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Silicosis

Kenneth Rosenman

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Introduction

The element silicon (Si) is the second most common element in the Earth's crust. Silicon in combination with oxygen is silica (SiO_2). Silicosis, an interstitial lung disease, is produced by the inhalation of silica-containing dust. Silicosis is the most common of the pneumoconioses (lung disease caused by inhalation of mineral dusts). Silicosis is an ancient disease, and its pathology was first described in the 1600s. The association of silica exposure with an increased risk of lung cancer, chronic obstructive pulmonary disease, connective tissue disease, renal disease, and tuberculosis was recognized in the 1900s. These conditions can occur after silica exposure in the absence of silicosis. Workers in many types of industry can be at risk of exposure to silica. Given it's abundance in the Earth's crust, workers in all parts of the mining industry, including quarry workers, have the potential for exposure to silica, which is commonly used in foundries and the manufacture of brick, cement, glass, pottery, sinks, and toilet bowls. Construction workers such as jackhammer operators, masons, and abrasive blasters are an additional high-risk group for silica exposure. New exposures to silica continue to be reported, such as in the manufacture or installation of stone countertops, abrasive blasting of denim clothes, dental laboratories, and hydraulic fracturing (fracking). Cristobalite is a polymorph of silica—it has the same chemical formula, SiO_2, but a different chemical structure. Cristobalite can be found in volcanic rock and when silica is heated, such as in foundries or diatomaceous production facilities. In animal models it is more fibrogenic than silica. The occurrence of silica exposure is worldwide. World production is estimated to be 121 million metric tons per year. The largest number of cases of silicosis are currently being recognized in rapidly growing economies such as China and Brazil.

General Overviews

General overviews on the topic of silicosis include Leung, et al. 2012; National Institute for Occupational Safety and Health 2002; Occupational Safety and Health Administration 2010; Steenland 2005; World Health Organization 2000; and Xia, et al. 2014.

Leung, C. C., I. T. S. Yu, and W. Chen. 2012. Silicosis. *Lancet* 379:2008–2018.

A summary on silica that covers the epidemiology, pathophysiology, adverse health effects, clinical management, and prevention of silicosis.

National Institute for Occupational Safety and Health. 2002. Health effects of occupational exposure to respirable crystalline silica. DHHS (NIOSH) Publication 2002-129. Cincinnati, OH: National Institute for Occupational Safety and Health.

A review of literature of effects of silica exposure with particular emphasis on the respiratory effects.

Occupational Safety and Health Administration. 2010. Occupational exposure to respirable crystalline silica—Review of health effects literature and preliminary quantitative risk assessment. Docket OSHA-2010-0034. Washington, DC: Occupational Safety and Health Administration.

The US Occupational Safety and Health Administration (OSHA), in conjunction with a proposal to update the US silica standard, conducted an extensive literature review and written a comprehensive report (483 pages) that includes quantitative exposure-risk assessments.

Steenland, K. 2005. One agent, many diseases: Exposure-response data and comparative risks of different outcomes following silica exposure. *American Journal of Industrial Medicine* 48:16–23.

Review of exposure-response data for silica and the development of three conditions: chronic renal disease; lung cancer; and silicosis. This paper calculates the quantitative risk for these three conditions based on the results of multiple studies.

World Health Organization. May 2000. Silicosis. WHO Fact Sheet 238. Geneva, Switzerland: World Health Organization.

The International Labor Organization and the World Health Organization in 1995 began the International Programme on the Global Elimination of Silicosis, the goal of which is the global reduction and eventual elimination of silicosis.

Xia, Y, J. Liu, T. Shi, H. Xiang, and Y. Bi. 2014. Prevalence of pneumoconiosis in Hubei, China from 2008 to 2013. *International Journal of Environmental Research and Public Health* 11:8612–8621.

This is a report from one province of 1,058 cases of silicosis over a six-year period.

