

Respiratory illness in agricultural workers

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Respiratory diseases have long been recognized in association with work in farming. Overall, only a small proportion of the population is employed in agriculture, so respiratory disease in farmers is not a major public health issue. However, farmers are known to have high morbidity and mortality from certain respiratory diseases, as shown by routinely collected statistics. Despite this, knowledge of the frequency, nature and risk factors for some respiratory disorders in agricultural workers is incomplete. Multiple exposures are common and some exposures can give rise to more than one specific disease. Moreover, the most common respiratory symptoms reported by farm workers (wheeze, dyspnoea and cough) are relatively non-specific and can be associated with several occupational respiratory disorders. This review describes the main occupational respiratory illnesses in farming and summarizes the current literature about epidemiology and prevention. The most important diseases are rhinitis and asthma, which, although common, are not usually fatal. Some non-allergic conditions, e.g. asthma-like syndrome and organic toxic dust syndrome, are not yet fully understood, but appear to be common among farm workers. The most serious respiratory diseases are hypersensitivity pneumonitis and respiratory infections, but these are rare. Most importantly, respiratory diseases are preventable by controlling harmful exposures to organic dust, toxic gases and chemicals on farms through improvements in animal rearing techniques, ventilation of animal accommodation, careful drying and storage of animal feed-stuffs, crops and other products, and use of personal protective equipment.

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Introduction

Respiratory disease is a well-recognized occupational problem among agricultural workers. Routinely collected statistics suggest that farmers have a higher morbidity and mortality from certain respiratory disorders than the general population [1–3] and other occupational groups [4], despite a lower prevalence of smoking. The Health & Safety Commission (HSC) has made agriculture one of the priority programmes set out in its *Strategic Plan for 2001–2004* [5], in part due to a relatively high incidence

of asthma in the industry, as highlighted in the HSC's *Health and Safety Statistics 2000/01* [4].

Although respiratory hazards in agriculture have been recognized for many centuries, a number of factors make it difficult to assess the size and nature of the associated risks in the UK. One problem is that much of the agricultural workforce in Britain is employed as casual labour and study populations can be difficult to define. Another factor is the wide variety of respiratory hazards (summarized in Table 1) and the potential for complex co-exposures in the same agricultural workplace. Working environments and techniques can vary markedly between farms and exposure can also be affected by climate, which influences infestation of crops or animal feed and pesticide use. Many of the epidemiological data on respiratory disease and exposure levels among agricultural workers

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Table 1. Respiratory hazards in farming

Organic dusts (grain, straw, hay)
• Moulds and spores
• Bacteria
• Mites and their excreta
• Animal dander
• Animal urine and faeces
• Animal feeds
Inorganic dusts
• Low molecular weight minerals (e.g. silicates)
Chemicals
• Pesticides
• Fertilizers
• Paints
• Preservatives
• Disinfectants
Gases and fumes
• Slurry (ammonia, methane, hydrogen sulphide, carbon dioxide)
• Silage (nitrogen dioxide, carbon dioxide)
• Welding fumes (oxides of nitrogen, ozone, metals)
• Engine exhaust fumes (oxides of nitrogen, particulate matter)
Infectious agents
• Bovine tuberculosis (<i>Mycobacterium bovis</i>)
• Psittacosis (<i>Chlamydia psittaci</i>)
• Q fever (<i>Coxiella burnetii</i>)

come from North America and elsewhere in Europe and care must be taken in extrapolating findings from studies in other parts of the world. Finally, aetiological mechanisms for occupational respiratory disease in farmers are incompletely understood. Whilst some conditions are well defined, the specific occupational cause and precise patho-physiological mechanisms for others have not yet been unravelled.

Despite these problems, several surveys have indicated that the prevalence of work-related respiratory symptoms such as wheeze, cough and dyspnoea is unusually high among farmers (23–50%) [6–8]. In general, these symptoms are non-specific and could arise from more than one disease. This review describes the main occupational respiratory illnesses that can give rise to symptoms in agricultural workers and outlines exposures known or suspected to be involved in the disease processes. Specific preventive measures are covered under each disease heading, but general control measures that are applicable to a range of respiratory disorders are addressed in a separate section.

Rhinitis

Of the well-defined respiratory disorders associated with farming, rhinitis is probably the most common. Grain farming, livestock breeding, dairy farming, and flax and hemp processing have all been associated with increased nasal symptoms [9]. Many type 1 allergens are present in these settings, and those that have been associated with allergic rhinitis in agricultural workers are shown in

Table 2. Case reports of allergic respiratory disease in farmers

Agent	Allergic disease	Reference
Hay	Asthma	[10]
Grain dust		
Moulds:	Asthma	[11]
<i>Alternaria tenuis</i>		
<i>Cladosporium herbarum</i>		
<i>Sporobolomyces</i>		
<i>Didymella exitialis</i>		[12]
Storage mites:	Rhinitis, asthma	
<i>Glycyphagus destructor</i>		[13]
<i>Glycyphagus domesticus</i>		[14]
<i>Tyrophagus longior</i>		[15]
<i>Tyrophagus putrescentiae</i>		[16]
<i>Acarus farris</i>		[17]
<i>Acarus siro</i>		[18]
Animal proteins		
Cow urine and dander	Asthma	[19]
Cow dander	Rhinitis	[16,20]
Pig urine	Asthma	[21]

Table 2. In addition, several components of organic dusts and some disinfectants and pesticides are irritants, and can cause non-allergic upper airways inflammation.

