Session No. 608

Settling the Dust: Silica Past, Present and Future

Dana M. Hollins, MPH, CIH
ChemRisk, LLC
San Francisco, CA

John L. Henshaw, MPH, CIH
ChemRisk, LLC
Sanibel, FL

Matthew H. Le, MPH, CIH, CSP
ChemRisk, LLC
San Francisco, CA

William D. Cyrs, MS, CIH
ChemRisk, LLC
San Francisco, CA

Introduction

Crystalline silica has been one of the most widely studied chemicals in the history of occupational disease and industrial hygiene. It is the most toxic form of silica, with estimates of approximately 1.7 million U.S. workers exposed to respirable crystalline silica in the mining, sandblasting, and construction industries. The National Institute of Occupational Safety and Health (NIOSH) estimates that over 15,000 silicosis deaths occurred over the last three decades (NIOSH 2005; NIOSH 2012). In addition, the numbers of workers exposed to crystalline silica could rise with the increasing prevalence of hydraulic fracturing, or fracking, in this country.

Despite the large number of workers exposed to crystalline silica, there is no comprehensive Occupational Safety and Health Administration (OSHA) standard requiring employers to assess employees' silica exposure, monitor potential health effects, and provide necessary worker training. Currently, silica exposure is regulated solely through an OSHA Permissible Exposure Limit (PEL) adopted when OSHA was established over 40 years ago (OSHA 2003). The current crystalline silica OSHA PEL for general industry is dependent on the amount of respirable quartz in the collected sample and is calculated using a formula proposed by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1968 (OSHA 2010):

$$PEL = \frac{10^{mg/m^3}}{(\%SiO_2) + 2}$$

The PEL for the construction and maritime industries is based on particle counting technology from the 1970s. These PELs for crystalline silica exposure are considered outdated or obsolete, and according to OSHA, workers are still at significant risk of developing silicosis at exposures below the current PELs (OSHA 2010). OSHA is expected to lower the PEL for silica as part of a comprehensive new standard, which is currently under review at the White House Office of Management and Budget (OMB).

Silica in the 20th Century (1900-1989)

Human Health Studies

Lung diseases resulting from dust exposure have long been recognized by health professionals as far back as the 16th and 17th centuries. Dusty work environments were prevalent during the industrial revolution in the United States due to the increasingly widespread mechanization and the use of pneumatic tools in industry and mining operations (Lanza 9, 360, 367). These dust exposures were uncontrolled for the most part, as dust suppression measures were not often utilized and respiratory protections were not typically worn by workers. As a result, disease and mortality rates due to silica exposures in the dusty trades significantly increased during this period, as compared to other occupations at this time (Air Hygiene Foundation of America, Inc. 16).

Silicosis was not recognized as a distinct disease until the early 1900s. Some of the first accounts linking silica exposure to severe lung disease were from studies of miners in the United Kingdom and Australia as well as published reports by the Miner's Phthisis Prevention Committee and the South African Institute for Medical Research (Miners' Phthisis Prevention Committee, 9-12; Lanza, 6-7). Although chest x-rays, in addition to other detection methods were being frequently used in the 1930s, early detection of silicosis was difficult. Even after significant medical advances in diagnoses of silicosis and tuberculosis, observations of increased rates of both diseases in the same populations led researchers to believe that exposure to crystalline silica increased the risk of contracting tuberculosis. These studies included miner cohorts from Great Britain, Australian and South Africa that mined a number of minerals including quartz, sandstone, gold, tin, coal and slate (Air Hygiene Foundation of America, Inc. 13-38). In 1915, Dr. E.L. Collis, a researcher in the United Kingdom, was one of the first to note that "free" crystalline silica caused serious lung injury, as well as concurrently increased susceptibility of tuberculosis (Lanza 8).