Health Disparity

There are multiple examples showing that the incidence of silicosis is not evenly distributed in the general population, and that minority populations have a higher incidence of the disease. This health disparity is related to the overrepresentation of minorities in industries and/or job titles and tasks with increased silica exposure. Additionally, barriers created by social, cultural, and economic issues, including language, literacy, and marginal economic status, cause differences in the availability of occupational health and safety prevention activity. The most blatant example of a silica-related health disparity was the effect of South Africa's apartheid laws on silica exposure and compensation levels among South African gold miners (Cowie and Mabena 1991). The workforce in the South African gold mines in the 1990s was 600,000, with 90 percent of the workers being black. The black workers were the laborers, while white workers were generally in supervisory jobs. The incidence of silicosis was ten times greater in black miners than in white miners. Examples in the United States include the Hawk's Nest Tunnel project in the 1930s, where an estimated 581 (63 percent) of the 922 black workers, who had been recruited to work on building a water tunnel in West Virginia, died from acute silicosis; and the recruitment of black workers from southern states to work in the hot, dirty jobs in Midwestern foundries, who were then not allowed to transfer to other jobs as they accumulated seniority (Cherniak 1986; Foote, et al. 2003; Rice, et al. 2002). These hot, dirty jobs also had higher air levels of silica, which is reflected in the higher rates of silicosis in black foundry workers.

Cherniak, M. 1986. *The Hawk's Nest incident: America's worst industrial disaster*. New Haven, CT: Yale Univ. press.

This book is the most comprehensive review of the background and epidemiology of the tunnel drilling operation that occurred in West Virginia in the 1930s and was responsible for causing 764 deaths from silicosis, and an unknown number of nonfatal cases.

Cowie, R. L., and S. K. Mabena. 1991. Silicosis, chronic airflow limitation and chronic bronchitis in South African gold miners. *American Review of Respiratory Disease* 143:80–84.

This was the first respiratory health study of black South African gold miners. This study found that 857 (71.6 percent) of 1,197 black miners had silicosis, and that 62 percent had chronic bronchitis, including 45 percent of the miners who had never smoked cigarettes.

Foote, C. L., W. C. Whatley, and G. Wright. 2003. Arbitraging a discriminatory labor market; Black workers at the Ford Motor Company, 1918–1947. *Journal of Labor Economics* 21:493–532.

Describes the discriminatory practices used to recruit and locate black workers in certain areas of the foundry.

Rice, C., K. D. Rosenman, M. J. Reilly, and V. S. Hertzberg. 2002. Reconstruction of Silica Exposure at a Foundry for Evaluation of Exposure-Response. *Annals of Occupational Hygiene* 46, Suppl 1.: 10–13.

This study in a Midwest foundry found that blacks workers who had the same duration of work as white workers had, on average, higher cumulative and average silica exposures metrics.

Epidemiology of Health Effects of Silica

This section is divided into subsections on Chronic Obstructive Pulmonary Disease (COPD), Connective Tissue Disease, Lung Cancer, Renal Disease, Silicosis (Interstitial Fibrosis), and Tuberculosis.

Chronic Obstructive Pulmonary Disease (COPD)

Silicosis has classically been described as a restrictive lung disease, although descriptions of the pathology of individuals with silicosis have always included the identification of emphysematous changes accompanying the interstitial fibrosis. Since the mid-1980s, there have been multiple studies that observed an increased occurrence of obstructive changes on pulmonary function tests, emphysema on chest CT scans, and chronic bronchitis and death from COPD in individuals exposed to silica with and without the radiographic changes of silicosis. These studies have also shown that these adverse effects were more common in silica-exposed individuals who had also smoked cigarettes. The studies that found that silica caused obstructive disease were conducted among South African gold miners, individuals seeking working compensation in Quebec, foundry workers, sand workers, crushed stone workers, tunnel workers, quarry workers, and cement factory workers (Begin, et al. 1995; Brüske, et al. 2014; Cowie, et al. 1993; Ehrlich, et al. 2011; Hertzberg, et al. 2002; Hnizdo 1990; and Hnizdo and Vallyathan 2003). The obstructive changes were more prevalent in individuals with the radiographic changes of silicosis than in silica-exposed workers without such radiographic changes. Emphysematous changes were increased among individuals exposed to silica with or with radiographic changes in comparison to a non-silica-exposed control population.

