ORIGINAL ARTICLE

Occupational asthma: an assessment of diagnostic agreement between physicians

David Fishwick, Lisa Bradshaw, Mandy Henson, Chris Stenton, David Hendrick, Sherwood Burge, Rob Niven, Chris Warburton, Trevor Rogers, Roger Rawbone, Paul Cullinan, Chris Barber, Tony Pickering, Nerys Williams, Jon Ayres, Andrew D Curran

Occup Environ Med 2007;**64**:185–190. doi: 10.1136/oem.2006.027722

Objectives: To investigate the levels of agreement between expert respiratory physicians when making a diagnosis of occupational asthma.

Methods: 19 cases of possible occupational asthma were identified as part of a larger national observational cohort. A case summary for each case was then circulated to 12 physicians, asking for a percentage likelihood, from the supplied information, that this case represented occupational asthma. The resulting probabilities were then compared between physicians using Spearman's rank correlation and Cohen's κ coefficients.

Results: Agreement between the 12 physicians for all 19 cases was generally good as assessed by Spearman's rank correlation. For all 66 physician–physician interactions, 45 were found to correlate significantly at the 5% level. The agreement assessed by κ analysis was more variable, with a median κ value of 0.26, (range –0.2 to +0.76), although 7 of the physicians agreed significantly ($p < 0.05$) with ≥ 5 of their colleagues. Only in one case did the responses for probability of occupational asthma all exceed the “on balance” 50% threshold, although 12 of the 19 cases had an interquartile range of probabilities not including 50%, implying “on balance” agreement. The median probability values for each physician (all assessing the identical 19 cases) varied from 20% to 70%. Factors associated with a high probability rating were the presence of a positive serial peak expiratory flow Occupation Asthma SYStem (OASYS)-2 chart, and both the presence of bronchial hyper-reactivity and significant change in reactivity between periods of work and rest.

Conclusions: Despite the importance of the diagnosis of occupational asthma and reasonable physician agreement, certain variations in diagnostic assessment were seen between UK expert centres when assessing paper cases of possible occupational asthma. Although this may in part reflect the absence of a normal clinical consultation, a more unified national approach to these patients is required.

See end of article for authors' affiliations

Correspondence to:
David Fishwick, Centre for
Workplace Health, Health &
Safety Laboratory, Harpur
Hill, Buxton, Derbyshire
SK17 9JN, UK; d.fishwick@
sheffield.ac.uk

Accepted
22 September 2006
Published Online First
9 November 2006

Consistent epidemiological data identify occupational exposures as important determinants of adult asthma.^{1,2} Recent population-based estimates suggest that at least 10% of all adult-onset asthma may relate to occupation.³ An accurate diagnosis is key to improving the prognosis in this condition,⁴ whereas false-positive diagnoses may be disastrous.

Previous studies of inter-rater reliability have dealt with individual diagnostic techniques. For example, a variation between experts in the interpretation of serial peak expiratory flow (PEF) in patients with a suspected diagnosis of occupational asthma⁵ has recently been shown. To our knowledge, there are no data relating to variation between experts when assessing all relevant clinical information from patients with possible occupational asthma.

This study describes the results of a national, multicentre study of diagnostic practices for occupational asthma, with the focus on levels of agreement between expert physicians.

METHODS

Twelve physicians agreed to participate. One respiratory physician in training was included, as were two physicians working for the Health and Safety Executive, within the employment medical advisory service. The remainder were consultant respiratory physicians with a clinical and research interest in occupational lung disease, assessing such cases in a specialist clinic.

The cases used in this study were selected at random from a larger series of patients with suspected occupational asthma,

referred to six secondary care respiratory centres in the UK with possible occupational asthma. Although the setting for the study could not replicate the actual referral process, each recruiting physician was allowed to enter any patient with suspected occupational asthma referred from a variety of sources, so that the patients included in the study were a true reflection of day-to-day clinical practice. It was not the intent of this study to create a group of perfectly investigated cases, but to record the normal level of investigation thought necessary by each attending physician. All diagnostic tests considered in this study were available to all study physicians, if thought necessary for the diagnostic process.

The local multicentre research ethics committee agreed to this study, and all patients gave informed consent for their inclusion.

Phase 1

Two researchers visited each centre, and transcribed from the hospital case notes all relevant clinical information, examination findings, investigations and diagnostic opinions on to a standard study proforma. Study physicians were allowed to investigate each patient according to their own judgement, and were not constrained by a specific protocol.

