Crystalline Silica: Human Epidemiological Evidence

RoseyFields Chemical-Specific Epidemiology Review

Respirable Crystalline Silica (RCS) | CAS 14808-60-7 (quartz) | Prepared July 23, 2026 | Weight-of-evidence assessment for causation analysis

> *RoseyFields evaluates the human epidemiological evidence for Crystalline silica as follows.*

The human evidence base for respirable crystalline silica (RCS) is among the largest in occupational medicine, spanning more than a century of observation across dozens of industries and countries. That breadth allows a graded assessment in which some outcomes are supported by strong, consistent, quantitatively-modeled exposure-response data, others are supported only by suggestive or inconsistent associations, and still others have been investigated and found null or inadequate. This review synthesizes the peer-reviewed epidemiological literature underlying the principal regulatory and consensus evaluations, organizes the investigated outcomes by body system and by strength of evidence, and explains how that evidence functions—together with toxicology—in supporting general and specific causation opinions.

1. Study Populations and Data Sources

The RCS epidemiological literature draws on several distinct study architectures, and the convergence of findings across these architectures is what gives the strongest conclusions their durability. The dominant design is the **occupational cohort**, in which workers in a dust-generating industry are enumerated from employment records and followed for morbidity or mortality. These cohorts cover a striking range of settings: hard-rock and metal mining (South African gold miners, Chinese tungsten, tin and iron/copper miners, U.S. coal miners), quarrying and stone work (Vermont granite, Spanish granite), the manufacture of building and ceramic products (British and Chinese pottery, German porcelain, Swedish and Chinese foundries), and specialized mineral processing (U.S. industrial sand, California diatomaceous earth, Norwegian silicon carbide). The largest single cohort—the pooled Chinese miners and pottery workers followed by Wang et al. (2020a, 2020b)—contributed roughly 1.15 million person-years and nearly 9,400 silicosis cases, while long-running Western cohorts such as the German porcelain workers (Mundt et al. 2011; Birk et al. 2025) and the diatomaceous earth workers (Checkoway et al. 1997; Gallagher et al. 2015) provide decades of follow-up with detailed quantitative exposure reconstruction.

A second design is the **nested case–control study**, drawn from within a defined cohort, which permits more detailed individual-level covariate assessment (notably smoking and respirator use) than the parent cohort supports. Examples include Hnizdo et al. (1997) among South African gold miners, Steenland and Sanderson (2001) and McDonald et al. (2005) among U.S. sand workers, and Vacek et al. (2011, 2019) among granite and sand workers. A third architecture is the **population- or registry-based study**, which links national census or cancer-registry data to job-exposure matrices to estimate risk across the general working population rather than a single plant. Pukkala et al. (2005), using the Finnish census and cancer registry with the FINJEM job-exposure matrix, and Preller et al. (2010), within the Netherlands Cohort

Study, exemplify this approach, as does the IARC SYNERGY pooled analysis of 14 European and Canadian case–control studies (Ge et al. 2020). Community and contamination-site studies are comparatively rare for silica but include Kreiss and Zhen (1996), who examined silicosis risk in a Colorado mining community. General-population biomonitoring cohorts of the NHANES type, and birth cohorts, have essentially not been applied to RCS because there is no validated systemic biomarker of silica dose—exposure is quantified as airborne respirable dust, not as a serum or urinary analyte—so the general-population evidence remains thinner than for chemicals with a measurable body burden.

The value of assembling such diverse populations is methodological, not merely additive. Cohorts in different industries carry different co-exposures (radon and diesel exhaust in mines; lead in potteries; asbestos and non-fibrous minerals in various trades), different smoking distributions, different diagnostic practices, and different healthy-worker selection pressures. When an exposure-response relationship reproduces across mining, ceramics, foundry and mineral-processing settings—as it does for silicosis—the concordance argues strongly against any single confounder or bias being responsible, satisfying the Bradford Hill considerations of consistency and coherence. Conversely, when a relationship appears in some populations but not others (as with lung cancer), the heterogeneity itself becomes informative, directing attention to what differs between the positive and null studies.