Beginning in the 1920s, numerous large-scale epidemiology studies were being conducted in the United States, particularly in the mining, granite and foundry industries. The mining studies focused on disease incidence and latency in lead and zinc metal mining in Missouri, Kansas, Montana and Oklahoma (Madl et al. 556). Additional studies focused on disease incidence of nonmetal mining such as coal (soft and hard) and hematite. Prevalence rates in these studies, however, varied considerably by industry and region. The nonmetal mining studies were among the first to show that the quartz content of dust was a significant risk factor associated with the mining cohorts, and thus explained the variability in disease rates among cohorts (Brundage & Frasier 1; Sayers 46, 48-49, 73; Air Hygiene Foundation of America, Inc. 93, 110). The importance of exposure duration and latency was also noted in these studies.

Perhaps the most important of the early epidemiological studies were conducted in granite mining regions of New England. Russell and colleagues conducted one of the most comprehensive epidemiological studies in 1929 related to exposure to granite dust and the incidence of silicosis (Russell et al. 1-5). The findings from this study were ultimately used as the basis for the first Occupational Exposure Limit (OEL) for silica. The granite studies confirmed that dose, duration and percent silica content of the inhaled dust were significant risk factors associated with the incidence of lung disease (Madl et al. 556-557). In addition, studies on foundry workers also confirmed the prevalence of silicosis among foundry workers associated with felting, cleaning, finishing and abrasive blasting operations (Sander 603; McLaughlin et al. 74-76).

By the 1970s and the passage of OSH Act, silica was identified as one of five key industrial exposure hazards in the United States. Epidemiologic studies conducted during the 1970s and 1980s began to look at disease rates and latency with silica exposed cohorts (Theriault et al. 16, 27; Rice et al. 78). According to these studies, the OSHA PEL of 0.1 mg/m3 was protective for silicosis.

Toxicology Studies

In the 1930s, animal studies involving silica exposure were conducted mainly at the Saranac Lake Laboratory and the results of which were shared during a series of symposia known as the "Saranac Lake Symposia" (Kuechle 40). These studies, among others, demonstrated that silica depressed the function of leukocytes and that silica was directly toxic to macrophages, the primary cells involved in immune responses in the lung, thus resulting in increased susceptibility to bacterial diseases such as tuberculosis (Mavrogordato 3, 7-9, Gardner 1928). In the late 1930s, it was first reported that the extent of tissue damage was inversely proportional to particle size. Particles greater than 10 μm in diameter had no appreciable effect on lung cells (Gardner 1928). The dose response relationship between silica exposure and lung disease, in addition to duration of exposure, were also confirmed via animal inhalation studies.

In 1953, King and colleagues (10, 12, 17) found that tridymite produced the most severe and rapid response in rat lungs. These studies subsequently formed the basis for development of OELs for the various forms of silica. It was also shown by numerous researchers that silica particles ranging from 0.5 to 8 μm produced fibrosis in the rat lung (Goldstein & Webster 72, 74, King et al. 10).

The primary focus of animal studies conducted during the 1980 timeframe was to evaluate the carcinogenic potential of crystalline silica; however, it was discovered that silica's ability to cause lung tumors was limited to rats (IARC 74-79).

Regulatory Actions/Guidelines

The first OEL for silica was recommended by the U.S. Public Health Service (USPHS) and based upon the granite, gold and anthracite miners (Russell, et al. 1929). The recommendation was for 9-20 millions of particles per cubic foot (mppcf), less than or equal to 10 μm in 1929 (Madl et al. 560). The recommended levels decreased as more research was conducted, and by 1946, ACGIH had recommended an OEL that included the percent of free silica within the Threshold Limit Value (TLV) calculation (ACGIH 55). By 1962, when the potencies of the various forms of silica were established, ACGIH's previously formulated maximum allowable concentrations (MACs) were subsequently changed to TLVs for crystalline and non-crystalline silica.

