

Mushroom worker's lung: organic dust exposure in the spawning shed

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Two people employed for several years in the spawning shed of a mushroom farm developed mushroom worker's lung. The first patient presented in respiratory failure, with radiological features characteristic of hypersensitivity pneumonitis. The condition of the second patient was subacute on presentation, with a computed tomography (CT) scan showing ground-glass opacities. With absence from the workplace and no steroid therapy, the symptoms of both patients subsided and the results of lung function tests and CT scans improved markedly. (MJA 2007; 186: 472-474)

Clinical record

Two employees of the same mushroom farm presented to our hospital within a 5-month period. The farm is a large commercial producer of *Agaricus bisporus* mushrooms. Both workers were employed in the spawning shed, where mushroom compost is tipped onto a conveyor belt for mushroom spawn (sterilised grain inoculated with mushroom mycelia) distribution. The process is associated with increased levels of ambient organic dust.¹ The principal means of minimising organic dust in the shed was local exhaust ventilation. Neither worker recalled receiving instructions about respiratory protection or the specific hazard of organic dust exposure during their employee-induction process.

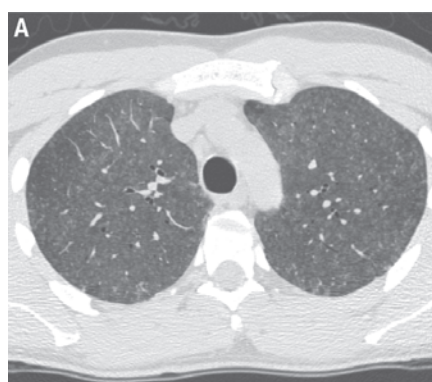
Case 1

A 36-year-old man, who was a non-smoker, had been employed at the mushroom farm for 8 years, and had worked in the spawning shed for 3 years. He described a 4-month history of non-productive cough that was noticeably worse in the afternoons at work and improved on weekends. Two weeks before presenting, he developed daily chills, sweats, myalgia, chest tightness and exertional dyspnoea. His symptoms consistently commenced 5 hours after arriving at work and persisted into the evening at home. They abated sufficiently by morning and over the weekend to allow him to return to work. He had lost 7 kg in weight over 4 weeks.

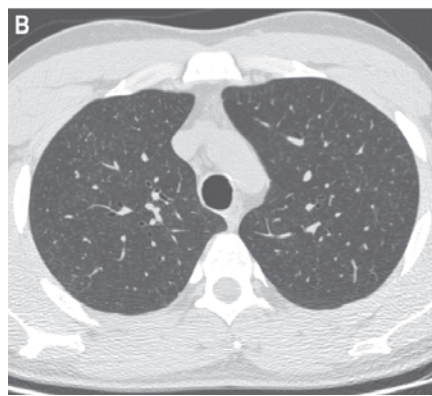
He presented to the emergency department with worsening dyspnoea after a day at work. He was pyretic (38.1°C) and had bilateral basal inspiratory crackles. Measurement of arterial blood gases confirmed hypoxaemia (PaO₂, 57 mmHg; reference range [RR], 80–100 mmHg). Inflammatory markers were elevated: C-reactive protein level, 92 mg/L (RR, <8 mg/L); and erythrocyte sedimentation rate, 17 mm/h (RR, 8–12 mm/h). Results of a full blood examination were within normal limits. Chest x-ray showed a diffuse bilateral reticulonodular infiltrate, and a high resolution computed tomography (HRCT) scan showed changes consistent with hypersensitivity pneumonitis (Box 1).

Mushroom worker's lung was diagnosed and the patient was admitted for observation and oxygen administration. Corticosteroid treatment was not administered. Respiratory function tests showed a borderline restrictive ventilatory defect, with moderately impaired transfer factor for carbon monoxide diffusion (TLCO) (Box 2). During 4 days of observation, there was an improvement in oxygenation, his fever abated, and C-reactive protein levels fell to 15 mg/L. He was discharged home and advised not to return to work.

