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Occupational asthma: measures of frequency from four countries

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How common is occupational asthma? The quick answer to the question is that no one knows. The frequency of occupational asthma in any population will depend on many factors including susceptibility to respiratory sensitisation, the types of industry in which people work, the numbers of people exposed to sensitising agents, and the levels of those exposures. Many epidemiological studies have estimated the prevalence of occupational asthma in various work forces but few have tackled the question of the frequency and distribution of the disease in the population.

Any measure of disease frequency will, of course, depend in part on the definition and diagnostic criteria used. While most would agree with Newman Taylor's definition of occupational asthma as variable airways narrowing causally related to exposure in the working environment to airborne dust, gases, vapours, or fumes, some would limit the diagnosis to new onset asthma and others to asthma caused by agents believed to induce sensitisation and not include asthmatic symptoms due to irritant effects such as occurs in the reactive airways dysfunction syndrome (RADS).²

The picture we have of the frequency and distribution of occupational asthma in populations is built up from surveys, surveillance schemes, disease registers, and compensation statistics, each of which may use different definitions and draw its cases from different populations. The picture is therefore patchy and fraught with inaccuracies and bias, but it is the best that can be done with the information currently available.

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Prevalence

Prevalence depends on both the frequency with which new cases occur and the duration of the illness. Follow up studies of patients with occupational asthma indicate that, for many, the development of the disease means that they become asthmatic and continue to have episodes of bronchoconstriction even when no longer exposed to the sensitising agent. Although many of these studies have been of patients attending specialist clinics with relatively short periods of follow up so the gen-

eralisability of the results as well as the longer term consequences are still in some doubt, it would seem that many people have symptoms that persist for five or six years, and in some cases longer.³ As occupational asthma is rarely fatal, its prevalence will therefore be considerably greater than its incidence.

It is frequently quoted that 15% of asthma is occupational in origin. This figure was based on the results of two studies in the 1970s; however, in neither study was the evidence strong. One was a study of 813 Japanese men, but it is not clear how the sample was selected or how the occupational aetiology was established.4 The second was a large survey of over 6000 people aged 18-64 in the USA in 1978 and was based on self-reported symptoms.⁵ It seems likely, however, that the population surveyed and the questions asked resulted in an overestimation of the prevalence of occupational asthma; two thirds of the sample were drawn from applicants for Social Security disability benefits, and attribution of disease to occupational exposure was on the basis of the somewhat leading question: "Was this condition caused by bad working conditions such as noise, heat, or smoke?"

Self-reported work-related symptoms may bear little relation to clinical diagnosis. Stenton et al found in a survey of shipyard workers that respiratory symptoms suggestive of asthma reported on questionnaire (wheeze, chest tightness, coughing, and breathlessness) correlated poorly with a clinical diagnosis of asthma.⁶ In addition, in a study of 94 adults of working age discharged from three hospitals in Michigan in 1990 with a diagnosis of asthma, three met the National Institute for Occupational Safety and Health (NIOSH) criteria for probable and 17 for possible occupational asthma, but of 24 patients who attributed their illness to bad working conditions only 11 were in the probable or possible occupational asthma categories.

From a survey of self-reported work-related illness in the preceding 12 months in a nationally representative sample of some 75 000 adults in England and Wales, which was an addition to the Labour Force Survey of 1990,8 it was estimated that 19 700 people (95% confidence interval (CI) 14 000 to 26 900) had

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asthma caused by their work and a further 47 400 (95% CI 38 200 to 58 200) had asthma made worse by work. The diagnosis of asthma was derived either from the respondents' reports of what their doctor said was the matter or from a detailed description of symptoms. If 5% of the working population have asthma (a reasonable crude estimate⁹), this would mean that approximately 2% of those have occupational asthma and that, in a further 4%, their illness is exacerbated by exposures at work. These figures are, of course, based on statistics that may not be valid and assumptions that may not be true, but they give us a rough idea of the prevalence of the disease and are probably more reliable than previous estimates.

Incidence

Prevalence is a measure of the burden of disease in the population, but incidence is of particular interest in occupational asthma for two reasons: firstly, duration of symptoms is likely to vary making prevalence figures hard to interpret but, more importantly, if the determinants of new cases are understood and further illness prevented, the numbers of new sufferers entering the disease pool will be reduced.