Workplace Controls

Beginning in the 1920s, engineering controls associated with abrasive blasting in the automotive industry were evaluated. Engineering controls such as enclosed units, positive-pressure air supplied helmets, wetting methods, ventilation controls and good housekeeping afforded the best protection of worker exposure (Winslow et al. 519-520; Bloomfield & Greenburg 188-189; National Silicosis Conference 1). In the 1950s and 1960s, a number of associations and other bodies including the American Industrial Hygiene Association (AIHA) and ACGIH, published recommendations regarding respiratory protection use during abrasive blasting.

Prompted by a number of European countries banning silica in abrasive blasting, alternatives began to be investigated starting in the 1950s and research into the toxicity of these alternatives was beginning to be conducted (Holmqvist & Swensson 254). Throughout the 1970s, NIOSH was actively involved in investigations of abrasive blasting practices and the resulting reports from these investigations often discussed what respiratory protection measures were necessary to adequately protect workers against silica exposures. These specifications included the use of a separate air supply (either as a supplied-air respirator or an air-supplied hood) (NIOSH 8).

Modern Years (1990-2013)

Current Health Hazard State-of-Knowledge

A major focus of health studies during the late 20th century and beginning of the 21st century was to further characterize the dose-response relationship between crystalline silica and silicosis. Improvements in exposure assessment and dose reconstruction methods, as well as follow-up with the established occupational cohorts, allowed scientific research to generate quantitative estimates of disease risk at different levels of silica exposure over a working lifetime (Madl 584). These new studies suggested that the current OSHA PEL for silica did not provide sufficient protection against the development of disease (Madl et al. 584). Most of these studies offered a quantitative estimate of risk for silicosis mortality, and at least one developed a model to estimate a no observed adverse effect level (NOAEL) at which illness would not be expected (Rice and Stayner 87; Madl et al., 584).

Human Health

In the years following the designation of silica as a group 2A carcinogen (probably carcinogenic to humans) by the International Agency for Research on Cancer (IARC) in 1986, various epidemiological studies were conducted to address the relationship between exposure to silica and the development of silicosis and lung cancer (Madl 585). These studies reported increased lung cancer risk; however, in many cases, these associations were not statistically significant, or statistically significant findings were limited to those workers clinically diagnosed with silicosis (Madl et al. 586). In fact, the issue as to whether silicosis is a necessary step in the development of lung cancer through exposure to silica has been a controversial issue (Checkoway and Franzblau 252-253).

In 1996, IARC re-evaluated the carcinogenicity of crystalline silica, in which the committee relied upon the least confounded (those with the least bias arising from the co-occurrence or mixing of effects of extraneous factors) epidemiological studies (IARC 86). IARC

concluded that "the epidemiological findings support increased lung cancer risk from inhaled crystalline silica (quartz and crystabolite) resulting from occupational exposure" that could not be explained by known confounders or any other biases (IARC 86). Therefore, IARC designated crystalline silica a group 1 carcinogen (carcinogenic to humans). However, IARC also noted "carcinogenicity in humans was not detected in all industrial circumstances studied" (IARC 210).

In 2002, NIOSH published a health hazard review for silica. The review focused on the same epidemiologic cohorts as the 1997 IARC assessment, with some exclusions based on confounding exposures (NIOSH 23-25). Overall, the review performed by NIOSH supported the conclusion made by IARC that there was an association between lung cancer risk and silicosis.

Toxicology

The mechanisms underlying silica-induced carcinogenesis in animals are still not fully understood. During the 1990s, researchers focused on characterizing the mechanisms by which silicosis occurs and its apparent role in lung cancer development (Madl 593). As reported by Madl (593), many of the toxicological studies at the time focused on how particle surface chemistry, intercellular signaling pathways, and oxidant stress may induce inflammation and stimulate the immune system leading to tissue fibrosis. Some of these studies also led to the investigation for potential biomarkers of exposure, effect, and susceptibility for silicosis (Gulumian et al., 357-386). The formation of lung tumors in rats exposed to silica may also be consistent with a non-specific response to persistent inflammation and increased cell proliferation (Mossman et al.1116).