2. Outcomes Investigated in Humans, by Body System and Strength of Evidence

The following table maps every major health outcome examined in the human literature to the body system involved and to a three-tier characterization of the evidence. The tiers are then discussed in turn.

Body system	Outcome	Evidence tier	Representative human studies
Respiratory (fibrotic)	Silicosis (chronic, accelerated, acute)	Strong / quantitatively modeled	Cherry 1998; Hughes 1998; Rego 2008; Vacek 2011, 2019; Wang 2020a; Birk 2025
Respiratory (malignant)	Lung cancer	Strong at high exposure; heterogeneous at low	Hnizdo 1997; Pukkala 2005; Ge 2020; Steenland & Stayner 1997; Gallagher 2015
Respiratory (obstructive)	COPD, chronic bronchitis, emphysema	Suggestive / inconsistent	Oxman 1993; Cowie & Mabena 1991; Hnizdo 1991; Cowie 1993
Respiratory (function/ mortality)	PFT decrement; non-malignant respiratory disease (NMRD) mortality	Suggestive (exposure-response in some)	Checkoway 1997; Costello 1995
Infectious	Pulmonary tuberculosis; non-tuberculous mycobacterial & fungal infection	Suggestive; strong when silicosis present	Cowie 1994; Corbett 1999; Kleinschmidt & Churchyard 1997; Hnizdo & Murray 1998
Immune / connective tissue	Scleroderma, rheumatoid arthritis, SLE, sarcoidosis	Suggestive / inconsistent	Cowie 1987; Klockars 1987; Rosenman & Zhu 1995; Rafnsson 1998; Steenland & Brown 1995b
Renal			

	End-stage renal disease, glomerulonephritis, subclinical tubular changes	Suggestive / inconsistent	Steenland 1990; Calvert 1997; Nuyts 1995; Ng 1992a; Boujemaa 1994; Hotz 1995
Digestive	Stomach / gastric cancer	Null / inadequate	Parent 1998; Cocco 1996 (review)
Other cancers	Esophageal, skin, brain, bladder, lympho-hematopoietic	Null / inadequate	Amandus 1991; various (no association established)
Respiratory (allergic)	Asthma	Not established / not investigated	Chan-Yeung 1994

2a. Outcomes with strong, consistent, quantitatively-modeled evidence

Silicosis is the paradigmatic silica-caused disease and carries the strongest evidence. Every occupational cohort that estimated cumulative RCS exposure reported increasing silicosis risk with increasing exposure, with a clear exposure threshold—although the numeric threshold varied by industry, diagnostic definition and exposure-reconstruction method (Mundt et al. 2025 systematic review). **Lung cancer** occupies a more nuanced position: the evidence is strong and quantitatively modeled at the high cumulative exposures characteristic of heavily-exposed historical cohorts and among silicotics, but heterogeneous and confounding-vulnerable at lower exposures. Both are treated in detail in Section 3.

2b. Outcomes with suggestive but inconsistent evidence

Autoimmune and connective-tissue diseases. More than a dozen post-1985 epidemiological studies reported statistically significant excesses of scleroderma (systemic sclerosis), rheumatoid arthritis, systemic lupus erythematosus and sarcoidosis among silica-exposed workers (Cowie 1987; Klockars et al. 1987; Rosenman and Zhu 1995; Rafnsson et al. 1998; Steenland and Brown 1995b), and a review by Steenland and Goldsmith (1995) found the pattern consistent enough to warrant a probable-association characterization by later panels. The evidence is nonetheless graded suggestive rather than strong because many contributing reports are case series or surveillance-registry analyses without individual exposure quantification, the outcomes are rare, and no consistent exposure-response function has been established.