SOURCES AND FREQUENCY

Estimates of the incidence of occupational asthma may be derived from compensation statistics and a small number of surveillance schemes, although each source has its own problems of diagnostic criteria, completeness, and selection bias.

Finland

The most complete ascertainment of cases is in Finland where official statistics on occupational diseases and injuries have been compiled since 1926. In 1964, in order to improve the timeliness and accuracy of the figures, a register was established of all reported cases of occupational disease; the register is maintained by the Finnish Institute of Occupational Health (FIOH) in Helsinki. Registered cases come from three sources: (1) reports from provincial medical officers to whom, since 1974, physicians have been required by law to report all cases of occupational disease they diagnose; (2) claims made of insurance companies; and (3) cases diagnosed at the Institute of Occupational Health.

All employees must be insured and compensation for a confirmed occupational disease is relatively generous; it includes costs of treatment and retraining as well as various allowances and pensions. There is therefore an incentive for people whose health has been affected by their job to come forward. As the insurance companies require good evidence before paying compensation, the diagnosis and causal link is usually well established and in most cases of occupational asthma serial respiratory function tests (peak expiratory flow rates at and away from work) or inhalation challenge tests are performed.

Insurance for self-employed people is voluntary, however, and only a minority are covered. The self-employed are therefore likely to be under-represented on the disease register. The one exception to that is farmers who have been eligible for compensation since a change in the law in 1982 which, as will be seen later, has had a substantial impact on the numbers of cases of occupational asthma diagnosed. There has been a steady rise in numbers of new cases of occupational asthma registered each year from 80 in 1976, 10 equivalent to a rate of 36 per million working people, to 379 in 1992 which represents approximately 153 cases per million workers.

United Kingdom

There are four sources of information on the incidence of occupational asthma in the United Kingdom which overlap to some extent. The two official sources are the numbers of cases accepted under the Industrial Injuries Scheme which are published annually by cause¹¹ and cases reported to the Health and Safety Executive under the Reporting of Injuries, Diseases and Dangerous Occurrences Regulations (RIDDOR). 12 RIDDOR requires employers to report cases of occupational asthma in their employees to the Health and Safety Executive if they are informed in writing by a doctor and the person concerned has been exposed to one of seven prescribed sensitising agents. However, the serious under-reporting under RIDDOR is widely recognised 11 13 and its statistics contribute little to our understanding of the incidence of the disease. The Industrial Injuries Scheme is also based on a limited list of prescribed causes of occupational asthma, although in 1990 the list was extended considerably¹⁴ and the so called "category Z - any other sensitising agent inhaled at work" was added to the list for which the Department of Social Security could award compensation. As in Finland, neither scheme includes selfemployed workers who made up 12% of the economically active population of the UK in 1991,15 and as only those with a disability assessed at 14% or more qualify for benefit, and even 100% disability was only valued at £91.60 per week in 1993,11 many people with occupational asthma may not bother to apply.

In addition to the two statutory sources of information there are two voluntary reporting schemes in the UK, both of which have been in existence since 1989: the Surveillance of Work-related and Occupational Respiratory Disease (SWORD) project, which obtains reports on newly diagnosed cases of occupational lung disease of all sorts from specialists in occupational and chest medicine from throughout the UK,16 and a more intensive scheme for occupational asthma in the West Midlands region known as SHIELD, which includes reports from the local Medical Boarding Centre in addition to cases reported by chest and occupational physicians. 1718 As many of the physicians from the West Midlands also participate in SWORD, the data for the region are likely to include many of the same cases, but Occupational asthma 437

the two schemes are complementary. SHIELD is able to assess the overlap with compensation statistics and, through local contacts, is in a position to investigate clusters of cases; SWORD is able to look at the national picture and, because of the larger numbers involved, produces more stable, albeit less complete, estimates of disease incidence.

