Occupational lung diseases in the 21st century: the changing landscape and future challenges

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Purpose of review
Occupational exposures remain an underrecognized and preventable cause of lung disease in high-income countries. The present review highlights the emergence of cleaning-related respiratory disease and the re-emergence of silicosis as examples of trends in occupational lung diseases in the 21st century.

Recent findings
Employment trends, such as the shift from large-scale manufacturing to a service economy, the growth of the healthcare sector, and changing consumer products have changed the spectrum of work-related lung diseases. Following decades of progress in reducing traditional hazards such as silica in U.S. workplaces, cases of advanced silicosis have recently re-emerged with the production of engineered stone countertops. With growth in the healthcare and service sectors in the United States, cleaning products have become an important cause of work-related asthma and have recently been associated with an increased risk of chronic obstructive pulmonary disease (COPD) in women. However, these occupational lung diseases largely go unrecognized by practicing clinicians.

Summary
The present article highlights how changes in the economy and work structure can lead to new patterns of inhalational workplace hazards and respiratory disease, including cleaning-related respiratory disease and silicosis. Pulmonary clinicians need to be able to recognize and diagnose these occupational lung diseases, which requires a high index of suspicion and a careful occupational history.

Keywords
cleaning products, medical surveillance, occupational lung disease, silicosis, work-related asthma

INTRODUCTION

Occupational exposures remain an under-recognized and preventable cause of lung disease in high-income countries, and world-wide. As observed in a recent American Thoracic Society (ATS) review, workplace exposures contribute substantially to the burden of chronic respiratory diseases and a pressing need to improve clinical recognition of occupational lung disease remains [1▪▪]. The present review highlights both the re-emergence of a traditional pneumoconiosis, silicosis, and an important but rarely recognized cause of asthma and likely also chronic obstructive pulmonary disease (COPD), cleaning products.

Following decades of progress in reducing inhalational hazards in U.S. workplaces, preventable well known occupational lung diseases such as coal worker's pneumoconiosis [2,3] and silicosis [4,5,6▪] have recently re-emerged. The respiratory hazards of common workplace exposures, such as cleaning solutions among nurses and traffic-related air pollution among urban drivers, are increasingly recognized as well. The increasing use of nonstandard work arrangements, the decline in union representation, and the shift from an industrial to a service-oriented economy have likely contributed to the fragmentation of traditional occupational health and safety practices, including workplace health surveillance programs [7–10]. The introduction of new products such as artificial stone, coupled with inadequate industrial hygiene controls and limited enforcement of existing occupational safety and health standards, have further contributed to the persistent burden of occupational lung disease [6▪,11▪,12,13▪].

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KEY POINTS

- Despite progress in occupational health, work exposures contribute to a substantial burden of chronic lung disease in high-income countries.
- Occupational exposure to cleaning products is an important cause of work-related asthma and is associated with an increased risk of COPD in women.
- Changing work structures and exposure patterns has resulted in the re-emergence of silicosis, an irreversible and potentially fatal occupational lung disease.
- There is a need for clinicians to have a high level of suspicion for occupational lung disease and integrate a thorough occupational history into their evaluation of patients with chronic lung diseases.

In this review, we focus on the emergence of cleaning-related respiratory disease and the re-emergence of silicosis as examples of these current trends in occupational lung disease. These conditions highlight how the changing structure of work, exposure patterns, and workplace hazards provide a challenge to the clinician to diagnose and prevent occupational lung diseases.

CLEANING-RELATED RESPIRATORY EXPOSURES AND DISEASE

Occupational exposure to cleaning products is common, especially among women. Population-based studies from Northern Europe have observed that 16% of the general population has been employed in the occupational cleaning sector, with a mean duration of 5.7 years [14]. One in five working women has reported exposure to industrial cleaning and sterilizing agents in a similar population-based survey [15]. With the growth of the healthcare sector, an increasing population of workers is being exposed to disinfectants and more potent sterilizing agents at work. Highly concentrated exposure can occur in poorly ventilated spaces such as bathrooms, and few exposure limits have been recognized.