Renal disease. Statistically significant associations have been reported between RCS exposure and end-stage renal disease morbidity (Steenland et al. 1990), incident ESRD from glomerulonephritis (Calvert et al. 1997), chronic renal-disease mortality (Steenland and Brown 1995b), Wegener's granulomatosis with glomerulonephritis (Nuyts et al. 1995), and subclinical tubular markers such as elevated urinary enzymes and low-molecular-weight proteins in exposed but non-silicotic workers (Ng et al. 1992a; Boujemaa et al. 1994; Hotz et al. 1995). The subclinical-marker studies are notable because they detected renal effects in the absence of silicosis, but the clinical significance and reversibility of those markers remains uncertain, keeping the outcome in the suggestive tier.

Tuberculosis and other infections. Silica impairs alveolar-macrophage killing of *Mycobacterium tuberculosis*, and the epidemiology shows clearly elevated TB risk among workers with silicosis (Cowie 1994; Corbett et al. 1999; Hnizdo and Murray 1998) and increased incidence in higher-dust jobs even among some workers without radiographic silicosis (Kleinschmidt and Churchyard 1997). The association is biologically coherent and, in silicotics, strong; it is graded suggestive at the population level chiefly

because background TB rates are driven heavily by socioeconomic and geographic factors that are difficult to disentangle from occupational dust in the available studies.

COPD, chronic bronchitis and emphysema. A structured overview of methodologically rigorous cohorts concluded that mineral (especially gold-mine) dust is an important cause of COPD, particularly in smokers (Oxman et al. 1993). Cross-sectional and mortality studies link exposure to chronic bronchitis and airflow limitation independent of smoking (Cowie and Mabena 1991), and emphysema has been associated with silica dust and with silicosis at autopsy (Hnizdo et al. 1991; Cowie et al. 1993). The inconsistency across non-gold-mining populations, and the dominant role of smoking, place these obstructive outcomes—and the related decrements in pulmonary-function testing and the broader category of non-malignant respiratory disease (NMRD) mortality, for which Checkoway et al. (1997) and Costello et al. (1995) reported exposure-response trends—in the suggestive tier.

2c. Outcomes investigated with null or inadequate findings

Gastric and other extra-pulmonary cancers. Excess stomach-cancer deaths have been reported in scattered occupational groups, and one Canadian case–control study found an odds ratio of 1.7 (95% CI 1.1–2.7) for "substantial" silica exposure after adjustment for smoking and other factors (Parent et al. 1998). However, a systematic review of gastric cancer in dusty occupations concluded that, because most studies neither adjusted for confounders nor examined dose-response, the evidence was insufficient to classify silica as a gastric carcinogen (Cocco et al. 1996). Associations reported for esophageal, skin, brain, bladder and lympho-hematopoietic cancers (e.g., Amandus et al. 1991) are isolated and unconfirmed, and no causal association has been established for any extra-pulmonary cancer. **Asthma** has not been identified as silica-related; crystalline silica is not recognized as an asthma-inducing (sensitizing) agent and has essentially not been the subject of dedicated epidemiological investigation for asthma induction (Chan-Yeung 1994).

3. Strong-Evidence Outcomes in Detail

3.1 Silicosis

Exposure metric. Across the literature, cumulative RCS exposure—expressed in mg/m³·years and reconstructed from job-exposure matrices linked to personal and area air-sampling—is the metric reported by essentially all quantitative studies, with average intensity (mg/m³) and duration used secondarily. Outcome ascertainment is predominantly radiographic, using the International Labour Organization (ILO) profusion score, with silicosis most often defined as ILO ≥ 1/0 or ≥ 1/1 read by trained, blinded B-readers; some studies rely on death certificates or national diagnostic criteria (Mundt et al. 2025).