This information was then converted into an anonymous summary document for each case; any investigations that could

Abbreviations: OASYS, Occupation Asthma SYStem; PEF, peak expiratory flow

Table 1 Content of each case

Case	Age, sex	Work-related history	Work-related onset of symptoms after first exposure (latency in years)	Airway obstruction documented (FEV ₁ /FVC <75%)	Exposure to "known" sensitiser* mentioned in history	Positive specific IgE to any workplace allergen (positive skin prick test)	Serial PEF chart OASYS-2 system, with WEI >2.5 generally considered positive	Airway hyper-reactivity present (fourfold difference in any measure†)	Positive workplace challenge‡	Specific allergen challenge
1	37, M	✓	✓ (2)	✓	✓ Flour	✓	✓ (1.88)	ND	ND	ND
2	43, M	✓	✓ (5)	✓	✓ Colophony	ND	ND	ND	ND	ND
3	43, M	✓	✓ (1)	ND	✓ Chrome	ND	✓ (1.8, 2.14)	ND	ND	ND
4	58, M	✓	✓ (2)	✓	—	×	ND	ND	ND	ND
5	60, F	✓	✓ (10)	×	—	ND	ND	×	ND	ND
6	59, M	✓	✓ (1)	✓	✓ Isocyanate	×	ND	✓ (✓)	✓	ND
7	54, M	✓	✓ (5)	✓	✓ Flour	×	ND	✓ (×)	ND	ND
8	34, F	✓	✓ (1)	✓	—	×	? ND	✓ (✓)	ND	ND
9	61, F	✓	✓ (7)	×	✓ Glut	×	? ND	ND	ND	ND
10	43, M	✓	✓ (22)	×	—	ND	ND	×	ND	ND
11	44, M	✓	✓ (2)	×	—	×	✓ (3.2)	✓ (✓)	ND	ND
12	59, F	✓	✓ (0)	×	✓ Glut	ND	✓ (1.38, 1.29 from 6 charts)	✓ (ND)	ND	ND
13	59, F	✓	✓ (5)	×	✓ Glut	×	ND	×	ND	ND
14	45, M	✓	✓ (0)	✓	—	ND	✓ (3.5, 1, 3.17 from 5 charts)	✓ (ND)	ND	ND
15	61, M	✓	?	✓	✓ Chrome	ND	✓ (2.17)	✓ (ND)	ND	ND
16	32, M	✓	✓ (1)	×	✓ Isocyanate	×	✓ (1, 2.5, 1.13)	×	ND	ND
17	44, M	✓	✓ (22)	✓	—	ND	✓ (1, 2.5)	×	ND	ND
18	57, F	✓	✓ (4)	×	—	×	✓ (2, 2.22, 1.25, 2)	×	ND	ND
19	64, M	✓	? ×	✓	✓ Isocyanate	×	✓ (3.75, 3.38)	ND	ND	ND

F, female; FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity; M, male; ND, not done; OASYS, Occupation Asthma SYStem; PEF, peak expiratory flow; WEI, work effect index; ✓, test performed and positive; ×, test performed and negative; ?, not clear from case notes; —, not exposed to common sensitiser.

*To one of the top nine known UK sensitisers—namely, isocyanate, laboratory animals, flour, paints, glutaraldehyde (glut), chrome, solder, enzymes and wood dust.

†Airway reactivity data. Information in brackets relates to sequential measures of airway reactivity if performed, and whether fourfold difference between periods of work and rest documented.

‡Defined as any PEF or FEV₁ falling below the 95% CI of the control days data.

not be summarised (eg, serial PEF measures) were photocopied and added to the case summary.

The summaries were circulated to all participating physicians, who were invited to rate, using the information available to them, the probability of occupational asthma for each case. They were also asked whether they would report the case to SWORD. Physicians were asked to work independently, and to complete the task within 1 week.

Phase 2

The physicians caring for each of the 19 patients were asked to prepare a narrative summary that was then appended to the clinical summary above. These were recirculated to the panel physicians who were asked to repeat the rating process as above. The individual scores from phase 1 were made available. Ten of the original panel physicians took part in phase 2.

Data presentation and analysis

All data were entered into and analysed using SPSS for Windows V. 12.0.1. The raw probabilities given for each case by each panel physician are presented, and a median and interquartile range for each case across all physicians, and for each physician across all cases has been calculated. Any missing data in cells were excluded from the calculation of the median.

The agreement between physicians was assessed using two statistical methods, Spearman's rank correlation and Cohen's κ analysis.

Spearman's rank correlation

All physician interactions over 19 cases were primarily assessed using a Spearman rank correlation coefficient. As all data were non-normal, this method of correlation assessment was used (SPSS V. 12.0.1). All interactions were systematically assessed

in this way, with a resulting correlation coefficient and p value quoted.

