Silica: A Lung Carcinogen

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Silica has been known to cause silicosis for centuries, and evidence that silica causes lung cancer has accumulated over the last several decades. This article highlights 3 important developments in understanding the health effects of silica and preventing illness and death from silica exposure at work. First, recent epidemiologic studies have provided new information about silica and lung cancer. This includes detailed exposure-response data, thereby enabling the quantitative risk assessment needed for regulation. New studies have also shown that excess lung mortality occurs in silica-exposed workers who do not have silicosis and who do not smoke. Second, the US Occupational Safety and Health Administration has recently proposed a new rule lowering the permissible occupational limit for silica. There are approximately 2 million US workers currently exposed to silica. Risk assessments estimate that lowering occupational exposure limits from the current to the proposed standard will reduce silicosis and lung cancer mortality to approximately one-half of the rates predicted under the current standard. Third, low-dose computed tomography scanning has now been proven to be an effective screening method for lung cancer. For clinicians, asking about occupational history to determine if silica exposure has occurred is recommended. If such exposure has occurred, extra attention might be given to the early detection of silicosis and lung cancer, as well as extra emphasis on quitting smoking.
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Introduction

Silica has recently been in the news because this past August the US Occupational Safety and Health Administration (OSHA) proposed a new rule lowering the occupational limit from 0.1 mg/m^3 (0.25 mg/m^3 for the construction industry) to 0.05 mg/m^3.[1] The current standard was set in 1971. Although regulation of occupational exposures at the current standard has substantially reduced silicosis death rates in the United States, new cases of silicosis continue to be diagnosed, some among younger individuals who entered the workforce well after the existing standard was in place. Risk assessments estimate that lowering occupational exposure limits from the current to the proposed standard will reduce silicosis and lung cancer mortality to about one-half of the rates predicted under the current standard.

OSHA estimates that 2.2 million US workers are exposed to silica, 1.85 million of these in the construction industry. This translates into approximately 1 to 2 workers per every 100 workers. In addition to workers currently exposed to silica, many more have been exposed to silica in the past. Patients may develop symptoms of silicosis years after their occupational exposure to silica has ended. Patients who smoke and have a history of silica exposure, and particularly those with silicosis, can reduce their risk of lung cancer by smoking cessation. Current and former long-term smokers with a history of silica exposure may meet lifetime risk guidelines for computed tomography (CT) screening for lung cancer, when their increased risk due to silica exposure is taken into account. For all these reasons, it is important for clinicians to be aware of occupations with potential silica exposure, the symptoms and diagnostic criteria for silicosis, and the risk of lung cancer associated with silica exposure and silicosis.

What Is Crystalline Silica?

Silica (silicon dioxide) exists in crystalline and amorphous forms. The latter is less toxic and a less common form of exposure; hereafter we will refer to "silica" and mean "crystalline silica." Silica, also known as quartz or cristobalite, is made up of fine particles much smaller than a grain of sand. Respirable silica refers to particles with a diameter less than 10 μm; these smaller particles are less likely to be trapped in the nose and throat and are more likely to reach the lungs.

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How Are Individuals Exposed to Silica?

According to OSHA, "exposures occur when workers cut, grind, crush, or drill silica-containing materials such as concrete, masonry, tile, and rock." Table 1 provides a list of common occupations and industries with silica exposure. While there is also some low-level silica exposure on beaches and in ambient air in general, there is no evidence such low-level exposure causes health effects.