Begin, R., R. Filion, and G. Ostiguy. 1995. Emphysema in silica- and asbestos-exposed workers seeking compensation: a CT scan study. *Chest* 108:647–655.

This study found an association between emphysematous changes observed on a CT scan and silica exposure in workers applying for compensation.

Brüske, I., E. Thuering, J. Heinrich, K. M. Huster, and D. Nowak. 2014. Respirable quartz dust exposure and airway obstruction: A systematic review and meta-analysis. *Occupational and Environmental Medicine* 71:583–589.

This paper reported on a meta-analysis that showed a statistically significant increase in measures of airway obstruction and with increasing occupational exposure to silica. Selected studies had quantitative data to calculate an exposure response effect.

Cowie, R. L., M. Hay, and R. G. Thomas. 1993. Association of silicosis, lung dysfunction, and emphysema in gold miners. *Thorax* 48:746–749.

This study of black South African gold miners found emphysema on CT scans in cigarette and non-cigarette smokers, and in individuals with and without radiographic changes of silicosis.

Ehrlich, R. I., J. E. Myers, J. M. te Water Naude, M. L. Thompson, and G. J. Churchyard. 2011. Lung function loss in relation to silica dust exposure in South African gold miners. *Occupational and Environmental Medicine* 68.2: 96–101.

This study quantitated the loss of pulmonary function by exposure levels of silica among black South African gold miners and found that silica dust levels had a greater impact on pulmonary function results than the development of silicosis.

Hertzberg, V. S., K. D. Rosenman, M. J. Reilly, and C. H. Rice. 2002. The effect of occupational silica exposure on pulmonary function. *Chest* 122:721–728.

This study quantitated an exposure response effect between silica exposure and obstructive changes on pulmonary function testing among silica-exposed foundry workers without radiographic evidence of silicosis.

Hnizdo, E. 1990. Combined effect of silica dust and tobacco smoking on mortality from chronic obstructive lung disease in gold miners. *British Journal of Industrial Medicine* 47:656–664.

This study calculated that among white South African gold miners, 5 percent of the attributable risk of mortality from COPD was from silica, 34 percent from cigarette smoking, and 59 percent from the combined effect of silica and cigarette smoking.

Hnizdo, E., and V. Vallyathan. 2003. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: A review of epidemiological and pathological evidence. *Occupational and Environmental Medicine* 60:237–243.

A review of the studies summarizing the occurrence of obstructive changes in pulmonary function, emphysematous changes on radiographs, chronic bronchitis, and mortality from COPD among workers with silica exposure.

Connective Tissue Disease

Connective tissue diseases (CTDs) feature inflammatory changes caused by the immune system acting against the individual's own body tissues (autoimmunity). Silica exposure with or without silicosis has been associated in case series, cohort, registry linkage, and case-control studies with rheumatoid arthritis, scleroderma, systemic lupus erythematosus, Sjogrens syndrome, and small vessel vasculitides, particularly antineutrophil cytoplasmic antibody (ANCA)–associated microscopic polyangiitis (Makol, et al. 2011; Gómez-Puerta, et al. 2013). The prevalence of polyclonal hypergammaglobulins, positive antinuclear antibodies, and rheumatoid factor have been found to be increased in silica-exposed individuals even in the absence of clinical CTD. Possible biological mechanisms proposed for the epidemiological association observed between silica exposure and CTD is that silica (1) acts as immunoadjuvant by activating macrophages, which then produce enzymes involved in the degradation and remodeling of extracellular matrix; (2) causes defective apoptosis leading to the prolonged survival of pathogenic lymphocytes; and (3) activation of T cells and lymphocytes. Surrogates of silica exposure such as duration of work in a silica-using industry or radiographic profusion of silicosis have not been found to be risk factors for development of CTD. No studies have examined the prevalence of CTD with exposures metrics such as cumulative or average silica exposure. See also Parks, et al. 1999 and Rocha-Parise, et al. 2014.

Gómez-Puerta, J. A., L. Gedmintas, and K. H. Costenbader. 2013. The association between silica exposure and development of ANCA-associated vasculitis: systematic review and meta-analysis. *Autoimmune Reviews* 12:1129–1135.

