

---



# Is Your Patient's Workplace Causing Lung Disease?

Occupational lung diseases not only have a significant health impact on the affected individual, but they often result in workplace changes and significant socio-economic impact.

By Susan M. Tarlo, MB, BS, FRCPC

---

The range and relative frequency of occupational lung diseases has changed significantly in Canada over the past 30 years. Occupational lung diseases that were relatively common before, such as silicosis and coal miners' pneumoconiosis, are now uncommon conditions in Canada. Although silicosis can still be caused by sandblasting and occasionally by other types of exposures, it has become uncommon in Canadian underground miners. This is due to much improved dust-control measures, such as spraying water to keep dust down while drilling and

blasting underground. Conversely, the diagnosis of occupational asthma caused by workplace sensitizers has risen, and this is now the most common chronic occupational lung disease in Canada.<sup>1,2</sup>

It is estimated that occupational asthma (usually due to an immunologic response to a work agent) accounts for about 7% of all adult-onset asthma,<sup>3</sup> and occupational factors may play a role in up to 30% of adult asthma.<sup>4</sup> There has been increased recognition of the role of workplace irritants in aggravating asthma and even, at times, causing asthma due to very high

respiratory irritant exposures (termed reactive airways dysfunction syndrome [RADS], or irritant-induced asthma).<sup>5,6</sup>

Besides causing asthma in some patients, workplace respiratory irritant exposures in accidental high levels (such as nitrogen oxides from silage, or spills of chlorine in chemical plants), can also induce other acute respiratory effects in any part of the respiratory tract.<sup>7</sup> These can include acute respiratory distress syndrome, pneumonitis, bronchiolitis, bronchiolitis organizing pneumonia (BOOP), bronchiectasis, bronchitis, tracheitis, laryngitis and rhinitis.

The effects are determined by several factors, including the odor threshold, solubility and hydrogen ion concentration (pH) of the irritant, the inhaled particle size, the duration and concentration of the exposure, and the presence or absence of underlying respiratory disease. For example, ammonia has a strong odor, which alerts the worker to relatively low exposure conditions, and is water soluble. The effects are, therefore, usually upper airway effects rather than lower airway



Figure 1. Acute pneumonia or hypersensitivity pneumonitis? Bilateral interstitial lung disease in a worker cleaning carrots.

effects. In contrast, nitrogen oxides are odorless and poorly soluble. The effects are less immediately apparent and occur mainly in the bronchoalveolar regions.

Unless a clear exposure history is obtained, the physician may find it difficult to determine whether the individual is suffering from the effects of an irritant or from coincidental non-occupational causes of respiratory disease, such as viral pneumonia or laryngitis.

Another source of respiratory symptoms in the workplace is endotoxin airborne exposure, which can be caused by gram-negative bacteria contaminating organic dusts on farms or in animal feed. Endotoxin exposure also can occur from contaminated cutting oils (i.e., metal working fluids) in factories and humidifiers in buildings.

This exposure can result in such flu-like symptoms as cough, chills, fever,



**Dr. Tarlo is professor of medicine and public health sciences, University of Toronto, and respiratory physician, University Health Network, Gage Occupational and Environmental Health Unit, Toronto, Ontario.**

### Asbestos-related manifestations continue to increase in prevalence due to their long latency period.

malaise and chest pain, which are usually self-limited.<sup>8</sup> Contamination of the same materials with organisms, such as fungi, can cause a similar self-limiting syndrome, or may cause an immunoglobulin E (IgE) antibody-mediated response, lead-

ing to either new-onset asthma or an exacerbation of pre-existing asthma. IgE and immunoglobulin G (IgG) antibody-mediated allergic bronchopulmonary mycoses (*e.g.*, allergic bronchopulmonary aspergillosis) also may present initially as an exacerbation of asthma. Thermophilic actinomycetes or fungi also can cause a Type IV hypersensitivity response (T-lymphocyte-mediated), resulting in hypersensitivity pneumonitis (*e.g.*, farmer's lung), which may clinically mimic pneumonia.<sup>9</sup>

True respiratory infections may have an occupational cause, such as tuberculosis in health-care workers; legionella pneumonia from contaminated workplace humidification systems; anthrax in farmers; and respiratory viral infections spread by close contact among office workers.

