The SHIELD scheme in the West Midlands Region, United Kingdom

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Abstract

***Objective*—To study the general and specific incidence of occupational asthma within a defined geographic area; to audit the diagnosis of occupational asthma; to determine proposed mechanisms of asthma; and to determine the employment state of workers at diagnosis.**

***Design*—A surveillance scheme of physicians likely to see cases of occupational asthma.**

***Setting*—The West Midlands Region of the United Kingdom.**

***Subjects*—Workers with occupational asthma diagnosed within the boundaries of the West Midlands Region.**

***Main measures*—Demographic data, employer, agent to which exposed, date of diagnosis, method of diagnosis, proposed mechanism of asthma, and employment state.**

***Results*—A recognised incidence of 43 (95% confidence interval CI 35–52) new cases per million general workers per year was detected. Specific occupational incidences varied from 1833 (95% CI 511–2990) per million paint sprayers to eight per million clerks. Specific incidence by District Health Authority varied from 103 in Solihull to 14 per million general workers in South Warwickshire. Agents to which workers were exposed at the time of diagnosis were generally well recognised (isocyanates 20·4%, flour 8·5%, colophony 8·3%). The most commonly used method of diagnosis was serial peak expiratory flow (PEF) measurement. Its use varied (specialist unit 72%, general chest physicians 50%, compensation board 48%). Workers were still exposed and therefore could have usefully performed PEF readings in 4% of cases where they were omitted from the specialist centre, 16% seen by chest physicians, and 2% seen by the Compensation Board. Other methods of diagnosis were used only infrequently outside the specialist unit. Fifty six per cent of reporting physicians considered that the mechanism of asthma was allergy compared with 18% who believed that it was irritation. Twenty eight per cent of workers were exposed to the suspected causative agent at the time of diagnosis, 38% were either on long term sickness absence, had retired, or had become unemployed. More workers (38%) who were exposed to agents recognised for statutory compensation before the 1991 changes seen at the specialist centre reach compensation and were reported to the scheme by the Compensation Board than those seen by chest physicians (9%).**

***Conclusions*—These recognised incidences are likely to be an underestimate of the true incidence. They highlight at risk occupations and suggest underdiagnosis in some District Health Authorities. They suggest that diagnostic methods are underused outside specialist centres and that the mechanism of asthma is generally considered to be allergic.**

(*British Journal of Industrial Medicine* 1993;50:791–796)

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Occupational asthma is asthma that is caused by or made substantially worse by agents inhaled in the workplace. It is a preventable disease. At present little is known about its incidence in the general working population or in specific occupations. Blanc[1] found a prevalence of occupational asthma of 12 000 per million of the disabled population in the United States and Keskinen *et al*[2] found an annual incidence of 35 per million of the general working population in Finland in 1978. Finland has a well organised national notification scheme. The most recent data available suggest a general incidence of 152 per million workers in 1990 (Keskinen H, Nordman H, personal communication). A study in a Zambian chest clinic found a prevalence of occupational asthma of 5900 per

million asthmatic patients.[3] Preliminary results from the Surveillance of Work Related and Occupational Respiratory Disease (SWORD) project have also been reported.[4] These suggested an incidence of 22 cases per million general workers for the whole United Kingdom. Regional incidences varied from 63 new cases per million general workers in the West Midlands Metropolitan County to eight new cases per million general workers in West Yorkshire. Specific incidences by occupation were as high as 639 cases per million, for coach and paint sprayers. A preliminary report of the Midland Thoracic Society's Rare Respiratory Disease Registry Surveillance Scheme of Occupational Asthma (SHIELD) has been published.[5] This described findings after one year of a surveillance scheme covering the West Midlands Region including the West Midlands Metropolitan County. This scheme is confined to occupational asthma alone and collects more detailed information on individual cases than SWORD, which collects information on various occupational lung diseases. It reported an annual incidence of 30 cases per million general workers per year for the whole region. Specific incidences were described for broad occupational groups and ranged from 154 to three per million workers in painters/assembly workers and clerical workers respectively.

Confirmatory investigations of a history suggestive of occupational asthma include serial measurement of peak expiratory flow (PEF),[6] measurement of specific IgE antibodies,[7] specific bronchial provocation testing,[8] and measurement of non-specific bronchial hyper-reactivity before and after periods at work.[9] Little is known about how the diagnosis of occupational asthma is made in practice.

