Crystalline Silica Exposure Issues in the Traditional Ceramic Industries

by

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Introduction

Crystalline silica in the form of quartz is a well-known component of many ceramic mixes in processing of traditional ceramic products. It is a measurable component of many clays due to geologic processes such as the kaolinization of granite leaving a SiO_2 residue in the kaolin deposit. It is an intentional additive in the form of "Potter's flint" in whiteware products serving as a filler and a framework to prevent excessive deformation in firing. It is a *nemesis* in some respects due to its relationship to cooling cracks in the firing process as different forms of silica have different densities leading to volume changes when changes are experienced.

The fact that the low temperature form of crystalline silica, quartz, "converts" to higher temperature forms with other crystal structures on heating is well known, and these new forms are known as tridymite and cristobalite (Table 1). It is now widely known, however, that tabulated data for these transformations is misleading as the conversions are complex and sluggish. Grimshaw states that "the changes (conversion of quartz to tridymite) are so slow that pure quartz can be maintained at temperatures well above 870^{o}C". He continues, " Almost invariably, cristobalite forms in preference to tridymite even in the temperature range of stability of the latter material" (1).

Added to the complexity in understanding crystalline silica, the forms of silica undergo polymorphic inversions producing new forms with the same crystal structure but slightly different interatomic spacing. Furthermore the conversions (Table 1) are not reversible. For example, on heating quartz above about 1250^{o}C, quartz converts "directly" to cristobalite with polymorphic inversions of cristobalite observed on cooling (and sometimes related to cracking known as "dunting"). As most technologists expect physiochemical changes to occur at exact temperatures, they are highly frustrated when they learn that the α to β cristobalite inversion usually is seen somewhere in the *range* of 210-280^{o}C. This means tabulated data on silica conversions and inversions can be viewed only as a guideline.

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Table 1: Summary of Important Conversions and Inversions of Silica

Process & (Volume Increase)	Type	Tabulated Temperature of the Process	Observed Temperature of the Process
β quartz → β tridymite (13%)	Conversion (not reversible)	870°C	Usually not observed*.
β tridymite → β cristobalite (5%)	Conversion (not reversible)	1470°C	Usually not observed*.
β quartz → β cristobalite (18%)	Complex Conversion (not reversible)	-	~1250°C (depends on impurities)
α quartz → β quartz (2%)	Inversion (Reversible)	573°C	573°C (but can be observed at higher crown thermocouple readings if the product size is appreciable).
α cristobalite → β cristobalite (5%)	Inversion (Reversible)	210-280°C	210-280°C (May result in "dunting")

* In some long term firing processes, such as firing of silica refractories, tridymite is observed usually together with cristobalite.

Now the ceramic industry is faced with decisions regarding health effects of crystalline silica exposure by the International Agency on Research For Cancer (IARC) in 1996. IARC, a component of the World Health Organization of the United Nations, reclassified crystalline silica as a "Class I Carcinogen" publishing its findings in the IARC *Monographs* in 1997 (2). While it is a fact that the IARC "Working Group" was divided on the vote to reclassify silica, the overriding fact is that the action was taken.

Concern over exposure to high levels of respirable silica has driven debates in the field of occupational health and safety since the early part of this century (3). Early concerns over "epidemics" of silicosis in the extractive industries of mining and quarrying and in metalworking, ceramic manufacturing and sandblasting led to the first attempts to regulate exposure to crystalline silica. In the past, the regulation of occupational exposure to crystalline silica involved more than an academic discussion of the etiology and pathology of silicosis among radiologists and pulmonary specialists. The imposition of clinical judgements on health issues in the workplace has always carried with it *policy implications*. The need for administrative procedures for making sound

judgements about acceptable exposure levels has also involved political action in the form of regulations and standards (4).

During the long debate over silica exposure, separation of fact and fiction has been complicated by differing exposure conditions amongst different industries and by technological changes in processing. As medical diagnostic procedures and sampling methods improved over time, there were adjustments to the established exposure standards that reflected the current understanding of the relationship between crystalline silica and health risks. The effects of prolonged exposure to high levels of respirable crystalline silica have been established (5,6,7). Yet there remains *controversy* concerning how crystalline silica produces pathological outcomes and under what conditions these pathological outcomes are most likely to occur. There are also uncertainties concerning the relationship between the level of exposure, duration of exposure, and the influence of other factors on the emergence of diseases related to crystalline silica exposure.