Effect sizes and direction. The direction is uniformly positive and the magnitudes are large. Among British pottery workers, Cherry et al. (1998) modeled an odds ratio of 1.37 (95% CI 1.24–1.53) per 1 mg/m³·year of cumulative exposure. In the U.S. diatomaceous-earth cohort, Hughes et al. (1998) reported relative risks rising from 4.35 (95% CI 1.7–11.1) at >1–3 mg/m³·years to 43.5 (95% CI 16.7–113.5) above 8 mg/m³·years, relative to <1 mg/m³·year. Spanish granite workers showed odds ratios of 10.16 (95% CI 3.3–31.0) at 6.75–11.71 mg/m³·years and 31.46 (95% CI 10.3–95.5) at the highest exposures (Rego et al.

2008). Nested case–control analyses of U.S. granite and sand workers found relative risks of 8.62 (95% CI 1.86–39.95) at 3.65–6.71 mg/m³·years (Vacek et al. 2011) and 12.57 (95% CI 2.08–55.47) at 3.01–5.50 mg/m³·years rising to 25.5 (95% CI 5.5–117.8) above 5.5 mg/m³·years (Vacek et al. 2019). The very large Chinese cohort reported tungsten-miner relative risks of 3.90, 11.24 and 19.50 across ascending exposure quartiles (Wang et al. 2020a), and the German porcelain cohort reported a hazard ratio of 16.3 (95% CI 6.1–43.4) in the highest category for ILO ≥ 1/0 silicosis (Birk et al. 2025). Zhang et al. (2010) reported an odds ratio of 5.38 (95% CI 3.82–7.57) per unit exposure in Chinese foundry workers.

Consistency and threshold. A PRISMA-compliant systematic review of the exposure-response literature (Mundt et al. 2025) found increasing silicosis risk with increasing cumulative RCS exposure in every one of the ten qualifying studies, with substantially and statistically significantly increased risk clear at or above roughly 1 mg/m³·year when silicosis is defined as ILO ≥ 1/0, and thresholds nearer 3 mg/m³·years for the more specific ILO ≥ 1/1 definition. Estimated apparent thresholds ranged from as low as 0.39 mg/m³·years (Lenander-Ramirez et al. 2022, based on only six cases) to above 6.75 mg/m³·years (Rego et al. 2008), a spread attributable to differing case definitions, exposure-reconstruction methods and reference-group construction rather than to any doubt about the underlying relationship. This is the model of a strong, consistent, quantitatively-characterized causal relationship: reproducible across mining, ceramics, foundry and mineral-processing populations on three continents, monotonic, and specific to the exposure.

3.2 Lung cancer

Exposure metric and outcome. As with silicosis, cumulative RCS exposure (mg/m³·years) from job-exposure matrices is the primary metric; lung-cancer outcomes are ascertained from death certificates, cancer registries or the National Death Index. The critical methodological feature distinguishing lung-cancer studies is the need to control tobacco smoking, the dominant cause of lung cancer, which in these cohorts carried relative risks of roughly 2.7 to 20.9 (Mundt et al. 2025).

Effect sizes and heterogeneity. The exposure-response results are mixed. Some studies show clearly elevated risk confined to the highest exposures: South African gold miners had a relative risk of 3.19 (95% CI 1.3–7.6) above 6.3 mg/m³·years (Hnizdo et al. 1997); the Finnish registry study found 1.42 (95% CI 1.2–1.7) at ≥10 mg/m³·years (Pukkala et al. 2005); and diatomaceous-earth workers showed hazard ratios of 1.98 (95% CI 1.11–3.54) and 2.36 (95% CI 1.25–4.46) in the two highest cristobalite-exposure categories (Gallagher et al. 2015). U.S. sand-worker analyses reported significant trends and a top-category relative risk of about 2.0–2.7 (Steenland and Sanderson 2001; McDonald et al. 2005). By contrast, several strong studies found no significant association at any exposure: British pottery workers (Cherry et al. 2013), U.S. coal miners (Graber et al. 2014), Dutch cohort participants (Preller et al. 2010), and the German porcelain cohort, which showed no lung-cancer excess at all (standardized mortality ratio 0.90, 95% CI 0.78–1.04 in men; Birk et al. 2025). The IARC SYNERGY pooled analysis (Ge et al. 2020)—the largest and one of the highest-quality studies—reported modestly elevated, statistically significant odds ratios across exposure categories in current and former smokers, and, critically, in never-smokers an odds ratio that reached significance only in the open-ended highest category (1.4, 95% CI 1.03–1.86 at ≥2.4 mg/m³·years), indicating that a threshold for lung-cancer risk in the absence of smoking lies somewhere above 2.4 mg/m³·years.

