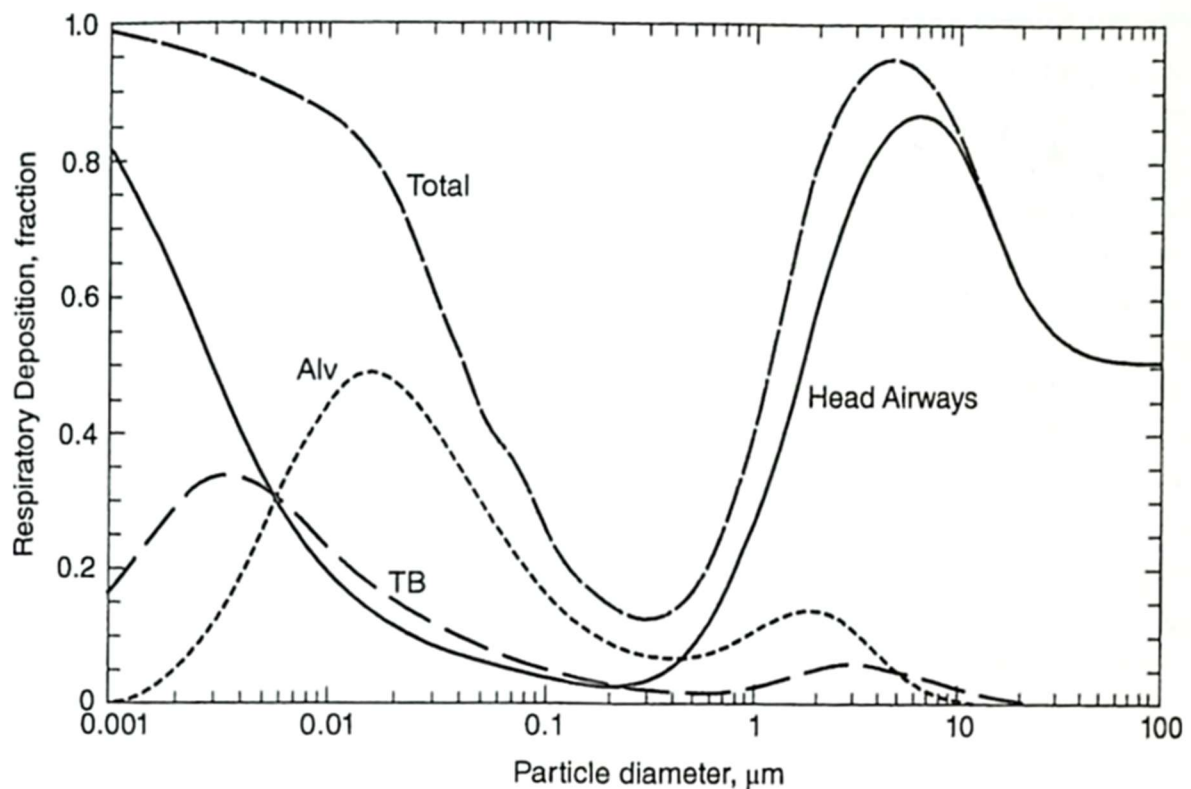


Figure 1. Predicted total and regional deposition for light exercise (nose breathing) based on ICRP deposition model based on average male and female data. TB=trachea-bronchial region; Alv=Alveolar region (Reproduced from Hinds and Zhu (2022) with permission). NOTE: Deposition fraction in the alveolar region, the relevant site for silicosis, does not increase linearly as particle size decreases.

iii. How do biological clearance processes (e.g., macrophage phagocytosis, mucociliary transport) relate with particle size?

Following deposition, the extent to which particles are retained, and therefore capable of eliciting biological effects is dictated by pulmonary clearance mechanisms. These include mucociliary transport in the conducting airways, translocation to regional lymphatics, and macrophage mediated clearance in the alveolar region (Agency for Toxic Substances and Disease Registry (ATSDR) 2019). Notably, macrophages are capable of engulfing equant particles that are considerably larger than those typically classified as respirable, suggesting that independent of surface area (which is discussed in a later section), differences in particle size within the respirable range are unlikely to be the principal determinant of clearance efficiency for these particles (Ishida et al. 2019; Castranova n.d.). Instead, repeated irritation to target tissues reflects a combination of deposition, persistence, and frustrated phagocytosis, with adverse outcomes occurring when normal macrophage-mediated clearance and repair processes are repeatedly challenged by a sufficient burden of reactive particles (Hamilton Jr et al. 2008; Hornung et al. 2008).

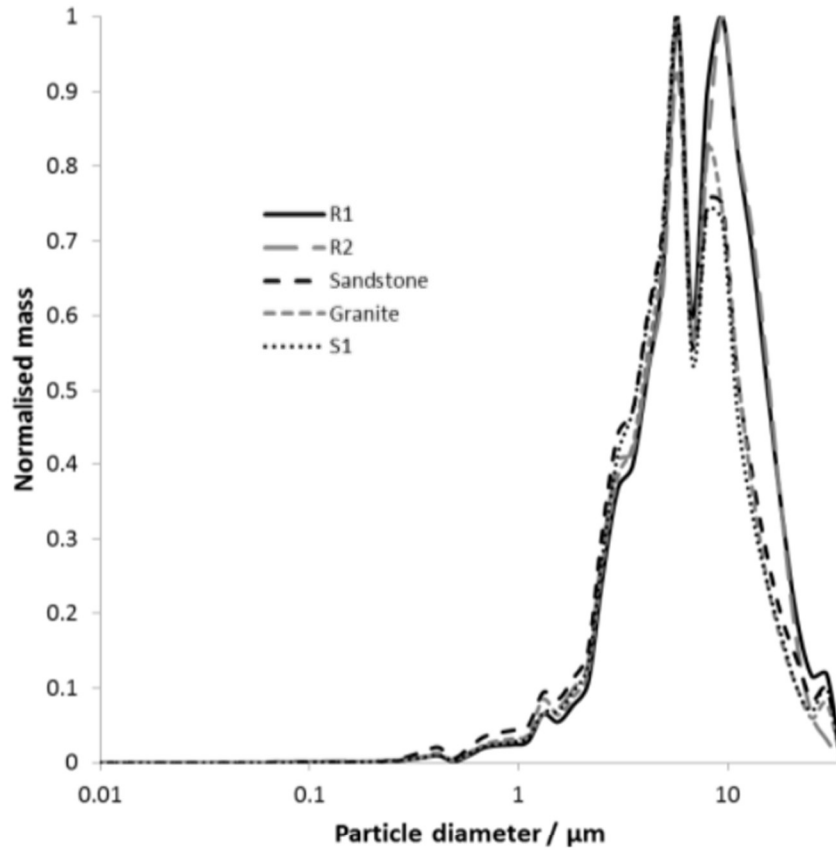
It should be noted that as particles enter the ultrafine or nanoparticle size range ($<0.1\ \mu\text{m}$), a fraction may translocate across the alveolar epithelium and air–blood barrier, with translocation generally increasing as particle size decreases (Kreyling et al. 2002; Geiser and Kreyling 2010). As such, their biopersistence in the alveolar region and role in the development of silicosis may be reduced proportional to this translocation. That said, some researchers have suggested that $<1\%$ of nanoparticles are translocated from the lungs to other organs (Kreyling et al. 2009; Kuempel et al. 2015).

iv. To what extent do fabrication processes (e.g., cutting, grinding, polishing, wet vs. dry methods) govern the size distributions of airborne particles, and do ES and NS materials release different particle size distributions under identical processes?

The available evidence indicates that particle size distributions generated during stone fabrication are governed primarily by the nature of the process, particularly task type and applied energy, rather than whether the material is ES or NS. As a result, attempting to distinguish or regulate RCS particles by particle size on the basis of material source is not scientifically or practically supportable.