Figures from the National Occupational Disease Register in Finland show a general increase in numbers of reported cases of occupational allergic rhinitis from 1981 to 1991. Cow epithelium and storage mites have shown increased importance as causal agents during that time period, and were the first and third most common causes, respectively, of reported cases of occupational rhinitis in Finland in 1991 [22]. The prevalence of self-reported nasal allergies (including hay fever) among farmers in mainland European countries has been found to be lower than estimates in the general population [6,23]. However, there are few reliable estimates of the prevalence or incidence of rhinitis specifically related to occupation. Nasal irritation was the most common work-related respiratory symptom (prevalence 23%) reported by farmers in Northern Germany [7]. In a study of US grain handlers, the prevalence of nasal symptoms was 64% and the prevalence of sensitization to grain dust antigens (defined by a positive specific skin test) was 2–18% [24]. Among very few studies which include a comparison group, one [6] reported a higher prevalence of work-related nasal symptoms in farmers (6.5%) than in healthy urban dwellers (0%), while another [8] found similar rates of nasal irritation (22–25%) among pig and dairy farmers and greenhouse workers. Whilst rhinitis is common, it is not generally considered to be a serious condition, although the development of immunological sensitization is associated with an increased risk of asthma [25].

The primary prevention of rhinitis is by exposure control, as described later in this review. Once sensitized, farm workers should avoid exposure if possible, or utilize

respiratory protective equipment to undertake tasks that are associated with particularly high exposure to organic dusts.

Asthma

Asthma can be induced or exacerbated by the same components of organic dusts that cause allergic rhinitis. There is some overlap between rhinitis and asthma symptoms in farmers, although rhinitis is approximately twice as common [25]. Of the agents shown in Table 2, storage mites, cow dander and urinary proteins are the most common causes of occupational asthma in farmers.

Data from the Surveillance of Work Related Respiratory Disease (SWORD project) give an indication of the frequency of asthma compared with other reported respiratory disorders in farmers in the UK. When cases of occupational respiratory disease of short latency reported by chest physicians in 1997/1998 were classified by occupation, asthma was the second most common diagnosis in farmers, comprising 17 of 38 notifications [26]. According to the Swedish Register of Occupational Disease for 1990–1992 [27], the self-reported rate of occupational asthma was high in male farmers—179 cases per million per year—compared with a crude annual rate of 80 cases per million per year. Female dairy and poultry farm workers had the second highest rate of self-reported occupational asthma by occupational group among Swedish women [27]. Asthma is not a major cause of mortality in farmers. The proportional mortality rate (PMR) for asthma in UK farmers is not significantly elevated [2], but this is not surprising since asthma is a common disease in the general population. In Sweden, the standardized mortality ratio (SMR) for asthma among farmers was 137 [3].

Most of the available epidemiological information about occupational asthma in farmers comes from cross-sectional surveys, which will tend to underestimate the problem due to self-selection of affected individuals out of the industry. Prevalence data from various farming populations are shown in Table 3. Risk estimates depend on exposure and on the outcome measure (symptoms, physician diagnosis, bronchial challenge testing, RAST or skin prick test). However, the prevalence of asthma diagnosed by a doctor or treated with medication has generally been ~3–10%. Very few studies give comparative figures from the general population or other working groups. However, in the European Community Respiratory Health Survey [37], information was collected from random samples of young adults from the general population. When the risk of asthma (defined as bronchial hyperresponsiveness and asthma symptoms or medication) was analysed by occupation, the highest risk was shown for farmers [odds ratio (OR) = 2.62, 95% confidence interval (CI) = 1.29–5.35]. This important

finding was consistent across nearly all the European countries in the study.

The main means of preventing occupational asthma in agricultural workers is through reducing exposure to organic dusts and other allergens. General measures to reduce allergen exposure are discussed later. As with other individuals who develop type 1 allergy, sensitized farmers should aim to avoid future contact with the specific allergen. However, because many farm workers are self-employed, they are often unwilling to consider a change of occupation. In these circumstances, respiratory protective equipment can be used as a last resort to enable farmers to continue working.