A challenge in the diagnosis and control of cleaning-related respiratory disease is the heterogeneity of exposure (Table 1). Common cleaning solutions include detergents, disinfectants such as bleach, caustic alkaline agents such as ammonia or sodium hydroxide, corrosion inhibitors, polishes, quaternary ammonium compounds, and perfumes [16]. A single cleaning solution is often a mixture of several different compounds, and many are applied by spraying, which increases the risk of inhalational exposures. Cleaners are also exposed to mineral [17] and biologic dusts and other environmental exposures, which are independently associated with respiratory symptoms [18].

Cleaning agents may act through immune and nonimmune mechanisms and a single agent may act as both a sensitizer and irritant [19]. Mixtures of common products can also result in significant toxicity: mixing bleach with acids or ammonia releases chlorine and chloramine, respectively, and can

Table 1. Selected cleaning-related exposures that may cause respiratory disease

Chemicals	Example
Disinfectants (alcohols, aldehydes, phenolics)	Ethanol, glutaraldehyde[a], hydrogen peroxide, phenolics, chlorhexidine[a], quaternary ammonium compounds (e.g. benzalkonium chloride)[a]
Chlorine disinfectants	Sodium hypochlorite (bleach), chloramine
Caustics and alkaline agents	Ammonia, sodium hydroxide, silicates, carbonates
Acids	Phosphoric, acetic, citric, and hydrochloric acid
Solvents	Glycol ethers, 2-buthoxyethanol
Corrosion inhibitors	Ethanolamine[a], other amines[a]
Complexing agents / water softeners	EDTA, tripolyphosphates
Scents and perfumes	Eugenol, terpenes such as pinene, limonene[a]
Detergents	Soaps, organic sulphonates
Preservatives	Isothiazolinones[a], formaldehyde[a], benzalkonium chloride[a]
Other cleaning-related exposures	
Natural rubber latex	Latex gloves
Biologic dusts	Fungi, bacteria, animal droppings
Construction dusts, particulates	Wood, silica, cement, plaster
Other contributing factors	Inadequate ventilation, spray application, use of multiple products, enclosed spaces

[a]Known or suspected chemical sensitizers.

result in an acute inhalational injury and other respiratory conditions, such as adult respiratory distress syndrome (ARDS), reactive airways dysfunction syndrome (RADS), or chemical pneumonitis [20,21]. Typical cleaning work is characterized by a mixture of different chemical and biologic or other environmental exposures, and may result in diverse phenotypes. Given the complexity of exposures that occur during cleaning, it can be challenging to identify which specific component is the causative agent.

The most well characterized respiratory disease associated with cleaning exposures is work-related asthma, which includes new occupational asthma and exacerbation of preexisting asthma [22–24]. Several recent epidemiologic and surveillance studies have documented that cleaning exposures and cleaning-related occupations account for a substantial fraction of work-related asthma [25,26▪▪,27▪▪]. A recent systematic review and meta-analysis of population-based studies across Europe, for example, estimated that work as a professional cleaner was associated with more than a 50% increase risk of asthma (odds ratio (OR) = 1.5; 95% confidence interval (CI), 1.3–1.8) [28]. A significant dose–response relationship between duration of cleaning in years and self-report of respiratory symptoms and asthma has also been observed among professional cleaners [14].

A notable surveillance study published in the past year reviewed cases of cleaning-related respiratory disease reported to three surveillance networks by physicians in the United Kingdom over more than 20 years (1997–2019) [27▪▪]. Among the Surveillance of Work-Related and Occupational Respiratory Disease (SWORD) cases, asthma (60% cases) and acute inhalation accidents (28%) were the most common cleaning-related diagnoses. Other diagnoses included rhinitis, bronchitis/emphysema, allergic alveolitis, and upper airway irritation, reflecting the broad spectrum of lung disease related to cleaning products. The highest risk occupations for cleaning-related respiratory disease included industrial and domestic cleaners, laundry and dry cleaning workers, nurses, medical assistants, and workers in athletics who are exposed to chlorinated pools (Table 2) [27▪▪].