Asbestos-related manifestations continue to increase in prevalence due to their long latency period, despite the reduced use of asbestos—especially the reduced use of the most hazardous types of asbestos: crocidolite, amosite and anthophyllite. Although most manifestations,

such as pleural plaques, pleural calcification and mesothelioma, are relatively easy to attribute correctly to asbestos exposure, some manifestations may mimic other lung diseases, (*e.g.*, pleural effusions, interstitial fibrosis and lung cancer). Lung cancer also can be caused by other occupational exposures, such as chromium salts, radon, coke production, arsenic, beryllium and nickel refining.

The above examples illustrate that occupational lung diseases may present in many different forms beyond the more traditional pneumoconioses. Often now, these diseases present to physicians in ways that mimic non-occupational lung diseases. Without an accurate occupational history in all patients with respiratory disease, the correct diagnosis will easily be missed.

The importance of a missed occupational diagnosis can have implications, not only for the individual patient, whose condition may worsen if he/she continues to work in the same conditions, but also for co-workers. Medical screening of other workers after diagnosis of an "index case" may lead to earlier diagnosis and treatment. Awareness of the "index case" and sending notification to the Provincial Ministry of Labour and/or workplace occupational hygienist, as appropriate with the consent of the patient, also may allow preventive measures to be taken in the workplace.

The following cases illustrate the importance of thinking and asking about workplace exposure in all patients, but especially in those with lung disease.

## Case 1

### *Recurrent viral infections, humidifier fever or mass psychogenic illness?*

Eight out of 40 underground nickel miners, seen by the same local family physician, presented with recurrent cough, chest discomfort, difficulty breathing, chills and malaise. These symptoms occurred within a few hours of working underground, and improved away from work within hours. No abnormalities were found on physical examination, chest radiographs or pulmonary function testing, although all tests were performed several hours after leaving the mine. Peak expiratory flow (PEF) readings were slightly reduced underground, but would be expected to fall somewhat due to

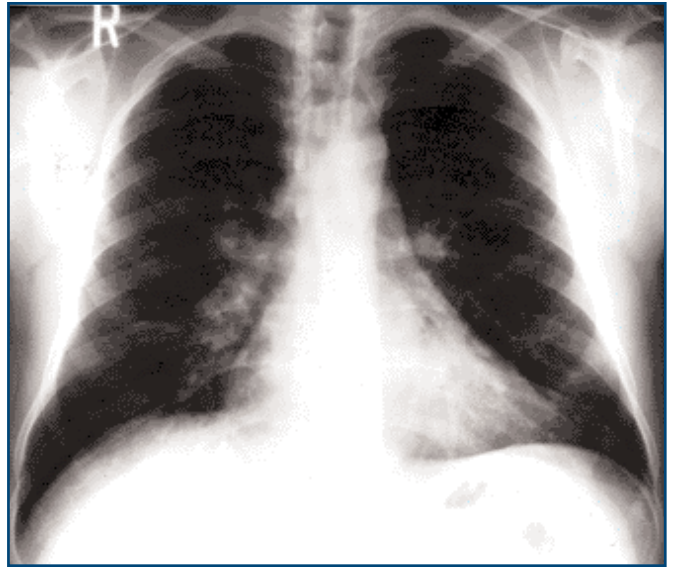


Figure 2. Sarcoidosis or berylliosis? Bilateral hilar lymphadenopathy and right lower lobe infiltrate.