Extrinsic allergic alveolitis (hypersensitivity pneumonitis)

Extrinsic allergic alveolitis is characterized by lymphocytic interstitial pneumonitis. It occurs in response to a variety of antigenic agents, including thermophilic *Actinomyces* spp. and certain *Aspergillus* fungi, particularly *A. fumigatus* and *A. umbrosus*. The most common form of the disease in farmers—farmers' hypersensitivity pneumonitis (FHP), or farmers' lung—is associated with exposure to *Saccharopolyspora rectivirgula*. All of these organisms can be found in mouldy hay, grain or straw dust. Symptoms are most prevalent in cold wet climates that favour fungal overgrowth and in the winter months when stored crops are used for animal feed. The aetiology of FHP is not fully understood. A recent review stated that, in addition to *S. rectivirgula*, exposure to unspecified cofactors was likely to be required to induce the disease [9]. This conclusion was based on evidence from animal models in which the lung response to *S. rectivirgula* was heightened in the presence of adjuvants (e.g. endotoxin, or previous infection with Sendai virus) and on a greater observed response in humans exposed to hay dust compared with simple *S. rectivirgula* extracts.

There are several distinct patterns of disease. The acute form is characterized by episodes of breathlessness, cough, fever and chills that occur several hours after exposure (typically in the late afternoon or evening) and subside within a few days. Other presentations include a subacute form (with gradual onset of dyspnoea over weeks or months), recurrent acute attacks and chronic productive cough.

The pathogenesis of FHP is complex. An allergic mechanism is most likely, but the disease does not fit neatly with one of the four types of allergic reaction. The presence of precipitating antibodies, immune complexes (suggestive of a type III reaction) and granuloma formation (more in keeping with a cell-mediated type IV reaction) have all been well documented. Lymphocytic infiltration of the alveoli is typical of the acute phase of the disease, but, with progression, fibrotic changes are seen

Table 3. Epidemiological surveys of occupational asthma in agricultural workers

<i>Ref.</i>	<i>Country</i>	<i>Year of study</i>	<i>Population</i>	<i>Outcome measure</i>	<i>Prevalence (%)</i>
[15]	Scotland	NS	Cattle farmers	Self-reported asthma RAST test and SPT for storage mites	Asthma = 15 Pos. RAST test = 17.4 Pos. SPT = 37.2
[28]	Finland	1980	Urban versus rural (farming) inhabitants	Diagnosed asthma Periodic wheeze	Diagnosed asthma = 4.1 (urban), 2.7 (rural) Periodic wheeze = 15.2 (urban), 17.5 (rural)
[29]	Denmark	NS	Farmers	Self-reported asthma Asthma medication Asthmatic symptoms	Asthma = 7.7 (pig farmers = 32) Medication = 3.5 SOB = 13.7, wheeze = 16.3, cough = 22.1
[30]	France	1992	Farmers, farm workers (retired)	Current asthma Lifetime asthma	Asthma (current) = 5.9 (OR = 5.35, 95% CI = 1.33–21.50) Asthma (lifetime) = 9.3 (OR = 2.3, 95% CI = 1–5.47)
[31]	Sweden	1994	Farmers	Physician-diagnosed asthma RAST test for common airway allergens, storage mites, cow epithelium	Diagnosed asthma = 10.5 (general population in region = 6) One-third of asthmatic farmers had pos. RAST test to any one allergen
[32]	New Zealand	NS	Farmers, farm workers	Wheeze, BHR	OR for prevalence of wheeze = 4.27 (95% CI = 1.28–14.29)
[33]	Norway	1991	Farmers and spouses	Self-reported asthma	Asthma (lifetime) = 6.3
[34]	France	1994	Dairy farmers	Self-reported asthma Physician-diagnosed asthma	Asthma = 5.3 Physician-diagnosed asthma = 4.9
[35]	Denmark	1992–1994	Farming students	Self-reported asthma, BHR	Asthma = 5.4–21.0 ^a ; asthma and BHR = 15.4–50.0 ^a
[36]	Denmark, Germany, Switzerland, Spain	1995–1997	Cattle, pig, poultry and sheep farmers	Self-reported asthma, asthmatic symptoms	Asthma = 2.8, SOB = 5.1, wheeze = 14.1

NS, not stated; OR, odds ratio; BHR, bronchial hyperresponsiveness; SOB, shortness of breath; SPT, skin prick test; RAST, radioallergosorbent test; pos., positive.

^aRange according to smoking status and gender.

on X-ray. Among studies that have followed up farmers with FHP, 30% of a group of cases in Orkney developed further acute attacks over 10 years [38] and 25% of a group of cases in the USA (with an average disease duration of 14 years) described shortness of breath when walking with people of their own age [39].