Opinions differ as to the mechanism of occupational asthma. Irritant, allergic, and pharmacological aetiologies have been suggested.[10]

Some studies have been performed following up the outcome of workers with a diagnosis of occupational asthma; no studies have looked at the employment state of workers at the time of diagnosis.

This article describes results from three years of the SHIELD scheme.

Method

The Midland Thoracic Society's Rare Respiratory Disease Registry circulates chest physicians in the West Midlands Region monthly. It asks if they have seen any new case of a number of respiratory diseases. The Occupational Asthma Surveillance Scheme started in January 1989. For occupational asthma the circulation of the Registry was expanded to include the Medical Boarding Centre (respiratory disease) (the body that awards statutory compensation for occupational asthma) and members of the West Midlands Group of the Society of Occupational Medicine. For the first year old and new cases were accepted. Physicians who had seen cases of occupational asthma were sent a questionnaire requesting detailed information about the case. This included demographic data, occupation, causative agents, employers, method of diagnosis, proposed mechanism, and employment state at the time of diagnosis.

An annual incidence of occupational asthma for the general working population was determined from the date of diagnosis and denominators from the 1990 Labour Force Survey.[11] Occupation was coded according to the Office of Population Censuses and Surveys 1980 job classification[12] allowing incidences for specific groups to be calculated from Labour Force Survey denominators. Home address post code allowed assignment to health authority of residence with the Post Code Address File on compact disc system.[13] Denominator data on working populations within health authorities were not available and this was estimated from total populations from the fraction of workers (2·2 million) out of the total population for the West Midlands (5·1 million). 95% confidence intervals (95% CIs) are given for incidence figures where numbers allow calculation.

Results

In three years 500 new and old cases of occupational asthma were reported, the earliest diagnosed case was in 1965. Three hundred and fifty (70%) cases were seen by the authors in a specialist occupational lung disease unit (including dual reports from other physicians), 110 (22%) by the Medical Boarding Centre (respiratory disease) (the Compensation Board), 104 (20%) by general chest physicians, and 14 (3%) by occupational physicians. Of the new cases reported 47 (56%), 57

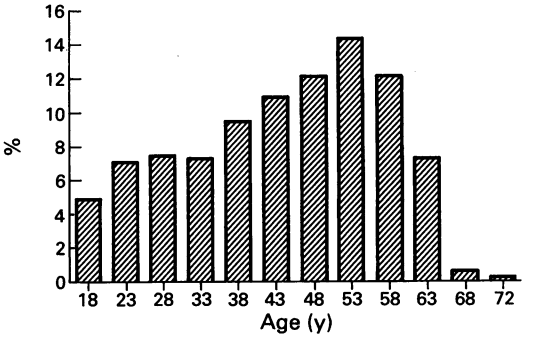

Figure 1 Distribution of cases of occupational asthma by age.

Table 1 Agents to which workers were exposed when they first developed occupational asthma

Agent	*No*	*%*
Isocyanates	101	20·4
Flour	42	8·5
Colophony	41	8·3
Oil mist	32	6·5
Wood dust	28	5·6
Epoxy resin	23	4·6
Metals	22	4·4
Air conditioning	13	2·6
Farm grain	9	1·8
Welding fumes	8	1·6
Cleaning fluids	7	1·4
Gluteraldehyde	6	1·2
Farm animals	5	1·0
Laboratory animals	5	1·0
Acid fluxes	4	0·8
Cobalt (hard metal)	4	0·8
Acid anhydride	3	0·6
Formaldehyde	3	0·6
Hairdressing chemicals	3	0·6
Chlorine gas	2	0·4
Other	135	27·2

Table 2 Attempted methods of diagnosis of occupational asthma for a specialist occupational lung disease unit and general chest physicians and the attempted methods used to diagnose occupational asthma accepted for compensation by the Medical Boarding Centre (respiratory diseases) (Compensation Board)

Method of diagnosis	*Specialist unit*	*General chest physicians*	*Compensation Board*
Serial peak flow measurement	72%	50%	48%
Specific IgE antibodies	53%	6%	19%
Specific bronchial provocation test	10%	3%	12%
Stop resume histamine testing	1%	0%	0%

(55%), and 62 (65%) were seen by the authors in the first, second, and third year respectively. Forty five cases in total (9%) were reported by two different sources and five cases (1%) were reported by three different sources. Three hundred and forty four (69%) were male. Figure 1 shows the frequency by age. The mean age was 44 years (range 16 to 72). Table 1 shows agents to which workers were exposed when symptoms developed; isocyanates, flour, and colophony were the most frequent. Two hundred and sixty (52%) were recognised for statutory compensation[14 15] in the United Kingdom before the 1991 changes.[16] In 161 (33%) cases the reporting physician considered that the specific causative agent had been identified.

