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Radiographic Screening Reveals High Burden of Silicosis among Workers at an Engineered Stone Countertop Fabrication Facility in California

To the Editor:

Silicosis is a progressive and incurable, but preventable, occupational respiratory disease caused by respirable crystalline silica exposure. Over the past decade, outbreaks of silicosis have been reported in several countries among workers who cut and finish engineered stone slabs for countertops (1–3). Many affected workers have been young, with rapidly progressive disease (4, 5). Engineered stone is a composite material made of crushed quartz bound together with polyester resins and pigments, with significantly higher silica content than natural stone materials; engineered stone typically contains >90% silica, compared with <45% in granite and <5% in marble (3, 6). Workers can be exposed to markedly elevated levels of respirable crystalline silica when cutting and finishing engineered stone materials (7, 8).

Approximately 98,000 people work in the stone fabrication industry in the United States (9); however, systems for silicosis surveillance and reporting are not well developed, and limited information is available about disease prevalence among industry workers. In early 2019, the California Department of Public Health (CDPH) identified three cases of silicosis, including two fatalities, among former workers at an engineered stone countertop fabrication company (company A) (10). During a 2009 inspection of company A, the California Division of Occupational Safety and Health had measured respirable crystalline silica levels 22 times higher than the permissible exposure limit of 0.1 mg/m^3 in effect at that time; in 2019, a repeat California Division of Occupational Safety and Health inspection again identified inadequately controlled silica dust exposures. We sought to determine prevalence of silicosis and related risk factors among current employees of this company. Some of the results of this investigation have been previously reported in the form of an abstract (11).

At CDPH's recommendation, company A provided silicosis screening to all current employees working in stone fabrication areas at the company's two locations. Screening included spirometry and chest radiography, with radiograph classification performed by a National Institute for Occupational Safety and Health–certified B Reader physician according to the International Labor Organization (ILO) system for pneumoconioses (12). Medical and employment records were reviewed by a CDPH physician, and silicosis was defined as ILO classification of 1/0 or higher. Global Lung Function Initiative reference values (13) were used to calculate lower limit of normal values for FEV_1, FVC, and FEV_1/FVC ratio.

All 43 currently employed fabrication workers underwent silicosis screening. All were Hispanic men; median age was 37 years (interquartile range [IQR], 24–45 yr). Five employees (12%) had silicosis, with ILO classifications of 1/0 for two employees, 1/1 for one employee, and 2/1 for two employees. Median age of employees with silicosis was 40 years (IQR, 38–53 yr), and job tasks of these employees included cutting, fabricating, and laminating stone slabs. Duration of employment was available for 36 (84%) employees, including 4 with silicosis. Median duration was 14.9 years among those with silicosis (IQR, 13.9–16.2 yr) and 6.5 years among those without silicosis (IQR, 3.1–15.2 yr). Three employees with silicosis had FEV_1 and FVC less than the lower limit of normal with normal FEV_1/FVC ratio, a restrictive pattern typical of silicosis; five employees with negative chest radiographs had similar restrictive spirometry patterns.

This investigation provides the first estimate of silicosis prevalence among a cohort of countertop fabrication workers in the United States. The only prior estimate in this industry came from Queensland, Australia, where the government began offering free silicosis screening to current and former industry workers in 2018. As of May 2020, among 1,047 workers screened, 204 (19.5%) had been diagnosed with silicosis, including 31 (2.9%) diagnosed with progressive massive fibrosis (14).

This investigation was subject to several limitations. First, chest radiographs may not have been sufficiently sensitive to identify all silicosis cases. Results from a series of 78 countertop fabrication workers with silicosis demonstrated that 43% had normal chest radiographs and were diagnosed instead by chest computed tomographic (CT) imaging, which has been found to have higher sensitivity for pneumoconioses (15, 16). Second, although the index

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cases at company A were identified among former employees, only current employees underwent screening; additional silicosis cases might have occurred among former workers, particularly given the lag time between exposure and disease onset. Information about prior employment was also unavailable for screened workers. The screening program in Queensland, which identified a higher prevalence of disease, assessed both current and former industry workers; some workers underwent chest CT imaging in addition to a chest radiograph (14). Finally, these results are limited to a single employer and may not be generalizable to other workers in this industry.

In the United States, federal and state occupational silica standards were updated in 2016 to include a lower permissible exposure limit of 0.05 mg/m^3 for respirable crystalline silica and requirements for employers to conduct exposure monitoring and provide medical surveillance to workers. Although well-established strategies exist for reducing silica dust exposure, including tools equipped with water feeds, well-designed exhaust ventilation, wet cleanup methods, and use of appropriate respiratory protection, the extent to which these control methods are used effectively in the countertop fabrication industry is not known. The high prevalence of this serious but preventable disease identified here and in Queensland, however, suggests that additional efforts are needed to adequately control silica dust exposures among countertop fabrication workers. In Queensland, dry cutting of engineered stone was banned in 2018 (17); no such ban currently exists in the United States. Recommendations have been made elsewhere to ban engineered stone materials altogether (4).