Interpretation. The consistent signal is that lung-cancer risk becomes demonstrable at high cumulative exposures—levels at or above those that also produce silicosis—and that the weaker, low-exposure associations are readily explained by residual confounding by smoking and by exposure misclassification, which attenuates exposure-response slopes. The systematic review concluded that lung-cancer risk may not be increased at cumulative exposures below the silicosis thresholds, and that preventing silicosis would be expected to prevent silica-related lung cancer (Mundt et al. 2025). This graded conclusion coexists with the firm hazard identification (Section 5): crystalline silica is a recognized human lung carcinogen, but the quantitative causation question at a given exposure level is exposure-dependent.

4. Study-Quality Considerations Affecting Confidence

Confounding by tobacco smoking is the single most consequential quality issue, and it bears far more heavily on lung cancer than on silicosis. Because smoking is such a powerful lung-cancer risk factor, any cohort in which more heavily exposed workers also smoked more will generate spurious or inflated associations; even studies with individual-level smoking data generally captured only crude ever/never categories rather than pack-years, leaving residual confounding that can bias small effect estimates away from the null (Mundt et al. 2025). The most persuasive lung-cancer evidence therefore comes from the never-smoker analyses (Ge et al. 2020), where confounding by smoking is structurally removed. For silicosis, smoking is a far weaker concern, though some studies reported modest smoking–silicosis associations that may reflect diagnostic bias (Birk et al. 2025; Zhang et al. 2010).

Reverse causation and diagnostic bias. Radiographic silicosis ascertainment is sensitive to reader bias: Mundt et al. (2011) documented systematic over-reading in compensation-oriented films that was corrected only by blinded systematic re-reading to ILO/B-reader standards. The choice of ILO ≥ 1/0 versus ≥ 1/1 materially changes both case counts and apparent thresholds, the more sensitive 1/0 cut-point being more susceptible to false positives. In lung-cancer work, the "healthy-worker survivor effect" and the observation that silicotics carry higher lung-cancer risk raise the question of whether fibrosis is a step on the causal pathway or a marker of the exposures that cause cancer independently.

Exposure misclassification is universal: no study possessed individual airborne measurements spanning each worker's full history, so all relied on job-exposure matrices and historical reconstructions carrying substantial uncertainty. Non-differential misclassification biases exposure-response slopes toward the null and can obscure genuine thresholds; differential misclassification (for example, sampling preferentially during upset conditions) can distort estimates in either direction. Widely differing exposure-category cut-points and reference-group definitions across studies further complicate direct comparison and partly explain the apparent threshold spread for silicosis.

Co-exposure to related agents limits attribution in specific cohorts. Silicon-carbide workers were simultaneously exposed to cristobalite, SiC dust and SiC fibers, so quartz-specific risk could not be isolated (Bugge et al. 2012); miners carry radon and diesel-exhaust co-exposures; pottery workers were exposed to lead; and various trades to asbestos. Studies that adjusted for or were free of these co-exposures —industrial-sand cohorts, for instance, are relatively free of radon and diesel—carry correspondingly greater weight. These considerations do not undermine the silicosis conclusion, which is robust across co-

exposure profiles, but they appropriately temper confidence in individual lung-cancer and extra-pulmonary findings.