One hundred and thirty four workers (38%) reported from the specialist centre were exposed to agents recognised for compensation under the old rules. Of these 35 (27%) were also reported by the Compensation Board. Forty four (40%) workers reported by chest physicians were exposed to agents recognised for compensation under the old rules. Of these four (9%) were also reported by the compensation board.

Table 2 shows the attempted method of diagnosis for the specialist occupational lung disease unit, compensation board, and general chest physicians. The small number of workers reported by occupational physicians precluded breakdown by method of diagnosis. Workers seen by chest physicians and the Compensation Board had less investigations than those seen at the specialist unit. Of the workers whose diagnosis did not involve the use of serial PEF measurement, three (4%) seen at the specialist centre, 10 (16%) seen by chest physicians, and one (2%) seen by the Compensation Board were still exposed at the time of diagnosis and could therefore have performed serial PEF measurement. Of the workers seen by the specialist centre who had not had specific antibody testing, 11 (9%) were exposed to large molecular weight compounds (flour or grain, wood dusts, animal proteins) with easily available radioallergosorbent tests (RASTS) and 19 (15%) were exposed to small molecular weight compounds (isocyanates and acid anhydrides) with available, but less readily accessible

Table 3 General and specific occupational group incidences for occupational asthma

Occupational group	*Population*	*Incidence per million workers (actual number of cases)*				
		1989	*1990*	*1991*	*Mean*	*95% CI*
General	2200 000	38 (84)	47 (104)	44 (96)	43	35–52
Paint sprayers	4000	2750 (11)	1250 (5)	1500 (6)	1833	511–2990
Rubber and plastics workers	12 000	1167 (14)	1167 (14)	830 (10)	1054	495–1670
Electroplaters	3000	1000 (3)	1000 (3)	1000 (3)	1000	
Foundry core makers and moulders	5000		400 (2)	1000 (5)	467	
Bakery workers	15 000	667 (10)	133 (2)	534 (8)	445	121–821
Chemical processing workers	7000	286 (2)	143 (1)		143	
Machine tool operators	43 000	163 (7)	140 (6)	117 (5)	140	28–821
Carpenters	46 000	130 (6)	152 (7)	109 (5)	130	26–235
Solderers	79 000	80 (6)	177 (14)	80 (6)	112	40–188
Farmers	61 000	66 (4)	49 (3)	16 (1)	44	
Clerks	353 000	6 (2)	8 (3)	8 (3)	8	

95% CIs are shown when numbers were sufficient to allow calculation.

Table 4 General and specific health authority incidence for occupational asthma

District Health Authority	Working population	Incidence per million workers			
		1989	1990	1991	Mean
General incidence	2200 000	38	47	44	43
Solihull*†	68 100	193	73	132	103
Sandwell*	87 900	34	114	46	65
Bromsgrove and Redditch*†	47 200	106	21	64	64
Hereford†	74 200	41	45	54	50
Central and South Birmingham*	290 700	54	41	41	45
Mid Staffordshire†	86 100	58	58	12	43
Coventry*	155 100	26	26	70	41
East Birmingham*	90 900	33	22	66	40
Kidderminster†	44 700	45	45	22	37
Dudley*	143 600	7	41	48	32
West Birmingham*	119 600	25	50	17	31
North Birmingham*	85 700	23	34	35	31
South East Staffordshire*†	96 200	31	52	10	31
Worcester†	99 100	20	40	30	30
Shropshire†	174 700	29	34	17	27
Wolverhampton*	146 800	27	7	48	27
Rugby and North Warwickshire*†	101 400		40	40	27
North Staffordshire*	221 100	5	41	14	20
Walsall*	101 100	10	10	30	20
South Warwickshire†	92 700	11	32		14

*Industrial area; †rural area; *†mixed industrial and rural area.

RASTs. The figures for chest physicians were 23 (21%) and 29 (27%), and for the Compensation board 19 (26%) and 38 (53%) respectively.

With incidence calculated from new cases reported per year there was a mean general incidence of 43 (95% CI 35–52) new cases per million general working population per year. Table 3 shows incidences by specific occupational group. These varied from 1833 (95% CI 511–2990) per million paint sprayers to eight per million clerks.