Strict diagnostic criteria are not specified in SWORD; participants are asked to report all cases of respiratory disease which they believe are due to exposure at work. Between 1989 and 1991 there were on average 509 cases of occupational asthma reported to SWORD each year, equivalent to an annual incidence of 19 per million working persons.¹⁹ The incidence of occupational asthma reported to SWORD from the West Midlands region, however, was about twice the national average after taking differences in occupation into account.20 This discrepancy was thought to be due to less complete ascertainment and reporting from other parts of the country. In 1992 a new system was introduced to SWORD whereby those chest physicians whose practice involved relatively little occupational disease, and who tended to report less regularly than those with a major interest in such conditions, were divided into 12 random samples, one of which was selected to report each month.¹⁹ This has resulted in much improved participation rates and increased incidence figures. In 1992 there were an estimated 1047 cases¹⁹ and in 1993 907 cases of occupational asthma were reported (SWORD project, personal communication), which together are equivalent to an average of 37 per million working persons per year, very close to the SHIELD estimate of 43 per million working persons per year for 1989-91.

The SWORD figures do not necessarily include cases awarded disablement benefit. Gannon and Burge found that, of 178 workers with occupational asthma exposed to agents recognised for compensation reported to SHIELD by chest physicians over a three year period, only 37 (21%) were also reported by participants from the compensation board.¹⁸ Some of this discrepancy may be due to delays in coming before the board, but if there was the same degree of overlap for SWORD cases it would mean that SWORD underestimates the incidence of diagnosed occupational asthma by at least a third which would put the annual incidence at nearer 50 than 40 per million working persons. This is without considering all the cases which are never diagnosed.

United States

In the United States surveillance has taken a different path from in Europe. Under the Sentinel Event Notification System for Occupational Risks (SENSOR) programme the emphasis is on identification of potentially hazardous sentinel cases for the purpose of investigation and preventive intervention in the workplace21 rather than obtaining an epidemiological overview of the distribution and determinants of disease in the population. There are SENSOR programmes for occupational asthma in six states, four of which have been in operation since 1988.²² Although the estimation of disease frequency is not one of the aims of the programme, and the number of cases reported from some states is very small, the incidence of disease in Colorado and Michigan, relative to the population at risk, is of a similar order to that in the UK based on Department of Social Security (DSS) figures and reports to SWORD in the early years (table 1). The sorts of cases reported, however, are not necessarily similar. To reach a diagnosis of occupational asthma in SENSOR the following are required: (1) a physician diagnosis of asthma, (2) an association between symptoms of asthma and work, and (3) exposure to an agent previously associated with asthma or evidence of an association between work exposure and either a significant decrease in lung function or increase in airways responsiveness.23

The SENSOR schemes in Michigan and New Jersey have identified the largest numbers of cases, mainly from reports by physicians but also from hospital discharges, and these have been described in some detail.²³ In addition to cases of definite occupational asthma, probable cases, cases of reactive airways dysfunction syndrome, and cases in which pre-existing asthma is made worse by work are included.

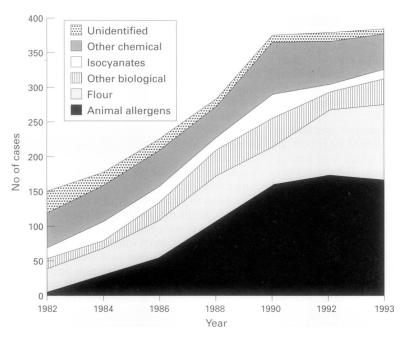
Ouebec

Lagier et al have published figures on successful new claims for occupational asthma from the province of Quebec between 1986 and 1988.²⁴ Occupational asthma is defined in Quebec as asthma resulting from exposure to a sensitising agent in the work place. Objective evidence is required, usually a specific bronchial challenge or serial lung function tests at work and away from work,²⁵ before a claim is accepted. As a measure of incidence, compensation statistics