A meta-analysis of six case-control studies that found a statistically significant risk of the ANCA-associated vasculitides with having ever been exposed to silica.

Makol, A., M. J. Reilly, and K. D. Rosenman. 2011. Prevalence of connective tissue disease in silicosis. *American Journal of Industrial Medicine* 54:255–262.

This paper is an analysis of the prevalence of connective tissue disease in a registry of individuals with the diagnosis of silicosis. Rheumatoid arthritis was the most frequently diagnosed connective disease, while antineutrophil cytoplasm antibody (ANCA) vasculitis was the condition with the highest risk.

Parks, C. G., K. Conrad, and G. S. Cooper. 1999. Occupational exposure to crystalline silica and autoimmune disease. *Environmental Health Perspectives* 107. Suppl. 5: 793–802.

A review of the epidemiological studies performed examining an association between silica exposure and CTD, with a discussion of possible biological mechanisms.

Rocha-Parise, M., L. M. Santos, J. G. Damoiseaux, et al. 2014. Lymphocyte activation in silica-exposed workers. *International Journal of Hygiene and Environmental Health* 217:586–591.

A study of immune activation in silica-exposed workers that examines the effect of silica exposure on lymphocyte activation.

Lung Cancer

Crystalline silica in the form of quartz or cristobalite has been classified by the International Agency for Research on Cancer (IARC) as a Group 1 carcinogen, carcinogenic to humans based on sufficient evidence from both human and animal studies. IARC work groups reviewed silica in 1986, 1987, 1996, and 2009 (International Agency for Research on Cancer 2012). The most recent review reaffirmed the 1996 classification of silica as a Group 1 carcinogen. The IARC review group considered the release of cytokines by silica particles that initiated inflammation as the mostly likely mechanism of carcinogenesis, but did not rule out the generation of free radicals by silica particles that either induced epithelial proliferation or had a direct genotoxic effect. Evaluation of the carcinogenic potential of silica has been complicated by the presence of other carcinogens in workplaces where silica was used (e.g., foundries where exposure to the carcinogens asbestos and benzopyrene also occurred), assessing whether silica itself or the development of silicosis was required for there to be an increased risk of cancer, controlling for the confounding effect of cigarette smoking, and the publication of studies with insufficient statistical power or where the workers had insufficient silica exposure or latency from initiation of silica exposure. See Lacasse, et al. 2005; Rice, et al. 2001; Steenland and Greenland 2004; Steenland, et al. 2001; and Yu and Tse 2007.

International Agency for Research on Cancer. 2012. *A review of human carcinogens: Arsenic, metals, fibres, and dusts*. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 100C. Lyon, France: International Agency for Research on Cancer.

The silica chapter in this monograph (pp. 355–456) is the most recent review by an IARC working group of the literature on silica exposure and cancer. It includes a succinct summary of the studies reviewed and the process for classifying silica as a human carcinogen.

Lacasse, Y., S. Martin, S. Simard, and M. Desmeules. 2005. Meta-analysis of silicosis and lung cancer. *Scandinavian Journal of Work, Environment & Health* 31:450–458.

A meta-analysis that concluded that the effects of cigarette smoking did not explain the increased risk of lung cancer from exposure to silica.

Rice, F. L., R. Park, L. Stayner, et al. 2001. Crystalline silica exposure and lung cancer mortality in diatomaceous earth industry workers: A quantitative risk assessment. *Occupational and Environmental Medicine* 58:38–45.

This report analyzed multiple different exposure models and found that trends of predictive risk were the same across all the models for a cohort of diatomaceous exposed workers.

Steenland, K., and S. Greenland. 2004. Monte Carlo sensitivity analysis and Bayesian analysis of smoking as an unmeasured confounder in a study of silica and lung cancer. *American Journal of Epidemiology* 160:384–392.

This is a sensitivity analysis of the effect of cigarette smoking that continued to show an increased risk of lung cancer among sand and gravel workers over a wide range of estimated prevalence of cigarette smoking in the cohort. The original study showing an increased risk of lung cancer mortality had not had data on who in the cohort smoked.