Table 4 shows the incidence by health authority. These varied from 103 per million general workers in Solihull to 14 per million workers in South Warwickshire. Six health authorities had incidences above the general incidence.

Figure 2 shows the employment state of the worker at the time of diagnosis. Most workers were still exposed but several were no longer exposed.

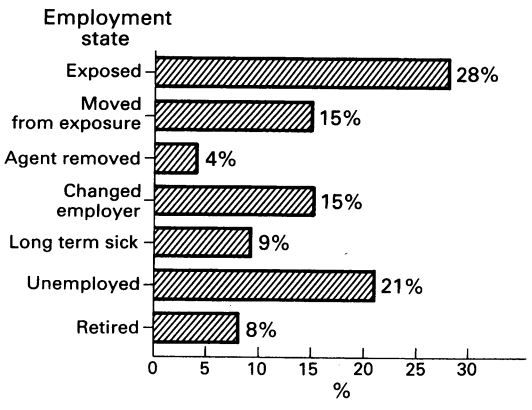

Figure 2 Employment state at the time of diagnosis.

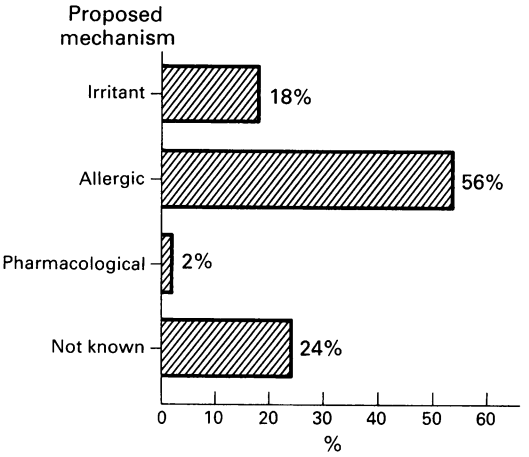

Figure 3 Proposed mechanism of occupational asthma suggested by reporting physician.

Figure 3 presents the proposed mechanism of occupational asthma suggested by the reporting physician.

Discussion

Fewer new cases of occupational asthma were reported in the first year of the scheme. The increased numbers in subsequent years is likely to reflect increased awareness of the scheme from twice yearly reports and the feedback of relevant information to reporting physicians. Most cases were reported from our own specialist unit; how-

ever, the number of new cases seen increased each year (47, 57, and 62 workers in 1989, 90, and 91). Similar numbers were reported by the Medical Boarding Centre and general chest physicians, but few cases were reported by occupational physicians. This was unexpected as the occupational physicians included some employment medical advisors. Employers have a statutory requirement to report cases of occupational asthma due to recognised agents under the Reporting of Injuries, Disease, and Dangerous Occurrences Regulation (RIDDOR) to the Employment Medical Advisory Service. Subsequent enquiries showed that there was a reluctance to report these cases because of possible breaches in confidentiality. The employment medical advisors are now reporting new cases seen without disclosing their identity. Most cases were men, and this is likely to reflect the greater number of men working in occupations associated with occupational asthma. The age distribution of reported cases suggested that occupational asthma may become more prevalent with age; this has also been noted by SWORD.[17] This may reflect greater duration of exposure to sensitising agents or cigarette smoke or the fact that younger workers with occupational asthma are more likely to remove themselves from exposure without ever seeking medical advice. Older workers have less opportunity to relocate away from exposure and they are therefore more likely to seek medical help and be reported.[18]

The agents to which workers were exposed when diagnosed reflect many of the most well known causes of occupational asthma. Less well recognised causes included oil mists[19], metals (chrome[20], nickel[21], and aluminium[22]) and glutaraldehyde[23]. Some workers developed occupational asthma while incidentally exposed to cleaning fluids, some of which contained ethylene diamine,[24] a known cause of asthma. In 66% of cases the reporting physician was unable to specifically identify the causative agent. This is likely to reflect the use of serial PEF measurement as the tool of diagnosis. This technique, although able to diagnose occupational asthma, cannot usually identify the exact causative agents.[25]

Comparison of the methods of diagnosis between reporting groups showed that serial PEF was underused by chest physicians and the Medical Boarding Centre. It was not appropriate, however, for use in all cases at our own specialist centre. Reasons for this include the worker having already left work, the worker suffering severe reactions on returning to work, or the worker being unable to take time off work. In 4% of workers seen at the specialist centre and 10% of those seen by chest physicians the worker was still exposed at the time of diagnosis, which would have allowed serial PEF to be performed. This was true of 1% of the cases seen by the Compensation Board who had not had serial PEF performed. This is in keeping with the timing of compensation claims, which usually occur after the worker has left the original job. Specific IgE antibodies were only used on a regular basis in the specialist unit. This may be partly due to the limited availability of tests. The results suggest however, that they were not performed by any physician in many cases when they were available. Specific bronchial challenge tests were seldom used in any centre.