**Without an accurate occupational history of patients with respiratory disease, the correct diagnosis will be missed.**

underground pressure changes. Some miners had a mild neutrophilia on blood counts performed a few hours after mining. The miners attributed their symptoms to diesel fumes underground. Subsequent investigations at the mine showed that water was sprayed underground

to reduce levels of silica and other dust, especially when blasting. The water had been obtained from a tailings pond produced by damming a river. The surrounding area had been used as a sewage dump site, and the water was treated to kill bacteria (but this would not destroy endotoxin). Measured levels of endotoxin in the water sample collect-

ed by the miners were initially elevated, though later samples collected in a sterile manner were normal. Mold counts underground were significantly higher than outdoor levels.<sup>10</sup>

It was considered most likely that symptoms were due to “humidifier fever,” induced by endotoxin and/or mycotoxins, or other fungal irritants, such as beta 1-4 glucans from fungal walls. However, viral infections or mass psychogenic illness could have caused symptoms in some of the miners. Occupational hygiene changes in the mine were associated with resolution of symptoms.

## Case 2

### *Pneumonia or hypersensitivity pneumonitis?*

A 24-year-old-man worked for five years in a plant where carrots and onions were cleaned and packed. He was seen in the

## Lung Disease

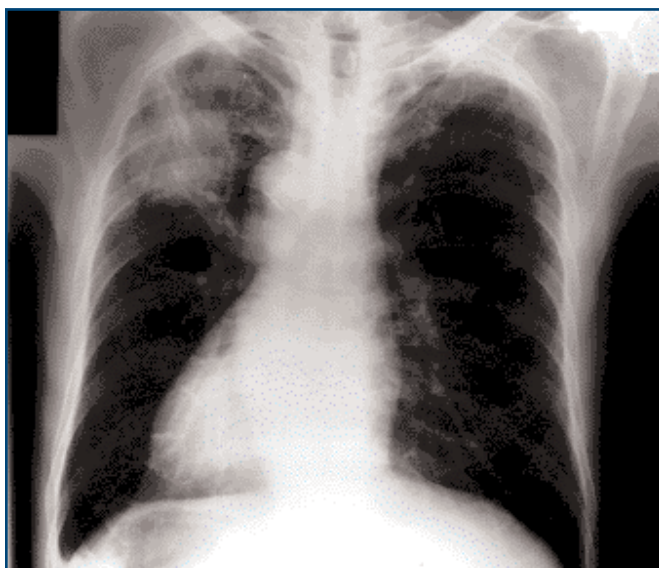


Figure 3. Left upper lobe lung cancer, due to smoking or chromium?



Figure 4. Left upper lobe lung cancer, due to smoking or chromium?

emergency department with a three-day history of dry cough, dyspnea and mild fever. He was found to have severe hypoxia and bilateral infiltrates on his chest radiograph (Figure 1). He was admitted to the intensive care unit and rapidly improved with supplemental oxygen. Five days later, he was discharged after treatment with intravenous fluids and antibiotics, showing a clear chest radiograph. He was readmitted two days later with similar findings. His white blood count was  $27.9 \times 10^3$ , with a marked neutrophilia and leftward shift. He improved without antibiotics, with only supportive treatment (fluids and supplemental oxygen).

Episodes had occurred on both occasions eight hours after working in the onion room, soon after plastic curtains had first been erected to prevent cold air from entering the area. He returned to work in the separate carrot room and had

no symptoms, except when he re-visited the onion room, where symptoms would recur three hours after even short visits. Pulmonary function tests at the time of symptoms showed restrictive changes with reduced diffusing capacity, clearing between episodes. Serum precipitins (IgG antibodies) were identified from the patient against *Aspergillus flavus* and *Aureo pullulans*. Fungal culture from uncleaned onions from the workplace showed a heavy growth of *A. fumigatus*, various penicillin species, and a lighter growth of *A. flavus* and *A. Fusarium* species.



A diagnosis of onion worker's hypersensitivity pneumonitis was made, due to fungal exposure at work. The patient moved to a different workplace. The plastic sheeting at work was removed and the other 40 workers had no ill effect.