1 Lung imaging — Patient 1



A: High resolution computed tomography (HRCT) scan of the chest of Patient 1 at presentation showing small, ill-defined centrilobular ground-glass nodules < 5 mm in diameter. Scans of the lower zones (not shown) revealed more confluent areas of ground-glass opacity, without discrete nodules.



B: Repeat HRCT scan performed 1 month later (1 month without workplace exposure) showing significant improvement, but with persistence of tiny centrilobular nodules, particularly in the upper zones. ◆

2 Respiratory function tests — Patient 1

Tests	Normal range	Time since exposure (percentage of mean predicted value)		
		Presentation	1 month	4 months
FEV ₁ (L)	> 3.34	3.24 (76%)	4.50 (107%)	4.54 (108%)
FVC (L)	> 4.27	4.11 (78%)	5.17 (99%)	5.51 (105%)
FEV ₁ /FVC (%)	> 72%	79%	87%	82%
TLCO (mL·min ⁻¹ ·mmHg ⁻¹)	> 30.3	19.8 (53%)	26.8 (72%)	32.7 (88%)
V _A (L)	> 5.8	5.3 (78%)	6.4 (95%)	6.9 (104%)

FEV₁ = forced expiratory volume in 1 second. FVC = forced vital capacity. TLCO = transfer factor for carbon monoxide diffusion. V_A = alveolar volume. ◆

Over the next 4 months, with ongoing avoidance of workplace exposure, ventilatory function, gas transfer and vital capacity improved significantly, and the abnormalities seen on HRCT scan partly resolved, with persistence of tiny centrilobular nodules (Box 1).

Precipitin testing for antibodies to *A. bisporus* was not available. Precipitin testing for antibodies to *Micropolyspora faeni* (a fungus of compost, hay and grain), done at the 1-month review, gave negative results.

Case 2

A 40-year-old man, an ex-smoker, who had worked in the spawning shed for 6 years, had experienced 3 months of non-productive cough, fatigue, exertional dyspnoea and weight loss. His working day in the spawning shed started at 6 am, with onset of symptoms usually occurring around midday.

He presented on two occasions to another hospital after a full day's work complaining of dyspnoea, cough, chest tightness, myalgias and fever. At the first of these two presentations, resting SpO₂ (oxygen saturation measured by pulse oximetry) was mildly reduced at 93%. Results of a full blood examination showed neutrophilia ($9.4 \times 10^9/L$; RR, $2.0\text{--}8.0 \times 10^9/L$), and the C-reactive protein level was 13.5 mg/L. No abnormalities were seen on chest x-ray. He was diagnosed with a respiratory tract infection, prescribed antibiotics and discharged. His symptoms abated during a period of sick leave, but recurred 2 hours after returning to work in the spawning shed.

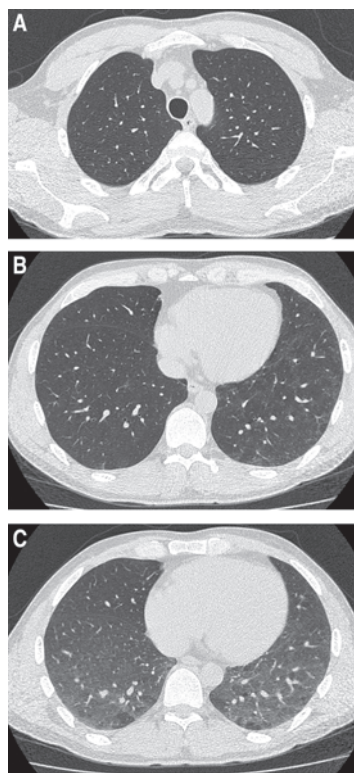
On presenting to our outpatient clinic, 9 days after his last work exposure, he reported that his cough and fever had abated, but exertional dyspnoea persisted. Spirometry tests showed no abnormality, but TLCO was impaired at $22.8 \text{ mL}\cdot\text{min}^{-1}\cdot\text{mmHg}^{-1}$ (58% predicted). Serum precipitin testing for *M. faeni* gave negative results. HRCT scan of the chest showed subsegmental air-trapping on expiratory scans and subtle patchy ground-glass opacities in both lower lobes (Box 3).