Regulatory Actions/Guidelines

The ACGIH TLV for quartz remained at 0.1 mg/m3 throughout the 1990s, and in 2000, the TLV was decreased to 0.05 mg/m3 based on studies suggesting that the risk of silicosis associated with exposure to 0.1 mg/m3 over a working lifetime was well above the established acceptable risk level of 1 in 1,000. In addition to decreasing the TLV, ACGIH also designated quartz as a group A2 carcinogen (suspected human carcinogen) In 2006, ACGIH chose to combine the TLV for quartz and crystabolite, and the combined TLV was set at 0.025 mg/m3 (ACGIH 71). The basis for the change was that epidemiologic studies in the diatomaceous earth industry (exposure to crystabolite) during the late 1990s and early 2000s reported that exposure-response risk for silicosis was similar to that for quartz (ACGIH 1-3).

There is a particular need for OSHA to modernize its occupational exposure limits for silica for all industries, The current OSHA PEL, which was based on the 1968 ACGIH TLV of [10/(% quartz +2)] mg/m^3 for respirable dust and [30/(% quartz +2)] mg/m^3 for total dust, still remains unchanged from its initial adoption in 1971. Furthermore, the current OSHA PEL for construction and shipyards (derived from the 1970 ACGIH TLV) is based on particle counting technology, which is considered obsolete. NIOSH and ACGIH recommend 50$\mu g/m^3$ and 25$\mu g/m^3$ exposure limits, respectively, for respirable crystalline silica (OSHA 2010). Both industry and worker groups have recognized that a comprehensive OSHA standard for crystalline silica is needed in order to provide for exposure monitoring, medical surveillance, and worker training (Iafallo 1-3).

Although OSHA was considering a comprehensive standard for occupational exposure to crystalline silica much earlier, it completed the Small Business Regulatory Enforcement Fairness Act (SBREFA) Report for the proposed rule in December, 2003, and peer review of silica health

effects and risk assessment in January, 2010 (OSHA 2010). Currently, the proposal is stalled in the OMB's Office of Information and Regulatory Affairs (OIRA). The OIRA review has lasted for approximately two years (Iafallo 1-2).

The proposed rule will have one of three alternative PELs, 50, 75, or 100 $\mu g/m^3$ as an 8-hour time-weighted average (TWA), although 100 $\mu g/m^3$ does not represent a deviation from the current general industry formula PEL in the vast majority of settings (OSHA 2003). The proposed standard will also include an action level, which would trigger additional provisions such as ones related to health screening and training (OSHA 2003).

Current Activities

Several organizations have voiced concerns recently regarding the delays in promulgating the new proposed silica rule. For example, the American Society of Safety Engineers (ASSE) wrote a letter to an OIRA administrator in September, 2011, encouraging OIRA to move forward with the review of OSHA's rulemaking on silica (Norris 1). In this letter, ASSE President Terri Norris noted that the "silica issue first appeared on OSHA's Unified Agenda in 1997 [and] OSHA completed the SBREFA Report at the end of 2003" (Norris 2). She added that inadequate resources for standard development, potential future litigation, and political motivations have contributed to OSHA's difficulty in setting timely standards. In addition, Ms. Norris expressed desire to contribute to the standard's development, along with other stakeholders, noting that the ASSE's membership has extensive expertise in protecting workers from the hazards of silica without burdening industry (Norris 2).

In addition, AIHA president Elizabeth Pullen wrote a letter to OMB Director Jacob Lew and Secretary of Labor Hilda Solis in November, 2011, encouraging them to avoid further delays so that the public rulemaking process can begin as soon as possible (Pullen 1-2). She asserted that although the AIHA recognized the merit of OIRA review with regard to costs/benefits and quality assessment, it considered an additional delay "unacceptable" in that it rendered the proposed rule more vulnerable to political influence (Pullen 1). Ms. Pullen also noted that there was a lack of transparency of the OMB review process, and that it was important for the rule to be released for publication so that industry, labor, and the safety and health community could provide meaningful input in the public forum. In addition, she encouraged the OMB to defer to "OSHA's scientific judgment as much as possible" (Pullen 2).