About 320,000 workers are exposed in general industry operations such as brick, concrete, and pottery manufacturing, as well as operations using sand products, such as foundry work. Workers are also exposed during sandblasting in general industry (available at osha.gov/silica/factsheets/OSHA_FS-3683_Silica_Overview.html). Worldwide, there are estimated to be tens of millions of workers exposed to silica,[2] many of whom are exposed to much higher concentrations of silica than current US workers. Although most occupations and industries with silica exposure have existed for decades, new exposure circumstances continue to be documented. For example, silica exposure from hydraulic fracturing (fracking) of oil and gas wells is a new phenomenon. In a recent survey, investigators from the National Institute for Occupational Safety and Health (NIOSH; part of the Centers for Disease Control and Prevention) obtained 116 silica samples at 11 different fracking sites and found that silica concentrations exceeded the current OSHA standard in 47% of the samples. More than three-quarters of the samples, or 79%, had concentrations that were greater than the new standard proposed by OSHA (available at osha.gov/dts/hazardalerts/hydraulic_frac_hazard_alert.html). A study in Turkey found high exposure levels and a prevalence of silicosis among workers who sandblasted denim[3] (sandblasting is a well-known source of high exposure, but sandblasting denim is new!). A study in Chile found that a switch from manual carving to the use of power tools for carving pottery resulted in extremely high levels of silica dust.[4] A report from the Centers for Disease Control and Prevention in 2004 noted cases of silicosis occurring among dental assistants possibly exposed while grinding casts and porcelains, which can contain a high percentage of silica (available at cdc.gov/mmwr/preview/mmwrhtml/mm5309a3.htm). As is discussed later in this article, the best approach to primary prevention when such high-exposure situations are identified is to reduce silica exposure hazards through process changes and engineering controls, such as the substitution of less hazardous materials, the use of water-based methods, and local exhaust ventilation. Respirators may be useful for workers in short-term high-exposure situations, but are generally not recommended as the primary means of exposure control due to worker discomfort, difficulties in communicating with others, lack of compliance and enforcement, and the fitting and maintenance requirements.

TABLE 1. Occupations and Industries With Silica Exposure

OCCUPATION	INDUSTRY
Sandblasting	Shipbuilding, ironworking, construction/painting (clearing painted surfaces)
Miner	Mining underground
Miller	Silica flour mills
Ceramic worker	Pottery and ceramics
Glassmaker	Glass producion
Granite quarry worker	Mining in quarries
Sand grinding	Industrial sand
Stone grinding	Granite industry (monuments)
Casting, shakeout, blasting	Foundry

What Health Problems Are Caused by Silica?

There is strong evidence that silica causes silicosis and lung cancer; suggestive evidence that it causes renal disease; and limited evidence that it causes autoimmune diseases, particularly scleroderma and rheumatoid arthritis.[5-7]

Silicosis

It has long been known that silica causes silicosis, a progressive, disabling, and incurable disease. NIOSH estimates that there has been a decrease from about 1200 US deaths due to silicosis per year in 1968 to fewer than 100 deaths per year in the early 2000s[7] based on death certificate data, which are likely to undercount silicosis deaths. There is no national surveillance system for silicosis, although a few states have such systems. Using state-based data, Rosenman et al estimated between 3600 and 7300 new cases annually in the 1990s.[8] Hospital discharge data indicated approximately 1000 discharges for silicosis annually and another 1000 discharges for pneumoconiosis (of which the most common is silicosis) in the early 2000s.[1] Estimates of the prevalence of silicosis from state-based surveillance systems are likely to be low because of underreporting, and because estimates from hospital discharge summaries reflect only those patients sick enough to be admitted to the hospital.

Silicosis is a worldwide problem, and is especially prevalent in low- and middle-income countries, in which the burden is often underreported because of poor surveillance. China appears to have the highest burden of silicosis, with more than 500,000 cases recorded between 1991 and 1995, and 6000 new cases and more than 24,000 deaths reported annually. The problem is particularly serious among workers in small-scale mines in developing countries.[2]

The clinical diagnosis of silicosis requires determination that silica exposure adequate to cause this disease has