Steenland, K., A. Mannetje, P. Boffetta, et al. 2001. Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. *Cancer Causes & Control* 12:773–784.

This quantitative assessment of lung cancer risk from ten diverse industries with silica exposure found that cumulative crystalline silica exposure (cumulative, unlagged and lagged; log cumulative, unlagged and lagged) showed highly significant trends for lung cancer risk.

Yu, I. T., and L. A. Tse. 2007. Exploring the joint effects of silicosis and smoking on lung cancer risks. *Int Journal of Cancer* 120:133–139.

A review of ten previously published studies that concluded that the risk of lung cancer for silica exposure was underestimated in those who had never smoked cigarettes. It also concluded the risk for lung cancer for silica exposure was overestimated in those who had ever smoked cigarettes but was still significantly increased.

Renal Disease

The prevalence and mortality of chronic renal disease is increased in cohorts of silica-exposed workers and among individuals with silicosis. Adverse outcomes associated with silica exposure have included mortality from renal disease among US gold miners, industrial sand miners, and quarry workers; increased prevalence of end stage renal disease; and measures of glomerular and tubal dysfunction. There has been sufficient exposure data to quantitate the excess risk of mortality from renal disease, with the calculated excess risk being as high as 5.1 percent in one cohort. Renal disease risk was most prevalent among workers with cumulative exposures of 0.5 mg/m^3 or more. The only specific renal clinical or pathological changes associated with silica exposure have been ANCA-associated vasculitis. This relatively rare cause of renal disease is not sufficient to explain the epidemiological findings of increased risk of end stage renal disease and increased renal mortality in silica-exposed individuals. Possible mechanisms of the renal disease associated with silica exposure include a direct toxic effect on the kidney, kidney deposition of immune complexes, or an autoimmune mechanism. Further work to identify specific pathological changes and changes in urine parameters that occur with silica exposure are needed. See Ibrahim, et al. 2011; Ng, et al. 1992; Millerick-May, et al. 2015; Steenland, et al. 2002; and Vuppurturi, et al. 2012.

Ibrahim, K. S., S. B. Ahmed, and N. M. Amer. 2011. Study of kidney dysfunction in non-silicotic Egyptian workers. *International Journal of Hygiene and Environmental Health* 214:53–58.

A cross-sectional study of ceramic workers with silica exposure, with an increase in markers of tubular and glomerular dysfunction in comparison non-silica-exposed control group.

Millerick-May, M., M. J. Reilly, S. Schrauben, K. D. Rosenman. 2015. Silicosis and chronic renal disease. *American Journal of Industrial Medicine* 58:730–736.

Twenty four percent of 1,072 individuals with silicosis in a population-based case registry had an increased prevalence of kidney dysfunction controlling for diabetes and hypertension.

Ng, T. P., Y. L. Ng, H. S. Lee, K. S. Chia, and H. Y. Ong. 1992. A study of silica nephrotoxicity in exposed silicotic and non-silicotic workers. *British Journal of Industrial Medicine* 49:35–37.

Significantly higher urinary excretions of albumin and macroglobulin were found in quarry workers exposed to silica.

Steenland, K., M. Attfield, and A. Mannejte. 2002. Pooled analyses of renal disease mortality and crystalline silica exposure in three cohorts. *Annals of Occupational Hygiene* 46:4–9.

Based on the results of three cohorts, the estimated excess risk of death from renal disease was calculated to be 1.8 percent (0.8 percent–9.7 percent). There was a statistically significant exposure-response trend for acute and chronic renal disease mortality.

Vuppurturi, S., C. G. Parks, L. A. Nylander-French, A. Owen-Smith, S. L. Hogan, and D. P. Sandler. 2012. Occupational silica exposure and chronic kidney disease. *Renal Failure* 34:40–46.

A case-control study of individuals with chronic renal disease that found a dose-response to silica exposure estimates based on industrial hygiene evaluation of lifetime work histories.