Conclusion

Knowledge regarding health hazards from crystalline silica exposure and how to best safeguard exposed workers has evolved drastically over the past century. However, regulatory uncertainties and the fact that many workers are still overexposed to silica dust in the U.S. and abroad have led to a call for a comprehensive OSHA standard. Some labor groups and occupational safety advocates have even called for a ban on all types of abrasive blasting, including methods that do not use sand or silica, as a means to safeguard workers. Promulgating a standard that contains requirements for engineering controls, respiratory protection, exposure monitoring, training, and medical surveillance might create an environment where a ban would not be necessary. In addition, such a standard would likely serve to protect workers on a global scale, since many countries would presumably adopt the standard.

Bibliography

Air Hygiene Foundation of America Inc. *Silicosis and Allied Disorders: History and Industrial Importance. Medical Series, Bulletin No. 1. April 15, 1937*. Pittsburgh, PA: Air Hygiene Foundation of America, Inc, 1937.

American Conference of Governmental Hygienists (ACGIH). "Report on the Sub Committee on Threshold Limits." Proceedings of the Eight Annual Meeting of the American Conference of Governmental Hygienists: Chicago, Illinois- April 7-13, 1946. Cincinnati, OH: American Conference of Governmental Hygienists, 1946. 54-56.

American Conference of Governmental Industrial Hygienists (ACGIH). *Documentation of Threshold Limit Values for Silica, Sixth Edition Supplement.* Cincinnati, OH: American Conference of Governmental Industrial Hygienists, 2000.

American Conference of Governmental Industrial Hygienists (ACGIH). *Documentation of Threshold Limit Values for Silica, Crystalline: Quartz and Cristobalite, 7th Edition*. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, 2006.

Bloomfield, J.J., and L. Greenburg. "Sand and metallic abrasive blasting as an industrial health hazard." *Journal of Industrial Hygiene*. 15. 1933: 184-204.

Brundage, D.K., and E.S. Frasier. *The Health of Workers in Dusty Trades. General Statement and Summary of Findings: III. Exposure to Dust in Coal Mining; IV. Exposure to Dust in a Textile Plant; V. Exposure to the Dusts of Silverware Manufacturing Plant; VI. Exposure to Municipal Dust (Street Cleaners in New York City). Public Health Bulletin No. 208*. Washington, DC: Government Printing Office, 1933.

Checkoway, H., and A. Franzblau. "Is silicosis required for silica-associated lung cancer?" *American Journal of Industrial Medicine*. 37. 2000: 252-259.

Gardner, L.U. "Etiology of pneumoconiosis." *Journal of the American Medical Association*. 11. 1938: 1925-1936.

Goldstein, B., and I. Webster. "Intratracheal injection into rats of size-graded silica particles." *British Journal of Industrial Medicine*. 23. 1966: 71-74.

Gulumian, M., P.J. Borm, V. Vallyathan, V. Castranova, K. Donaldson, G. Nelson, and J. Murray. "Mechanistically identified suitable biomarkers of exposure, effect, and susceptibility for silicosis and coal-worker's pneumoconiosis: a comprehensive review." *Journal of Toxicology and Environmental Health. Part B, Critical Reviews*. 9. 2006: 357-395.

Holmqvist, I., and A. Swensson. "The fibrogenetic effect of a granulated iron silicate intended for use in sand blasting: An experimental study." *Int. Arch. Gewerbepathol. Gewerbehyg.* 20. 1963: 253-260.