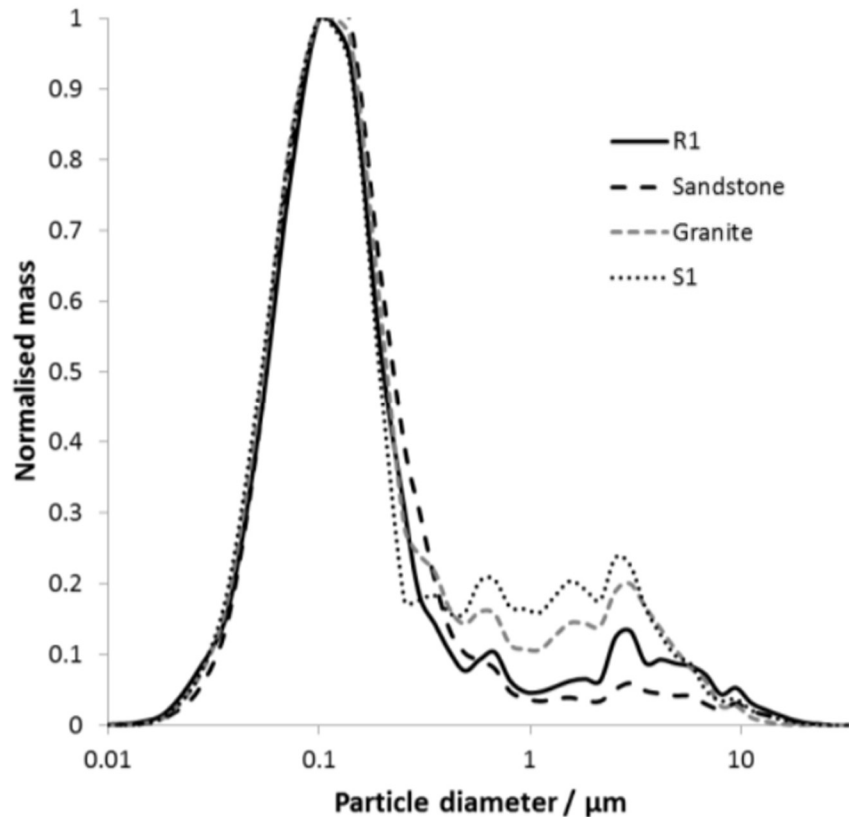
Comparing ES and NS materials directly, studies consistently show similar particle size distributions across material types when subjected to identical processes of cutting, grinding, or polishing (Ramkissoon et al. 2022; Thompson and Qi 2023; Figure 2; Mandler et al. 2026).

Controlled comparative studies are the gold standard for this research because particle size distributions may be affected not only by the process task, but also by blade size, blade material, applied force, and other factors. For example, peak particle sizes in Hall et al. (2022) following polishing activities were 0.1 μm , while they appear closer to 1 μm in Bahri et al. (2022) during the same general task. Process conditions such as the use of wet versus dry methods have a pronounced effect on airborne concentrations, with wet techniques substantially reducing total particle counts, including ultrafine particles, due mostly to agglomeration. However, when using the same cutting tool, it was observed by Pavan et al. (2016) and Bahri et al. (2022) that distributions and mean particle sizes did not appear to differ significantly under controlled wet or dry conditions.



(a)

Figure 2. Normalized size distributions of emissions, by mass, during (a) cutting and (b) polishing. R1 and R2 are rein-based engineered stones, while S1 is a sintered engineered stone. (Reproduced from Hall et al. (2022)). NOTE: Size distribution is heavily influenced by fabrication task, however, is similar across natural and engineered stone types.



(b)

From a risk assessment perspective, these findings indicate that particle size is more meaningfully controlled by task and tool rather than the type of stone that is being fabricated. It should be noted, however, based on our review of the literature, no one has isolated and performed measurements of specifically RCS particles within the respirable airborne fraction of dust: rather total dust (which includes a large fractions of non-crystalline silica materials) was evaluated.

4.2. Surface Area and Surface Chemistry

- i. Do ES and NS materials release RCS particles with morphological differences under identical manipulation (e.g., cutting, grinding, or polishing)?

Surface area is dictated by a combination of two characteristics: particle size and surface morphology. As discussed in the previous section, based on the available data, there is no reason to suspect meaningful differences in particle size for ES RCS particles compared to NS where the tool and task drive this characteristic instead.

Qualitative morphology differences have been reported between ES and NS fabrication dusts, such as more angular, highly fractured, and agglomerated particles in some ES dusts compared to smoother, more conchoidal particles in certain natural stones. However, these studies are limited in that they do not specifically evaluate RCS particles and have limited sample sizes (Hall et al. 2022; Ramkissoon et al. 2022). The only quantitative observations in the literature, to our knowledge, were performed by Ramkissoon et al. (2022; 2025), but are analyses of total bulk settled dust rather than the airborne RCS fraction. The presence of many non-RCS particles (that may vary in surface area, but are not relevant for silicosis risk assessment) in these samples complicates interpretation.

Importantly, surface area measurements of crystalline silica-containing dusts, across NS and ES sources, seem to be strongly correlated with crystalline silica content, suggesting the ratio of RCS to non-RCS particles drives differences in the specific surface area of settled dusts (Figure 3). This does not suggest that there are differences between RCS particle surface area based on material source. In Ramkissoon et al. (2025), after dry cutting, an engineered stone that produced a 7% RCS by weight airborne dust had a specific surface area of 0.79 m²/g. This was nearly identical to that of a natural stone (white granite) that produced a 3.5% RCS by weight airborne dust (0.88 m²/g). Dry cut reference quartz (n = 1) had a far greater specific surface area than any of the silica-containing ES or NS manipulated in the same manner.

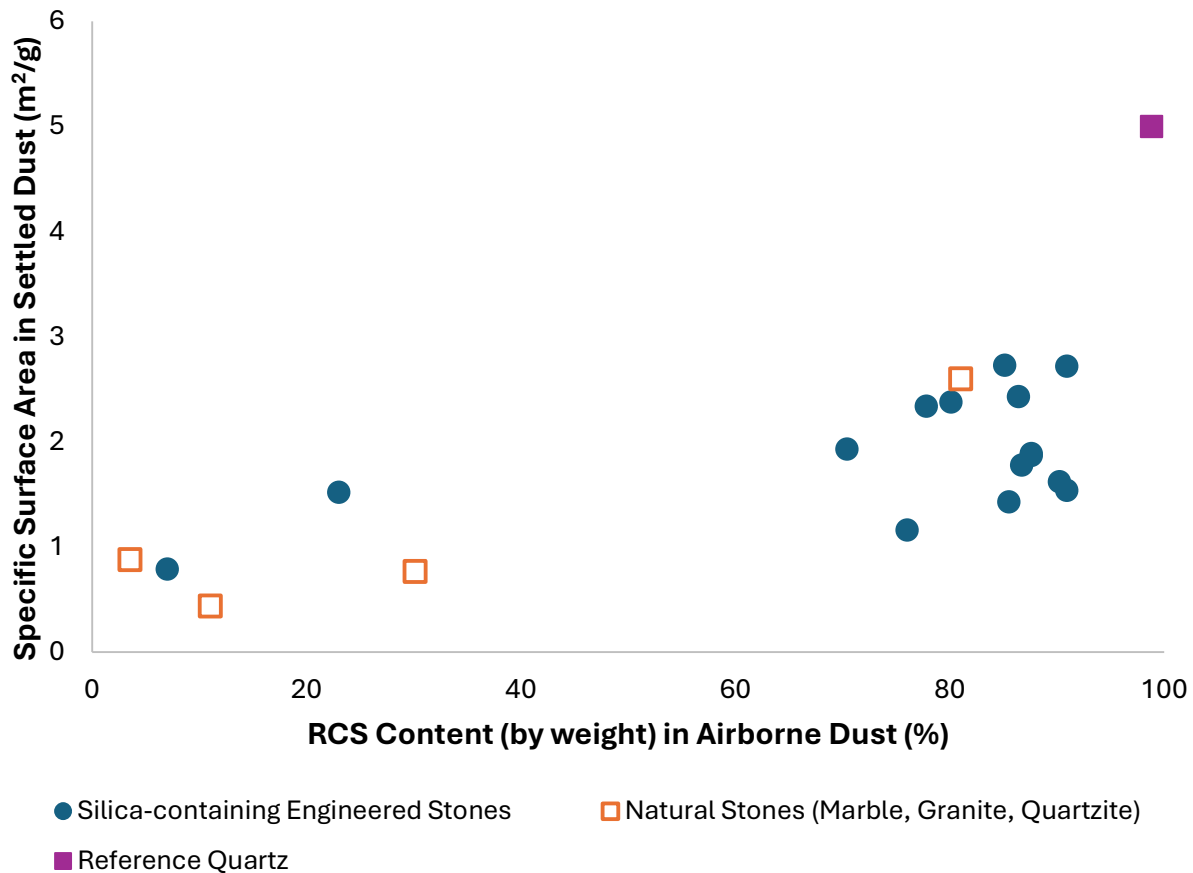


Figure 3. Airborne dust RCS content (% by weight) compared to the specific surface area (m^2/g) measured in total settled dusts after dry cutting [data from (Ramkissoon et al. 2022; Ramkissoon et al. 2025)]. NOTE: Surface area is associated the silica content in these dusts, and these data do not suggest that surface area differences between airborne RCS particles from NS or ES exist.

ii. What is the effect of surface area on the silicotic potency of RCS?

