

EDITORIAL

The world wide problem of occupational asthma

In this issue of *Clinical and Experimental Allergy* there is a report from a new national reporting programme of occupationally-induced lung disease [1]. The Surveillance of Work-related and Occupational Respiratory Diseases project in South Africa (SORDSA) began in 1996, and this report evaluates data obtained over the initial 2 years, with a particular focus on asthma. It was modelled on similar surveillance programmes that have been operating in the UK over the last 10 years at national (SWORD) or local levels (SHIELD), though inevitably has differences [2,3]. Perhaps the most important are the exclusion of disorders arising in current mine workers (these have been monitored independently for many years because mining, especially gold mining, is a dominant industry in South Africa) and the use of nurses as reporters, in addition to respiratory and occupational physicians. The nurses are not, however, responsible for making diagnoses; they record the diagnoses reached by attending physicians.

Case ascertainment and accuracy

The organization of both SORDSA and SWORD programmes depends on close cooperation between relevant professional and government bodies, though both are fundamentally independent of government. For both, reports are filed at monthly intervals from volunteer participants. This leaves the possibility that regional differences in case ascertainment might arise (at least partly) because of bias attributable to differences in organization, enthusiasm and compliance. A further, but inevitable, source of potential inaccuracy lies with the strength of the diagnostic evidence from case to case, and in particular the common difficulty in attributing a given disorder in an individual to a specific occupational cause rather than to non-occupational (coincidental) factors.

While some respiratory diseases are almost invariably occupational in origin (for example mesothelioma or pleural plaques from asbestos exposure, and toxic bronchitis/pneumonitis from chemical inhalation accidents), many arise commonly in the population at large regardless of occupational exposures (asthma, COPD, chronic bronchitis, tuberculosis, lung cancer, diffuse interstitial fibrosis, and pleural effusion/thickening). Although the epidemiological evidence may be strong that a given occupational exposure causes or contributes to a particular disease, personal judgement becomes critical when attribution is considered

in the individual case. In practice the consulted physician simply decides on the evidence available whether an occupational cause is more likely than not, and if he/she has sufficient enthusiasm, time and organizational ability, any case of probable occupational origin is reported nationally to SORDSA or SWORD. The consistency of SWORD reports over recent years and its comprehensive level of coverage suggest that in practice there is a high level of reporting whenever cases are recognized.

Most reporting schemes (others have been active in Australia, Canada, Finland, France, Italy, New Zealand and the USA) are consequently vulnerable to inaccuracy from limited diagnostic information and to bias from the prejudices of the reporters, and all are likely to underestimate true incidences because not all cases come to light [4–10]. The information obtained nevertheless fills an uncomfortable void, since there are otherwise few clues to the importance or otherwise of occupational environments to lung disease at national and international levels. While the data provide crude estimates of overall incidence, for which wide confidence intervals are appropriate, they are the best that are available and they provide invaluable assistance to understanding (and ultimately to preventing) lung disease of occupational origin. What is more, lung disease arising in a particular circumscribed setting, where relevant exposures may be quantified and manipulated, and relevant individuals may be studied, allows its mechanisms and natural history to be elucidated with greater ease than in the population at large. In short, an occupational example of a particular disease provides a useful model for its investigation. This can be illustrated no better than by occupational asthma.

Relative importance of asthma of occupational origin

The SORDSA report identified asthma as the second most commonly reported respiratory disease of occupational origin arising over the 2 years, 1996–1998. It accounted for 7% of the total of 3285 cases. Most common (76%) was pneumoconiosis, whether alone or associated with tuberculosis or COPD. This was despite the exclusion of current miners from the denominator (the survey population at risk). Although SORDSA is in its infancy and currently is most vulnerable to incomplete recognition of incident cases, the outcome immediately suggests that South Africa's particular economic dependence on the mining

industry is indeed reflected by an overwhelming dominance of mining-related disorders among the total of incident cases of occupational lung disease.

The situation is considerably different in Britain, and other western industrially developed countries. Over the last 10 years SWORD has consistently identified asthma as the single most common respiratory disorder of occupational origin, accounting for 25–30% of all incident cases. Many different occupational agents appear to be responsible, however, and in many different occupational settings. By contrast, exposure to asbestos is reported to cause a number of different disorders (mesothelioma, benign diffuse pleural thickening, pleural plaques, asbestosis, lung cancer) and these together account for about 50% of SWORD reports. Thus asthma and asbestos dominate occupational lung disease in Britain, as they do in most industrially developed countries. Table 1 summarizes the overall distribution by disease type over the last 3 years for which there are published SWORD data [11–13].

Occupational disorders of short and long latency

Asthma and asbestos illustrate well a further issue of major epidemiological importance. Occupational asthma generally produces symptoms within a matter of months after first exposure and, like most infections, inhalation accidents and allergic alveolitis, is considered a disorder of short latency. The effects of asbestos exposure, however, are rarely evident for 20 or more years after exposure onset, and like pneumoconiosis in general, and other diseases related to chronic dust exposure (mesothelioma, lung cancer, benign pleural disease, COPD) are diseases of long latency. The relevant period(s) of exposure and the relevant populations at risk may consequently differ considerably, and the most useful reporting schemes take this into account in assessing risk within particular occupations.