In addition, without standardized silicosis screening and surveillance, industry-wide prevalence remains unknown. The estimate from our limited sample of workers in California, along with the higher prevalence seen among Australian workers, suggests there might be many more cases yet to be identified among the nearly 100,000 U.S. workers in this industry. Understanding the burden of silicosis among these workers is important for addressing this preventable cause of occupational morbidity and mortality. Clinicians should obtain an occupational history and consider a silicosis diagnosis in patients with compatible clinical findings and occupational exposure; identified silicosis cases should be reported to public health and occupational health and safety authorities. Enhanced surveillance using techniques such as chest CT imaging and lung diffusing capacity should also be considered, especially in workers with known or suspected high levels of exposure, as has been suggested elsewhere (18). Given the number of potentially affected workers, these actions are urgently needed to identify at-risk workers and control silica dust exposure to prevent silicosis. ■

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The Impact of Hospital-Ward Ventilation on Airborne-Pathogen Exposure

To the Editor:

The coronavirus disease (COVID-19) pandemic has brought into sharp focus the importance of protecting patients and healthcare workers (HCW) from nosocomial infection (1). Hospital-associated infection can occur through contact with contaminated surfaces, either by droplets generated by coughing or sneezing settling on exposed mucous membranes or through direct inhalation of aerosols. Although the contribution of each route differs according to the setting and type of infection, the ventilation within hospitals is a key determinant of infection risk from airborne pathogens (2, 3), with low ventilation rates associated with increased risks of transmission of, for example, tuberculosis and Middle East respiratory syndrome (4, 5). For patients with cystic fibrosis (CF), acquisition of multidrug-resistant pathogens from other patients represents one of the greatest threats to health (6), with both experimental data (7) and room sampling (8) highlighting the risk of cross-infection through long-lived infectious aerosols.

As a direct response to these challenges, Royal Papworth Hospital installed a bespoke ventilation system during its relocation from a 100-year-old facility to a new state-of-the-art cardiothoracic hospital in Cambridge, United Kingdom. Whereas the old CF center relied on passive, natural ventilation, with airflow created by opening doors and windows, the mechanical ventilation in the new hospital provides 15 air changes per hour (ACH) in the CF center (both patient rooms and corridors), and 6 ACH in other clinical areas.

We examined the impact of high-frequency air changes within patient rooms by monitoring the dispersal, mixing, and ventilation of an injected tracer gas (CO_2) using eight CozIR-A sensors (Gas Sensing Solutions) in the new and old hospitals, just before occupation and just after vacation respectively, while the heating and ventilation systems were operational. Although CO_2 persisted in passively ventilated rooms for over 58 minutes, rooms actively ventilated at 6 or 15 ACH experienced exponential decay of tracer concentrations to 10% of peak concentration within 26.7 and 11.7 minutes, respectively (Figure 1A).

The type of ventilation determines the fraction of airborne droplets that settle in the patient's room before being removed. Because the fall speeds of droplets vary with particle size, the impact of changes in ventilation between old and new hospitals is particularly marked for particles in the respirable size range produced by coughing (9) (1–10 μm in diameter) (Figure 1B), indicating that high-frequency air changes may reduce transmission risks from aerosols.

We next explored air mixing and dispersal along hospital-ward corridors, again using a CO_2 tracer with sensors deployed 1.7–14.7 m from the CO_2 source (Figure 1C). With passive ventilation at the Old Papworth site, CO_2 spread rapidly along the corridor and persisted for 30 minutes. At the new site, active ventilation (at 6 ACH) limited dispersal distances and improved clearance rates (although detectible amounts of CO_2 remained after 30 min), whereas high ventilation with 15 ACH initially caused faster dispersal than conventional ventilation but led to clearance of CO_2 to below 10% of peak concentration by 13.3 minutes.

To examine the impact of ventilation on patient exposure to airborne pathogens, we deployed static NIOSH BC 251 two-stage cyclone aerosol samplers (9), positioned at 90 cm and 180 cm from the floor, to sample the air in unoccupied patient rooms and ward corridors in both the old and new CF centers during periods of similar ward–bed occupancy (92% ± 3.5% new ward vs. 94% ± 7.9% old ward; mean ± SD, $P = 0.43$). The amounts of total bacteria (quantified by *16S* quantitative PCR) were reduced in rooms and corridors with high-frequency air changes compared with those with passive ventilation (Figure 1D), suggesting reduced patient and HCW exposure to nosocomial airborne infections.

We also considered how people walking might influence air mixing and residence times in corridors. Given the high Reynolds number (*Re*) associated with a person walking ($Re = \rho u L/\mu \approx 0.5 \times 10^5$; based on a speed [$u$] of 1 m/s, size [$L$] of 0.5 m, air viscosity [μ] of 10^{-5} Pa·s, and density [ρ] of 1 kg/m^3), we expect the resultant wake to drive highly turbulent mixing. When measured in hospital corridors with high ventilation, a person walking along the corridor every 30 seconds considerably increased the residence time of CO_2 after a pulse release (Figure 1E), lengthening the half-life from 1.3 minutes to 6.7 minutes (Figure 1E).

Finally, we explored the potential for movement of infectious aerosols between corridors and rooms. We found that CO_2 released in the corridor rapidly penetrated into patient rooms with open doors (Figure 1F), despite the fact that both corridor and rooms had their own balanced system of inflow and outflow ducts and were at the same pressure. We saw a steep relationship between the duration of door opening and the resulting CO_2 concentrations in the room (Figure 1G), suggesting that limiting door opening could greatly reduce corridor-to-room pathogen spread.

We also noticed that movement of CO_2 into rooms increases with temperature contrasts. Airflow speeds across a doorway based on video recordings of tracer smoke show air flowed out of the room near the top (with mean speeds of 15–16 cm/s); whereas at lower heights, air flowed into the room at 16–18 cm/s ($P = 0.002$) (Figure 1H). The associated exchange flow driven by a 3°C temperature difference between the room and corridor (0.16 m^3/s) is comparable

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