Table 2. Selected occupations with cases of cleaning product-related asthma

Cleaners, janitors, housekeepers
Maintenance workers, building and grounds workers
Nurses, other healthcare workers
Medical and dental technicians, dental nurses
Clerical workers
Teachers

A recent review and analysis of 3634 cases of confirmed work-related asthma in the state of Michigan over 31 years (1988–2018) noted marked changes in the type of industries and exposures over this time period [26▪▪]. Comparing the most recent to the earliest decade, cleaning agents became the most commonly identified causative exposure, increasing from 5 to 20% of cases, and healthcare became the second most common industry (after all manufacturing) [26▪▪]. The study also highlighted the important need for physicians to consider workplace exposures in their patients with asthma.

Accumulating evidence suggests that cleaning also contributes to the development of COPD [28]. In several large cross-sectional studies from Northern Europe, cleaning work has been associated with higher risk of COPD based on self-report of physician diagnosis [14] and obstructive physiology on pulmonary function testing [29], as well as higher risk of death from COPD [30]. A recent prospective analysis of the European Community Respiratory Health Survey (ECRHS), an international multicenter population-based longitudinal cohort study conducted over 20 years, found that women who cleaned either at home or professionally had an accelerated decline in lung function that was comparable to 10–20 pack-years of tobacco smoking, as measured by decreases in both forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) [31▪]. Impairment in lung function was more pronounced in women who cleaned more frequently, and was independent of asthma or asthma treatment. Exposure to biologic dusts at work has also been associated with higher risk of chronic bronchitis, emphysema, and COPD, with a greater risk in women compared to men [18].

The results from these European cohort studies were recently supported by analysis of a large prospective U.S. cohort, the Nurse's Health Study. This analysis of 73 262 nurses demonstrated that exposure to commonly used disinfectants, including alcohol, bleach, hydrogen peroxide, glutaraldehyde, and quaternary ammonium compounds was associated with a 25–36% increased risk of COPD, depending on the compound, and estimated that as much as 12% of COPD may be attributed to occupational exposure to disinfectants [32▪▪].

As with work-related asthma in general, early recognition of cleaning-related asthma is essential, as persistent work exposures can lead to more advanced disease and substantial adverse health and socioeconomic consequences [33,34]. A recent cohort study, for example, demonstrated a two- to three-fold increased risk of severe asthma (OR = 2.8;

95% CI, 1.5–5.6) among individuals with asthma and persistent occupational exposures [35▪]. Asthma can persist even away from the causative work exposures, which may limit future job opportunities, especially for less-skilled workers [36,37▪].

Cleaning-related asthma should be considered in any patient with new onset or worsening asthmatic symptoms and potential cleaning-related exposures. The diagnosis and management of work-related asthma has been reviewed [38▪,39,40]. As noted in these reviews and guideline documents, a detailed clinical and occupational history is essential to diagnosing work-related asthma, including documenting the onset of asthma, work tasks, exposures, and the temporal relationships between work and asthmatic symptoms. Management focuses on exposure reduction, and if needed, complete removal from work, as well as addressing the patient's work status and employment options [38▪,39,40].

Acute inhalational events following exposures to cleaning products, such as accidental spills, are more easily recognized as work-related than chronic conditions such as COPD, which may present years after employment in the causative work environment, requiring a high index of suspicion.