Silicosis (Interstitial Fibrosis)

The lung pathology of silicosis is well described with the presence of a pathognomonic silicotic nodule on microscopic examination. Typically, the individual has been exposed to silica for ten to twenty or more years before development of silicosis (chronic silicosis), but accelerated silicosis, which has the same pathology as chronic silicosis, can occur within a few years of heavier exposure. Even heavier exposure can cause acute silicosis, which has a different pathological finding than accelerated or chronic silicosis. The pathology of acute silicosis is identical to acute alveolar proteinosis. The absolute risk of silicosis has ranged from 47 percent to 75 percent in cohort studies that included adequate follow-up after employment. Studies of active workers did not reflect the true burden, since the incidence of silicosis increases with duration and latency since first exposure. The absolute risk of death from silicosis is estimated at 1.9 percent (0.8 percent–2.9 percent), based on a pooled analysis of six cohort studies. Mortality from silicosis has been decreasing in the United States since 1968. This decrease partially reflects improvement in workplace controls but is heavily affected by the decrease in the number of workers exposed to silica from increased automation and the reduced use of silica in abrasive blasting. At the same time that mortality where silicosis was the underlying cause of death has decreased, other respiratory causes of death have increased, COPD and the annual number of hospitalizations for silicosis has stayed constant, and the ratio of living to dead individuals with silicosis in surveillance systems has increased. There have been conflicting studies on whether individuals having the low-profusion radiographic changes of simple silicosis have clinically important changes on pulmonary function tests. Differences in whether a study reported there was a relationship between spirometry and radiographic changes can be attributed to whether the studies used non-exposed controls or silica-exposed controls. No relationship was found in the latter studies, which is consistent with silica-causing emphysema and changes on pulmonary function tests independently of the silicotic nodules seen on radiographs. Additionally, different latency periods after the initiation of silica exposure for the development of spirometry and radiographic changes would cause the results from studies of current and retired workers to differ. See Chen, et al. 2001; Kreiss and Zhen 1996; Mannetje, et al. 2002; Rosenman, et al. 2003; Rosenman, et al. 2010; and Steenland 2005.

Chen, W., Z. Zhuang, M. Attfield, et al. 2001. Exposure to silica and silicosis among tin miners in China: Exposure-response analyses and risk assessment. *Occupational and Environmental Medicine* 58:31–37.

This study found an exposure-response relation for silicosis in a cohort of 3,010 Chinese tin miners and predicted a 55 percent cumulative risk of silicosis for a forty-five-year lifetime exposure at the current OSHA standard.

Kreiss, K, and B. Zhen. 1996. Risk of silicosis in a Colorado mining community. *American Journal of Industrial Medicine* 30:529–539.

Analysis of the results of a radiographic survey of a community, with a large population of long-term and former molybdenum, lead, zinc, and gold miners, predicted that 75 percent of workers exposed to the current OSHA silica standard would develop silicosis.

Mannetje, A., K. Steenland, M. Attfield, et al. 2002. Exposure-response analysis and risk assessment for silica and silicosis mortality in a pooled analysis of six cohorts. *Occupational and Environmental Medicine* 59:723–728.

A pooled analysis of 18,000 workers from six cohort studies of silica-exposed workers found an increased risk of death from silicosis at the current OSHA standard.

Rosenman, K. D., M. J. Reilly, and J. Gardiner. 2010. Results of spirometry among individuals in a silicosis registry. *Journal of Occupational and Environmental Medicine* 52:1173–1178.

This paper examines spirometric results by radiographic profusion among 526 individuals in a silicosis case registry. The literature of previous studies that examined pulmonary function testing by radiographic profusion were reviewed.

Rosenman, K. D., M. J. Reilly, and P. K. Henneberger. 2003. Estimating the total number of newly-recognized silicosis cases in the United States. *American Journal of Industrial Medicine* 44:141–147.

This paper describes the use of data from a state-based surveillance system and national mortality data to estimate the incidence of silicosis in the United States. The analysis used the ratio of living to deceased individuals with silicosis and capture-recapture analyses to derive the estimate.

Steenland, K. 2005. One agent, many diseases: Exposure-response data and comparative risks of different outcomes following silica exposure. *American Journal of Industrial Medicine* 48:16–23.