Despite a large number of epidemiological studies of this disease, it has been difficult to assess the extent of the problem. There is a lack of consistency in case ascertainment and prevalence estimates vary according to whether cases are defined by symptoms alone, by tests for precipitating antibodies in serum or by more sophisticated investigations, including chest X-ray and broncho-

alveolar lavage. Prevalence surveys based solely on symptoms are likely to overestimate the problem, since the symptoms can be relatively non-specific and some misdiagnosis of toxic febrile reactions will occur. On the other hand, surveys based on modern imaging and biopsy or lavage specimens are likely to lead to underestimation, since milder cases are unlikely to be admitted to hospital for investigation or treatment.

In the early 1970s, a pilot study of farmers in Scotland found a prevalence rate of 86 cases per 1000 of the working population in some regions [40]. The incidence of acute cases in Swedish farmers has been estimated as 2–5 per 10 000 farmers per year [41,42]. According to

SWORD, allergic alveolitis is the most commonly reported respiratory disorder in UK farmers, comprising 20 out of 38 notifications in farmers in 1997 [26]. By far the highest annual rates of this disease reported to SWORD (1998–2000) were in farming or veterinary jobs [4]. Because of the inconsistencies in research definitions and the general lack of incidence data, it is difficult to identify any clear trends in the incidence of disease with time. Chronic disease can be fatal, but deaths are rare. Only 56 deaths were recorded in the UK over an 11 year period between 1979–1980 and 1982–1990 [2].

Acute extrinsic allergic alveolitis can be treated with low dose oral steroids and this has been found to be as effective as avoidance of exposure. Specific preventive measures include adequate drying and conditioning of crops prior to storage, sufficient ventilation in storage facilities and use of commercial anti-mould preparations. These last contain acid-forming bacteria (commonly *Lactobacillus* spp.), the products of which inhibit mould growth. General measures to reduce organic dust levels are mentioned later. Avoidance of allergens is usually recommended to prevent chronic lung damage, although some studies have shown that those who experience the disease and remain in farming have comparable long-term pulmonary function to those who leave the industry [43]. Thus, change of occupation may not be necessary, although affected workers should be encouraged to avoid certain activities (e.g. cleaning barns, silos, grain bins, etc.) to minimize contact with mouldy hay, grain or straw and to use respirators for high-risk activities.

Organic dust toxic syndrome (ODTS)

The syndrome known as ODTS is an acute inflammatory reaction in the airways and alveoli. Although the main features of aetiology and pathology have been defined, the precise disease mechanisms remain unclear. It is likely that more than one agent can give rise to the syndrome and it is possible that ODTS represents a group of disorders. The symptoms (breathlessness, fever, cough and malaise occurring 4–6 h after exposure to organic dusts) resemble the acute form of extrinsic allergic alveolitis. However, in general, the individual recovers quickly from ODTS (usually within 36 h) without the need for treatment. Transient decrements in forced expiratory volume in 1 s (FEV1) [44,45], vital capacity (FVC) [44,46] and peak expiratory flow [47,48] have been observed in symptomatic cases, but unlike extrinsic allergic alveolitis, there is no progressive impairment in lung function.

The local inflammatory response in ODTS occurs in the absence of immunological hypersensitivity and is believed to be a toxic reaction. This is supported by clustering of cases where exposures have been very high (e.g. removing mouldy material from silos), including

individuals who have not been exposed previously [46–48]. The agents best documented to be associated with ODTS are endotoxins. These lipopolysaccharide protein complexes are released from Gram-negative bacteria, which are present in mouldy vegetable material. However, the role of other agents found in organic dusts is still debated.

ODTS appears to be very common in farmers. Because of its self-limiting nature, ODTS does not feature in routinely collected statistics and most information about the scale of the problem comes from epidemiological surveys in farming populations. Among Swedish farmers, the lifetime prevalence of ODTS has been estimated at 6% [49] and the annual incidence at 1% [42]. A study from The Netherlands [50] using a symptom questionnaire found a lifetime prevalence of ODTS among pig farmers of ~6%, compared with 2.6% among non-farming controls from the same rural community. The authors pointed out that, although the controls did not work in agriculture, some of them may have worked in occupations possibly associated with febrile reactions. The occurrence of ODTS is likely to vary according to climatic and other conditions, and some studies have reported higher lifetime prevalences (10–15%) for fever associated with farming activity [42,51,52]. Since these estimates are based on reported symptoms, they are likely to include some cases of hypersensitivity pneumonitis. However, because FHP is rare, it is unlikely to distort the prevalence estimates for ODTS by more than a small amount.

The methods of preventing ODTS are the same as for hypersensitivity pneumonitis. Education of farmers is crucial. Because of its link to heavy exposures, clusters of ODTS should alert farmers to poor practice and the need for local improvements in dust control.

Asthma-like syndrome/acute non-allergic respiratory symptoms

Inhalation of organic dust in the agricultural environment may also give rise to another non-allergic respiratory response, which has been described as 'asthma-like syndrome'. The main clinical feature is wheeze and breathlessness that tends to improve as the working week progresses. The prevalence of symptoms in agricultural populations has been reported in a few studies from Europe [53,54] and is of the order of 20–50%. Acute cross-shift reductions in FEV1 (usually ~10% or less) have been observed among both grain and livestock workers [55–57]. The distinguishing feature from asthma is the absence of persistent airways hyper-responsiveness. Grain and cotton dust are the most well-established causes, but ammonia and endotoxins have also been implicated. Increasingly, studies have used personal sampling methods in order to assess exposures [55–57].