occurred, the presence of chest radiographic abnormalities consistent with silicosis, and the absence of other diseases that may mimic silicosis.[5] The presentation and severity of silicosis are influenced by the level and duration of exposure.[6] Classical silicosis, the most frequent presentation in the United States, generally results from low to moderate exposure to silica dust for 20 or more years.[6] The characteristic radiographic pattern of simple silicosis is the presence of rounded opacities that range in size from 1 to 10 mm and are typically distributed in the upper zones of both lungs in a near symmetric pattern. Hilar lymph nodes are often enlarged with a distinctive peripheral calcification, described as eggshell calcification.[5,6] Mild classic silicosis does not cause respiratory impairment and the primary health concerns for patients are increased susceptibility to mycobacterial infections and the development of progressive massive fibrosis (PMF). PMF involves the coalescence of silicotic nodules to form a mass of dense hyalinized connective tissue with minimal silica content, minimal cellular infiltrate, and negligible vascularization, the center of which often cavitates due to mycobacterial infection or ischemic necrosis.[6] PMF is a highly debilitating disease that results in impairment in pulmonary function, dyspnea, cor pulmonale, and an increased risk of spontaneous pneumothorax. This form of complicated silicosis has the potential to be fatal. Another form of silicosis, accelerated silicosis, results from higher exposure to silica, usually over a period of 5 to 10 years. Progression of this form of silicosis is virtually certain even if the worker is removed from the workplace.[6] Acute silicosis, the most rapidly fatal form of silicosis, is due to extraordinarily high exposure to small silica particles. In one of the most tragic industrial disasters in US history, approximately 3000 miners employed to build the Hawks Nest Tunnel through Gauley Mountain in West Virginia starting in 1927 were exposed to massive amounts of silica dust, resulting in at least 476 deaths.[9] This form of silicosis has not been reported to occur in the United States for many years; however, patients continue to be diagnosed with simple, progressive, and accelerated silicosis. Tuberculosis is a known complication of silicosis. An increased incidence of tuberculosis has been observed among workers in the mining, quarrying, and tunneling industries and in workers in steel and iron foundries.[9] The primary prevention of silicosis through exposure controls is important because there is no proven effective therapy for this disease.[6]

Review of Epidemiologic Evidence Regarding Silica and Lung Cancer

There have been over 100 epidemiologic studies of silica and lung cancer. The large number of studies is a product of the fact that occupational silica exposure is widespread and of public health importance, and because the risk of lung cancer found in epidemiologic studies is low compared with other classic lung carcinogens such as arsenic and asbestos, requiring many studies to detect it. When the relative risk (the disease risk of the exposed vs the risk of the nonexposed or, alternatively, the risk of individuals with high exposure vs the risk of those with low exposure) associated with a potential carcinogen is low, it is more difficult to rule out the possibility that the risk was caused by a confounding exposure (such as tobacco smoking or an occupational exposure other than silica), or to demonstrate a dose-response relationship, two important factors considered in evaluating whether there is a causal association. The carcinogenicity of silica was reviewed by the International Agency for Research on Cancer (IARC) Monographs Program in 1997 and silica was classified as a known human carcinogen (group 1).[10] The evaluation of human carcinogenicity in 1997 was largely based on the findings of epidemiologic studies which were judged to have the least opportunity for confounding. Although an excess risk of lung cancer was not found in all studies, the majority of studies found an increased risk and some demonstrated increasing risk gradients in association with increasing exposure. For these reasons, the IARC concluded that overall the epidemiological findings supported an increased lung cancer risk from inhaled crystalline silica from occupational exposure and that the observed associations were not explained by confounding or other biases. Some experts disagreed with this conclusion.[11] After the IARC's 1997 evaluation, residual questions remained about whether silicosis was a prerequisite for the development of silica-related lung cancer, the role of smoking, and the exact nature of the exposure-response relationship between silica exposure and lung cancer.

The IARC rereviewed a number of carcinogens, silica among them, in 2012 (one of the present authors, E. W., participated in this meeting).[12] There were considerably more epidemiologic data. One of these was a pooled analysis by Steenland et al of 10 large silica-exposed cohorts, all of which had good-quality exposure data during the entire follow-up period.[13] Together, these cohorts included over 1000 lung cancer deaths. The pooled analysis found a significant positive exposure-response relationship between cumulative silica exposure and lung cancer mortality. The increase in the relative risks (disease risk for workers with a given level of exposure compared with that for those with the lowest exposure) with increasing cumulative exposure to silica, divided into quintiles, were 1.0, 1.0, 1.3, 1.5, and 1.6, with silica exposure categories defined as less than 0.4, 0.4 to 2.0, 2.0 to 5.4, 5.4 to 12.8, and 12.8 or more mg/m^3-years. For comparison, an exposure over a 45-year working lifetime at the current OSHA standard of 0.1 mg/m^3-years results in 4.5 mg/m^3-years. Other studies available to OSHA were meta-analyses combining data from a number of studies of silica and lung cancer. These yielded overall

relative risk estimates within the same range.[12] A meta-analysis of studies with exposure-response data found results that were similar to the earlier pooled analysis, with relative risks approaching 2 for the highest category of cumulative exposure to silica, and with a similar slope of the exposure-response curve.[14] This meta-analysis also found that studies with and without controls for smoking yielded similar relative risks, suggesting that confounding from smoking (eg, the silica-exposed individuals smoked more that those not exposed to silica) was not likely to explain the elevations in the relative risk.[12]