Table 1 Reports of occupational asthma in four countries

Country	Region (source)	Years	No. of cases	Employed persons		Annual rate	95% CI
				Year	(No. × 10³)	per 10 ⁶	(per 10 ⁶ /year)
Finland	All (FIOH)	1988, 1990, 1992	1038	1990	2470	140	132 to 149
UK	All (SWORD) All (SWORD) West Midlands (SHIELD) Great Britain (DSS)	1989–91 1992–93 1989–91 1989–92	1528 1954 284 1282	1989-90 1989-90 1990 1989-90	26533 26533 2200 25940	19 37 43 12	18 to 20 35 to 39 38 to 48 12 to 13
Canada	Province of Quebec	1986-88	214	1986	2796	25	22 to 29
USA	Colorado Massachusetts Michigan New Jersey	1988–91 1988–91 1988–92 1988–92	102 31 381 154	1990 1990 1990 1990	1633 3028 4166 3869	16 3 18 8	13 to 19 2 to 4 17 to 20 7 to 9

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Number of cases of occupational asthma registered by the Finnish Institute of Occupational Health by year.

in Quebec underestimate disease frequency: as in the UK and Finland self-employed persons are not included and many people with occupational asthma do not make claims, perhaps because they do not judge it to be in their financial interest. The Quebec group have compared numbers of claims with cases diagnosed in special surveys in three industries24: snowcrab processing in which there were 10 claims compared with 46 diagnosed cases, spray painters exposed to isocyanates (a ratio of three claims to six cases), and guar gum workers (a ratio of one to three). If these ratios were representative of all industries the true incidence of occupational asthma in Quebec would be some four times greater than the number of compensated cases, and would be of the order of 100 per million working population per year.

Table 2 Number (%) of suspected agents from surveillance schemes in four countries

Suspected agent	SWORD 1989–91	Finland 1992	Quebec 1986–88	Michigan and New Jersey SENSOR 1988–92	
Animal allergens	150 (9.8)	172 (45·4)	31 (14·5)	4 (0.7)	
Enzymes	7 (0.5)	2 (0.5)	- ` ´	3 (0.6)	
Flour and grain	108 (7.1)	93 (24.5)	30 (14.0)	11 (2·1)	
Wood dust	63 (4·1)	6 (1.6)	27 (12-6)	7 (1.3)	
Moulds	14 (0.9)	2 (0.5)	1 (0.5)	3 (0.6)	
Other plants	41 (2.7)	18(4.7)	7 (3.3)	8 (1.5)	
Isocyanates	336 (22.0)	11 (2.9)	54 (25.2)	104 (19.4)	
Solder	85 (5·6) [´]	_ ` ´	5 (2.3)	4 (0.7)	
Welding fumes	35 (2·3)	17 (4.5)	- ` ´	10 (1.9)	
Cobalt	9 (0.6)	2 (0.5)	-	15 (2·8)	
Chrome	9 (0.6)	- ` ′	_	5 (O·9)	
Platinum salts	9 (0.6)	1 (0.3)	_	2 (0.4)	
Aldehydes	49 (3·2)	9 (2.4)	2 (0.9)	28 (5·2)	
Chlorine	9 (0.6)	_ ` `	- ` `	13 (2.4)	
Amines	12 (0.8)	_	4 (1.9)	8 (1.5)	
Styrene	7 (0.5)	_	- ` ´	9 (1.7)	
Resins and glues	80 (5.2)	9 (2.4)	_	35 (6.5)	
Paints	29 (1.9)	4 (1·1)	2 (0.9)	- ` ´	
Inks and dyes	21 (1.4)	- ` ´	3 (1.4)	4 (0.7)	
Drugs/pesticides	31 (2.0)	_	11 (5·1)	11 (2·1)	
Oil mists	22 (1.4)	_	- ' '	53 (9.9)	
Other	281 (18.4)	19 (5.0)	4 (1.9)	89 (16.6)	
Unknown	121 (7.9)	14 (3·7)	33 (15·4)	109 (20.4)	
Total	1528	379	214	535	

Values in parentheses are percentages of total.

CAUSES

The causes of reported occupational asthma may vary over time because of social and legal changes rather than because of real alteration in the pattern of disease, as the data from Finland demonstrate. The distribution of cases registered between 1982 and 1993 by broad category of causal agents is shown in the figure. It seems likely that the increase in numbers of cases due to animal allergens and flour is the result of inclusion of agricultural workers in the compensation scheme from 1982; numbers in the other categories changed little over the decade. Knowledge of hazards may also affect diagnosis; for example, in 1989 there were only two cases of asthma attributed to glutaraldehyde reported to SWORD but over the next two years, as it became more widely known as a possible cause, a further 28 cases were reported.