Results indicating a significant dose-response relationship between symptoms and dust, ammonia and endotoxins have been reported [56].

Chronic obstructive airways disease

It is not clear whether chronic obstructive airways disease should be regarded as an occupational disease of farmers. Agricultural exposures to organic and inorganic dusts, gases and fumes can cause irritation and inflammation of the airways. In theory, prolonged exposure might lead to chronic airflow limitation through airway obstruction or loss of elastic recoil in damaged parenchyma. Cross-sectional surveys [58–63] using standardized symptom questionnaires (Medical Research Council, American Thoracic Society) to ascertain chronic bronchitis have found prevalence rates of 7–26% among various farming populations. In some studies [58,59], symptom rates have been higher among farmers who work with confined animals as compared with farmers using non-confinement methods or non-farming controls, but others [60] have found similar symptom rates in all groups. In studies that adjusted for smoking status, some [61,63] found an increased rate of symptoms of chronic bronchitis in farmers. The association between work in farming and impaired (obstructive) lung function has been variable. Several studies [58,60,64] have found no significant reduction of lung function in farming populations compared with controls, despite increased symptom rates. Overall, there is no excess mortality from chronic bronchitis in farmers compared with the rest of the working population in the UK, although this is likely to be confounded by a low prevalence of smoking in farmers [2].

Toxic gas inhalation

High concentrations of oxides of nitrogen are produced by decomposing organic matter in freshly filled silos and can cause severe but self-limiting respiratory irritation (silo-filler's disease). Acute exposure to extremely high levels can cause pulmonary oedema and bronchiolitis obliterans and is sometimes fatal [65]. High levels of other toxic gases, such as ammonia (NH_3) and hydrogen sulphide (H_2S), are generated from liquid manure storage pits and the growth in high-density animal farming has led to an increased risk of exposure to these gases among livestock workers. In the UK, occupational exposure standards (OES) of 25 and 10 p.p.m. (8 h time-weighted average) have been set for ammonia and hydrogen sulphide, respectively, although a recent study of poultry workers [56] found that concentrations of 12 p.p.m. of ammonia were associated with a significant decrease in lung function and the authors recommended that new exposure limits should be set.

Occupational measures to safeguard workers from noxious gas inhalation include education about asphyxiation accidents, engineering controls such as storing silage and manure in leak-proof structures, and routine monitoring of gas levels in silos and animal confinement units. Good hygiene in animal houses, including reduction of the number of animals per unit area, increasing frequency of manure removal and use of litter materials that limit ammonia release, can help reduce long-term exposures.

Respiratory infections

Zoonoses are a rare cause of respiratory morbidity in farmers. In the UK, bovine tuberculosis (TB) is the most important. The number of infected cattle in the UK has risen in recent years [66], although the number of humans contracting bovine TB is low. In 1999, 40 human cases were notified [67], but these were thought to be related to the consumption of unpasteurized milk. Other agricultural zoonoses include psittacosis and Q fever. Psittacosis follows inhalation of desiccated droppings or secretions of poultry infected with *Chlamydia psittaci*. Q fever, caused by inhalation of *Coxiella burnetii* from infected cattle, goats and sheep, can result in pneumonitis, and up to 20% of the dairy herd in England and Wales may be infected [67]. Both conditions are uncommon, with 230 laboratory-confirmed human cases of psittacosis and 124 cases of Q fever reported throughout the UK in 1999 for all occupations [67]. However, SWORD data suggest that respiratory zoonoses occur mainly among food processing operators and veterinary practitioners, with no reported cases in farmers in 1997 [27].

Good occupational and personal hygiene practices, which aim to control diseases among livestock and therefore reduce the risk of transmission to humans, are essential. Herd surveillance and animal vaccination, if available, may also contribute to reducing human exposure.

General preventive measures

Among the respiratory disorders described, several have common agricultural exposures and a similar approach to risk reduction is appropriate. General preventive strategies have focused upon reducing the generation of dusts and gases in farms, and improving methods of storage and ventilation to reduce the growth of moulds, fungi and bacteria. A recent study [57] demonstrated that workers in animal houses that were equipped with humidity sensors and automatic ventilation achieved significantly higher lung function results than those workers in buildings with poor ventilation control. The use of tractors and combine harvesters that have enclosed

cabs equipped with air filtration can reduce inhalable grain dust levels. Other measures include spraying enclosed areas to prevent dust from rising, adding dust suppressants (e.g. molasses) to animal feed, using automatic feeding systems, installing extractor fans and industrial vacuums to remove dust, and covering or screening equipment used in the transfer or storage of grain.