After 4 months of avoiding further exposure, he noted no recurrence of symptoms, slow improvement in exercise tolerance, and improved gas transfer.

Discussion

Mushroom cultivation in Australia is a large agricultural industry, employing over 2500 people,² yet mushroom worker's lung has not previously been reported in the Australian medical literature, nor to an occupational lung disease notification scheme.³ It is likely that there is considerable under-recognition of this condition, as it is estimated that 5%–15% of those exposed to the causative antigens may develop hypersensitivity pneumonitis.⁴ In the largest cross-sectional study of workers at an *Agaricus* mushroom farm, 20% of those heavily exposed to organic dust reported experiencing symptoms consistent with mushroom worker's lung.⁵

3 Lung imaging — Patient 2



High resolution computed tomography scan of Patient 2 at presentation, showing normal upper lobes (A) and patchy, centrilobular ground-glass opacities, with expiratory subsegmental air-trapping at the lung bases (B, C). This is a non-specific pattern, compatible with hypersensitivity pneumonitis. ♦

Unfortunately, with no thorough epidemiological studies, specifically cohort studies, it is not possible to estimate the true incidence of respiratory disease in mushroom farm workers.

Several outbreaks of mushroom worker's lung have been reported in the international literature since the 1950s. Workers with high exposure to organic dust from mushroom compost, such as spawners and compost handlers, are commonly affected and hence the more specific term "mushroom compost worker's lung" is occasionally used.^{1,6} In this form of mushroom worker's lung, *M. faeni* (currently known as *Saccharopolyspora rectivirgula* of the class Thermoactinomycetes), which is present in mushroom compost, is the most commonly implicated allergen.^{1,5,7} Organic dust from mushroom compost consists of a vast array of microorganisms and organic antigens; failure to demonstrate precipitins to *M. faeni*, as in the patients reported here, does not exclude the diagnosis of mushroom worker's lung.^{1,4,8}

Japanese mushroom farm workers have been reported to suffer an alarmingly high incidence of allergic respiratory disease.^{9,10} In a 3-year follow-up study by Tanaka et al, 40% of workers left the industry due to intolerable respiratory symptoms.¹⁰ Japanese mushroom varieties such as *Hypsizygus marmoratus* (Bunashimeji) are grown on wet wood dust rather than compost and release billions of spores 4–6 µm in diameter before being harvested.^{10,11} The inhaled mushroom spore (rather than Thermoactinomycetes) is the causative allergen in this setting, with the term "mushroom picker's lung" used to more accurately describe the group of workers at risk of this form of mushroom worker's lung.¹¹ Although commercial cultivation of "exotic" mushrooms in Australia is small (1000

tonnes per year compared with 52 250 tonnes of *Agaricus* mushrooms), as demand for and cultivation of these mushrooms increases, employers must be aware of the significant hazard posed by these varieties when developing safe work practices.²

The most important component of identifying hypersensitivity pneumonitis is recognition of exposure to a causative antigen, reinforcing the importance of a thorough occupational history, and identification of workplace hazards (Box 4).^{7,12} A temporal relationship between the development of symptoms (cough, fever, chills, dyspnoea, chest tightness and malaise) 4–8 hours after the start of exposure, and an improvement during weekends or vacations, is quite indicative of this condition.^{4,8} Organic dust toxic syndrome, a form of inhalation fever, may be difficult to differentiate from acute hypersensitivity pneumonitis and is estimated to be 30–50 times more common.¹² Organic dust toxic syndrome may result from a single heavy exposure to organic dust, and is self-limiting, with symptoms rarely exceeding 36 hours.⁸