Incidence of occupational asthma

SWORD learns of a total of approximately 1000 new cases of occupational asthma each year in Britain, which amounts to about 40 cases per million who are employed and hence at some risk per year. If 2–3% of the general public develops asthma during the usual 60 years of adult life (16–75 years), as currently seems likely, then the general incidence is of the order 333–500/million/year and occupation is responsible for a minimum of 7–12%. This agrees well with other estimates that occupation accounts, on average, for some 9–10% of new cases in adults in industrialized western countries.

However, reported regional and national incidences vary very considerably from 5 per million per year in Massachusetts to 175 in Finland, though most fall within

Table 1. Reported cases of occupational lung diseases to SWORD over 3 years, 1996–1998

Disease	Number	%
Asthma	2989	29
Benign pleural disease	2178	21
Mesothelioma	2175	21
Pneumoconiosis	925	9
Inhalation accidents	621	6
Lung cancer	343	3
Infection	257	2
Bronchitis/emphysema	185	2
Allergic alveolitis	155	1
Others	649	6

From refs 11–13.

the range 25–100 per million per year [4]. Regular reporting has been particularly comprehensive in Finland for many years and so may be the most accurate, but even in Finland cases arising among the self-employed who are not farmers may not readily come to attention. The question arises therefore whether the differences are artefactual depending on inaccuracies and failures to recognize and report, or due to real differences in incidence.

Asthma risk from different agents and different industries

Differences are, of course, to be expected in different regions according to differences in the patterns of local employment, and differences in risk between different industries. Much of the high rate of occupational asthma in Finland is thus a consequence of the high proportion of the population engaged in farming. Data addressing the risks from different agents and different industries are therefore useful in assessing what might be expected in any given location, though exposure levels will additionally exert a critical influence even when regions and populations are closely matched otherwise. Thus an agent with low potency but encountered at high levels of exposure may have a greater impact than one of high potency encountered only at low levels. Table 2 summarizes the importance to occupational asthma of different occupational agents in Britain, and Table 3 summarizes the industries that are principally affected [14].

A model for asthma in general

To the allergist and clinical immunologist occupational asthma is possibly of most interest as a model of

Table 2. Agents causing occupational asthma by presumed hypersensitivity (SWORD 1998)

Agent	1998
Enzymes	14
Isocyanates	13
Laboratory animals and insects	12
Colophony, fluxes and solders	9
Flour/grain	7
Latex	6
Glutaraldehyde	5
'Others'	34

These most recent figures suggest a relative increase in the importance of laboratory animals and latex, and a decrease in that of isocyanates. While the increases may be real, they may also reflect increasing awareness of the reporters. The prominence of enzymes in 1998 was largely a consequence of new cases being discovered in a single detergent enzyme manufacturing plant. Wood dusts and resins/glues represent groups of some importance that are concealed among the 'other' reports; they have each accounted for around 5% of reports over previous years. From refs 4, 11–14.

'environmental' asthma in the population at large, since in comparison the occupational environment can be characterized more readily by exposure to specific asthma-inducing agents, and the associated 'captive' workforce can be investigated more readily for any ensuing physiological or immunological response. A number of lessons have emerged, quite apart from the early demonstration in subjects with occupational asthma that distinctly different

Table 3 Annual incidence of occupational asthma within selected industries in Britain per million employed (SWORD 1992–7)

Occupation	/mill/yr
Coach and spray painting	1367
Baking	958
Metal treatment	643
Chemical processing	641
Plastics manufacture	447
Welding and soldering	279
Food processing (exc. baking)	256
Laboratories	214
Wood working	147

The overall risk among the employed was 38/mill/yr [4], and so the relative risk of developing occupational asthma in these particular industries ranged from 4 to 40 times the average. Taken from Table 8.5 of [14].

early and late reactions may follow the inhalation of allergens.

First, it is clear that immunological hypersensitivity is not the only pathway to the development of asthma, since acute toxicity from industrial inhalation accidents is complicated in a minority of survivors by the development of asthma [15]. Designated, perhaps confusingly, the reactive airways dysfunction syndrome (RADS), it may persist for weeks, months, or even indefinitely. It can be considered a model for asthma that is commonly reported to arise in association with an acute viral insult to the respiratory tract. In the most recent annual analysis of SWORD data (those for 1998) RADS accounted for as much as 11% of incident cases of occupational asthma [13].