For an equivalent deposited mass of otherwise comparable particles, a smaller particle size or a “rougher” particle generally provides a greater specific surface area and, therefore, more surface available to interact with pulmonary surfactant, alveolar macrophages, and epithelial cells. It has been suggested that particle surface area may influence the silicotic or fibrogenic potency of RCS because the initial toxicologic interactions occur at this particle–lung interface.

Several authors have considered the effects of surface area on particle reactivity *in vitro*, and based on these findings, some have theorized that RCS concentrations would be more appropriately characterized based on total surface area, rather than mass (as is conventional) (Rishi et al. 2024). Based on our review of the literature, it is unclear from a

mechanistic toxicological standpoint that surface area is a more reliable predictor of silicosis outcome than mass. The scientific evidence for this is lacking in the current literature and predominately based on non-RCS evaluations for non-silicosis endpoints.

Comparative animal studies have found that quartz-induced inflammation, cytotoxicity, and early fibrotic responses do not consistently track with particle size or Brunauer–Emmett–Teller (BET)-measured surface area (Warheit et al. 2007; Warheit et al. 2009; Skuland et al. 2014). It is also important to recognize that much of the comparative literature evaluates acute or subchronic endpoints *in vitro*—such as hemolysis, cytotoxicity, or reactive oxygen species—rather than the actual development of silicosis. The presence and configuration of silanol groups, especially nearly free silanols (NFS), appear to be directly implicated in membranolytic activity and downstream inflammatory signaling *in vitro*, but it is unclear whether bulk surface area metrics meaningfully translate to silicosis potency (Pavan et al. 2016; Brinker et al. 2020; Pavan et al. 2020).

Mechanistically, silicosis is essentially the scarring of epithelial tissue in the gas exchange region of the lungs. The immune system responds to deposited RCS particles in this manner for a few reasons. In the alveolar region, a foreign body is recognized via the surface receptors on macrophages (Dezube 2025; British Society for Immunology n.d.). Macrophages then get signaled to attempt to engulf the foreign body. Once phagocytosed by macrophages, the surface reactivity and biopersistence of crystalline silica can damage phagolysosomal membranes, promoting inflammation and macrophage death (Pollard 2016). Persistent particles may subsequently be re-phagocytosed, sustaining inflammation and ultimately promoting collagen deposition and formation of silicotic nodules around the particle (Pollard 2016).

Even if a particle has comparatively more reactive surface sites due to increased surface area, which *in vitro* may enhance the cascade of inflammation response, the extent to which the nodule differs in size remains uncharacterized. As such, to what degree, if any, the silicosis response of an equal mass of RCS in the alveoli may vary based on surface area is unclear.

Practically speaking, the wealth of epidemiology data that are relied on for deriving dose-response relationships for RCS and silicosis cannot reliably be retrospectively converted from mass to surface area. Determining a dose-response for silicosis based on surface area, rather than mass, and measuring exposures based on this metric is not practically achievable, and the potential benefits from attempting to do so are unclear. It has not been shown that on an equal mass basis, smaller RCS particles are causing a greater response of silicosis *in vivo*. Such an assessment based on epidemiology seems unlikely

given the practical limitations of dust characterization, situational variability, and exposure in the workplace.

iii. Do freshly fractured silica surfaces exhibit greater silicosis potency than aged surfaces due to transient increases in reactive surface sites and radical activity?

It is difficult to parse out the importance of biopersistence relative to surface chemistry, although both are clearly necessary for silicosis outcomes. As an example, respirable amorphous silica dust is regulated like nuisance dust under an OSHA permissible exposure limit (PEL) that is one hundred times greater than the PEL for its crystalline silica dust counterpart ($5,000 \mu\text{g}/\text{m}^3$ compared to $50 \mu\text{g}/\text{m}^3$). Nonetheless, *in vitro* studies have acknowledged similar and sometimes greater reactivity of amorphous silica in comparison to crystalline silica (Turci et al. 2016; Pavan et al. 2020). Ultimately, this indicates that the surface of amorphous silica, specifically the density of NFS groups capable of interacting with cell membranes, behave similar to that of crystalline silica at a certain point in time. That said, amorphous silica is generally considered non-potent for silicosis based on extensive *in vivo* and epidemiological information (Marrocco et al. 2024). This is because the amorphous structure of these particles is far less resistant to dissolving in the acidic environment of a macrophage in comparison to its crystalline counterpart and repeated frustrated phagocytosis does not occur as described in the section above (Agency for Toxic Substances and Disease Registry (ATSDR) 2020; Baum and Arnold 2023).

There is relatively consistent evidence to suggest that freshly fractured silica surfaces are more biologically active than aged ones at a given point in time (Marrocco et al. 2024). Fracturing of crystalline silica generates highly reactive surface species (e.g. Si^+ and SiO^-) through cleavage of silicon-oxygen bonds, which have been associated with increased generation of reactive oxygen species and pro-inflammatory responses (Mossman and Churg 1998; Fubini and Hubbard 2003; Dutta Gupta et al. 2024; Marrocco et al. 2024). However, these reactive surface features are not stable; experimental data indicate that radical activity decays with a half-life on the order of ~30 minutes in air (Porter et al. 2002), and substantially more rapidly in aqueous environments (Vallyathan et al. 1988; Meldrum and Howden 2002).

Given the time and number of macrophage deaths it takes for silicotic nodules to form around a particle as well as the transient nature of surface silanol groups in the aqueous environment of the lungs, it is the opinion of the authors that the presence of NFS groups, or the “ever fractured” nature of the RCS particle, is far more relevant than the “freshness” of the particle (personal communications with Drs. Pavan and Turci). This appears to remain debated amongst the scientific community.

iv. Are metrics such as zeta potential or total surface charge reliable predictors of silica-induced cytotoxicity and inflammation?

Metrics such as zeta potential (total surface charge) are not reliable standalone predictors of silica-induced cytotoxicity and inflammation, as their relationship to biological effects is indirect and inconsistent. *In vitro* analysis by Ramkissoon et al. (2024) found no significant correlation between zeta potential and inflammatory or cytotoxic responses. Findings from the broader nanotoxicological literature indicate that surface charge density, rather than absolute zeta potential, may be more relevant (Weiss et al. 2021). In the case of crystalline silica, the weight of evidence points to specific surface functionalities, particularly NFS groups, as more proximate determinants of biological activity (Pavan et al. 2016; Brinker et al. 2020).

Accordingly, reliance on zeta potential as a primary metric of hazard would appear to be of limited value. Ramkissoon et al. (2024) has noted that particle charge may play a role in the adhesion of particles to cells, but overlapping zeta potential ranges have been reported for ES and NS dusts, despite crystalline silica content differences (Thredgold et al. 2022; Ramkissoon et al. 2024).

4.3. Resins

i. How do resin coatings, and their potential degradation or detachment/reattachment during fabrication, affect the surface chemistry, bioavailability, and toxicity of RCS particles?

Resin coatings on ES particles, if present, appear to act as a physical barrier that limits direct interaction between reactive surface sites (e.g., silanol groups and surface radicals) and biological membranes, thereby attenuating hemolytic activity and epithelial cell injury *in vitro*. In controlled settings, removal of these coatings by thermal treatment increased bioavailability of the surface groups, the dominant determinant of toxicity (Pavan et al. 2016; Pavan et al. 2026). Thus, it is logical to expect that the presence of resins would reduce the potency of RCS particles for silicosis.

On the contrary, some have hypothesized that resin encapsulation could preserve reactive surface species, prevent aging, and thereby prolong toxicity after the resins degrade on the surface of RCS particles in the lung (Di Benedetto et al. 2019). Available data does not substantiate this hypothesis. The *in vitro* studies by Pavan et al. did not compare ES to NS crystalline silica particles to compare reactivity, nor did they determine whether resin stripped ES RCS particles behave like freshly fractured or aged particles. Given how quickly these particles age in an aqueous environment like the lung and the importance of

biopersistence in causing silicosis, this is likely of minimal relevance to long-term health outcomes (see section 4.2.iii).

Dissolution studies indicate that ES resins are relatively persistent under simulated physiological conditions, with only 1–20% degradation over two months in artificial lysosomal fluid (ALF) and negligible degradation in lung-lining-fluid analogs (Pavan et al. 2026). Furthermore, the rate of degradation decreased from the first to the second month in this solution (Pavan et al. 2026).

Under non-overloaded conditions, poorly soluble and low-reactive particles deposited in the alveolar region are cleared primarily through macrophage-mediated transport via the mucociliary escalator and lymphatic system. If unable to be removed by a macrophage, in humans, there is a modeled half-time of ~250 days for these particles to travel from the alveolar compartment to the bronchial tree (ICRP 2015). Based on the limited resin degradation reported by Pavan et al. (2026), resin occlusion, if present on the RCS particles deposited in the alveoli, likely persists during the time frame which a substantial fraction are cleared from this region, suggesting a meaningfully reduced or lack of silicosis-potency from these particles.