### Case 3

#### *Sarcoidosis or beryllium lung disease?*

A 39-year-old woman worked for six years in a company casting and grinding aluminum alloys. Her job was to clean the castings. She presented with a six-month history of dry cough and exertional dyspnea. Chest radiograph showed mediastinal lymphadenopathy and a lower lobe infiltrate (Figure 2). Biopsy showed non-caseating granulomas, and bronchoalveolar lavage showed mainly lymphocytes—especially T cells suggestive of sarcoidosis. The workplace material safety data sheets showed that the aluminum alloys included small amounts of zinc, copper, beryllium and other metals. Her sister, who also worked there, had similar findings.<sup>11</sup> Both were found to have a positive serum beryllium lymphocyte transformation test, supporting a diagnosis of beryllium lung disease. They were removed from further exposure and treated with oral corticosteroids. Other workers underwent a medical surveillance program for berylliosis, as is recommended in Ontario.

Chronic beryllium disease can exactly mimic sarcoidosis. A genetic component has been recognized. Exposure to beryllium may occur in metal working, especially in the manufacture of aerospace components, nuclear energy applications,

electronics, metal extraction, dental alloys and sporting goods, such as golf clubs. If beryllium is suspected as a cause because of the patient's history, then a beryllium lymphocyte transformation test is appropriate. This is performed on blood or bronchoalveolar lavage fluid lymphocytes, and is only available in a few specialized centers.

**Chronic beryllium disease can exactly mimic sarcoidosis.**

### Case 4

#### *Lung cancer: Caused by cigarette smoking or occupation?*

A 60-year-old man died within six weeks after diagnosis of oat cell carcinoma of the lung (Figures 3 and 4). He had a 40-year history of smoking one pack per day. He had worked for 17 years in the aircraft industry with exposure to spray paint, aluminum, lubricants and small amounts of an anticorrosive spray, which contained hexavalent chromium. He had no history of skin rash or nasal septal preparation. Did hexavalent chromium exposure contribute to his lung cancer?

Hexavalent chromium has been recognized as a carcinogen since 1948 on the basis of animal studies and human epidemiologic studies. As with other occupational carcinogens, there is a latent period between exposure and cancer (*i.e.*, the cancer appears many years after the exposure). The risk increases with the extent of expo-

### Early referral to an occupational medicine or respiratory specialist with expertise in occupational lung disease is helpful.

sure (duration and exposure concentration), and the history is essential in assessing this.<sup>12</sup> Cigarette smoking can be synergistic for cancer risk with occupational carcinogens,

and smoking alone increases the relative risk of lung cancer 10- to 100-fold. In calculating the relative risk of an occupational carcinogen contributing to lung cancer one must consider the above factors and published epidemiologic studies.

### Case 5

#### *Occupational asthma or asthma coincidental to the workplace and aggravated by work?*

A 45-year-old man had a history of nasal polyps, but no asthma symptoms until five years after he began working in a rubber tire manufacturing plant. His job involved working with a heated glue containing pine resin. The man described his workplace as dusty and smoky. He said there was no respiratory protection at work, and that he noted an improvement in his symptoms on weekends and during holidays.

Serial PEF readings and spirometry were worse at work, as compared to periods off work. Methacholine challenge while away from work confirmed the presence of airway hyper-responsiveness, but this test could not be repeated within

24 hours of a working day due to a markedly reduced forced expiratory volume in one second (FEV<sub>1</sub>). Specific challenge testing with the glue was positive, further confirming the diagnosis of true occupational asthma due to sensitization to the pine resin in the glue.<sup>13</sup>

He moved to a different workplace and his asthma markedly improved, but did not completely resolve.


### Conclusion

These cases illustrate how occupational lung disease can very closely resemble non-occupational lung diseases. Unless a careful occupational history is taken, the diagnosis will likely be missed.<sup>14</sup> Important aspects of the history's occupational component include:

1. Details of workplace exposure at the time of symptom onset and during symptom exacerbations (especially for diseases, such as asthma, where symptoms relate to exposures in the preceding minutes to hours);
2. Details of previous workplace exposures (especially for conditions, such as lung cancer- or asbestos-related diseases, which have a long latency period);
3. Understanding the job description and exposure by supplementing the history with information from workplace materials safety data (MSDS) sheets, which are available from the employer;
4. For diseases, such as asthma, obtaining a history of the timing of symptoms in relation to workplace expo-

tures *versus* periods off work; and

5. Assessing non-occupational factors in the history, which may contribute to, or completely account for, the respiratory disease (*e.g.*, smoking, atopy, hobbies).