Iafolla, R. "Silica: Petition for Action on Silica Rule Falls Short AS OMB Review Stretches to Two Years. 43 OSHR 152." *Occupational Safety & Health Reporter*. Feb 14. 2013.

IARC Working Group. "Silica and some silicates." *IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans*. Volume 42. 1987: 39-143.

International Agency for Research on Cancer (IARC). *Volume 68: Silica, Some Silicates, Coal Dust and Pararamid Fibrils*. Lyon, France: International Agency for Research on Cancer, 1996.

King, E.J., G.P. Mohanty, C.V. Harrison, and G. Nagelschmidt. "The action of different forms of pure silica on the lungs of rats." *British Journal of Industrial Medicine*. 10. 1953: 9-17.

Kuechle, B. *Symposium on Silicosis. An Unofficial Transcript of the Silicosis Symposium Held in Connection with the Trudeau School of Tuberculosis at Saranac Lake, N.Y., June 18 to 22, 1934*. Wausau, WI: Storey-Bellack Co, 1934.

Lanza, A.J. *Miners' Consumption: A Study of 433 Cases of the Disease Among Zinc Miners in Southwestern Missouri. Public Health Bulletin, No. 85. January 1917*. Washington, DC: Government Printing Office, 1917.

Lanza, A.J. *Silicosis and Asbestosis*. New York: Oxford University Press, 1938.

Lanza, A.J., and E. Higgins. *Pulmonary Disease Among Miners in the Joplin district, Missouri and Its Relation to Rock Dust in the Mines: A Preliminary Report. Technical Paper 105*. Washington, DC: Government Printing Office, 1915.

Madl, A.K., E.P. Donovan, S.H. Gaffney, M.A. McKinley, E.C. Moody, J.L. Henshaw, and D.J. Paustenbach. "State-of-the-science review of the occupational health hazards of crystalline silica in abrasive blasting operations and related requirements for respiratory protection." *Journal of Toxicology and Environmental Health. Part B, Critical Reviews*. 11. 2008: 548-608.

Mavrogordato, M.A. "The Aetiology of Silicosis." IVe Réunion de la Commission Internationale Permanente Pour les Maladies Professionnelles, Lyon (3-6 avril 1929). Lyon: A. Rey, 1929.

McLaughlin, A.I.G., E.A. Goodman, J. Garrad, S.R. Gloyne, K.L. Goodall, H.E. Harding, M.H. Jupe, W.B. Lawrie, K.M.A. Perry, C.L. Sutherland, and H. Woods. *Industrial Lung Diseases of Iron and Steel Foundry Workers*. London: His Majesty's Stationery Office, 1950.

Miners' Phthisis Prevention Committee. *General Report of the Miners' Phthsis Prevention Committee*. Johannesburg, South Africa: The Government Printing and Stationary Office, 1916.

Mossman, B.T., L.A. Jimenez, K. BeruBe, T. Quinlan, and Y.M.W. Janssen. "Possible mechanisms of crystalline silica-induced lung disease." *Applied Occupational and Environmental Hygiene*. 10. 1995: 1115-1117.

National Institute for Occupational Safety and Health (NIOSH). *Occupational Exposure to Crystalline Silica*. Cincinnati, OH: National Institute for Occupational Safety and Health, 1974.

National Institute for Occupational Safety and Health (NIOSH). *Health Effects of Occupational Exposure to Respirable Crystalline Silica. April 2002*. Washington, DC: National Institute for Occupational Safety and Health, 2002.

National Institute for Occupational Safety and Health (NIOSH). 2005. *NIOSH Safety and Health Topic: Chest Radiography - The NIOSH B Reader Certification Program*. (retrieved October 5, 2005). (http://www.cdc.gov/niosh/topics/chestradiography/breader.html).

National Institute for Occupational Safety and Health (NIOSH). 2012. *Silica*. (retrieved March 14, 2013). (http://www.cdc.gov/niosh/topics/silica/).

