Asthma

Farming and asthma

M B Schenker

Where do we stand?

At the beginning of the 20th century, farmers were believed to be healthier than the general population. Exposure to fresh countryside air and physical work were thought to be the source of this improved health. During the 20th century this agrarian myth was shown to be incorrect—farmers not only had significantly higher rates of mortality from occupational injuries, but they also had higher rates of many chronic diseases such as cardiovascular and chronic obstructive pulmonary disease.[1] Higher rates of chronic diseases were observed despite the generally lower rates of cigarette smoking among farmers, suggesting that workplace exposures were contributing factors.[2] In the 1980s and 1990s there was markedly increased attention to the occupational health of farmers, and to respiratory effects of agricultural exposures. These studies confirmed that farming causes a wide range of respiratory diseases, including airway diseases such as asthma and COPD, hypersensitivity pneumonitis, interstitial fibrosis, infectious pneumonias, and toxic injuries such as silo filler's disease and tracheo-bronchitis from numerous irritant chemicals.[3] Indeed, the spectrum of respiratory diseases from agricultural exposures reads like a textbook of occupational lung disease, and is very different from many classic occupational lung diseases such as asbestosis that are associated with a narrow pulmonary response.[4]

The range of lung diseases resulting from agricultural work is not surprising when one looks at the breadth and high concentration of toxic exposures on the farm. Pulmonary toxins include organic dusts (animal, vegetable products, pollens), infectious agents (bacteria, fungi, viruses, mycobacterium), endotoxins and glucans, toxic chemicals (solvents, fuels, disinfectants), pesticides (paraquat, fungicides, organophosphates), gases from silos, welding and animal waste (NH_3, Cl_2, H_2S, CO_2, CO, NO, NO_2, etc), inorganic dusts (silica, silicates, clays), fertilisers, and feed additives. Exposures to these agents occur at concentrations significantly higher than in other occupational settings or from ambient sources. For example, endotoxin concentrations from agricultural exposures are commonly observed in the thousands (EU/m^3), one or two orders of magnitude higher than levels seen in industrial occupational settings.[5] Similarly, inhalable particle concentrations of farmers are frequently over 5 mg/m^3 and respirable particle concentrations of 1–5 mg/m^3 are commonly seen, well above levels in other occupations and dwarfing ambient particle exposures.[6 7]

While agricultural respiratory disease has been of interest to a relatively small group of investigators, general interest in the health of farming populations has diminished as the percentage of the population farming has declined in developed countries, now less than 2% in many countries. Replacement of farm families by hired farm workers, usually immigrants, did not alter the decreasing interest in this population. All of this changed at the end of the 20th century, at least from a scientific perspective. This resulted from the juxtaposition of two important observations. First, the incidence of asthma was unequivocally shown to be increasing around the world over the past four or five decades.[8] Second, it was observed that children exposed to farming environments in early life had a reduced incidence of atopy and asthma.[9–14] The observation of reduced asthma among children raised on a farm is a fascinating story, originating from the clinical observation that these children had fewer allergies, and subsequently being confirmed in several epidemiological studies. Numerous studies have confirmed lower rates of atopy among children raised on a farm, and additional, but not all, studies have observed lower rates of asthma. Critical observations from these studies include the farm exposures occurring in the first year of life, and exposures to farm animals. The so-called ''hygiene hypothesis'' has been suggested to explain the global increase in asthma, but that debate is beyond the scope of this editorial.[15]

More relevant is the apparent contradiction that exposures to farm environments in early life reduce the incidence of asthma, but adult exposures on the farm are a known cause of occupational asthma. One might refer to this as the ''agricultural asthma paradox''. There are many well described agricultural sensitisers causing asthma, including grain-dust mites, soya bean powder, cow epithelium, and various arthropod exposures.[3] However, numerous respiratory irritants in agriculture cause an ''asthma-like'' syndrome, an acute non-allergic airway response arising from inhalation of various agents such as grain and cotton dust. Is the farm good or bad for asthma, or is timing everything? So far, there has been no clear answer to the question, but increasing attention to the issue is beginning to increase our understanding of this paradox.