In addition to meta-analyses of silica-exposed workers, the IARC reviewed 5 meta-analyses summarizing the results of studies of lung cancer among workers with silicosis. Here the summary relative risks were higher, ranging from 1.74 to 2.37.[12] Silicosis is a strong indicator of high exposure. The higher relative risks among those with silicosis stimulated continued debate about whether lung cancer should be interpreted solely as a consequence of the fibrotic process rather than a direct effect of silica exposure, or if the higher risk among patients with silicosis was simply a marker of higher exposure. This controversy could only be resolved by a study or studies with data regarding who did and who did not have silicosis within a cohort, and that were large enough to have sufficient power to detect excess risks in both groups.

A recently published cohort study from China has been able to address this question, and has added significantly to the literature of silica studies with exposure-response data. Liu et al studied 34,000 tungsten miners, iron miners, and pottery workers.[15] This is a subcohort of the Chinese cohort included in the earlier pooled analysis of Steenland et al,[13] but with tin miners excluded because of potential confounding by arsenic exposure, and with follow-up extended by 10 years. Data regarding silicosis (based on a medical surveillance program) and smoking were available for all cohort members. There were 546 lung cancer deaths and 5297 cases of silicosis. A positive statistically significant exposure-response trend for lung cancer was again noted, which was similar to that found in the earlier pooled analysis. The relative risks (vs a nonexposed reference category) were 1.26, 1.54, 1.68, and 1.70, respectively, for quartiles of cumulative exposure, focusing on exposure 25 years before disease occurrence (exposure categories of 0, 0.01 to 1.2, 1.12 to 2.91, 2.91 to 6.22, and 6.22 or more mg/m^3-years).[15]

The study by Liu et al[15] was able to address the question of whether silicosis was a necessary precursor of lung cancer. After excluding individuals with radiographic evidence of silicosis from the analysis (representing 427 of the 546 lung cancer deaths), the relative risks were 1.12, 1.41, 1.58, and 1.70, respectively, by quartile of cumulative exposure (same exposure categories as noted above), demonstrating that silicosis was not a requirement for lung cancer. Liu et al also were able to address the question of effect modification by smoking, because their sample size was large enough to include a relatively large number of never-smokers (n = 12,177) and, more importantly, a number of never-smokers who developed lung cancer (n = 77). The lack of lung cancers among never-smokers is typically a key limitation in most studies addressing effect modification by smoking. In the study by Liu et al, never-smokers were divided into categories with low (less than 1.12 mg/m^3-year) and high (1.12 mg/m^3-years or higher) cumulative exposure, with a relative risk of high exposure versus low exposure of 1.60 (95% confidence interval, 1.01-2.55).[15] The analogous relative risk for highly exposed ever-smokers versus ever-smokers with low exposure was 1.48; these data indicate that the relative risk for exposure to silica is similar in smokers and nonsmokers. Nonetheless, because smoking is such a strong risk factor for lung cancer, the risks for silica exposure and smoking together are high. For example, looking at these data with the never-smokers with low exposure as the referent group for the other 3 categories, the relative risks for highly exposed never-smokers, ever-smokers with low exposure, and highly exposed ever-smokers were 1.60, 3.43, and 5.07, respectively. For those with high exposure to silica, stopping smoking (after some time) will decrease one's excess risk from 5-fold to 1.6-fold.

Both silicosis and lung cancer are believed to result from the strong inflammatory response that silica evokes in the lung. Inhaled silica causes both silicosis and lung tumors in rats. When rat macrophages attempt to digest silica, they are themselves killed, and their disintegration results in the release of oxidants and cytokines and leads to persistent inflammation with elevated neutrophils. This in turns causes epithelial cell injury and proliferation, resulting in fibrosis (silicosis).[12] The chronic inflammation and release of oxidants is also thought to cause genotoxic damage to the lung epithelium, thereby increasing the risk of lung cancer. These inflammatory cells also release several growth factors that may contribute to the pathogenesis of silicosis and lung cancer. It seems likely that these mechanisms also cause lung disease in humans. It is also thought the strong immune response in the lung may trigger autoimmune diseases in humans (including scleroderma, rheumatoid arthritis, and some forms of renal disease).

