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Occupational Lung Disease

From Case Reports to Prevention

Flock worker's lung disease presents a useful paradigm for identifying new occupational causes of lung disease. It is an unusual interstitial lung disease characterized by lymphocytic bronchiolitis and peribronchiolitis in workers exposed to flock fibers in manufacturing velvet-like fabrics, fuzzy greeting cards and wall papers, and automotive gaskets and glove box surfaces. Flock is made by cutting short synthetic fibers from bundles of parallel monofilaments of nylon or other polymers for application to adhesive-coated substrates. Unlike respirable mineral fibers such as asbestos, synthetic flock is visible, as illustrated in its typical applications. Since 1975, published case reports have raised suspicion of a respiratory hazard associated with various synthetic fibers, including polyester, nylon, and acrylic dust.¹⁻³ With regard to synthetic flock, early reports in 1974 and 1981 of workplace eval-

uations by National Institute of Occupational Safety and Health (NIOSH) investigators attributed respiratory symptoms among workers to irritant properties of nonrespirable flock fibers on the upper airways but did not pursue the possibility of lung disease associated with flock work.^{4,5} Systematic workplace investigation of lung disease in workers that flock with synthetic fibers awaited the recognition of case clusters of interstitial disease in small workforces, first in Kingston, Ontario, Canada, and then in Rhode Island, as detailed in the background of the follow-up study by Turcotte et al⁶ published in this issue of *CHEST* (see page 1642). Subsequently, additional cases or subclinical morbidity have been found in relation to nylon flock in two Massachusetts plants, to polyethylene flock in Spain, to polypropylene flock in Turkey, and to rayon flock in Kansas.⁶

The discovery of this new cause of interstitial lung disease required multidisciplinary investigations. Environmental scientists found a previously unsuspected nylon dust of respirable size in the air of the Rhode Island and Massachusetts workplaces. Physician-epidemiologists identified risk factors in exposed workforces, and animal toxicologists demonstrated biologic plausibility in rodents by intratracheal instillation of workplace dusts and components thereof. Working in production and maintenance, on a flocking range, and more days and hours per week; cleaning with compressed air; and a higher current and cumulative respirable dust exposure were found to be strongly associated with symptoms of the disease. Finally, several observations affirmed that the exposure preceded the disease and that the disease improved with exposure cessation and recurred in several workers with re-exposure to the implicated work environments. Together, the investigations have reasonably fulfilled Bradford Hill's criteria for demonstrating causation.⁷ Thus, one lesson of flock worker's lung disease is that clinical suspicion of new causes of occupational lung disease requires multidisciplinary public health investigation to generate actionable preventive measures in workplaces.

Another lesson is that follow-up after workplace intervention is useful in evaluating a suspected cause. In Ontario, investigators noted that the adhesive used to adhere nylon flock to backing fabric had become contaminated with a fungal growth. Knowing that flock fibers are too large to be respirable and unaware of the presence of much smaller respirable-sized nylon particles in the work environment, they initially attributed the disease cluster to suspected mycotoxins, a previously known cause of lung disease. Replacing the adhesive, however, did not prevent new cases. Workforces generally lack systematic medical surveillance, and public health resources for in-depth investigations and long-term follow-up vary by agency and over time.

At the Ontario plant, limited investigation and lack of intervention follow-up delayed recognition of the preventable cause for years.

The reexamination of cause awaited recognition of a new outbreak in Rhode Island. This recognition depended on the vigilance and curiosity of an occupational physician who saw two young patients within 15 months of each other who worked in the same plant, both with interstitial lung disease.⁸ Had interstitial lung disease been common or had this physician not recognized a common place of employment, the opportunity for motivating the company to seek assistance from NIOSH would have been missed. This temporospatial cluster was challenging to NIOSH investigators, whose initial efforts were guided by evaluating causes of hypersensitivity pneumonitis and hampered by the lack of standard methods for identifying the respirable dust found in the plant. But eventually, the dust was found to have the precise melting point of nylon, clinching its identity. Its source was discovered by examining cut ends of flock with a scanning electron microscope. In cutting nylon filament to make flock and subsequently milling the cut flock to break up clumps of flock fibers fused together during cutting, fibrils of respirable size were created and released.⁹ The indicated intervention then was to exhaust key operations so that respirable fragments of nylon could be eliminated from workers' breathing zones. The company was not interested in follow-up of exposures or medical monitoring; thus, as with the Ontario plant, no public health follow-up was done for the Rhode Island plant, and no effective medical surveillance appears to have been implemented.¹⁰ In light of a report of an excess of lung cancer in the Rhode Island cohort,¹⁰ there are two lung disease outcomes for which workers might be monitored.

A third lesson for pulmonary physicians is that identification of a cause or a new occupational disease is not itself sufficient to preclude continued exposure and risk to workers. In the United States, systematic medical surveillance of workers rarely occurs in the absence of regulation by the Occupational Safety and Health Administration. Regulation of hazards is a slow and contested process, even for substances affecting many more workers than are exposed in flock operations and even when a quantitative risk assessment is available to support exposure limits. In the current study by Turcotte et al,⁶ the high incidences of wheeze and pulmonary function changes in unaffected workers in the up to 10-year follow-up in 2001 warrant further follow-up and remind us that we have insensitive tools for diagnosing bronchiolar diseases.^{11,12}

In summary, the discovery of flock worker's lung and its cause is a great success story seldom replicated, despite the likelihood that many occupational lung disease risks remain to be identified. The ingredients

for such a success are vigilance and reporting of sentinel cases by clinical practitioners, carrying out in-depth public health investigations (eg, by involving NIOSH at 1-800-232-2114), maintaining openness to new causes, and following up preventive interventions to evaluate their effectiveness. Ultimately, primary prevention requires elimination or control of causative exposures.

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