The current effort by the Occupational Safety and Health Administration to drastically reduce exposure standards by one half reflects modifications in the evaluation of the carcinogenic properties of crystalline silica. The *possible link* between exposure to crystalline silica and lung cancer has driven efforts to reduce the permissible exposure levels (PEL's) and the threshold limit values (TLV's). OSHA has indicated that the previous pattern of "negotiated rulemaking" will not be employed by the agency unless the recent "stakeholder meetings" offers compelling reasons to change this policy. Thus, compelling reasons exist for reviewing health information on silica pertaining to the traditional ceramic industries.

The Health Threat Posed By Crystalline Silica

The inhalation of *"free silica"* (quartz, tridymite, or cristobalite) can produce fibrotic lung disease known as silicosis. Free silica is a distinct particle or phase that exists as the mineral or compound SiO_2. Ceramic materials and products also contain *"combined silica"*, i.e. silica in combination with other species (forming different minerals) or as a vitrified (glassy) phase.

Of crucial importance for respiratory exposure are the size, nature, and chemical composition of the silica particles. The size of the particles, their concentration, and the duration of exposure to the dust containing the particles are important factors in determining:

- the attack rate (how many workers develop disease in a given time period),
- latency period (how long it takes disease to manifest itself after exposure begins),
- incidence rate (how rapidly new cases occur in a given time period), rate of progression (how quickly the disease moves into an advanced stage), and
- the outcome of disease (morbidity, disability, or death) (8).

Respirable particles (smaller than 10μ in diameter) can be inhaled into the conducting airways of the lungs and the gas exchange regions of the lung. Lung disease develops through three processes: 1) free silica induces a direct cytoltoxic effect on the alveolar cells; 2) damaged macrophages release oxidants and protolytic enzymes and directly cause lung damage. Macrophages then secret factors that recruit and activate polymorphoneuclear leukocytes which in turn release oxidants and enzymes which cause further parenchymal (functional) damage; 3) stimulated macrophages also secret numerous factors that induce fibroblast proliferation and stimulate collagen synthesis by fibroblasts.

Cytotoxic effects are the result of the chemical characteristics of the silica crystal surface involving the presence of free radicals—"loose" molecular bonds—that are highly reactive to cell membranes within the lung. Microphages represent an immune system response to the presence of the silica particles. Eventually fibrosis (the abnormal formation of fibrous tissue) begins occurring, reducing pulmonary functioning (9). When lung tissues are exposed to free crystalline silica, alveolar macrophages (part of the body's immune system) release tumor necrosis factor (TNF), an extracellular factor that controls the body's inflammatory responses. TNF triggers the production of a range of secretions and chemicals that stimulate the growth of fibroblasts (10). Once fibroblasts are activated, fibrogenesis (the creation of fibrous masses in the lung) begins (11). Fibrotic degeneration in the lungs is accelerated by the presence of free radicals, which are highly reactive and encourage the production of proteins and molecular secretions that further encourage fiber production (12). The fibers are made up of collagen, a family of proteins that can form large fibers. Increased collagen deposits destroy normal lung structure and decrease the lung's effectiveness in exchanging gases.

In addition to silicosis, inhalation of free crystalline silica has been associated with pulmonary tuberculosis, industrial bronchitis with airflow limitations, and severe extrapulmonary diseases. The American Thoracic Society reports that exposure to working environments contaminated by silica at dust levels that appear not to cause silicosis can nevertheless cause chronic airflow limitations and/or mucus hypersecretion and/or pathogenic emphysema (13).

The concentration of crystalline silica in respirable dust and its ability cause adverse reactions in the lungs is determined by:

- the silica content and composition of the materials used,
- the nature of the manufacturing process and/or processing the quartz materials undergo, and
- the environmental conditions in which these processes take place.