It is important to note that Pavan et al. (2026) evaluated ES dust that was generated by crushing ES, rather than solely grinding, polishing, or sawing as part of countertop fabrication. Fabrication processes introduce far greater mechanical energy than crushing. Thus, the Pavan et al. (2026) data may not be very useful for characterizing airborne fabrication dust.

Two recent investigations by Rishi et al. (2024) and Hall et al. (2022), based on advanced optical photothermal infrared (O-PTIR) spectroscopy and elemental mapping of carbon signals, respectively, suggest that fabrication energies are sufficient to promote detachment of resin from respirable airborne particles, particularly in smaller particle fractions. These two studies, despite also looking at dry cutting, grinding, and polishing generated dusts, could not identify resin in their airborne analyses (Hall et al. 2022; Rishi et al. 2024).

Furthermore, the in vitro analysis by Mandler et al. (2026) did not find significant toxicity differences between ES and NS (granite) dry cutting particles when silica content was close to equivalent, suggesting resins were either not present or not affecting silanol reactivity in the manner observed in the study by Pavan et al. (2026). Similarly, Thredgold et al. (2022) evaluated engineered stone dust generated under controlled cutting conditions (not specified whether dry or wet) and reported that “... *no correlation between resin content*

and dust reactivity was observed” in a cell free deoxyguanosine assay (Thredgold et al. 2022).

Consequently, the respirable dust fraction to which workers are exposed may be largely composed of “free” crystalline silica particles, which would effectively negate any biological effects of resin occlusion. The conditions in which resin is or is not present on airborne RCS particles as a result of different countertop fabrication techniques remain understudied.

On an equal-RCS basis, based on the limited available research, the authors expect that the toxicity or inflammatory potential of particles from engineered stone would be similar to, if not lower than, that of particles from natural stone as a result of resin occlusion. This evidently remains debated in the scientific community.

ii. Are VOC emissions during stone fabrication of sufficient magnitude to contribute to pulmonary toxicity or modify the dose–response relationship for silicosis?

VOC emissions associated with engineered stone fabrication arise from the thermal decomposition of organic resins where their airborne concentrations are highly dependent on process temperature. Under wet cutting conditions ($\leq 60^{\circ}\text{C}$ or $\leq 140^{\circ}\text{F}$), VOC generation is negligible, whereas high energy dry cutting can produce elevated temperatures ($\geq 120^{\circ}\text{C}$ or $\geq 248^{\circ}\text{F}$) that increase emissions (Hall et al. 2022). Appropriately designed ventilation can also reduce airborne vapor concentrations in worker breathing zones. Modeled and experimental data indicate that, regardless of controls, upper-bound exposure scenarios for VOCs remain well below established regulatory exposure limits (Hall et al. 2022; Ramkissoon et al. 2023).

The study by Ramkissoon et al. (2023) conducted dry cutting in an enclosed cabinet ($60 \times 80 \times 80$ cm) and, while *“the purpose of the study was not to determine a workplace exposure assessment to VOCs”*, it produced some interesting results. Of the many VOCs that were generated, only one peak VOC (phthalic anhydride) concentration of particles and vapors (0.32 mg/m^3) exceeded the ACGIH TLV STEL (0.005 mg/m^3), but did not exceed the OSHA PEL (12 mg/m^3 8-hour TWA). It should be noted that the ACGIH TLV is driven by respiratory sensitization for atopic individuals, rather than upper respiratory, eye, and skin irritation, as the OSHA PEL and historic TLV was. This reported concentration in Ramkissoon et al. (2023) was observed in an ES product that was chosen for its relatively high organic composition of phthalic anhydride and styrene. As evidenced by Hall et al. (2022), VOC emissions and emission products vary across ES products. Additionally, in a space more typical of a fabrication environment, they identified that the concentration of airborne VOCs,

even during dry cutting of a resin-based material, did not approach regulatory limits (Hall et al. (2022) Figure SF7).

From a toxicological perspective, the available evidence does not indicate that VOCs, including phalatic anhydride, are fibrogenic, so the presence of these chemicals should not increase the silicosis risk posed by crystalline silica (at low or moderate concentrations). The literature discussing VOCs has tended to characterize them as transient upper respiratory irritants (U.S. Environmental Protection Agency (EPA) 2025) that mechanistically are not key to the pathway leading to silicosis, which is driven by persistent crystalline silica surface reactivity and macrophage-mediated responses in the alveoli (American Chemistry Council (ACC) 2019). To be considered irritants, the concentrations need to be much greater than those observed during cutting. Accordingly, there is no evidence that the presence of VOCs meaningfully modifies the dose–response relationship for silicosis.

5.4. *Cristobalite v Quartz*

- i. Do differences in silica polymorph (cristobalite vs. quartz) result in meaningful differences in toxicological potency or slope of the dose–response for silicosis?

Natural and engineered stones do not contain a single, uniform crystalline silica polymorph profile. However, recent investigations have found that cristobalite tends to make up a larger fraction of the total silica content in ES than in some natural stone (Guarnieri et al. 2020; Hall et al. 2022; Ramkissoon et al. 2022; Thompson and Qi 2023). Across 12 engineered stone samples evaluated by Ramkissoon et al. (2022), cristobalite accounted for an average of 23.5% of the silica content, with the remainder being quartz. Cristobalite content ranged from 0 to 65.5% by weight in this study (Ramkissoon et al. 2022).

A careful review of the available data suggests that, while cristobalite and quartz differ crystallographically, these distinctions have not been shown to translate into meaningful differences in toxicological potency or the dose-response relationship for silicosis. This conclusion is reflected in regulatory practice. OSHA (2016) established a single permissible exposure limit encompassing quartz, cristobalite, and tridymite; NIOSH (2002) similarly recommended a unified exposure limit; and HSE (2002, 2003) and related international bodies have adopted comparable approaches.

Although it has been recently proposed that cristobalite may be more fibrogenic than quartz (Martinez-Gonzalez et al. 2024), this hypothesis is not well supported when considered alongside the broader body of evidence. Early studies suggesting greater toxicity of cristobalite (King et al. 1953) have not been consistently supported by later investigations showing comparable cytotoxicity (Mossman and Glenn 2013; Nattrass et al. 2017). In

particular, findings from animal inhalations studies (Johnston et al. 2000), a range of *in vitro* investigations (Shukla et al. 2001; Albrecht et al. 2002; Perkins et al. 2012; Peeters et al. 2013), and epidemiologic studies of exposed worker populations (Hughes et al. 1998; Park et al. 2001) have been evaluated collectively, with Mossman and Glenn (2013) concluding that the two polymorphs produce similar pulmonary outcomes and do not warrant separate exposure standards. Other analyses (Hemenway et al. 1990; Housley et al. 2002; Horwell et al. 2012; Nattrass et al. 2017; Ramkissoon et al. 2024) reached the same conclusion, suggesting comparable or even slightly lower potency for cristobalite in some settings. Thus, in engineered stone countertops, where cristobalite may comprise a larger proportion of RCS compared to some natural stones, there is no solid evidence that this polymorph composition materially alters the exposure–response relationship.

While quartz (trigonal) and cristobalite (tetragonal) differ in crystal structure, surface properties, and associated elemental composition (International Agency for Research on Cancer (IARC) 2012; Thompson and Qi 2023), these differences are not sufficient to produce clinically meaningful changes in biological reactivity. Cristobalite has been reported to exhibit a more negative zeta potential, particularly at higher pH (Júnior and Baldo 2014). As noted previously, available evidence indicates that zeta potential is a poor predictor of silicosis potency and overlapping zeta potentials are observed between ES and NS dust. This highlights the importance of not assuming that any individual physicochemical difference necessarily results in potency differences among RCS particles.

It is important to consider that potential physicochemical differences between the RCS from ES and NS may reflect these polymorph characteristics. Empirically, Ramkissoon et al. (2022) concluded that no correlation was observed *in vitro* between cristobalite content or zeta potential in engineered stone dust samples ($r < 0.5$), nor were consistent relationships identified between surface area, or other physicochemical parameters. An updated analysis by Ramkissoon et al. (2024) further suggested that, although no individual correlations were observed between quartz or cristobalite and A549 cellular response or THP-1 cytotoxicity, cristobalite was negatively correlated with the THP-1 cytokine response, whereas quartz exhibited a positive correlation ($p < 0.05$), suggesting a plausible lower or similar potency of cristobalite. Additionally, in the correlation matrix (Fig. 5 of the study), the authors did not find any positive correlation of the cristobalite content with the toxicity parameters PCA1, PCA2, or PCA3 (Ramkissoon et al. 2024). In fact, the correlation was negative with PCA1, although not with 95% confidence.