SILICA-RELATED LUNG DISEASE

Crystalline silica, one of the earliest recognized and most studied occupational inhalational exposures, has re-emerged in the United States and other high-income countries as an important, persistent occupational hazard. Inhalation of respirable crystalline silica dust causes silicosis, a chronic fibrotic lung disease, but is also associated with an increased risk of COPD, lung cancer, and tuberculosis. Exposure to silica, or silicone dioxide, is ubiquitous – it is the most abundant mineral in nature and the main constituent in 95% of rocks [11▪]. Hazardous exposure to silica occurs when respirable size particles (<10 μm) of crystalline silica dust are generated, aerosolized, and deposited in distal airways. The cumulative dose of crystalline silica dust retained in the lungs is a primary determinant in the pathogenesis of silica-related lung disease [41].

OSHA estimates that 2.3 million U.S. workers are exposed to silica at work [42]. Although hazardous exposure to silica has occurred across a wide spectrum of occupations for centuries, the offshoring of high-risk industries to low-income countries and the transition from industrial-type employment relations to nonstandard work arrangements has contributed to new patterns of exposure [7]. Common work settings with exposure to silica in the United States now include residential, nonresidential and road construction, roofing, demolition work, concrete manufacture, stone masonry, sandblasting, artificial stone manufacturing and finishing, and pottery and ceramic production [42] (Table 3). Fewer U.S. workers belong to labor unions, and many of these job tasks are now performed by independent contractors. Workers employed in these 'nonstandard' and/or nonunion work arrangements have been observed to be at increased risk for work-related injuries [43] and fatalities [10] that may be because of insufficient safety training, inappropriate personal protective equipment, or assignment to more hazardous job tasks [7].

Table 3. Selected industries and occupations with risk of silicosis

Construction of buildings (cement, concrete, and gypsum/drywall work, demolition)
Road construction and repair
Mining (coal, metals), tunneling, quarrying
Sandblasting / abrasive blasting in many settings
Manufacturing (engineered countertops, glass, pottery, ceramics, dental labs, jewelry)
Stone work, masonry (production, installation)
Foundries, metal work
Petroleum and natural gas extraction

Silicosis is an irreversible, potentially fatal lung disease and the most recognized outcome of silica exposure [44]. Chronic silicosis has historically been the most common presentation, and is typically diagnosed in older workers exposed to silica dust for more than 10 years [4]. Both chronic and accelerated silicosis, which develops within 5–10 years of a higher intensity exposure, are characterized by upper-lobe predominant pulmonary nodules that can eventually become confluent, described as progressive massive fibrosis.

The development of new industries and commercial products, changing work practices, and inadequate workplace monitoring and enforcement have contributed to multiple outbreaks of silicosis in the past 20 years, including among workers sandblasting of denim jeans [45], dental laboratory technicians [4,42], and engineered stone workers [6▪,12,13▪,46▪▪].

Recent case series in the United States have documented accelerated silicosis and end-stage coal worker's pneumoconiosis in younger workers [6▪,11▪,13▪], reflecting high exposures to silica dust even in industries that previously controlled exposure, such as U.S. coal mining. Progressive massive fibrosis, for example, had been nearly eradicated among miners in central Appalachian states prior to 2000, but the prevalence increased approximately 10-fold in the region between 2000 and 2015 [3,47].

The shifting epidemiology of silicosis in the United States was recently highlighted in a review and analysis of more than 1000 confirmed cases of silicosis in the state of Michigan over three decades [48▪▪]. Although the overall incidence of silicosis decreased with time, much of this decrease was attributed to a two- to three-fold reduction in the number of foundry workers. In comparing three time periods (1988–1997, 1998–2007, 2008–2016), the proportion of companies with respirable silica levels above the new OSHA limit did not change (62% of companies inspected), but OSHA workplace inspections decreased from 57 companies to 6 companies, mandatory reporting of silicosis cases decreased by 81%, and applications for worker's compensation among silicosis cases decreased from 42 to 16%. Across all time periods, workplace medical surveillance was identified in only 11% of companies inspected, and death certificates documented silicosis in only 8% of confirmed cases. These findings highlight how under-recognition of silicosis, reduced OSHA enforcement, and a shifting pattern of silica exposure, from large foundries to smaller businesses, has proved challenging to the diagnosis and prevention of silicosis.

