

Occupational asthma cases notified to OSH from 1996 to 1999

Christopher Walls, *Departmental Medical Practitioner, Occupational Safety and Health*; Julian Crane, *Associate Professor, Wellington Asthma Research Group, School of Medicine, Wellington*; John Gillies, *Respiratory Physician, Christchurch Hospital, Christchurch*; Margaret Wilsher, *Respiratory Physician, Green Lane Hospital, Auckland*; Colin Wong, *Respiratory Physician, Dunedin Hospital, Dunedin*.

Abstract

Aims. To update notifications to the Occupational Safety and Health Service of the Department of Labour (OSH) Notifiable Occupational Disease System (NODS) from June 1996 to the beginning of 1999.

Methods. All notifications received for non-asbestos related occupational respiratory disease were reviewed to confirm the clinical diagnosis, occupational causation, and to identify the causative agent where possible.

Results. 54 cases of asthma were notified, of which 21 (39%) were accepted as being occupationally caused. These cases arose from 'predictable' industries.

NZ Med J 2000; 113: 491-2

Conclusions. NODS offers sentinel data from interested practitioners and workplaces. Occupational asthma and other occupational respiratory diseases remain poorly notified to this system. NODS confirms the presence of occupational asthma in New Zealand from predictable and preventable causes not dissimilar to other countries. This data collection system needs supplementation by other mechanisms.

The OSH NODS scheme has operated since 1992. Briefly, NODS is a voluntary system that allows OSH to review the patient's medical information and the workplace's occupational hygiene data and decide whether the notified disease has arisen from workplace factors. Advice is offered to the employer, employee and medical providers so as to prevent the occurrence of new cases and the aggravation of current symptoms by workplace factors. Information so gathered is then distributed to similar workplaces to improve work practices and reduce employee exposures to all occupational hazards. The first report from this scheme dealing with work related respiratory illness reviewed notifications up to June 1996.¹ This report summarises the notifications made from the time of the first report (July 1996) until January 1999.

Methods

Notification was made from a number of sources, including the general practitioner, the occupational health nurse, the union official, the employer or the employee themselves. Information concerning the clinical problem was then collected by the OSH occupational health nurse. The nurse or an OSH occupational hygienist reviewed the workplace processes, practices and hygiene (including exposure measurements if appropriate). The OSH Departmental Medical Practitioner (DMP) carried out a file review and forwarded the case to the NODS asthma panel. Only occasionally was the patient examined by the DMP.

The Asthma Panel comprised of four respiratory physicians and one occupational physician (a DMP with an interest in occupational respiratory problems) reviewed the cases.

Cases were allocated to one of three categories according to criteria published in the OSH Occupational Asthma Guide.² These categories were Confirmed (a case of asthma with a convincing workplace cause), Not Valid (not asthma or no convincing workplace cause) and Unproven (either the diagnosis of asthma or the proof of a workplace cause was uncertain, though some of the diagnostic criteria were fulfilled). Commonly, the latter occurred where a patient had a history suggestive of asthma and a suspicious exposure, but was unable to provide a peak flow diary of acceptable quality. Cases had the ability to be reviewed again by the panel if further evidence made this desirable.

Results

A further 54 cases of possible occupational asthma or other occupational respiratory disease were notified to OSH and investigated, making a total of 331 cases since 1992 (Table 1).

The 21 confirmed occupational asthma cases (39% of the sample) are presented in Table 2 by occupational categories and causative agents. The most common aetiological agents

were isocyanates (5) and fumes from primary aluminium smelting (5). The age of confirmed cases ranged from 24 to 61 years, with the majority (57%) falling between the ages of 35 to 49. 85% of confirmed cases were male.

No other non-asbestos related respiratory diseases were notified to the NODS system in this time period.

Table 1. Classification of cases after reviewing workplace and clinical factors.

	Asthma Confirmed	Asthma Unproven	Asthma Not Valid	Other Respiratory Disease
Classified before Panel Formed	38 (22.5%)		123 (72.5%)	8 (5%)
Classified by Panel 1992-June 1996	35 (32%)	29 (27%)	33 (31%)	11 (10%)
Classified by Panel July 1996-Jan 1999	21 (39%)	20 (37%)	13 (24%)	0

Of the 99 cases of possible occupational asthma notified to NODS by doctors (out of a total of 151 cases reviewed by the panel), only nine were notified by the specialist physician community.

Discussion

The cases notified to the NODS system during the time covered by this report arose from continued exposure to recognised, potent asthma causing agents, or from the results of workplace surveillance programmes by a few conscientious employers (eg the primary aluminium smelting industry). This scheme does not capture all recognised cases of occupational asthma occurring in New Zealand. Many general practitioners and specialists remain unaware of the scheme, despite extensive publicity by OSH.³ Ideally, occupational notification could be simplified for doctors by combining NODS with workplace compensation systems. Job security remains a frequently reported reason for doctors or their patients to decline notification. This system does, however, provide better certainty of diagnosis and occupational causation than such population studies as that of Kogevinas.⁴

Once again, there is a commonality of the reported asthma causing agents (eg isocyanate paint and foam exposure), but

Table 2. Agents identified as causing occupational asthma.