The industries with the greatest risk for the development of silicosis and other related diseases are heavy construction, sandblasting, painting and

refinishing, pottery making, mining, segments of refractory manufacturing, and quarrying (14). Three areas of manufacturing are of special attention due to increase risk in exposure:

- industries using materials with high concentrations of free silica in the materials used,
- industries in which the quartz material is processed in such a way as to produce ragged crystals with freshly cleaved surfaces, and
- industries in which the crystalline silica is combined with other minerals which also contribute to disease (15).

Some mitigating processing factors are well known. The conditions surrounding the processing of materials are also a factor. The higher the moisture content or the "wetter" the processing, the lower the dust levels and the lower the risk of respirable free silica.

Crystalline Silica and Lung Cancer

In 1997, IARC noted that the carcinogenicity of silica was not detected in all industrial settings and it may be dependent on external factors affecting its biological activity or the distributions of its polymorphs. Weill and McDonald (6) concluded that in the absence of fibrotic disease, evidence that exposure to crystalline silica causes cancer must be considered *scanty and inconsistent* but biologically plausible. Conflicting claims and contradictory results mark the research literature on the relationship between crystalline silica exposure and the development of lung cancer.

The major problem with much previous research is the confounding of lung carcinogens such as smoking and radon exposure and of selection bias in the detection of cases of pneumoconiosis, a chronic inflammation of the lungs (13). The research literature is also characterized by: differences in risk estimates across studies.

Factors contributing to differences in risk estimates (16) include:

- *extrapolation* of the results of studies in one or two industries to all industries using silica,
- *errors* in exposure estimation,
- *differences* in measurement techniques,
- *differences* in the physiochemical properties of the silica and quartz content of the dust; and
- *cohort differences.*

A major weakness in most previous research is the use of disease registries for the selection of the study population. Weill and McDonald (6) have noted that, although studies using silicosis registries have raised the question of

a link between the risk of lung cancer and exposure to crystalline silica, they cannot contribute to any formal risk assessment because of unquantifiable selection bias. Subjects selected from silicosis registries are likely to have respiratory symptoms and impaired function related to *other risk factors* such as smoking or exposure to other suspected carcinogens. Until more studies that control effectively for these other carcinogens are conducted, the link between exposure to free crystalline silica and increased risk for lung cancer remains *biologically plausible* but *unconfirmed*. Even though the research results are *mixed,* several health organizations have declared crystalline silica to be a substance that acts as a carcinogen.

"A" Causes "B", and "B" Causes "C", but Does "A" Cause "C"?

Although stating that the carcinogenicity of silica is not detected in all industrial settings, the IARC (1997) has categorized crystalline silica as a Group I carcinogen – sufficient evidence of carcinogenicity in humans. In 1988, the National Institute for Occupational Safety and Health (NIOSH) recommended that crystalline silica be considered a *potential* occupational carcinogen. In 1992, the National Toxicology Program (NTP) of the United States Department of Health and Human Services listed respirable crystalline silica as among the substances which may reasonably be anticipated to cause cancer in its Sixth Annual Report on Carcinogens (1992). The American Thoracic Society (1997) stated that the disease silicosis should be considered a condition that *predisposes* workers to an increased risk for lung cancer. While the epidemiological evidence for a direct link between exposure to crystalline silica and the development of lung cancer contains many contradictory and controversial findings, the research literature linking silicosis to lung cancer is less ambiguous.

Several research studies suggest that while there may be no direct link between crystalline silica exposure and lung cancer, there is a link between exposure to crystalline silica and the development of silicosis. There is also a strong relationship between the development of silicosis and increased risk for lung cancer (6, 7,17,18,19). It has been suggested that the fibrogenesis created by silica exposure may predispose a person to carcinogenesis. It is also possible that fibrogenesis, cigarette smoking and other causal agents interact to raise the risk for lung cancer (15).

Other studies have established a relationship between lung cancer and silicosis *but not between lung cancer and exposure to silica*. The mere exposure to silica may not be sufficient to cause lung cancer unless the silica exposure eventually produces the onset of silicosis.