Eduard and colleagues have analysed data on asthma from a Norwegian cohort of 2169 farmers.[16] Asthma was determined by self-report, and atopy by positive RAST test. Additionally, exposures to major respiratory toxins were measured for different types of farming. Overall the prevalence of current asthma was low (2.7%), probably in part reflecting the well described healthy worker effect among farmers.[17] Most interesting was the observation that only 20% of farmers with asthma were atopic, and asthma in atopic farmers was significantly less prevalent among farmers of $\geqslant$2 types of livestock. Conversely, asthma among non-atopic farmers was significantly increased among those who farmed $\geqslant$2 types of livestock. Analysis of specific exposures suggested that fungal spores were more significantly associated with reduced asthma in atopic farmers than endotoxin or ammonia, but this analysis was likely limited by the low prevalence of asthma and collinearity of exposures.

These findings are important because they suggest some insight into the agricultural asthma paradox. Early farm exposures, especially those in the first year of life, reduce the incidence of atopy and atopic asthma, and exposures later in life increase the risk of non-atopic asthma. Careful study of non-atopic asthma among farmers and agricultural workers may reveal frequent occurrence of the ''asthma-like syndrome'' resulting from numerous agricultural exposures to respiratory irritants. The specific exposures responsible for reduced atopic asthma remain to be identified, although they appear to be most strongly associated with livestock farming, and not with crop farming. For the adult farmer, exposure to respiratory irritants is a significant risk factor for non-atopic asthma. Atopic asthma from

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exposure to well known agricultural antigens also exists, although it may be much less common than non-atopic asthma among farmers.

As the farm continues to be an important laboratory for understanding the causes, and possibly prevention, of asthma, it is important to remember that agriculture is still the most common occupation in the world, and many respiratory diseases including asthma are caused by exposures on the farm. As with all diseases, primary prevention is the preferred approach to reduce disease morbidity. On the farm this involves reducing exposures to many known respiratory toxins, an area of work in which there has been some recent progress in engineering controls,[18] but in which progress is difficult because of farmer behaviours.[19] Insights into the hygiene hypothesis should not decrease efforts to prevent the large burden of asthma and other respiratory diseases among farmers and other agricultural workers.

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Respiratory medicine

The role of occupation in the development of chronic obstructive pulmonary disease (COPD)

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A discussion of current issues

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death worldwide.[1] In the UK, COPD is given as the cause of death on about 30 000 death certificates annually.[2] This represents 5.1% of all deaths. The prevalence of COPD is difficult to determine because the condition does not usually manifest until mid-life, when it is already moderately advanced. In England and Wales, it is estimated that there are currently 900 000 diagnosed cases, and allowing for under-diagnosis the true prevalence is estimated to be 1.5 million.[3] The total annual cost to the National Health Service for the treatment of COPD is thought to be £491 652 000 in direct costs, and £982 000 000 including indirect costs, causing 21.9 million working days to be lost in 1994–95 as a result of this condition.

A recent clinical study from the UK noted that in a random sample of COPD patients, 44% were below retirement age, and 24% reported that they were completely prevented from working by their disease.[4] A further 9% were limited in their ability to work, and patient carers also missed time from work. Established disease clearly interferes with work capability.

Cigarette smoking is undoubtedly the main cause of COPD in the population, but the link between harmful workplace exposures and COPD has been debated for many decades. Indeed, awareness of a link between work in dusty trades and chronic bronchitis (termed industrial bronchitis[5]) can be traced back to the 19th century. In 1984, the US Surgeon General's report concluded that the only accepted cause of COPD was tobacco smoke; occupational exposures were characterised as putative rather than established causes.[6] Since 1993 British coalminers with chronic bronchitis and emphysema (COPD) have been eligible for compensation if they have worked underground for at least 20 years.[7] This would appear to acknowledge coalmine dust as a cause of COPD. This conclusion was subsequently endorsed following deliberations by the High Court in 1999, although not without controversy. In toxicological terms, there is nothing particularly harmful about coalmine dust compared to other workplace dusts, the key difference perhaps relating simply to generally higher and more prolonged exposures compared to most ''above-ground'' exposures. Certainly there is biological plausibility to the view that daily inhalation exposures over many years to high enough concentrations of dusts and irritants could cause lung damage eventually predisposing to COPD.

While the evidence for occupational exposures and asthma is more