Exposure Category	Putative Agent	Occupation (Numbers Affected)
Organic Materials (6) Animal Proteins Cereal Dusts Wood dusts	? Mussel Protein Flour Western Red Cedar	Mussel Processing Plant Operators (1) Baker (2) Manager (1) Carpenters & Joiners (2)
Chemicals (6) Aldehydes (1) Isocyanates (5)	Formalin all types	Laboratory Technician (1) Spray Painter (4), Process Worker (1)
Metal Processing (6) "Potroom Asthma"	Aluminium smelting fumes Welding fumes	Primary Aluminium Smelter Workers (5) Fitter welder (1)
Miscellaneous (3)	Colophony solder Detergent enzymes Irritant mineral dusts	Baker (1) Process Worker (1) Fertiliser plant operator (1)

Numbers of cases are shown in parenthesis.

a great variety of occupations (spray painters, boat-builders) that use these materials, making occupational category a poor predictor of possible asthma causation. Clinicians need to query their patients as to the occupational tasks undertaken and the materials used in these tasks when considering a possible case.

OSH investigations of workplace practices and hygiene continue to support the idea that occupational factors remain, in most cases, and as is found overseas, a totally preventable cause of asthma in New Zealand.^{5,6} Too often, OSH investigations found an undue reliance on inadequate and poorly maintained personal protection, instead of such effective control mechanisms as adequate ventilation.

Occupational asthma is potentially fatal,^{7,8} and its clinical control difficult in the presence of continued exposure.^{8,9} Patients with a longer history of symptoms are more likely to develop chronic persistent asthma, even after removal from exposure.⁸⁻¹⁰ This diagnosis is therefore of practical importance, not just an academic pursuit.

The failure of the specialist respiratory community to utilise the NODS system has encouraged OSH to plan the launch of an adaptation of the UK SWORD (Surveillance of Work Related and Occupational Respiratory Diseases) scheme,^{11,12} which involves polling respiratory physicians on a regular basis.

The data obtained from this system will be less useful to OSH from a preventive viewpoint, in that the SWORD system does not identify the employer nor the employee, making practical interventions impossible.

The panel continues to recommend, in keeping with the ACCP Consensus Statement,⁹ the use of four peak flow measurements per day (both at work and at home) over a period of two weeks, including work and non work periods, to elucidate the diagnosis of occupational asthma. It is vital that the subject has been correctly instructed in peak flow technique and that these records are undertaken carefully.

OSH branch offices and technical resources are available to doctors and other health professionals to help investigate possible occupational causation.

Correspondence. Dr CB Walls, Departmental Medical Practitioners, Manukau Branch, Occupational Safety and Health, PO Box 63010, Papatoetoe South. Email: Chris.Walls@osh.dol.govt.nz

1. Walls CB, Crane J, Gillies J et al. Occupational asthma and other non asbestos occupational respiratory diseases notified between 1993 and 1996. *NZ Med J* 1997; 110: 246-9.
2. A guide to the management of occupational asthma. Occupational Safety and Health Service, Department of Labour. ISBN 0-477-035590; Feb 1995.
3. Gavaghan S. Responsibilities under the NZ Health and Safety in Employment Act and the NZ Notifiable Occupational Diseases System. General Practice Awareness and Compliance. A good practice? MFOM Thesis, Occupational Safety and Health Service, Department of Labour Wellington; August 1996.
4. Kogevinas M, Anto JM, Sunyer J et al. Occupational asthma in Europe and other industrialised areas: a population based study *Lancet* 1999; 353: 1750-4.
5. Beckett WS. The epidemiology of occupational asthma *Eur Respir J* 1994; 7: 161-4.
6. Meredith S, McDonald C. Surveillance systems for occupational disease. *Ann Occup Hyg* 1995; 39: 257-60.
7. Hendrick DJ. Management of occupational asthma. *Eur Respir J* 1994; 7: 961-8.
8. Chan-Yeung M, Malo J. Occupational asthma. *N Engl J Med* 1995; 333: 107-11.
9. Chan-Yeung M. ACCP Consensus Statement. Assessment of asthma in the workplace. *Chest* 1995; 108: 1084-117.
10. Gannon PF, Weir DC, Robertson AS, Sherwood BP. Health, employment, and financial outcomes in workers with occupational asthma. *Br J Ind Med* 1993; 50: 491-6.
11. 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: 177-82.
12. Ross DJ, Sallie BA, McDonald JC. SWORD '94 surveillance of work related and occupational respiratory disease in the UK. *Occup Med* 1996; 45: 175-8.

Experts in urologic oncology have almost come to blows when discussing the merits of using serum PSA (Prostate Specific Antigen) to screen for prostate cancer, which surely indicates that supporting evidence for benefit is rather controversial, and that if there is gain, it is not very great. A serum marker is useful if it can be used to detect disease earlier than by symptoms or signs, and thereby increase the probability of cure or the duration of survival. Certainly, screening by serum PSA can detect prostate cancer in asymptomatic men. Since widespread introduction of PSA screening in the United States, the incidence of prostate cancer in white men has doubled from about 55/100 000 in 1983-87 to about 110/100 000 in 1993-95. In contrast, it has increased only slightly in the UK where PSA screening has been used less frequently. It has been estimated that about 75% of prostate cancers would never be diagnosed in the absence of PSA screening.

Ian F Tannock. *The Lancet Oncology* 2000; May: 17-19.