4.5. Metals

- i. To what extent do metals, and which metals, alter dose-response outcomes for silicosis?

Metals are more appropriately viewed as potential modifiers of RCS surface aging—through binding to surface silanol groups—than as primary drivers of the dose-response relationship for silicosis. Some evidence suggests that their influence depends on their chemical form and bioavailability (Occupational Safety and Health Administration (OSHA) 2016). Among metals studied in the context of co-exposure with RCS, aluminum has been the most extensively characterized. A substantial body of experimental work indicates that aluminum, whether present as a lattice constituent or surface-associated species, can passivate reactive silanol sites, thereby diminishing membrane interactions and downstream inflammatory signaling (Fubini and Mollo 1995; Meldrum and Howden 2002; Leung et al. 2012; Natrass et al. 2017).

Based on this mechanistic rationale, mid-20th-century occupational hygiene programs intentionally co-exposed silica-exposed workers to aluminum powders (e.g., “McIntyre Powder”) in an effort to passivate RCS surfaces, diminish fibrogenicity, and thereby lower the risk of silicosis. Due to a lack of detailed exposure characterization, the effectiveness of this practice was not measurable. Additionally, adverse neurological effects (Parkinson’s disease) at these extraordinary doses of aluminum were observed (Martell and Guidotti 2022). More recently, cases of “lower-than-expected” silicosis in the epidemiology of persons exposed to cristobalite-rich volcanic ash have also been attributed to lattice substitution by cations such as Al^{3+} or Na^+ , which may reduce surface reactivity (Horwell et al. 2012).

Iron presents a more nuanced case, as its toxicological role is highly dependent on oxidation state and speciation. Fortunately, iron is generally inert at any anticipated dose and has low toxicity. Bioavailable iron capable of redox cycling may enhance ROS generation and inflammatory responses, whereas iron present as a relatively inert surface coating may partially obscure reactive silica sites and attenuate toxicity (Fubini and Hubbard 2003). The potential effect of aluminum (decreased toxicity) and iron (increased or decreased toxicity depending on state and quantity) are acknowledged by OSHA; however, it was ultimately concluded that metal content was not a reasonable way to classify RCS exposures or derive a separate dose-response curve for silicosis as an endpoint (Occupational Safety and Health Administration (OSHA) 2016).

Other metals, including cobalt, titanium, chromium, nickel, and manganese, may contribute to oxidative stress pathways or interact with silanol sites under certain conditions, but there is limited evidence that they materially shift the silicosis dose-

response in either direction. Most importantly, the presence of these metals alone, is not indicative of a meaningful effect on disease outcome.

Ramkissoon et al. (2024) concluded that cobalt and aluminum (when evaluated in combination) appeared to explain 32.4% of the variance in macrophage cytotoxicity *in vitro*, and the supplementary information from Mandler et al. (2026) concluded that a combination of metals, primarily cobalt and titanium, appeared to be associated with overall toxicity based on an *in vitro* analysis. That said, it is unclear how, if at all, these studies controlled for silica content and how these toxicity endpoints—LDH release and cytokine production in Ramkissoon et al. (2024) and an unspecified combination of histopathology, blood cell parameters, BALF cytokines, and BALH LDH activity collectively called “Component 1” and “Component 2” in Mandler et al. (2026)—translate into any potentially meaningful shift in the dose-response for silicosis. Perhaps most important, there is not convincing evidence of a role of these metals in producing silicosis long-term.

Notably, the metals that were deemed most “significant” in these studies were often present only in a fraction of stones evaluated and at trace concentrations, while the limited number of dust samples also differed in several potentially confounding physiochemical characteristics. For example, in Mandler et al. (2026), titanium was reported at 0% in the only two silica-containing ES dusts evaluated, and cobalt was reported at 0% in one material (ESB) and 0.01% in the other (ESA). Similarly, Ramkissoon et al. (2024) reported measurable cobalt in ES dusts at an average concentration of only 0.01%. Thus, Mandler et al. (2026)’s conclusion that titanium played a “significant” role appears to rest largely on a comparison between one granite material containing 0.05% Ti and one non-silica-containing ES material containing 0.03% Ti, even though the latter would not be expected to have appreciable potency for silicosis.

By contrast, silica content was the clearest and most obvious predictor of toxicity outcomes in Mandler et al. (2026). After 84 days, lung inflammation scores for reference quartz, ESA (60% silica by weight in dust), granite (30%), ESB (23%), and non-silica-containing ES were 6.9, 5.6, 3.4, 2.6, and 2.4, respectively. Fibrosis scores followed a similar pattern, at 5.4, 4.1, 2.0, 1.0, and 1.25, respectively. Overall, consistent with OSHA’s evaluation, metal content does not appear to provide a reliable basis for distinguishing hazard across RCS sources.

ii. **Are metal concentrations in ES dust exceptionally high compared to other sources of RCS?**

Both ES and NS contain trace metals that vary across product lines, NS often showing greater variability and, in some cases, higher overall elemental metal content (reported as 29-37%, excluding silicon) than ES (reported as 1-8%) (Ramkissoon et al. 2022; Ramkissoon

2023). It is also worth noting that, given the low concentration of many metals in these dusts, metal like cobalt or nickel contributed from the saw itself should be considered when making cross-study or cross-experimental comparisons (Costa et al. 2017). As an example, Ramkissoo et al. (2024) found a positive correlation between cobalt, tin, and nickel presence ($p < 0.05$ for all three) in the ES compositions, while all three components are commonly found in diamond saws, the tool operated during the study (Borowiecka-Jamrozek and Konstanty 2025; Mexco Diamond Tools 2025). As noted previously, material hardness, which may affect wear on the saw, differs between stone products.

Ramkissoo et al. (2024) evaluated fabrication dust generated during cutting, whereas Mandler et al. (2026) evaluated dust generated during grinding.

For aluminum (Al), Ramkissoo et al. (2022); (2023) reported that ES dust samples ($n = 41$) contained trace levels (0.1%) to 3.4% Al, with a mean concentration of 0.41%. In contrast, white granite ($n = 1$) and white marble ($n = 1$) contained no detectable Al, whereas black granite ($n = 1$) contained 8.4% (mistakenly listed as 0.84% in the paper). Two building material samples included for reference contained 0.93% and 5.95% Al, respectively. Mandler et al. (2026) reported 0.42% Al for granite dust (which is very similar to the average reported across the 41 ES materials in Ramkissoo et al. (2022; 2023) and Al concentrations of 0.03% and 0.08% in the two silica-containing ES products evaluated in that study.

For iron (Fe), the Ramkissoo et al. (2022; 2023) reported that ES dust samples contained 0% to 1.0% Fe, with a mean concentration of 0.09%. White granite and white marble again showed no detectable Fe, whereas black granite contained 6.6%. The two reference building materials contained 1.13% and 0.37% Fe, respectively. Mandler et al. (2026) reported Fe concentrations of 0.02% and 0.03% for the two silica-containing ES dusts, compared with 1.20% for the granite dust evaluated in that study.

Ramkissoo et al. (2022; 2023), based on a very limited sample size ($n = 1$ for each of the NS materials), reported that dust from white granite contained a relatively substantial amount of magnesium (12%), whereas black granite, white marble, and engineered stone contained 3%, 0.4%, and 0.22%, respectively. By comparison, Mandler et al. (2026) reported 0.15% magnesium in the granite material they evaluated. White granite and white marble dust also contained substantial calcium concentrations in Ramkissoo et al. (21% and 37%, respectively), compared with engineered stones (0.36% on average) and black granite (6.2%). In Mandler et al. (2026), however, the granite dust contained only 0.15% calcium, illustrating substantial variability either by material type, by individual product, or by fabrication task.

The remaining metal compositions reported by Ramkissoon et al. (2022; 2023) were minimal, including 0.67% Ti, 1.8% Na, and 1.0% K in black granite, compared with average concentrations in ES dusts of 0.36% Ti, 0.52% Na, 0.04% K, and less than 0.01% for most other metals. Likewise, all remaining metals reported by Mandler et al. (2026) were less than 0.1% in ES dusts and less than 0.5% in the granite dust.

Table 1. Reported metal concentrations in the dust of different materials as reported in Ramkissoon et al. (2022); (2023) or Mandler et al. (2026) for the metals best characterized in the literature (Al, Fe) or those deemed relevant in some way by Ramkissoon et al. (2022; 2023) and Mandler et al. (2026).