A study by Amandus, et al. (18) examined 760 cases of silicosis collected between 1940 and 1983 and found that even when controlling for age and smoking, lung cancer rates were 3.9 times higher among miners with silicosis than among miners without silicosis. It has been suggested that fibrotic and

silicotic lesions in the lungs resulting from inhalation exposure to crystalline silica establish a “threshold” for lung cancer and that lung cancer is secondary to the development of these lesions in the lung (19). A meta analysis of 16 studies with well-documented silica exposure and low probabilities of confounding by other occupational exposures found the highest relative risk (2.3) for lung cancer among those with silicosis (7).

The Ceramic Industry, Silicosis, and Lung Cancer

A review of research on the nature of crystalline silica exposure in the ceramic industry reveals several interesting trends. There is a tendency in many studies to combine brick manufacturing with pottery making and fire brick or refractory brick manufacture (2, 20, 21, 22, 23). This distorts the nature of the risks posed by any one industry.

Another pattern in research findings is that some industrial segments in the ceramic industry consistently appears to produce levels of dust exposure that exceed the given exposure standard at the time the study is conducted (24, 25, 26, 27, 28, 29). This pattern persists in studies from the 1930s through the 1970s and 1980s. *In spite of excessive dust levels within those plants, the incidence of silicosis is relatively low.* There is, in the words of Rajhans and Budlovsky (29), a “discrepancy” between the environmental data and the medical findings.

Possible explanations for the combination of high exposure levels and low levels of silicosis in some ceramic industries involve the following:

- the alumina content of the clays (29, 31),
- the pH value of the materials (29, 31),
- the moisture content of the materials (24, 31),
- the silica content of the clays and rock (16), and
- the grinding and processing of the silica materials (47 Fubini, et al., 1995).

The presence of alumina may inhibit the reaction of lung tissue to crystalline silica (31, 32). In similar fashion, the pH value also affects reactivity - with alkaline levels above seven being associated with lower levels of fibrogenic activity in the lungs. Fubini, et al., (31) found that polymorphs of silicon dioxide exhibit different biological responses depending on the processes used to grind, heat, or etch them during manufacture. Grinding in the presence of water, using acids to etch the material, and thermal treatments all seem to reduce the presence of free radicals. Absorption of water was associated with reduced fibrogenicity.

The age of the dusts also affects reactivity. Freshly ground silica has a higher degree of toxicity due to the reactivity of the newly created surfaces. Stable surface radicals present in aged dusts do not seem to be involved in the pathogenic process (33). Some free radicals undergo rapid decay in the first

hours after grinding. Thus, in ceramic processing, where the process takes some time and other factors intervene, there may be time for the surfaces to stabilize. This is unlike the situation found in sandblasting, where exposure to freshly fractured surfaces is almost immediate and patterns of acute silicosis are often observed among workers (34). Acid etching smoothes crystal surfaces and eliminates surface radicals. Alumina may affect the acidity of the surface and thus lower biogenic reactivity. Thus, as pointed out by Donaldson and Borm (35), the hazard posed by quartz is not *a constant entity*, but one that may vary dramatically depending on the origin of the silica sample or its contact with other chemicals or minerals.

The growing evidence of a link between silicosis and elevated risk for lung cancer is a concern of the ceramic industry. Epidemiological studies have shown no excess cancer mortality among workers who have not developed silicosis. However, the presence of silicosis plays a significant role in the pathogenesis of silica-induced lung cancer.

Research results link the onset of pneumoconiosis and pulmonary malignancies to silicosis (17). The disease processes resulting in silicosis carry an increased risk for lung cancer. Researchers have called for further studies to determine if the Environmental Protection Agency should establish a reference concentration for crystalline silica based on a "no observable effect level" (NOAEL) for fibrogenesis. Levels suggested for NOAEL have ranged from 0.02 $\mu g/m^3$ to 0.04 $\mu g/m^3$ (19). Rice and Stayner (16) have called for further research to define the dose-response relationship between silica exposure and silicosis.

Unless studies are completed comparing the ceramic industry with other industries, there is the possibility that a universal LOAEL will be developed that may not reflect the lower risk for the development of silicosis observed in some ceramic industries as compared non-ceramic industries.