5. Independent Expert-Panel and Agency Weight-of-Evidence Conclusions

Multiple independent bodies have issued formal weight-of-evidence determinations. The **International Agency for Research on Cancer** classifies inhaled crystalline silica (quartz and cristobalite) from occupational sources as a **Group 1 human carcinogen**, concluding there is "sufficient evidence in humans" for its carcinogenicity, while explicitly cautioning that carcinogenicity "was not detected in all industrial circumstances studied" and may depend on inherent characteristics of the silica or on external factors affecting its biological activity (IARC 1997; IARC 2012). The **American Thoracic Society** concluded that lung cancer is associated with occupational silica exposure and that silicosis increases the risk of bronchogenic carcinoma, while noting the evidence is "less clear" as to whether silica causes lung cancer in the absence of silicosis (ATS 1997). The **U.S. National Institute for Occupational Safety and Health** designated respirable crystalline silica a potential occupational carcinogen and, on the noncarcinogenic side, concluded that the evidence supports silica as a cause of silicosis, renal and autoimmune disease, mycobacterial infection, and chronic obstructive airway disease.

On the **quantitative** side, meta-analyses summarized a modest overall lung-cancer summary relative risk of about 1.3 among silica-exposed workers and higher summary relative risks of roughly 2.2 to 2.8 among silicotics (Smith et al. 1995; Steenland and Stayner 1997), and IARC's later monograph identified seven meta-analyses of lung cancer among silicotics with relative risks between 1.69 and 3.27 (IARC 2012). For silicosis, three exposure-response analyses estimated a 45-year working-lifetime silicosis risk of 47–90% at the historical OSHA/MSHA permissible limits and roughly 10–30% at the more protective NIOSH recommended limit (Hnizdo and Sluis-Cremer 1993; Steenland and Brown 1995a; Kreiss and Zhen 1996). The convergence of these independent evaluations—hazard identification for lung cancer, quantitative dose-response for silicosis, and "probable link" characterizations for autoimmune and renal disease—forms the consensus backdrop against which any individual causation opinion is measured.

6. Use of the Epidemiological Evidence in Causation Analysis

In litigation, epidemiology performs two logically distinct functions, and RCS illustrates both cleanly. **General causation**—whether the substance is capable of causing the disease in humans at all—is established for silicosis and, as a hazard, for lung cancer by the combination of consistent, exposure-responsive human cohort data and the coherent toxicological mechanism (macrophage-mediated inflammation, oxidative stress and fibrogenesis). The IARC Group 1 classification and the concordant ATS and NIOSH conclusions supply the recognized-authority anchor that courts commonly look to. For the suggestive-tier outcomes (autoimmune disease, renal disease, TB, COPD), general causation is supportable but more contestable, and an opinion must acknowledge the weaker, less quantitative evidence honestly.

Specific causation—whether this substance caused this claimant's disease—turns on the quantitative exposure-response relationship and on the individual's reconstructed cumulative exposure measured against the thresholds the epidemiology defines. Here the graded nature of the evidence is decisive. Because silicosis rises steeply above roughly 1 mg/m³·year and because lung-cancer risk in the human data is demonstrable mainly at or above the silicosis-producing range, a defensible specific-causation opinion requires estimating the claimant's cumulative RCS dose and comparing it to those benchmarks, while accounting for smoking history (particularly for lung cancer), latency, and competing causes. The same body of evidence that supports causation at high occupational doses simultaneously constrains it: direct extrapolation from these high-exposure occupational cohorts to ambient or low-level exposures is not epidemiologically valid, since a century at typical ambient concentrations (on the order of 0.3 mg/m³·years cumulatively) falls well below the observed silicosis thresholds (Mundt et al. 2025). Epidemiology does not stand alone in this analysis—it is integrated with toxicological dose-response, mechanistic data on particle surface reactivity and fibrogenesis, and the individual exposure reconstruction—but it provides the quantitative human anchor without which a causation opinion cannot bridge from hazard to attribution.

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