Stone Type	Al Content	Fe Content	Co Content	Ti Content
White Granite (n = 1)	0%	0%	0%	0%
White Marble (n = 1)	0%	0%	0%	0%
Granite- Unspecified (n = 1)	0.42%	1.20%	0.05%	0.05%
ES (n = 43)	0.41% (0.03-3.4%)	0.09% (0-1%)	0.01% (0-0.02%)	0.36% (0-1.4%)
Black Granite (n = 1)	8.4%	6.6%	0%	0.67%
Building Materials (n = 2)	0.93%; 5.95%	1.13%; 0.37%	0%; 0.03%	0.12%; 0.28%

Ultimately, the available data indicates that specific metal content in ES countertop fabrication dust is unexceptional relative to other occupational RCS sources. The most comprehensive studies suggest that metal concentrations vary substantially across both engineered and natural materials, and that these differences do not provide a consistent or reliable basis for suggesting that they have any impact on the likelihood of developing silicosis.

4.6. RCS Threshold/Biologically Safe Dose

i. Is silicosis the most reliable and sensitive adverse endpoint for RCS risk?

Silicosis has long been regarded as the hallmark disease of RCS exposure and, among the chronic adverse effects associated with RCS, is generally considered the most sensitive endpoint for risk assessment purposes.

Although OSHA's quantitative risk assessment (QRA) for the 2016 crystalline silica rule may appear to suggest that estimated risks for lung cancer and non-malignant respiratory disease (NMRD) other than silicosis can be similar to or greater than those for silicosis at the same exposure level, this is better understood as a function of epidemiologic uncertainty and precautionary modeling rather than as evidence that silicosis is not the most sensitive or reliable endpoint. The use of life-table methods in the OSHA QRA, along with the assumptions embedded in those models, has been criticized in public comments on the rulemaking (Occupational Safety and Health Administration (OSHA) 2016).

In particular, NMRD is a heterogeneous category that may include silicosis, influenza, pneumonia, bronchitis, emphysema, chronic obstructive pulmonary disease, asthma, and other residual respiratory diseases, many of which have multiple alternative causes and are more susceptible to confounding by co-exposures and personal risk factors such as smoking and obesity. OSHA itself noted that, in the Park et al. (2002) study used for its NMRD risk estimates, smoking data were available for only about one-third of the NMRD decedents (Occupational Safety and Health Administration (OSHA) 2016). By contrast, silicosis is a pathologically distinctive disease with a well-established causal relationship to occupational RCS exposure.

The same general concern applies to lung cancer. As summarized by Mundt et al. (2025), epidemiological studies with relatively detailed smoking information have *“generally reported [lung cancer] risks close to background levels except in the highest cumulative RCS exposure categories”*. Lung cancer epidemiology is further complicated by the influence of polycyclic aromatic hydrocarbons, dioxins, fine particulate matter, and numerous other inhaled substances. These factors make lung cancer a heavily confounded endpoint for crystalline silica.

The epidemiology of Vermont granite workers employed between 1947 and 1998 provides strong support for the conclusion that silicosis is the most sensitive and reliable adverse health endpoint for cumulative RCS exposure. Despite very high cumulative exposures, divided into five exposure bands with the highest exceeding 4 mg/m³-years, no association was observed between cumulative RCS exposure and lung cancer, kidney cancer, or non-malignant kidney disease as reported in Vacek et al. (2011). In contrast,

cumulative RCS exposure was positively associated with silicosis and other NMRD mortality, with odds ratios of 1.13 and 1.10, respectively, for each 1 mg/m³-year increase in cumulative exposure. Importantly, Vacek et al. (2011) noted that the findings for “other NMRD” were less robust than those for silicosis, that the linear model tended to overestimate risk except at very high exposures, and that a statistically significant quintile trend was observed only for silicosis, not for “other NMRD”.

Accordingly, for RCS risk assessment, silicosis remains the most pathologically specific, and both sensitive and reliable endpoint under well controlled epidemiology. As ATSDR has stated, in consultation with EPA, among the major chronic effects of crystalline silica, “*silicosis is considered to be the most sensitive effect*” (Agency for Toxic Substances and Disease Registry (ATSDR) 2019).

ii. Does smoking or genetics affect silicosis risk and what other confounding factors for silicosis are there in epidemiology?

Confounding by smoking remains a persistent challenge in the epidemiology of many respiratory hazards, especially for alleged carcinogens, because even modest errors in smoking classification can materially affect risk estimates for respiratory outcomes. With respect to RCS specifically, there is mechanistic plausibility for smoking to influence silicosis risk, as smoking promotes inflammation and oxidative stress, including in the alveoli, the same tissues involved in crystalline silica-induced lung injury (Lugg et al. 2022; Cha et al. 2023). Consistent with this, synergistic effects between smoking and silica exposure, such as impaired pulmonary function, are documented (Quan et al. 2022). A retrospective cohort study published in 2024 also reported that heavy smoking (≥ 10 years) was significantly associated with advanced-stage (stage ii and iii) silicosis (Kang et al. 2024).

It should be noted that the possibility of additivity or synergy is a dose dependent phenomenon. It takes biologically significant doses of both agents for these effects to occur. Without distinction by ILO classification or severity, relationships between smoking and silicosis as a binary ‘yes’ or ‘no’ outcome are not consistently observed. Thus, while smoking may likely influence severity or progression, the available literature does not suggest that it displaces exposure to RCS as the principal determinant of silicosis causation. For example, smoking six cigarettes a day plus daily exposure to 0.15 $\mu\text{g}/\text{m}^3$ of RCS will not produce a synergistic response. The threshold estimates presented in later sections of this document are based on cohorts that included a significant fraction of smokers.

Genetic susceptibility has been proposed as a contributor to severity of the silicosis, including polymorphisms in inflammatory mediators, inflammasome components, and non-coding RNA pathways (Salum et al. 2020; Castro et al. 2022; Cheng et al. 2022; Wang et al. 2022; Chair et al. 2023). Yet the available evidence remains limited and is best interpreted

as indicating that genetic factors may, to some degree, modify response among exposed individuals rather than serve as primary determinants of the threshold risk of silicosis.

From a risk assessment standpoint, host factors do not supersede exposure. Threshold analyses for RCS and silicosis, discussed below, generally reflect mixed working populations that already include smokers and persons with varying degrees of underlying susceptibility. Other important confounding issues in the silicosis literature include co-exposures to other dusts and airborne contaminants, exposure misclassification, incomplete historical exposure reconstruction, and differences in particle characteristics across industries and tasks. These limitations do not undermine the causal relationship between silica and silicosis, but they do complicate attempts to derive precise quantitative risk estimates, especially at low exposure levels.

- iii. [How well do OSHA's linear and cumulative exposure assumptions reflect the biological reality of RCS toxicity, and do mechanistic and dose-rate evidence support a threshold model for silicosis?](#)

Despite OSHA's reliance on no-threshold models, when the totality of the evidence is considered, in the opinion of the authors, there is substantial support for the existence of a threshold or threshold-like behavior for RCS and its endpoints including silicosis. This remains heavily debated in the scientific community, especially for carcinogenic endpoints.

Additionally, OSHA's QRA effectively treats risk as a simple function of concentration multiplied by time, implying that equivalent cumulative exposures produce equivalent risks regardless of whether those exposures accrued gradually at low concentration or rapidly at high concentration. Although this cumulative exposure framework has long been used in crystalline silica epidemiology, it does not fully capture biological reality.

Available evidence suggests that dose rate matters. For example, in the diatomaceous earth cohort evaluated by Park et al. (2002), workers with similar cumulative exposures experienced substantially different (over 13 times greater) risks depending on exposure intensity, with higher disease incidence associated with shorter periods of higher exposure (American Chemistry Council (ACC) 2019). These findings are consistent with a broader toxicological understanding that the lung possesses clearance and repair mechanisms capable of managing particle burdens up to a point, beyond which macrophage-mediated clearance is impaired, inflammatory pathways (e.g., NLRP3 inflammasome activation), and oxidative stress responses become self-sustaining and lead to fibrosis (Cox 2017). Several recent analyses identify daily TWAs for establishing minimum threshold doses, rather than cumulative lifetime exposures.

Experimental studies describing a pre-fibrotic, reversible phase (Castranova et al. 2002), along with animal data indicating no fibrosis at lower concentrations and effects only at higher or prolonged exposures (Wagner 1986; Muhle et al. 1989), further support the view that silicosis exhibits a threshold or threshold-like behavior. When homeostasis is maintained, one would not expect disease (American Chemistry Council (ACC) 2019).