The incidence figures determined by this scheme are likely to be a considerable underestimate of the true incidence of occupational asthma in the West Midlands Region. Reasons for incomplete ascertainment of the true incidence include the fact, as mentioned earlier, that some workers with occupational asthma never seek medical advice. The relatively low percentage of duplicate notification suggests that not all recognised cases are being reported. This is because workers who seek statutory compensation from the Medical Boarding Centre have usually been diagnosed elsewhere in the West Midlands. We would therefore have expected to know about most of these cases from the investigating physicians had there been complete ascertainment of cases. Despite these problems the general incidence of 43 new cases per year remained higher than that reported by SWORD (22 per million workers) but below that reported from Finland (152 per million workers).

Specific incidences for occupational groups above the general incidence reflected those occupations with well recognised causes of occupational asthma. A less well recognised occupation using a well recognised sensitising agent was that of foundry core makers and moulders (mean incidence 467 per million workers). These workers make sand cores bound together with an isocyanate resin and the cores are used for casting hollow metal objects. A less well recognised occupation with a less well recognised agent was electroplaters who work with chrome and nickel (mean incidence 1000 per million workers). The specific incidence was considerably reduced below the general incidence in clerks (mean incidence eight per million workers) who are not exposed to any well recognised causes of occupational asthma (with perhaps the exception of air conditioning contaminants).

The incidence of occupational asthma by district health authority showed considerable variation. The Solihull district with the highest incidence of 103 new cases per million workers is served by our own specialist occupational lung disease unit. Even here the incidence still falls below that reported in Finland, suggesting that overdiagnosis is not a factor. On the contrary relatively low incidences in

heavily industrialised areas such as North Staffordshire suggest underdiagnosis.

Employment state at the time of reporting shows that 28% of workers remain exposed to the sensitising agent. Only 15% are relocated away from exposure within the workplace and in only 4% is the offending agent removed to allow the worker to continue in his job and also prevent his fellow workers from developing symptoms. A more likely outcome was to become unemployed, stay away from work on sick leave, or seek early retirement through ill health. Only 15% were able to find an alternative employer. Several studies have shown that continued exposure after diagnosis is associated with a poorer prognosis. In one study,[26] the workers removed from exposure who no longer complained of breathlessness had been diagnosed significantly earlier after the onset of their first symptom (48 *v* 66 months, $p = 0{\cdot}001$) and had a significantly higher forced expiratory volume in one second at presentation (90% *v* 73% predicted, $p = 0{\cdot}008$). They had developed symptoms earlier after first exposure (48 *v* 66 months, $p > 0{\cdot}05$) and had been removed from exposure sooner (eight *v* 12 months, $p > 0{\cdot}05$).

Most reporting physicians (56%) considered that the mechanism of occupational asthma was allergic, 18% thought that it was irritant, and some thought that it was a combination of these two mechanisms. Only a small percentage thought that it was a pharmacological effect. Many believed that the mechanism was not known.

Two cross sectional studies were carried out after a number of workers were reported to the scheme from the same workplace. One took place in an electroplating factory where workers were exposed to chrome and nickel[27] after two workers had been reported. Six new cases of occupational asthma were identified. The other study took place in a post office sorting office where workers were exposed to paper and mail bag dust after the report of three workers from different sorting offices. Six new cases of occupational asthma were identified. A further study resulting from SHIELD was of the provision of occupational health at workplaces from which workers had been reported as being exposed to isocyanate.

Conclusion

These figures are likely to be a substantial underestimate of the true incidence of occupational asthma. The general incidence and specific incidences by occupation however, provide a guide as to how commonly and where this condition occurs. The results suggest that serial PEF measurements and specific IgE measurement are underused outside specialist centres. Health authority incidence figures suggest that underdiagnosis is a problem. Increased use of investigations may lead to improved diagnosis. After diagnosis many workers remain exposed because unemployment is often the alternative; further exposure is likely to worsen their prognosis.

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Accepted 30 November 1992