Second, extraordinarily high prevalences of occupational asthma have occurred historically in some industrial settings when there were high levels of exposure to very potent agents (e.g. approximately 50% of workers exposed to platinum salts and detergent enzymes) [16,17]. This indicates that susceptibility to asthma is not restricted to a small minority of the population at large, a point of some concern in view of the current escalation in asthma prevalence world wide. While this lesson from occupational asthma is disturbing, the good news is that if the causal agent is identified speedily and exposure then ceases completely, the asthma commonly resolves.

A last point of particular interest is the interactive effect of smoking. It is generally believed that smoking increases the risk for occupational asthma, and in the platinum refining industry it has been suggested that smoking poses a greater risk than atopy [18]. With western red cedar workers, however, smoking appears to exert a protective effect (as it does for farmer's lung and sarcoidosis). Again there is a model for asthma in general; while smoking generally appears to worsen asthma, in a small minority of cases smoking cessation is associated with aggravation or even disease onset [19–21].

References

- 1 Hnizdo E, Esterhuizen T, Rees D et al. Occupational asthma as identified by the Surveillance of Work-related and Occupational Respiratory Diseases programme in South Africa. *Clinical and Experimental Allergy* 2001; 31:32–9.
- 2 Meredith SK, McDonald JC. Work-related respiratory disease in the United Kingdom 1989–92: report on the SWORD project. *Occup Med* 1994; 44:183–9.
- 3 Gannon PFG, Burge PS. A preliminary report of a surveillance scheme of occupational asthma in the West Midlands. *Br J Indust Med* 1991; 48:579–82.
- 4 Meredith SK, Blanc PD. Surveillance: clinical and epidemiological perspectives. In: Hendrick DJ, Burge PS, Beckett WS, Churg A. eds. *Occupational Disorders of the Lung*:

- Recognition, Management, and Prevention. London: WB Saunders, 2001: (in press).
- 5 Provencher S, Labrèche FP, de Guire L. Physician based surveillance system for occupational respiratory diseases: the experience of PROPULSE, Québec. Canada Occup Environ Med 1994; 51:272–6.
- 6 Contreras GR, Rousseau R, Chan-Yeung M. Occupational respiratory diseases in British Columbia, Canada, in 1991. Occup Environ Med 1994; 51:710–2.
- 7 Keskinen H, Alanko K, Saarinen L. Occupational asthma in Finland. Clin Allergy 1978; 8:569–79.
- 8 Ameille J, Calastreng-Crinquand Kopfersmitt MC, Blaumeiser M et al. Incidence of occupational asthma in France in 1996. In: Chiyotani K, Hosoda Y, Aizawa Y, eds. Advances in the Prevention of Occupational Respiratory Diseases. Amsterdam: Elsevier Science BV, 1998:430–3.
- 9 Bena A, d'Errico A, Mirabelli D. A system for the active surveillance of occupational bronchial asthma: the results of 2 years of activity of the PriOR program. Medicina del Lavoro 1999; 90:556–71.
- 10 Baker EL. Sentinel Event Notification System for Occupational Risks (SENSOR): the concept. Am J Public Health 1989; 79(Suppl.):18–20.
- 11 Ross DJ, Keynes HL, McDonald JC. SWORD '96: surveillance of work-related and occupational respiratory disease in the UK. Occup Med 1997; 47:377–81.
- 12 Ross DJ, Keynes HL, McDonald JC. SWORD '97: surveillance of work-related and occupational respiratory disease in the UK. Occup Med 1998; 48:481–5.
- 13 Meyer JD, Holt DL, Cherry NM et al. SWORD '98: surveillance of work-related and occupational respiratory disease in the UK. Occup Med 1999; 47:485–9.
- 14 Newman Taylor A. Non-malignant diseases. In: McDonald JC, ed. Epidemiology of Work Related Diseases 2nd edn. London: BMJ Books, 1999:149–74.
- 15 Brooks SM, Weiss MA, Bernstein IH. Reactive airways dysfunction syndrome (RADS); persistent asthma syndrome after high level irritant exposures. Chest 1985; 88:376–84.
- 16 Hunter D, Milton R, Perry KMA. Asthma caused by complex salts of platinum. Br Med J 1945; ii:92.
- 17 Mitchell CA, Gandevia B. Respiratory symptoms and skin reactivity in workers exposed to proteolytic enzymes in the detergent industry. Am Rev Respir Dis 1971; 104:1–12.
- 18 Venables KM, Dally MB, Nunn AJ et al. Smoking and occupational allergy in workers in a platinum factory. Br Med J 1989; 299:939–42.
- 19 Chan-Yeung M, Lam S, Koener S. Clinical features and natural history of occupational asthma due to western red cedar (*Thuja plicata*). Am Rev Respir Dis 1982; 72:411–5.
- 20 Warren CPW. Extrinsic allergic alveolitis: a disease commoner in non-smokers. Thorax 1977; 32:567–9.
- 21 Valleyre D, Soler P, Clerici C et al. Smoking and pulmonary sarcoidosis: effect of cigarette smoking on prevalence, clinical manifestations, alveolitis, and evolution of the disease. Thorax 1988; 43:516–24.

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