Compounding the shape of the model with potential exposure misclassification, especially in studies relying on job exposure matrices without direct measurement of RCS, introduces further uncertainty especially as risk estimates get further from the observable range. Low confidence exposure estimates greatly limit the conclusions that can be drawn from several studies (McDonald and Oakes 1984; Muir et al. 1989; Ng and Chan 1994; Steenland and Brown 1995; Chen et al. 2001; Churchyard et al. 2004; Wang et al. 2025). Taken together, these limitations complicate efforts to precisely define a threshold, but they also suggest that apparent risks observed at low exposure levels should be interpreted cautiously, particularly when derived from datasets with significant potential for confounding and misclassification.

Recent advances in statistical modeling continue to enable more precise identification for RCS acting as a threshold carcinogen and where this threshold takes place. In the past two decades, several epidemiological reviews (Morfeld et al. 2013, 2014; Cox Jr. 2017; Dickison 2019; Mundt et al. 2025; Wu et al. 2025). appear to agree that a most likely estimate of a threshold lies above what the current OSHA PEL (0.05 mg/m³ as an 8-hour TWA) allows. These studies are discussed in the next section. As expressed by one of these authors in their comments to *Safe Work Australia's Proposed Revision of the Workplace Exposure Standard for Silica, Crystalline (Respirable Dust)*:

“For respirable crystalline silica ... the threshold for turning on possible disease processes appears to be above 0.1 mg/m³ and may be as high as 0.4 mg/m³. Consequently, if long-term average silica exposures are maintained at or below 0.1 mg/m³, the risk of developing silicosis or other fibrotic lung pathologies should be negligible or nonexistent.” (American Chemistry Council (ACC) 2019)

Overall, OSHA's QRA projected lifetime silicosis risk at the PEL, ~7 in 1,000, is best viewed as conservative, policy relevant upper-bound estimates, rather than a “most likely estimate” or MLE of risk at low exposure levels. OSHA acknowledged that its approaches incorporate “... conservative assumptions [those that overpredict “true” risk] in interpreting the data ... risking error on the side of overprotection” (Occupational Safety and Health Administration (OSHA) 2016). Recent mechanistic evidence and dose-rate data support a threshold or threshold-like model for silicosis.

- iv. What are the most likely estimates for a threshold dose of RCS and how does that compare to the OSHA PEL of 0.05 mg/m³?

Regulatory limits are not synonymous with biological thresholds. At the same time, it is noteworthy that several recent independent analyses have suggested that a threshold for silicosis may exist at or above the current PEL, raising the possibility that the OSHA exposure limit, while derived using a precautionary QRA, may reflect a *de minimis* or effectively zero level of risk over the course of a worker's career.

Recent epidemiologic analyses employing improved statistical techniques, such as threshold Cox ("hockey-stick") and change-point models, have identified exposure levels at which risk begins to increase, with estimates often falling in the range of ~0.05 to 0.25 mg/m³ (Morfeld et al. 2013, 2014; Cox Jr. 2017; Dickison 2019; Mundt et al. 2025; Wu et al. 2025). While differences in study design and exposure reconstruction contribute to the variability in these estimates, the overall pattern is one of convergence toward a threshold TWA near or above the current OSHA PEL, including for smokers, suggesting that a *de minimis* risk persists when exposures are compliant with this standard.

Considering Morfeld et al. (2013, 2014) was published before OSHA's 2016 Silica Standard, it is not entirely clear based on OSHA's documentation why these studies did not hold more weight in their decision making (Occupational Safety and Health Administration (OSHA) 2016). Nonetheless, with the help of additional publications since the standard went into effect, which support the conclusions set forth by Morfeld et al., there is enough evidence to stand behind the current OSHA PEL as a scientifically defensible and appropriate standard.

One of the more recent reviews identifying a threshold for RCS was Mundt et al. (2025). In determining these thresholds, the authors conducted a systematic review examining exposure–response results from epidemiologic studies and identifying the point at which risk first became elevated (Mundt et al. 2025). Based on this synthesis, the authors reported that the most influential studies:

"... reported statistically significantly increased relative risk estimates for workers in exposure categories at and above 1.0 mg/m³-years [approximately 0.02 mg/m³ as an 8-hour TWA], with some threshold estimates at or above 3.0 mg/m³-years [0.07 mg/m³ as an 8-hour TWA over a working lifetime]." (Mundt et al. 2025) This analysis was based on the totality of the evidence reviewed, not taking each individual cohort and determining the weight each dataset held against each other to determine an "overall" threshold estimate.

Instead, Wu et al. (2025) provided the context necessary for a cumulative exposure of RCS that, based on the weight of evidence, is most likely the threshold for silicosis. Applying a change-point Cox modeling approach incorporating penalized likelihood methods based on exposure metrics from the epidemiological study Birk et al. (2025), the authors identified a cumulative exposure threshold of 4.038 mg/m³-years (95% CI: 3.109–4.967 mg/m³-years) Wu et al. (2025). When translated into a lifetime equivalent 8-hour TWA exposure (assuming a 45-year working lifetime), the resulting value is approximately 0.09 mg/m³ [in agreement with previous analyses by Morfeld et al. (2013) and Cox (2011a); (2011b; 2019)] and above the PEL of 0.05 mg/m³.

v. **How does the OSHA PEL compare to other national and international standards?**

It is important to recognize that OSHA's PEL was never intended to represent a definitive concentration below which no adverse effects could occur. Rather, it reflects a policy judgment regarding acceptable risk informed, but not exclusively driven by scientific evidence, feasibility considerations, and regulatory discretion. This is not unique to crystalline silica and is consistent with OSHA's broader approach to standard setting across most regulated substances) (Paustenbach and Cyrs 2021; Marrocco et al. 2024). The residual risk levels at the PEL are roughly consistent with other similarly derived estimates of adverse effects for other regulated chemicals like asbestos, hexavalent chromium, benzene, and others (U.S. Environmental Protection Agency (EPA) 1986; Occupational Safety and Health Administration (OSHA) 1987; Fairfax 1999; Proctor et al. 2016).

Other regulatory agencies across the globe (EU, UK, Japan, Sweden, Australia, and others) have adopted limits ranging from 0.05 to 0.1 mg/m³, and in some cases higher (e.g., China up to ~0.35 mg/m³), highlighting that the OSHA standard for RCS is among the strictest in the world. In a more recent analysis in late 2023, Cal-OSHA upheld the OSHA PEL despite the review process and opportunity to issue a new one (State of California Department of Industrial Relations 2024). The regulatory history in California has been to set aggressive limits.

5. Discussion

5.1 The Term “AS-Silicosis”

There has been a suggestion in the medical literature and in court proceedings that Artificial Stone-Silicosis (AS-silicosis) is a disease entity that is distinct from classic silicosis. Because silicosis was first described over 300 years ago, caution should be used when making the claim that the lung manifestations constitute a new condition. Despite that caveat, a review of the assertions that AS-silicosis represents a new, and more deadly, disease state is warranted.

An appropriate place to start is with the American Thoracic Society document entitled *Silicosis in the Artificial Stone Countertop Industry: An Official American Thoracic Society Workshop Report* (Gandhi 2026). Within the Report, AS-silicosis is defined as:

“Silicosis caused by exposure to AS dust, most commonly among countertop fabrication and installation workers. Compared to silicosis in many other industries, AS silicosis is notable for its shorter latency, more rapid progression, and poorer outcomes.” (Gandhi et al. 2026)

To our knowledge, this definition in Gandhi et al. (2026) is not derived from, nor endorsed by, any internationally recognized disease classification system or professional organization. We have been unable to identify a formal definition adopted by the World Health Organization (WHO) (Wagner 1996), National Institute for Occupational Safety and Health (NIOSH) (National Institute for Occupational Safety and Health (NIOSH) 2002), Occupational Safety and Health Administration (OSHA) (Occupational Safety and Health Administration (OSHA) 2016), or other recognized authorities. Rather, the definition appears to represent a descriptive summary developed by the Workshop authors (Gandhi et al. 2026).

Current disease-classification systems do not assign AS-silicosis a separate diagnostic code. Under ICD-10-CM, silicosis attributable to silica-containing dust other than the specifically enumerated categories is classified under J62.8: pneumoconiosis due to other dust containing silica (Centers for Disease Control and Prevention (CDC) 2024). This category encompasses several recognized forms of silica-related pneumoconiosis, including simple and complicated silicosis, pulmonary fibrosis attributable to silica-containing dust, and stonecutter's or stonemason's pneumoconiosis (Centers for Disease Control and Prevention (CDC) 2024). A separate code for AS-Silicosis does not appear in the ICD-10-CM classification (World Health Organization (WHO) 2019). This coding status does not determine whether a clinically useful exposure-associated phenotype may be described, but it does show that AS-silicosis is not presently classified as a distinct disease entity within that system nor does it appear on recent ICD-10-CM committee agendas to be considered for such a distinction.