One notable and concerning new exposure across high-income countries has been from the manufacture of artificial stone countertops, which can have substantially higher silica content than natural stone such as granite [6▪,12,13▪,46▪▪]. A recent case series from the United States documented 18 cases of severe silicosis and two fatalities among stone fabrication workers in the past 2 years [13▪]. Although the incidence of disease in the stone fabrication industry remains unknown, a screening program that was recently implemented in Australia identified silicosis in more than 12% of artificial stone workers [12]. Substantial exposures to respirable silica have also been observed in other emerging industries and jobs, including the hydraulic fracturing of oil and gas (fracking) [49].

More recent research has also expanded our understanding of the spectrum of silica-related lung disease. Since being classified as a class 1 carcinogen by IARC in 1997 [41], more recent meta-analyses have demonstrated that the risk of lung cancer is dose-related to silica exposure, elevated even in those without silicosis, and observed across heterogeneous occupational exposures [50–53]. It is also now clear that silica exposure increases the risk of tuberculosis infection even in the absence of silicosis [41], and silicotuberculosis has been recognized as an occupational health risk in construction, mining, and other industries, especially in tuberculosis endemic regions [54–56]. Exposure to silica is also associated with elevated risk of chronic renal disease and diverse autoimmune diseases including rheumatoid arthritis, systemic lupus erythematosus, and systemic sclerosis [41,57].

Because of ongoing concern for the hazards of silica, in June 2018, OSHA implemented a new silica standard for the first time since 1971, lowering the permissible exposure limit from 100 to 50 mcg/m^3 and mandating medical surveillance of workers with exposures above the permissible exposure limit. Surveillance should include medical examination and occupational questionnaire, chest x-ray with B read interpretation, tuberculosis screening, spirometry, and specialist referral for concern of silica-related lung disease. With lower exposure limits, the number of workers requiring evaluation for silica-related lung disease is expected to increase.

The approach to diagnosis of silica-related lung disease is well established [41]. A high level of suspicion and a thorough occupational history is essential, including all prior jobs given the long latency. For each job, estimates of the intensity and duration of exposure to silica dust, and the use of particulate respirators, should be noted. Pulmonary function testing is variable, and can show either obstructive or restrictive physiology, often with reduced DLCO. Imaging of silicosis can appear as small nodules in upper lobes, diffuse interstitial fibrosis, or calcified lymphadenopathy [41]. Biopsy is usually unnecessary and the diagnosis is made clinically based on imaging, history of silica exposure, and exclusion of more probable diagnoses. There is no treatment other than lung transplant for severe cases. Patients diagnosed with silicosis should be followed and assessed for comorbidities associated with silicosis, most notably rheumatologic disease and tuberculosis [41,58].

CONCLUSION

The present review has focused on the emergence of cleaning-related respiratory disease and the re-emergence of silicosis as two examples of occupational lung disease in the 21st century. The changing economy and structure of work, growth in the healthcare sector, and novel workplace hazards have contributed to the persistent burden of occupational lung diseases. Workplace exposures to cleaning products are becoming increasingly common with the expansion of the healthcare and service industries, and pulmonologists should have a high index of suspicion for cleaning-related asthma in any patient with new or worsening asthma and potential exposures to cleaning. With the new lower OSHA standard for silica and mandated medical surveillance, pulmonologists will likely see an increase in referrals for evaluation of possible silica-related

disease. Given the substantial occupational contribution to the major chronic lung diseases (asthma, COPD, interstitial lung disease), pulmonary clinicians need to maintain a high level of suspicion for work-related lung disease and integrate a thorough occupational history into their evaluation.

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Conflicts of interest

There are no conflicts of interest.

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