Review of exposure-response data for silica and the development of silicosis. This paper calculates the quantitative risk for developing silicosis based on the results of multiple studies.

Tuberculosis

The association between *Mycobacterium tuberculosis* (TB) and silicosis has been recognized since the 1500s. More recent studies have found an increased risk of TB in those with silica exposure in the absence of the radiographic changes of silicosis. The increased risk has been found in both active and retired workers. There is also an increased risk for the atypical mycobacteria such as *Mycobacterium avium-intracellulare*. The increased risk of TB from silica is thought to be secondary to silica's toxic effect on the macrophage, which allows the tubercle bacillus to grow more rapidly in macrophages and increases the likelihood of progression from latent to active TB after exposure. The risk is multiplicative in populations with increased HIV prevalence and silica exposure, such as South African gold miners. Treatment with directly observed therapy has proven effective for active TB in these high-risk individuals. The US Centers for Disease Control and Prevention (CDC) recommends treatment for latent TB for individuals with silicosis who have a positive Interferon Gamma Release Assay (IGRA) or a tuberculin skin test of ≥5 mm with silicosis. Despite the medical literature showing an increased risk of TB in individuals with silica exposure but without silicosis, the CDC recommendations do not address interpretation of tuberculin skin tests in silica exposed individuals. See Rees and Murray 2007, teWaterNaude, et al. 2006, and Cowie 1994.

Cowie, R. L. 1994. The epidemiology of tuberculosis in gold miners with silicosis. *American Journal of Respiratory and Critical Care Medicine* 150:1460–1462.

This report describes the increased risk of tuberculosis in black South African gold miners with and without silicosis even before the spread of HIV.

Rees, D., and J. Murray. 2007. Silica, silicosis and tuberculosis. *International Journal of Tuberculosis and Lung Disease* 11:474–484.

A review of the association between tuberculosis, silicosis, and silica exposure with discussion of management and prevention.

teWaterNaude, J. M., R. I. Ehrlich, G. J. Churchyard, et al. 2006. Tuberculosis and silica exposure in South African gold miners. *Occupational and Environmental Medicine* 63:187–192.

This study found that the risk of tuberculosis was related to quantitative estimates of silica exposure regardless of the radiographic presence of silicosis.

Clinical Guidelines for Diagnosis and Management of Silicosis

Simple silicosis is the presence of small rounded opacities on the chest radiograph or on a biopsy specimen with or without abnormalities on pulmonary function tests. On lung section, there are multiple, circular, hard nodules of varying size scattered throughout the lung parenchyma, but usually maximal in the upper zones. In addition, there are almost always areas of focal emphysema and bronchiectasis and the pulmonary arteries show hypertensive thickening, often with atheroma. On microscopic exam, the nodules consist of concentric, laminated layers of hyaline collagen around central collections of particles of silica. Examination of the nodules by polarized microscopy shows birefringent silica particles. With increased dust exposure, simple silicosis may progress to progressive massive fibrosis (PMF) with conglomeration of the nodular lesions of simple silicosis. The PMF lesion is usually found in the upper lobes and is a mass of dense, hyalinized connective tissue with minimal silica content, a small amount of anthracotic pigment, minimal cellular infiltrate, and negligible vascularization with cavitated centers of ischemic necrosis. Chest radiographs of simple silicosis will show hundreds of nodules 1.5 to 10 mm, which are first seen in the upper zones. With time the nodules may be seen in all zones or consolidate to large opacities >10 mm to fill the whole upper zone. The radiograph is indistinguishable from the radiograph of coal workers' pneumoconiosis. Egg shell calcification is fibrosis in the hila lymph nodes surrounded by a thin layer of calcification. Smooth rounded opacities may be seen in individuals with silicosis and rheumatoid arthritis (Caplan's syndrome) in contrast to PMF, where the nodules are typically not smooth and cause contraction and distortion of the surrounding lung. Both egg shell calcification and Caplan's syndrome are uncommon radiographic findings. The diagnosis of silicosis is made by obtaining a history of silica exposure and a radiograph consistent with silicosis. Biopsy is rarely needed. Breathing tests may be normal, show restriction, obstruction, or a mixed pattern. Given the increased risk after silica exposure, evaluation for tuberculosis, connective tissue disease, and renal disease should be performed. Progression of radiograph changes should heighten concern for these conditions as well as lung cancer and may suggest the need for a biopsy. Ongoing care for patients with silicosis or significant silica exposure should include periodic assessment for connective tissue disease, lung cancer, renal disease, and tuberculosis.