The distribution of reported cases by agent from SWORD, Finland, Quebec and SENSOR Michigan and New Jersey is shown in table 2. There are striking differences, some of which may be real, due to international variation in the types and size of industries, working practices, and environmental protection in the work place. Some of the variation may be spurious, however, due to differences in source of reports, referral patterns, and diagnostic criteria. For example, 70% of cases registered in Finland in recent years were due to animal allergens, flour and grain, compared with 29% from Quebec, 17% from the UK, and less than 3% from Michigan and New Jersey. Comparison of proportions is unsatisfactory, but there are clearly relatively many more cases attributed to these causes in Finland than in Michigan and New Jersey. Some of the differences may be due to there being a higher proportion of farmers in Finland and to different farming practices, and some to the fact that occupational asthma in farmers is compensated in Finland, whereas SENSOR is based on reports from hospitals and physicians whom self-employed and possibly uninsured farmer workers may be unlikely to consult. In 1981, prior to the change in legislation in Finland, only two of the 156 cases of occupational asthma reported were due to animal allergens, which is similar to the proportion reported to SENSOR. It is interesting that in the UK most of the cases attributed to animal allergens are due to work with laboratory animals, whereas this form of asthma is rarely reported in Finland.

There are, however, also some striking similarities, in particular in the proportion of cases caused by enzymes, moulds and platinum salts, all of which are well recognised causes of occupational asthma and clearly are quite rare. The proportion of cases attributed to isocyanates is very similar in the UK, Quebec, and Michigan and New Jersey, where they appear to be the most common cause of occupational asthma, but they are remarkably rare in Finland considering their widespread use. In the early 1980s, however, there were some 25 cases of isocyanate asthma per year registered with the FIOH (18% of the total), a level very similar to the other countries for

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Table 3 Rates of occupational asthma by occupational group

Occupation	Finland 1990		SHIELL	1989–1991	SWORD 1989-1991	
	Cases	Rate (95% CI) (per 10 ⁶ /year)	Cases	Rate (95% CI) (per 10 ⁶ /year)	Cases	Rate (95% CI) (per 10 ⁶ /year)
Baker	32	4000 (2737 to 5647)	20	444 (272 to 686)	64	285 (220 to 364)
Spray painter	14	3111 (1701 to 5220)	22	1833 (1149 to 2776)	108	729 (598 to 880)
Farmer	180	1401 (1204 to 1621)	8	44 (19 to 86)	43	34 (25 to 46)
Welder/solderer/electronic assembler	30	1035 (698 to 1477)	26	110 (72 to 161)	106	158 (130 to 191)
Plastics making		,	38	1056 (747 to 1448)	81	387 (307 to 481)
Chemical processing			3	143 (30 to 417)	77	346 (273 to 433)
Woodworker			18	130 (77 to 206)	65	45 (35 to 58)
Other	119	52 (43 to 62)	149	25 (21 to 30)	984	13 (12 to 14)
Total	375	152 (137 to 168)	284	43 (38 to 48)	1528	19 (18 to 20)

which there is information. Since then the numbers have declined by more than 50% to only 11 in 1992. Two explanations have been offered. Firstly, there is thought to have been an improvement in working practices in the polyurethane industry. Secondly, and perhaps more importantly, unemployment in Finland has risen to 19% and, with the economic recession, many small car repair shops have been put out of business thus reducing the frequency of isocyanate exposure; in addition, it may be that persons in employment are less likely than previously to jeopardise their job by seeking a diagnosis of occupational asthma. Indeed, since 1990 the total numbers of registered cases in Finland ceased their previously steady increase (figure).

OCCUPATION

In order to compare the incidence of disease in different communities in a meaningful way, the size of the population at risk must be taken into account. Unfortunately, only SWORD, SHIELD, and the Finnish register have attempted to estimate rates of work-related asthma by occupation. As has already been discussed, SWORD and SHIELD both suffer from incomplete ascertainment of cases and therefore any rates quoted are minimum estimates. Occupation-specific rates in high risk jobs from SHIELD, SWORD, and Finland are shown in table 3. A detailed comparison of rates may lead to erroneous conclusions because of differences in the classifications of jobs in the three registers. Despite classification differences, however, it is apparent that Finnish rates are consistently some five times those from SWORD (1989-91) and 2-3 times those from SHIELD, with the exception of rates for farmers which differ for reasons already discussed. A high rate in spray painters and bakers is a universal finding and highlights the urgent need for improved protection for people engaged in such work. In the UK the importance of chemical and plastics processing is also apparent.