Occupational lung diseases not only have a significant health impact on the affected individual, but they often result in workplace changes and significant socio-economic impact. These patients often will be considered for workers' compensation. Early referral to an occupational medicine or respiratory specialist with expertise in occupational lung disease will help provide an accurate diagnosis and assist the patient in appropriate disease management. This should include decisions about appropriate future workplace conditions and workers' compensation, if applicable. For diseases where there is a potential acute ongoing effect at work, as in the case of asthma and other allergic responses, it is important, where possible, to refer the patient while he/she is still working. This will allow the workplace relationship to be objectively investigated further, as recommended by Canadian guidelines.<sup>14</sup> In addition, diagnosis of occupational lung disease should be considered to be a "sentinel event," which should lead to a review of the risks for similar exposure and disease in co-workers by the provincial Ministry of Labour, or by the workplace physician, as appropriate. 

## Acknowledgements

*The author wishes to thank Dr. Ken Roberts for permitting the report of Case 2, which he astutely diagnosed, and Dr. Gary M. Liss for his helpful review of this article.*

## References

1. Provencher S, Labreche FP, Guire L: Physician based surveillance system for occupational respiratory diseases: the experience of PROPULSE, Quebec, Canada. *Occup Environ Med* 1997; 54:272-6.
2. Liss GM, Ellinson L: Recognition of occupational asthma. *Pract Allergy Immunol* 1994; 9(6):252-60.
3. Blanc P, Toren K: How much asthma can be attributed to occupational factors? *Am J Med* 1999; 107:580-7.
4. Johnson AR, Dimich-Ward HD, Manfreda J, et al: Occupational asthma in adults in six Canadian communities. *Am J Resp Crit Care Med* 2000; 162:2058-62.
5. Brooks SM, Weiss MA, Bernstein IL: Reactive airways dysfunction syndrome (RADS): Persistent asthma syndrome after high level irritant exposure. *Chest* 1995; 88:376-84.
6. Tarlo SM, Broder I: Irritant induced occupational asthma. *Chest* 1989; 96:297-300.
7. Tarlo SM: Workplace respiratory irritants and asthma. In: Banks D, Wong ML (eds.): *Occupational Medicine: State of the Art Review*. Hanley and Belfor, Philadelphia, 2000, pp. 471-83.
8. Clapp WD, Thorne PS, Frees KL, et al: The effects of inhalation of grain dust extract and endotoxin on upper and lower airways. *Chest* 1993; 104:825-30.
9. Pickering CAC, Newman Taylor AJ: Extrinsic allergic bronchioloalveolitis (hypersensitivity pneumonitis). In: Parkes WR (ed.): *Occupational Lung Disorders*. Third Edition. Butterworth-Heinemann Ltd., London, 1994, pp. 667-709.
10. Summerbell RC, Staib F, Ahearn DG, et al: Household hyphomycetes and other indoor fungi. *J Med Vet Mycology* 1994; 32S:277-86.
11. Tarlo SM, Rhee K, Powell E, et al: Marked tachypnoea in siblings with chronic beryllium disease due to copper-beryllium alloy. *Chest* 2001; 119:647-50.
12. Begin R, Christman JW: Detailed occupational history: The cornerstone in diagnosis of asbestos-related lung disease. *Am J Resp Crit Care Med* 2001; 163:598-9.
13. Tarlo SM: Occupational asthma induced by tall oil in the rubber tyre industry. *Clin Exp Allergy* 1991; 22:99-101.
14. Tarlo SM, Boulet LP, Cartier A, et al: Canadian Thoracic Society Guidelines on Occupational Asthma. *Can Resp J* 1998; 5:289-300.