American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health. 1997. Adverse effects of crystalline silica exposure. *American Journal of Respiratory and Critical Care Medicine* 155:761–768.

Official statement of the American Thoracic Society on the adverse effects of silica and their management.

Hong Kong Chest Service/Tuberculosis Research Centre, Madras/British Medical Research Council. 1992. A double-blind placebo-controlled clinical trial of three antituberculosis chemoprophylaxis regimens in patients with silicosis in Hong Kong. *American Review Respiratory Disease* 145:36–41.

This paper describes the reduction of risk of active TB after treatment of latent TB with six months of isoniazid in one hundred patients (relative risk 0.58) and three months of isoniazid and rifampicin (relative risk 0.64) in 87 patients with silicosis in Hong Kong.

Raymond, L. W., and S. Wintermeyer. 2006. Medical surveillance of workers exposed to crystalline silica. *Journal of Occupational and Environmental Medicine* 48:95–101.

The American College of Occupational and Environmental Medicine developed guidelines for the medical surveillance of workers exposed to crystalline silica.

Rosenman, K. D. 2014. Pneumoconiosis. BMJPoint-of-Care.

Web-based review of diagnosis and treatment of major pneumoconioses, including silicosis.

Hazard Control and Disease Prevention

Primary prevention by elimination and reduction of silica exposure includes substitution, a change to work practices that use wet rather than dry methods, engineering controls to enclose sources of silica, and ventilation. Many European countries have banned the use of silica for abrasive blasting and require that alternative abrasives be used. It has been recommended by the American Conference of Governmental Industrial Hygienists that the allowable silica level be 0.025 mg/m^3. The current US allowable standard is 0.1 mg/m^3. An alternative to obtaining air sampling for evaluating risk has been proposed; control banding, a qualitative risk assessment and management strategy uses task-based hazard data and potential exposure information to determine appropriate controls. Secondary prevention with medical surveillance is recommended for exposed workers. Individuals diagnosed with silicosis should be removed from further exposure to silicosis. Reduction of morbidity from silicosis is limited to standard medical therapy of individuals with chronic lung disease. There is no cure for silicosis.

Beaucham, C. C., T. J. Lentz, and F. L. Rice. 2012. Expanding control banding for workplace silica exposures throughout the Americas. *International Journal of Occupational and Environmental Health* 18:344–347.

A description of a NIOSH program to assess, implement, and provide tools to evaluate the use of control banding methodology in Chile, Peru, Colombia, and Brazil.

Center for Construction Research and Training.Work Safely with Silica.

This website contains information on the hazards and approaches to prevent the development of silica related adverse health effects in the construction industry.

Centers for Disease Control and Prevention. *Evaluation of substitute materials for silica sand in abrasive blasting*. Morgantown, WV: National Institute for Occupational Safety and Health, 1998–1999.

This report provides useful information for selection of an alternative to silica for performing abrasive blasting.

Health and Safety Executive. Control of Substances Hazardous to Health (COSHH) Essentials guidance publications. London: Health and Safety Executive.

This is the website for the English regulatory agency; it contains publications and guidelines for controlling silica exposure in a wide range of industries.

National Institute for Occupational Safety and Health. Silica. Workplace Safety and Health Topics. Atlanta, GA: National Institute for Occupational Safety and Health.

This website contains information on methods for air sampling and laboratory analysis, the B-reader program, recommendations for preventing silicosis, respirators, and surveillance data.

Occupational Safety and Health Administration. Silica, Crystalline. Safety and Health Topics. Washington, DC: Occupational Safety and Health Administration.

This website contains information on OSHA regulations, health effects, hazard recognition, and control measures for silica.

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