AGE AND SEX

Not surprisingly, considering the jobs associated with occupational asthma, the disease occurs more commonly in men than in women with over two thirds of cases reported to SWORD²⁰ and SHIELD¹⁸ being in men. The difference is slightly less pronounced when the size of the male and female working populations

is taken into account; the rate in working men from SWORD was 1·8 times the rate for women, and the Labour Force Survey study of self-reported work-related symptoms also found a male to female rate ratio for occupational asthma of 1·8 in persons aged 16–44 years, although it was 2·7 in the older age group. When occupational differences between the sexes were taken into account in an analysis of SWORD cases there was no real or consistent difference in disease incidence between men and women. ²⁰ A similar picture has been noted in Finland where exposure to cow dander is common in both sexes and similar numbers of cases are reported in men and women.

Analysis of cases reported to SWORD has shown an apparent increase in the risk of occupational asthma with age in both sexes.20 In women the gradient in annual incidence was 11 per million employed aged 16-29 years, 12 per million aged 30-44 years, and 15 per million in those 45 years or more; in men the figures were 17 per million, 23 per million, and 31 per million, respectively. This trend could not be explained by differences in occupation by age; indeed, adjustment for occupation made the age gradient steeper in both sexes. Although age-specific rates were not available for Finland, of the 384 cases of occupational asthma registered with the FIOH in 1993, 14% were in the 15-29 year age group, 39% were aged 30-44 years, and 41% aged 45-59 years; it seems most unlikely that these proportions reflect the age distribution of the working popu-

Such findings may be due to referral patterns, with young people perhaps being less likely to seek medical attention for work-related illness than older people who may be hoping for compensation or may wish, despite symptoms, to stay in a job where they have security and experience. Certainly, the Labour Force Survey study of the prevalence of self-reported asthma attributed to work found that the age gradient was in the opposite direction. However, the question of diagnostic accuracy is a potential source of error in the study; it seems quite possible that, whereas young people with chronic respiratory symptoms are given a diagnosis of asthma, older persons are more likely to be labelled as having chronic obstructive airways disease. Consistent with this, the survey found a very steep age-related increase in the prevalence of "other lower respiratory illness".8

It is possible that susceptibility to respiratory sensitisation may increase with age, perhaps because of previous exposures or behavioural 440 Meredith, Nordman

> factors such as smoking. In support of this argument, when persons attending emergency rooms with asthma on epidemic days during the Barcelona soybean asthma outbreaks were compared with those attending at other times, the age-specific rate ratios increased with age group26 and age was found to be an independent risk factor.27 Men were more likely to be affected than women on epidemic days, but this difference could be explained by smoking.²⁷

Conclusions

Disease registers and surveillance schemes are not necessarily designed to measure incidence and prevalence, but their data can provide useful epidemiological insights. Their main functions are the detection and identification of hazards so that further cases can be prevented, and to provide a database for research to improve our understanding of the determinants and consequences of disease.

Occupational asthma probably accounts for only a small proportion of adult asthma, of the order of 2-6% in the UK. On the other hand, asthma is very common and so thousands of people in the UK and in other western countries have asthma as a result of their work. The frequency of the disease in less developed countries is unknown but is potentially very large. Information on incidence is patchy but from Finland, where ascertainment is most complete, there are approximately 140 per million working people affected each year, and even there disease in the self-employed is probably missed. Data for the UK are limited to cases seen by specialist physicians and the best estimate is that the annual incidence of such cases is about 50 per million. Through internal comparisons and analysis it is possible to judge the extent to which variation in ascertainment and reporting have affected the frequency of reported disease, but so far it has not been possible to measure the underestimation due to persons with work-related symptoms not seeking medical attention or not being referred to a specialist.

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