Hill made a classic list of criteria with which to judge whether an observed association is causal. Among the key ones were: 1) the consistency of findings across studies; 2) the strength of the association (higher, more likely to be causal); 3) the temporal sequence (exposure precedes disease); 4) biological plausibility; and 5) increasing effect with increasing exposure (positive exposure-response trend).[16] The association between silica and lung cancer fulfills criteria 1 (many positive studies), 3 (silica exposure preceded

lung cancer), 4 (inflammatory response in the lung may lead to genetic damage to epithelial cells), and 5 (positive exposure-response trend). The only one of the 5 Hill criteria not clearly met by the silica evidence is the strength of the association, which is only modest for those exposed versus those not exposed. It should be noted that a positive exposure-response trend is considered among the most important criteria for assessing causality, especially if the increasing trend is monotonic (consistently increasing as exposure increases), even if the slope of such a trend is modest.

What Do National and International Agencies Say About Crystalline Silica and Lung Cancer?

There are 2 agencies that are usually considered to be authoritative regarding whether a substance causes cancer in humans. One is the IARC, which is an agency of the World Health Organization. The second is the National Toxicology Program, which is part of the National Institute of Environmental Health Sciences, part of National Institutes of Health. As noted previously, the IARC in 1997 determined that crystalline silica causes lung cancer, based on sufficient evidence in humans and animals. The IARC reaffirmed their conclusion regarding silica in 2012 (available at monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-5.pdf). In the United States, the National Toxicology Program determined that crystalline silica was a human lung carcinogen in 2000, and reaffirmed this judgment in 2011 (available at ntp.niehs.nih.gov/ntp/roc/twelfth/profiles/Silica.pdf).

How Can Clinicians Help Patients Avoid or Minimize Silica Exposure?

The most effective measures for the control of occupational silica exposures, including substitution and engineering controls, are the responsibility of the employer. Source control can be achieved by banning sandblasting, substituting metal grits for abrasive blasting, and modifying processes and equipment, including wet methods. Control of dust transmission includes isolation of the source or workers by enclosed processes, air curtains, water spray, local exhaust ventilation, general ventilation, enclosed cabs, and air supply systems. Employers should also provide silica warning signs, training and education about work practices, and personal protective equipment. Workers should be strongly encouraged by their personal physicians to comply with work practice guidelines, including the use of personal protective equipment, and to participate in the medical surveillance program.

More information about the hazards of silica and protection against exposure to silica in the workplace can be found at osha.gov/OshDoc/data_General_Facts/crystalline-factsheet.pdf. NIOSH has a number of publications outlining specific precautions to minimize silica exposure in a variety of industries (available at cdc.gov/niosh/topics/silica/). Workers who believe that they are exposed to hazardous conditions on their job have the right to contact OSHA (osha.gov/workers.html) and/or to request a Health Hazard Evaluation from NIOSH (www.cdc.gov/niosh/hhe/).

Health Care Recommendations For Individuals Exposed to Silica

For workers who are currently exposed to silica at air concentrations of 0.05 mg/m^3 or higher, the new recommended OSHA standard, the American College of Occupational and Environmental Medicine recommends enrollment in a workplace medical surveillance program involving baseline evaluation and annual follow-up, including occupational and medical history (questionnaire), physical examination, a purified protein derivative test, chest radiography, and spirometry. If a worker is suspected of having silicosis during the surveillance examination, he or she should be removed from any further exposure and promptly referred to a physician experienced in the diagnosis and treatment of silicosis. A new case of silicosis in the workplace should trigger a thorough assessment of silica exposure and control measures by a qualified industrial hygienist.

Workers no longer exposed to silica may still be at risk of developing silica-related diseases, the probability of which depends in large part on the level and duration of exposure. Although the diagnosis of silicosis has become relatively rare in the United States, the likelihood of a clinician encountering patients with past or present silica exposure is higher in communities with historical or current concentrations of high-risk industries and occupations. Radiographic surveillance programs for the development of silicosis should adhere to International Labor Organization (ILO) standards, which require interpretation by a NIOSH-certified B reader. The ILO classification relies mainly on a posteroanterior radiograph at full inspiration on a 14 × 17-inch film. The ILO recently made standard digital radiographic images available and has published guidelines on the interpretation and classification of these images. OSHA notes that recent studies and reviews suggest that CT or high-resolution CT (HRCT) may be superior to chest x-ray in the early detection of silicosis and the identification of PMF, but has requested comment on whether CT and HRCT should be considered "equivalent diagnostic studies" to plain chest x-rays under the standard for mandated medical surveillance programs, citing concerns about a lack of standardized methods for interpreting and reporting silicosis based on CT or HRCT scans and the higher radiation doses associated with these tests.