Fact and Fiction

Just as the conversions and inversions of crystalline silica are complex to understand, the epidemiological data on silicosis, lung cancer, and the ceramic industries must be studied carefully before conclusions are drawn. A further complicating factor is that many individuals outside of the ceramic industry just don’t understand the difference between “free” and “combined” silica. This all contributes to a scenario where regulators, who don’t completely understand the issues, are proposing “one size fits all” regulations on crystalline silica exposure.

The facts are that exposure to crystalline silica in segments of the ceramic industry is of a *significantly lower risk* to workers than in other industries where freshly cleaved particles are present and clay is absent in respirable dust. Silicosis is much less prevalent in ceramic workers than levels predicted based

on exposures. And silicosis, by the body of evidence, is a precursor to lung cancer.

The facts don't dispute that crystalline silica exposure causes silicosis in some industries. The fiction, which seems to predominate in regulatory thinking, is that all silica exposure is equally dangerous. These are complex issues, but they are no so complex that the industrial and scientific communities can't recognize and develop regulations based on *good science*.

References

1. The Chemistry and Physics of Clays, Rex W. Grimshaw, Techbooks (1971).
2. International Agency for Research on Cancer (1997) IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Silica, some silicates, dusts, and organic fibers. Vol. 68, Lyon, France: World Health Organization.
3. Keatinge, G. and N. Potter (1949) Health and environmental conditions in brickworks. British Journal of Industrial Medicine, 1(31):31-44.
4. Markowitz, G. and D. Rosner (1992) The illusion of medical certainty: Silicosis and the politics of industrial disability, 1930-1960. Pp. 185-226 In Rosenberg, C. and J. Golden (Eds.) Framing Disease: Studies in Cultural History. New Brunswick, New Jersey: Rutgers University Press.
5. Gregorini, G., Tira, P., Frizza, J., Haese, P., Elseviers, M., Nuyts, G. Maioria, R., and M. De Broe (1997) ANCA-Associated diseases and silica exposure. Clinical Reviews in Allergy and Immunology, 15:21-40.
6. Weill, H., and J. McDonald (1996) Exposure to crystalline silica and risk of lung cancer: The epidemiological evidence. Thorax, 51(1):97-102.
7. Steenland, K., and L. Stayner (1997) Silica, asbestos, man-made mineral fibers, and cancer. Cancer Causes and Control, 8:491-503.
8. Occupational Safety and Health Administration ( ) Special Emphasis Program, Silicosis. Washington, D.C.: U. S. Department of Labor.
9. Gregorini, et. al., op. cit.
10. Agostini, C., Siviero, M. and G. Semensato (1997) Immune effector cells in idiopathic pulmonary fibrosis. Current Opinion in Pulmonary Medicine, Sept. 3(5):348-355.
11. Deheinzelin, D., Jatene, F., Saldiva, P. and R. Brentani (1997) Upregulation of collagen messenger RNA expression occurs immediately after lung damage. Chest, Nov. 5, 112(5):1184-1188.
12. Poli, G. and M. Parola (1997) Oxidative damage and fibrogenesis. Journal of Free Radicals In Biology And Medicine, 22(1-2):287-305.
13. American Thoracic Society (1997) Adverse effects of crystalline silica exposure. American Journal of Respiratory and Critical Care Medicine, 155:761-763.
14. Starzynski, Z., Marek, K., Kujawska, A., and W. Szymczak (1996) Mortality among different occupational groups of workers with pneumoconiosis:

Results from a register-based cohort study. American Journal of Industrial Medicine, 30:718-725.
15. Dufresne, A., Begin, R., Dion, C., Vagridar, J., Rom, W., Loosereewanich, P., Muir, D., Ritchie, A., and G. Perrault (1998) Angular and fibrous particles in lung in relation to silica-induced diseases. International Archives of Occupational and Environmental Health, 71(4):263-269.
16. Rice, F. and L. Stayner (1995) Assessment of silicosis risk for occupational exposure to crystalline silica. Scandinavian Journal of Work and Environmental Health, 21, Suppl. 2:87-90.
17. Meijer, J., Sauen, G., and J. Slangen (1996) Mortality and lung cancer in ceramic workers in the Netherlands: Preliminary results. American Journal of Industrial Medicine, 30:26-30.
18. Amandus, H., Shy, C., Castellan, R. Blair, A. and E. Heineman (1995) Silicosis and lung cancer among workers in North Carolina dusty trades. Scandinavian Journal of Work and Environmental Health, 21, Suppl. 2:81-83.
19. Klein, K. and J. Christopher (1995) Evaluation of crystalline silica as a threshold carcinogen. Scandinavian Journal of Work and Environmental Health, 21, Suppl. 2:95-98.
20. Collis, E. and G. Yule (1933) The mortality experience of an occupational group exposed to silica dust, compared with that of the general population and an occupational group exposed to dust not containing silica. The Journal of Industrial Hygiene, 15(6):395-417.
21. Sayers, R., Dallavalle, J. and S. Bloomfield (1937) Occupational and environmental analysis of the cement, clay, and pottery industries. Public Health Bulletin No. 238, Sept. 1937, Division of Industrial Hygiene, National Institute of Health.
22. Maxfield, R., Alo, C., Reilly, M., Rosenman, K., Kaolinoswski, D., Stanbury, M., Valiente, D., Jones, B., Randolph, S., Socie, E., Gromen, K., Migliozze, A., Willis, T., Schnitzer, P., Perotta, D., Gretzmacher, G., Anderson, H., Jajosky, R., Castellan, R., Game, S., ILL Dept. of Public Health, MI State U., MI Dept. of Consumer and Industry Services, NJ Dept. of Health, NC Dept. of Health and Natural Resources, Ohio Dept. of Health, TX Dept. of Health, WI Dept. of Health and Social Services, NIOSH (1997) Surveillance for silicosis, 1993--Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin. MMWR, 46(SS-1):13-28.
23. Linch, K., Miller, W., Althouse, R., Groce, D., and J. Hale (1998) Surveillance of respirable crystalline silica dust using OSHA compliance data 1979-1995. American Journal of Industrial Medicine, 34:547-558.
24. Bureau of Industrial Hygiene, West Virginia State Health Department (1939) Preliminary report of a study in the brick and tile industry in West Virginia.
25. Colorado School of Mines Research Institute (1978) Industrial hygiene study.
26. Carstens, C. (1972) Silicosis potential in North Carolina brick industry survey, 1969-1972. North Carolina Board of Health.
27. National Institute of Occupational Safety and Health (1980) North Carolina brick industry industrial hygiene and respiratory disease morbidity survey,

1974-1975. Cincinnati, Ohio: National Institute for Occupational Safety and Health.
28. Freeman, C. and E. Grossman (1995) Silica exposures in workplaces in the United States between 1980 and 1992. Scandinavian Journal of Work and Environmental Health, 21, Suppl. 2:47-49.
29. Rajhans, G. and J. Budlovsky (1972) Dust conditions in brick plants of Ontario. American Industrial Hygiene Association Journal, Apr:258-268.
30. Keatinge, G. and N. Potter (1949) Health and environmental conditions in brickworks. British Journal of Industrial Medicine, 1(31):31-44.
31. Fubini, B., Bolis, V., Cavenago, A., and M. Volante (1995) Physiochemical properties of crystalline silica dusts and their possible implication in various biological responses. Scandinavian Journal of Work and Environmental Health, 21, Suppl. 2:9-14.
32. Burger, C. (1953) Experiences with aluminum therapy--silica brick industry paper. Presented at the 5th Conference of McIntyre Research Foundation On Silicosis, Chicago, Illinois.
33. Love, R., Waclawski, E., MacLaren, W., Porteous, R., Groat, S., Wetherill, G. Hutchinson, P., Kidd, M., and C. Soutar (1994) Cross sectional study of risks of respiratory disease in relation to exposures of airborne quartz in the heavy clay industry. IOM Report: Technical Memorandum Series 94/07, Edinburgh:Institute of Occupational Medicine.
34. Castranova, V. (1994) Generation of oxygen radicals and mechanism of injury prevention. Environmental Health Perspectives, 102, Suppl. 10:65-68.
35. Donaldson, K. and P. Borm (1998) The quartz hazard: A variable entity. Annals of Occupational Hygiene, Jul 42(5):287-94.