From a toxicological perspective, the proposed features identified by Gandhi et al. (2026) are consistent with established dose-response principles. Higher exposure intensity, greater cumulative RCS dose, and shorter periods of very high exposure drive earlier disease onset and more severe progression (Marrocco et al. 2024). Thus, the occurrence of an aggressive disease among stone fabricators who work with engineered stone does not, by itself, distinguish a new disease distinct from acute or accelerated silicosis caused by high-intensity crystalline silica exposure (National Institute for Occupational Safety & Health

(NIOSH) 2004). Claims of a distinct disease based on latency, progression, or outcome, without considering dose, are inappropriate. RCS exposure from ES and NS fabrication, and the dose-response of this dust on an equal RCS basis is addressed in the background and results of this document, respectively.

Related publications involving Workshop authors have described diffuse centrilobular-predominant nodules, superimposed ground-glass opacities, lower-lung involvement, cavitary large opacities, and concomitant infections as atypical features observed among stone fabricators who work with engineered stone (Lateef et al. 2026). Lateef et al. (2026), however, also described substantial overlap between these findings and recognized manifestations or complications of acute, accelerated, and chronic silicosis.

These papers' conclusions are therefore more limited than a claim of radiologic specificity (Gandhi et al. 2026; Lateef et al. 2026). The papers characterize stone fabricators who work with engineered stone as having imaging findings that may differ in distribution, combination, or severity from traditional descriptions but do not identify a single pathognomonic imaging feature that distinguishes AS-silicosis from other recognized forms of silicosis (Akira 2002; Chong et al. 2006; International Labour Organization 2022; Gandhi et al. 2026; Lateef et al. 2026).

Establishing disease specificity would ordinarily require demonstration that the proposed features reliably distinguish engineered stone-exposed workers from appropriately selected silica-exposed comparison groups, including workers exposed to natural stone at comparable cumulative and peak respirable crystalline silica doses. The available evidence supports the conclusion that engineered stone fabrication can be associated with severe and rapidly progressive silicosis under conditions of intense or inadequately controlled RCS exposure. It does not, however, establish that AS-silicosis is a separately classified disease with unique diagnostic, radiologic, or pathologic criteria. The proposed distinguishing characteristics—short latency, rapid progression, poor outcome, ground-glass opacity, nodular disease, progressive massive fibrosis, cavitation, and infection—substantially overlap with established manifestations and complications of acute, accelerated, and chronic silicosis (MedlinePlus 2025; Lateef et al. 2026; Merck Manual 2026).

Presenting AS-silicosis as an established and distinct disease entity before its diagnostic specificity has been confirmed, risks converting a scientific hypothesis into an apparent medical fact with potentially significant consequences for clinical diagnosis and occupational health policy. Such a designation should therefore be reserved until appropriately designed studies show that the proposed phenotype can be distinguished from established forms of silicosis through controlled comparisons with natural stone-

exposed workers matched for cumulative silica dose, exposure intensity and duration, latency, and disease severity. To date, there have been no cohorts of isolated engineered stone or natural stone fabricators supporting novel RCS potency or disease outcomes. In practice, workers in this field nearly always work with both types of stone over the course of their career and, at times, within the same day. Assuming that there exists a disease due to working specifically with artificial stone is not supported by the currently available data.

The weight of scientific evidence does not indicate that ES-derived RCS is uniquely potent for silicosis compared with other common occupational sources of RCS, nor does it support treating it as distinct for a risk assessment or from a regulatory standpoint. From an occupational health perspective, disease prevention depends primarily on controlling RCS exposure (regardless of the material source). Employers can do so through the use of engineering controls, administrative controls, and respirators.

5.2 Elimination/Substitution

Replacing materials to reduce or eliminate a workplace hazard is the most direct means by which an employer can control exposure. However, such decisions may take time to implement and are influenced by existing inventory, cost, availability of suitable alternatives, product performance, client demands, supply-chain reliability, and applicable contractual constraints. As it has for the last one hundred years, it is almost certain that the demand for crystalline silica-containing materials will persist in a number of industries in the United States and globally. It may be problematic to try to ban these materials without addressing poor workplace conditions and considering the hazards of alternative materials.

Based on the available exposure and toxicity data, the effective (in Australia) or proposed bans regulating ES materials $\geq 1\%$ silica that do not similarly apply to NS materials are inconsistent from a risk assessment perspective. Furthermore, the substitution of materials based solely on by-weight crystalline silica content can have unintended negative consequences if applied universally to ES and NS countertop materials. As mentioned previously, crystalline silica content by volume may be substantially lower than by weight for engineered stone products, given that resin and other fillers are lighter than crystalline silica and the other contents in natural stone. Additionally, the use of resins that are substantially softer than other constituents of NS have been evidenced to release substantially less total dust per cut compared to a NS like granite.

5.3 Engineering Controls, Administrative Controls, and PPE

Numerous studies show that the use of wet methods or local exhaust ventilation (LEV) greatly reduces RCS exposures in the stone fabrication industry (Phillips et al. 2013; Cooper et al. 2015; Qi and Lo 2016; Zwack et al. 2016; Qi and Echt 2021). The effectiveness

of wet-fabrication techniques and LEV, and the combined effects of both controls, are dependent on the design of these systems (e.g., they need to be properly designed to be effective). Just saying that one uses a wet method is not sufficient without confirming the effectiveness with air sampling. For wet fabrication techniques, a recent NIOSH study suggests that proper water-fed tools (either center or edge fed) used in combination with sheet-flow techniques can be highly effective at reducing airborne concentrations to be below the PEL (Qi and Echt 2021). When airborne concentrations are not within regulatory limits, even with engineering controls (LEV and the use of wet methods), the use of respirators is relied on as part of long-term safety strategies in many industries and for many potential hazards (including RCS).

The recent evaluation by McGowan et al. (2025) identifies real world exposures across North American fabrication shops. While a disproportionate number of small unregulated shops may have been excluded from this dataset, the results (91% of 8-hr TWAs below the OSHA PEL; 76% of shops having zero PEL exceedances) indicates that the vast majority of worker exposures were well controlled. Consistent with other evaluations (Surasi et al. 2022), these authors identified that short-term tasks of dry fabrication using handheld tools were the source of nearly all overexposures.

The decision to implement a respiratory protection program and the selection of which respirator is right is a task-dependent decision by the employer, although several national and local regulatory frameworks apply. If necessary, or yet to be evaluated via workplace sampling, these authors recommend the use of power air purifying respirators (PAPR) to minimize discomfort to the workers. On the administrative side, the rotation of potentially high-exposure tasks (such as grinding or cutting) with lower exposure tasks (such as measurement, layout, housekeeping, or on-site installation) is a tool that employers can implement to further reduce TWA exposures for individual workers and improve compliance with respiratory protection programs. Implementing proper training, policies, and procedures can also greatly reduce worker exposures. For example, wetting down the floor of the workplace in the morning rather than just evenings.

Disciplined use and maintenance of the engineering controls and respirators at a worker's disposal is crucial for their effectiveness. Whenever crystalline silica-containing materials are manipulated with the potential to exceed the OSHA PEL, engineering and administrative controls must be used in tandem to comprise a comprehensive exposure control framework.

6. Limitations

Although crystalline silica is one of the most extensively studied occupational hazards, as is the case with nearly any topic, gaps in the scientific literature remain. Efforts

to translate mechanistic hypotheses into consistent *in vitro*, *in vivo*, and epidemiologic evidence with respect to comparing ES and NS RCS are still evolving. There is a lack of detailed characterization (particle size distribution, surface chemistry, metal contents) of airborne respirable fractions (rather than dust on the floor) beyond RCS mass characterization, which is the metric in most historical epidemiology for crystalline silica-exposed cohorts.

With respect to engineered stone, these materials have become increasingly popular over the past decade and, until recently, have not received the level of scientific attention we see today. For the entirety of the countertop fabrication industry, cohorts with robust exposure assessment and disease tracking are limited. Long-term epidemiologic data involving ES dust exposures, in the absence of natural stone dust co-exposures, have not been conducted. These limitations do not undermine the broader conclusions and reasonable certainty that, on an equal RCS basis, ES dust is not more potent for silicosis than NS or concrete. There is no question that all types of available countertops can be safely cut, manipulated, and installed when informed professionals are involved.

7. Conclusion

In summary, an evaluation of the available literature on particle size, surface area and surface chemistry, resins, crystalline silica polymorphs, VOCs and metals do not indicate that ES RCS should be treated as a distinct hazard compared to other occupational sources of RCS. If exposures are limited to be at or below the OSHA PEL, disease is not expected.

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