Optimal management of hypersensitivity pneumonitis requires early recognition and complete avoidance of further exposure to the causative antigen,^{8,7,12} a change of occupation may be necessary. Although corticosteroid therapy has been shown to result in more

rapid improvement in lung function and may be warranted in severely unwell patients, it has not been shown to improve long-term outcomes.⁹ Recurrence of acute hypersensitivity pneumonitis is more common in patients treated with steroids; this may be due to their improved sense of wellbeing and less stringent adherence to antigen avoidance.^{8,13}

The natural history of hypersensitivity pneumonitis has been poorly described, primarily due to a lack of longitudinal studies.⁸ With repeated acute or chronic low-level exposure in farmer's lung, permanent lung damage caused by pulmonary fibrosis and emphysema has been shown to occur, with associated chronic dyspnoea and permanent impairment.^{8,9} Even patients who remain asymptomatic may have long-term physiological sequelae.⁸

Australian occupational health and safety legislation describes in broad terms employers' responsibilities to ensure every reasonable action is taken to preserve the health and safety of workers. Obligations to control hazardous non-organic substances, such as isocyanates and silica, are further described by subordinate Occupational Health and Safety (Hazardous Substances) Regulations (Vic) and the accompanying Hazardous Substances Code of Practice. Despite organic dust clearly having the potential to harm human health, the requirement to control organic dust falls outside the domain of hazardous substance legislation in Australia. Therefore, for their duty of care to be discharged, employers in the agricultural sector must demonstrate due diligence in their identification and control of all workplace hazards, including organic dust.

The National Occupational Health and Safety Commission (now known as the Australian Safety and Compensation Council) has established limits for some organic dusts, such as cotton.¹⁴ However, organic dust in most agricultural settings is a complex and variable mixture of constituents, impairing the ability to set useful standards.⁸ Episodic high concentrations of dust exposure, rather than static ambient levels, may precipitate respiratory diseases, further increasing the difficulty of determining "safe" exposure standards. These factors impair our ability to advise employers how best to control this hazard. It is also difficult to determine what can reasonably be expected of employers as far as monitoring is concerned. Urgent research has been called for in this area by the American Thoracic Society.⁸

Employers in agricultural industries should demonstrate awareness of the hazard of organic dust, and aim to reduce exposure levels using the "as low as reasonably practicable" (ALARP) principle. Mushroom farm workers specifically should be educated about the risk of developing hypersensitivity pneumonitis and be advised of the symptoms and warning signs.⁵

4 Occupational causes of hypersensitivity pneumonitis — disease and source of exposure^{9,12}

Farmer's lung: mouldy hay, grain; compost

Bagassosis: mouldy sugarcane

Mushroom worker's lung: mushroom compost, mushroom spores

Ventilation pneumonitis: humidifier, air conditioner

Machine operator's lung: contaminated metal working fluids

Humidifier lung: ultrasonic cool-mist humidifiers

Floor finisher's lung: mouldy wood floors

Malt worker's lung: mouldy malt dust (brewing)

Compost lung: compost

Tobacco worker's lung: mouldy tobacco

Sequoiosis: contaminated red-wood dust

Wood worker's lung: mouldy wood dust

Wood trimmer's disease: mouldy wood trimmings

Wine grower's lung: mouldy grapes

Suberosis: mouldy cork dust

Cheese worker/washer's lung: cheese mould

Salami worker's lung: salami seasoning

Saxophonist's lung: mouldy saxophone reed

Bird fancier/breeder/handler's lung: pigeon, duck, chicken, turkey, parrot

Furrier's lung: cat hair, fur dust

Laboratory worker's lung: laboratory rat or gerbil urine

Oyster shell lung: shell dust

Tobacco grower's lung: tobacco dust

Coffee worker's lung: coffee bean dust

Tea grower's/worker's lung: tea leaves

Streptomyces hypersensitivity pneumonitis: contaminated fertiliser

Detergent worker's disease: detergent ◆

Competing interests

None identified.

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