Conclusion

Farming is associated with a variety of respiratory hazards, and respiratory symptoms are highly prevalent among farmers. In the UK, respiratory disease in farming is not likely to be important in public health terms, since only a small proportion of the population is employed in the industry. However, occupational respiratory disease does represent an important cause of morbidity among farmers, as shown by routinely collected statistics in the UK and elsewhere. The most important diseases are asthma and rhinitis, but although common, these are not generally life-threatening. The precise mechanisms for a considerable proportion of respiratory symptoms are unclear, but in general the contribution of type 1 allergy to respiratory symptoms in farmers is modest [9]. Some non-allergic conditions, including ODS and asthma-like syndrome, appear to be very common in farmers, but are not yet fully understood. The most serious diseases include hypersensitivity pneumonitis and respiratory infections, but these are rare. Most importantly, these conditions are preventable by practical measures to reduce harmful exposures. Careful investigation of work-related symptoms is necessary to establish a correct diagnosis, since respiratory conditions often exhibit similar non-specific symptoms. Finally, improving knowledge among farm workers and raising diagnostic awareness in hospital physicians and general practitioners in rural areas are key components in addressing the problem of respiratory disease in the farming industry.

References

- Heller RF, Kelson MC. Respiratory disease mortality in agricultural workers in eight member countries of the European Community. *Int J Epidemiol* 1982; **11**: 170–174.
- Office of Population Censuses and Surveys, Health & Safety Executive. *Occupational Health Decennial Supplement*. London: HMSO, 1995.
- Toren K, Horte L-G, Jarvholm B. Occupation and smoking adjusted mortality due to asthma among Swedish men. *Br J Ind Med* 1991; **48**: 323–326.
- Health & Safety Commission. *Health and Safety Statistics 2000/01*. HSE Books, 2001.
- Health & Safety Commission. *Strategic Plan for 2001–2004*. HSE Books, 2001 (www.hse.gov.uk/action/content/strat2001.htm).
- Skórska C, Mackiewicz B, Dutkiewicz J, et al. Effects of exposure to grain dust in Polish farmers: work-related symptoms and immunologic response to microbial antigens associated with dust. *Ann Agric Environ Med* 1998; **5**: 147–153.
- Radon K, Opravil U, Hartung J, Szadkowski D, Nowak D. Work-related respiratory disorders and farming characteristics among cattle farmers in Northern Germany. *Am J Ind Med* 1999; **36**: 444–449.
- Rylander R, Essle N, Donham KJ. Bronchial hyper-reactivity among pig and dairy farmers. *Am J Ind Med* 1990; **17**: 66–69.
- American Thoracic Society. Respiratory health hazards in agriculture. *Am J Respir Crit Care Med* 1998; **158**: S1–S76.
- Sallie BA, Ross DJ, Meredith SK, McDonald JC. SWORD '93. Surveillance of work-related and occupational respiratory disease in the UK. *Occup Med* 1994; **44**: 177–182.
- Darke CS, Knowelden J, Lacey J, Milford Ward A. Respiratory disease of workers harvesting grain. *Thorax* 1976; **31**: 294–302.
- Harries MG, Lacey J, Tee RD, Cayley GR, Newman Taylor AJ. *Didymella exitialis* and late summer asthma. *Lancet* 1985; **1**: 1063–1066.
- Ingram CG, Jeffrey IG, Symington IS, Cuthbert OD. Bronchial provocation studies in farmers allergic to storage mites. *Lancet* 1979; **2**: 1330–1332.
- Cuthbert OD, Brostoff J, Wraith DG, Brighton WD. 'Barn allergy'. Asthma and rhinitis due to storage mites. *Clin Allergy* 1979; **9**: 229–236.
- Cuthbert OD, Jeffrey IG, McNeill HB, Wood J, Topping MD. Barn allergy among Scottish farmers. *Clin Allergy* 1984; **14**: 197–206.
- Terho EO, Husman K, Vohlonen I, Rautalahti M, Tukiainen H. Allergy to storage mites or cow dander as a cause of rhinitis among Finnish dairy farmers. *Allergy* 1985; **40**: 23–26.
- Blainey AD, Topping MD, Ollier S, Davies RJ. Respiratory symptoms in arable farmworkers: role of storage mites. *Thorax* 1988; **43**: 697–702.
- Blainey AD, Topping MD, Ollier S, Davies RJ. Allergic respiratory disease in grain workers: the role of storage mites. *J Allergy Clin Immunol* 1989; **84**: 296–303.
- Ylönen J, Mäntylä R, Taivainen A, Virtanen T. IgG and IgE antibody responses to cow dander and urine in farmers with cow-induced asthma. *Clin Exp Allergy* 1992; **22**: 83–90.
- Rautiainen M, Ruoppi P, Jägerroos H, Nuutinen R, Mäntylä R, Virtanen T. Nasal sensitization of dairy farmers to bovine epithelial and urinary antigens. *Rhinology* 1992; **30**: 121–127.
- Harries MG, Cromwell O. Occupational asthma caused by allergy to pig's urine. *Br Med J* 1982; **284**: 867.
- Kanerva L, Vaheri E. Occupational allergic rhinitis in Finland. *Int Arch Occup Environ Health* 1993; **64**: 565–568.
- Danuser B, Weber C, Kunzli N, Schindler C, Nowak D. Respiratory symptoms in Swiss farmers: an epidemiological study of risk factors. *Am J Ind Med* 2001; **39**: 410–418.
- doPico GA, Reddan W, Flaherty D, et al. Respiratory