National Silicosis Conference. *Report on Engineering Control: Final Report of the Committee on the Prevention of Silicosis Through Engineering Control*. Washington, DC: U.S. Department of Labor, Division of Labor Standards, 1938.

Norris, T.S. *Memorandum from Terrie S. Norris, CSP, ARM (ASSE) to Cass R. Sunstein (White House Office of Information and Regulatory Affairs). RE: Allowing OSHA Rulemaking on Occupational Exposure to Crystalline Silica to Move Forward. Dated September 22, 2011*. Des Plaines, IL: American Society of Safety Engineers, 2011.

Occupational Safety and Health Administration (OSHA). *Draft. Preliminary Initial Regulatory Flexibility Analysis of the Draft Proposed OSHA Standard for Silica Exposure for General Industry and Maritime. Docket H006A. Ex. 4-3. October 3, 2003*. Washington, DC: Office of Regulatory Analysis, Occupational Safety and Health Administration, U.S. Department of Labor, 2003.

Occupational Safety and Health Administration (OSHA). *Proposed Rule Stage. 110. Occupational Exposure to Crystalline Silica. Fed Reg 75: 79603-79604*. 2010.

Pullen, E.L. *Memorandum from Elizabeth L. Pullen, CIH (AIHA) to Honorable Jacob J. Lew (White House Office of Management and Budget). RE: Executive Order 12866 Review of OSHA's Proposed Crystalline Silica Standard*. Fairfax, VA: American Industrial Hygiene Association, 2011.

Rice, C., R. Harris, H. Checkoway, and M. Symons. "Dose-response relationships for silicosis from a case-control study of North Carolina dusty trades workers." Silica, Silicosis, and Cancer. Volume 2. D. Goldsmith, D. Winn, and M. Shy, editors. New York: Praeger Publishers, 1986. 77-86.

Rice, F.L., and L.T. Stayner. "Assessment of silicosis risk for occupational exposure to crystalline silica." *Scandinavian Journal of Work and Environmental Health*. 21. 1995: 87-90.

Russell, A.E., R.H. Britten, L.R. Thompson, and J.J. Bloomfield. *The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry). Public Health Bulletin No. 187. July 1929*. Washington, DC: Government Printing Office, 1929.

Sander, O.A. "Lung findings in foundry workers: A four year survey." *American Journal of Public Health*. 28. 1938: 601-609.

Sayers, R.R. *Anthro-Silicosis Among Hard Coal Miners. Public Health Bulletin No. 221. December 1935*. Washington, DC: Government Printing Office, 1936.

Sayers, R.R., F.V. Meriwether, A.J. Lanza, and W.W. Adams. *Silicosis and Tuberculosis Among Miners of the Tri-State District of Oklahoma, Kansas and Missouri. I: For the Year Ended June 30, 1928. Technical Paper 545*. Washington, DC: U.S. Dept. of the Interior, Bureau of Mines, 1933.

Theriault, G.P., W.A. Burgess, L.J. DiBerardinis, and J.M. Peters. "Dust exposure in the Vermont granite sheds." *Archives of Environmental Health*. 28. 1974a: 12-17.

Theriault, G.P., J.M. Peters, and L.J. Fine. "Pulmonary function in granite shed workers of Vermont." *Archives of Environmental Health*. 28. 1974b: 18-22.

Theriault, G.P., J.M. Peters, and W.M. Johnson. "Pulmonary function and roentgenographic changes in granite dust exposure." *Archives of Environmental Health*. 28. 1974c: 23-27.

Vigliani, E.C., and G. Mottura. "Diatomaceous earth silicosis." *British Journal of Industrial Medicine*. 5. 1948: 148-160.

Winslow, C.-E.A., L. Greenburg, and E.H. Reeves. "The efficiency of certain devices used for the protection of sandblasters against the dust hazard." *Public Health Reports*. 35. 1920: 518-534.