The publication of the results of the National Lung Screening Trial demonstrating a 20% reduction in lung cancer mortality in patients undergoing low-dose CT (LDCT) led a number of organizations, including the American Cancer Society, to publish recommendations for screening with LDCT for individuals aged 50 years and older with smoking histories of at least 30 pack-years. Most guidelines regarding LDCT screening, including draft recommendations from the US Preventive Services Task Force, do not make specific recommendations for individuals with occupational exposure to lung carcinogens (uspreventiveservicestaskforce.org/bulletins/lungcandrftbulletin.pdf). An exception is the National Comprehensive Cancer Network guidelines, which recommended that individuals with significant occupational exposure to a carcinogen be offered screening beginning at age 50 years if they have a smoking history of at least 20 pack-years (nccn.org/professionals/physician_gls/f_guidelines.asp#lung_screening). Clinicians should apply the National Comprehensive Cancer Network lung cancer screening guidelines to patients with a history of smoking and employment in silica-exposed occupations.

What Is the Expected Impact of OSHA's Proposed Regulatory Changes?

Risk assessment for a standard setting requires translating these epidemiologic findings into lifetime excess risk at given levels of exposure. OSHA generally seeks a level of exposure that results in no more than a 1/1000 lifetime risk of disease, in excess of existing background risk. Ideally, with silica, OSHA would seek a level of exposure through a working lifetime (45 years) that would increase the general population's lifetime risk of lung cancer (about 6% by age 85 years or 60/1000) by no more than 1/1000 (eg, no increase beyond a lifetime risk of 61/1000). This means that one expects that 60 cases of lung cancer will occur over the lifetime of 1000 silica-exposed workers, based on the experience of the general nonexposed population. OSHA's goal is to set a standard such that no more than one extra case occurs (ie, that no more 61 cases occur over the lifetime of 1000 silica-exposed workers).

However, OSHA is also constrained to consider the technologic and economic feasibility of lowering the standard to a new level. In the case of silica, OSHA has determined that the exposure at the current standard (0.1 mg/m^3) results in between 13 to 60 excess lung cancer cases over a lifetime, 11 to 83 cases of silicosis (all silicosis cases are excess cases) over a lifetime, and 39 excess cases of chronic renal disease per 1000 workers exposed (osha.gov/silica/Combined_Background.pdf). Lowering the standard to 0.05 mg/m^3 is feasible and is expected to reduce these numbers to 6 to 26 excess lung cancer cases, 7 to 43 cases of silicosis, and 32 excess cases of chronic renal disease. While the proposed new standard still falls short of OSHA's goal of limiting excess lifetime risk to no more than 1/1000, OSHA estimates that the new standard will save 700 lives per year, as well as avoiding 1700 cases of silicosis. In addition, OSHA's analysis indicates that reducing the permissible exposure limit results in benefits that substantially exceed costs (osha.gov/silica/Silica_PEA.pdf for their silica cost-benefit analysis).

Summary

In conclusion, silica has been established as a cause of lung cancer in humans, a finding that is supported by a large body of epidemiologic evidence. Compared with some other lung carcinogens such as smoking or asbestos exposure, the relative risks for lung cancer are smaller, making it more difficult to differentiate the contributions of silica exposure, silicosis, and tobacco smoking to the cancer risk. Nonetheless, after over 100 epidemiologic studies, there is strong and consistent evidence that silica exposure increases lung cancer risk. This has both regulatory implications and implications for clinicians. For OSHA, it is appropriate to lower existing standards. Clinicians should inquire about occupational history to determine if silica exposure has occurred; if such exposure has occurred, it is essential to implement the early detection of silicosis and lung cancer, as well as place an extra emphasis on quitting smoking and to provide education on the importance of learning and following occupational safety recommendations. In cases of obvious high exposure, communication to relevant authorities may be in order, while conserving worker confidentiality. ■

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