- abnormalities among grain handlers. *Am Rev Respir Dis* 1977; **115**: 915–927.
25. Siracusa A, Desrosiers M, Marabini A. Epidemiology of occupational rhinitis: prevalence, aetiology and determinants. *Clin Exp Allergy* 2000; **30**: 1519–1534.
 26. Ross DJ, Keynes HL, McDonald JC. SWORD '97: surveillance of work-related and occupational respiratory disease in the UK. *Occup Med* 1998; **48**: 481–485.
 27. Torén K. Self-reported rate of occupational asthma in Sweden 1990–92. *Occup Environ Med* 1996; **53**: 757–761.
 28. Heinonen OP, Horsmanheimo M, Vohlonen I, *et al.* Prevalence of allergic symptoms in rural and urban populations. *Eur J Respir Dis* 1987; **71**: 64–69.
 29. Iversen M, Dahl R, Korsgaard J, Hallas T, Jensen EJ. Respiratory symptoms in Danish farmers: an epidemiological study of risk factors. *Thorax* 1988; **43**: 872–877.
 30. Nejari C, Tessier JF, Letenneur L, Dartigues P, Barberger-Gateau P, Salamon R. Prevalence of self-reported asthma symptoms in a French elderly sample. *Respir Med* 1996; **90**: 401–408.
 31. Johnston N, Rask-Andersen R, Lundin Å, Talbäck M. Asthma increase in farmers: a 12-year follow-up. *Eur Respir J* 1996; **9**(Suppl. 23): 378s.
 32. Fishwick D, Pearce N, D'Souza W, *et al.* Occupational asthma in New Zealanders: a population based study. *Occup Environ Med* 1997; **54**: 301–306.
 33. Melbostad E, Wijnand E, Magnus P. Determinants of asthma in a farming population. *Scand J Work Environ Health* 1988; **24**: 262–269.
 34. Dalphin JC, Dubiez A, Monnet E, *et al.* Prevalence of asthma and respiratory symptoms in dairy farmers in the French province of the Doubs. *Am J Respir Crit Care Med* 1998; **158**: 1493–1498.
 35. Omland Ø, Sigsgaard T, Hjort C, Pedersen OF, Miller MR. Lung status in young Danish rurals: the effect of farming exposure on asthma-like symptoms and lung function. *Eur Respir J* 1999; **13**: 31–37.
 36. Radon K, Danuser B, Iversen M, *et al.* Respiratory symptoms in European animal farmers. *Eur Respir J* 2001; **17**: 747–754.
 37. Kogevinas M, Antó JM, Sunyer J, Tobias A, Kromhout H, Burney P and the European Community Respiratory Health Survey Study Group. Occupational asthma in Europe and other industrialised areas: a population-based study. *Lancet* 1999; **353**: 1750–1754.
 38. Cuthbert OD, Gordon MF. Ten year follow up of farmers with farmer's lung. *Br J Ind Med* 1983; **40**: 173–176.
 39. Braun SR, doPico GA, Tsiatis A, Horvath E, Dickie HA, Rankin J. Farmer's lung disease: long-term clinical and physiologic outcome. *Am Rev Respir Dis* 1979; **119**: 185–191.
 40. Grant IWB, Blyter W, Wardrop VE, Gordon RM, Pearson JCG, Mair A. Prevalence of farmer's lung in Scotland—a pilot survey. *Br Med J* 1972; **1**: 530–534.
 41. Terho EO, Heinonen OP, Lammi S. Incidence of clinically confirmed farmer's lung disease in Finland. *Am J Ind Med* 1986; **10**: 330.
 42. Malmberg P, Rask-Andersen A, Höglund S, Kolmodin-Hedman B, Read Guernsey J. Incidence of organic dust toxic syndrome and allergic alveolitis in Swedish farmers. *Int Arch Allergy Appl Immunol* 1988; **87**: 47–54.
 43. Cormier Y, Bélanger J. Long-term physiological outcome after acute farmer's lung. *Chest* 1985; **87**: 796–800.
 44. Cockcroft AE, McDermott M, Edwards JH, McCarthy P. Grain exposure—symptoms and lung function. *Eur J Respir Disease* 1983; **64**: 189–196.
 45. doPico GA, Flaherty P, Bhansali P, Chavaje N. Grain fever syndrome induced by inhalation of airborne grain dust. *J Allergy Clin Immunol* 1982; **69**: 435–443.
 46. Vogelmeier C, Krombach F, Münzing S, *et al.* Activation of blood neutrophils in acute episodes of farmer's lung. *Am Rev Respir Dis* 1993; **148**: 396–400.
 47. Malmberg P, Rask-Andersen A, Rosenhall L. Exposure to micro organisms associated with allergic alveolitis and febrile reactions to mold dust in farmers. *Chest* 1993; **103**: 1202–1209.
 48. Larsson K, Eklund A, Hansson LO, Isaksson BM, Malmberg P. Swine dust causes intense airways inflammation in healthy subjects. *Am J Respir Crit Care Med* 1994; **150**: 973–977.
 49. Rask-Andersen A. Organic dust toxic syndrome among farmers. *Br J Ind Med* 1989; **46**: 233–238.
 50. Vogelzang PFJ, van der Gulden JWJ, Folgering H, van Schayck CP. Organic dust toxic syndrome in swine confinement farming. *Am J Ind Med* 1999; **35**: 332–334.
 51. Husman K, Terho EO, Notkda V, Nuutinen J. Organic toxic dust syndrome among Finnish farmers. *Am J Ind Med* 1990; **17**: 79–80.
 52. Malmberg P, Rask-Andersen A, Palmgren U, Höglund S, Kolmodin-Hedman B, Stalenheim G. Exposure to micro-organisms, febrile and airway-obstructive symptoms, immune status and lung function of Swedish farmers. *Scand J Work Environ Health* 1985; **11**: 287–293.
 53. Haglind P, Rylander R. Occupational exposure and lung function measurements among workers in swine confinement buildings. *J Occup Med* 1987; **29**: 904–907.
 54. Bongers P, Houthuijs D, Remijn B, Brouwer R, Bierskaker K. Lung function and respiratory symptoms in pig farmers. *Br J Ind Med* 1987; **44**: 819–823.
 55. Chan-Yeung M, Wong R, Maclean L. Respiratory abnormalities among grain elevator workers. *Chest* 1979; **75**: 461–467.
 56. Donham KJ, Cumro D, Reynolds SJ, Merchant JA. Dose-response relationships between occupational aerosol exposures and cross-shift declines of lung function in poultry workers: recommendations for exposure limits. *J Occup Environ Med* 2000; **42**: 260–269.
 57. Radon K, Weber C, Iversen M, Danuser B, Pedersen S, Nowak D. Exposure assessment and lung function in pig and poultry farmers. *Occup Environ Med* 2001; **58**: 405–410.
 58. Holness DI, O'Brien FL, Sass-Kortsak A, Pilger C, Nethercott JR. Respiratory effects and dust exposures in hog confinement farming. *Am J Ind Med* 1987; **11**: 571–580.
 59. Donham KI, Merchant JA, Lassise D, Popendorf WJ, Burmeister LF. Preventing respiratory disease in swine confinement workers: Intervention through applied

- epidemiology, education and consultation. *Am J Ind Med* 1990; **18**: 241–261.
60. Choudat D, Goehen M, Korobaef M, Boulet A, Dewitte JD, Martin MH. Respiratory symptoms and bronchial reactivity among pig and dairy farmers. *Scand J Work Environ Health* 1994; **20**: 48–54.
 61. Dosman JA, Graham BL, Hall D, Van Loon P, Bhasin P, Froh F. Respiratory symptoms and pulmonary function in farmers. *J Occup Med* 1987; **29**: 38–43.
 62. Dalphin JC, Pernet D, Dubiez A, Debieuvre D, Allemand H, Depierre A. Etiologic factors of chronic bronchitis in dairy farmers: case control study in the Doubs region of France. *Chest* 1993; **103**: 417–421.
 63. Babbott FL, Gump DW, Sylvester DL, MacPherson BV, Holly C. Respiratory symptoms and lung function in a sample of Vermont dairymen and industrial workers. *Am J Public Health* 1980; **70**: 241–245.
 64. Iversen M, Pedersen B. Relation between respiratory symptoms, type of farming and lung function disorders in farmers. *Thorax* 1990; **45**: 919–923.
 65. Lowry T, Schuhman LM. ‘Silo-filler’s disease’—a syndrome caused by nitrogen dioxide. *J Am Med Assoc* 1956; **162**: 153–160.
 66. Department for Environment, Food & Rural Affairs (DEFRA). *TB in Cattle. Protecting Human Health*, PB5118B. DEFRA, 2000.
 67. Department for Environment, Food & Rural Affairs (DEFRA). *Zoonoses Report United Kingdom*. 1999, PB5577. DEFRA, 2001.