Cohen's κ analysis

The κ values were also calculated after categorising each probability scored by each physician as either high (>50) or low (<51). Each physician was then systematically compared with all other physicians in turn, generating a series of κ values representing all physician interactions. The probabilities generated from phases 1 and 2 were separately analysed in this way.

All cases were objectively assessed for content (eg, presence of bronchial hyper-responsiveness, presence of atopy) by one observer (DF), blinded to the probabilities generated by each case. These objective markers of content were used, along with physician identity, in an attempt to identify key features associated with either low or high probability assessment by the physicians.

RESULTS

Table 1 shows the clinical content of each case summary; these were highly variable. Only the documentation of a work-related respiratory symptom and the lack of specific allergen challenge test were common to all cases. Two cases seemed to have a history consistent with acute irritant-induced asthma (previously referred to as reactive airway dysfunction syndrome, cases 12 and 14), and all others but one had a clear latency between the onset of relevant exposures and the onset of work-related respiratory symptoms. Documented exposures were generally represented in the top current UK causes, but other exposures included solvents and thinners (case 4), tea dust (case 8) and latex (case 18).

Phase 1

Table 2 depicts the probability of occupational asthma for each case as individually rated by each panel physician in phase 1. In addition, this table notes whether each case was thought to represent occupational asthma by the attending physician. With regard to specific features of the case summaries that dictated a consistent probability rating, no clear consistent pattern emerged. Of the four case summaries (cases 6, 8, 11 and 14) with a median probability rating of $\geq 70\%$, all had documented airway hyper-reactivity, three with demonstrable variability between periods of work and rest. Of the four, two had an Occupation Asthma SYStem-2 (OASYS-2) serial PEF chart, both of which noted a work effect index >2.5 . The remaining two cases had no serial PEF data. Three did not list a commonly reported cause of occupational asthma in the occupational history. The remaining case mentioned exposure to isocyanate.

Of the four case summaries (cases 4, 5, 10 and 16) with median probability ratings of 25% or lower, none had evidence of hyper-reactive airways (although it was measured in three). Only one case had an OASYS-2 serial PEF assessment, and this chart had a work effect index of 2.5, generally considered as being evidence of a relevant work relationship.⁶ Again, three of the cases did not list a commonly reported cause of occupational asthma in the occupational history, and the remaining case mentioned exposure to isocyanate.

There seemed to be little influence of latency on probability ratings, although those with low probability tended to have longer latency periods documented. Only one individual underwent a workplace challenge (which was positive), and therefore any influence on the probability assessment of a specific bronchial challenge could not be assessed in this study.

The highest median probability was reported for case 11, whose summary documented a latency period of 2 years, a positive OASYS-2 chart, and sequential relevant changes in airway reactivity associated with periods of work and rest.

There was clear variation in the range of probability scored for each case. For example, case 16 (whose case summary is reproduced in box 1) generated a range of probabilities between 1% and 85%, with a median of 15%. By contrast, cases 3 and 11 generated narrow ranges (with a range width of 45% in each case).

It was also evident that certain physicians consistently reported either generally high probabilities (physicians 5, 6 and 10) or generally low probabilities (physicians 3 and 9).

Nevertheless, when agreement was assessed for all 66 physician-physician interactions, 45 of these were found to be significant to a value of $p < 0.05$. All but one Spearman rank correlation coefficient was positive. All but one physician agreed significantly with ≥ 5 of their colleagues.

When probability ratings were stratified to above or below the arbitrary cut-off of 50%, to assess agreement with the κ analysis, agreement was worse. The median κ value for all 66 physician interactions was 0.26 (range -0.2 to $+0.76$). In all, 24 of these interactions, as judged by individual κ coefficients were significant ($p < 0.05$); 7 of the 12 physicians agreed significantly with ≥ 5 of their 11 colleagues.

To assess whether the amount of information provided increased the absolute probability, or increased agreement between the physicians, ranges of probability for each case were compared between the 13 cases, where either bronchial responsiveness measures or OASYS-2 serial PEF measures were measured and supplied for the case summary, denoted good information, and the 6 cases where these data were absent (or just a single negative airway reactivity measure was documented) denoted limited information. There were no clear differences in the ranges of probability between those cases

with good or limited information. Specifically, the geometric mean difference between maximum and minimum probability rated for the 6 cases with limited information was 64.8 compared with 63.7 for cases with good information. If the three cases with neither bronchial responsiveness nor OASYS-2 data were compared with the 16 with some data, the geometric mean of the range was 69.3, compared with 63 for the rest. Both these comparisons were not statistically different at the 5% level.

With regard to the individual study physicians, a differing background did not seem to influence the rated probabilities. In particular, the three physicians not currently working as consultant respiratory physicians (but all with considerable experience of occupational lung disease) had median probabilities for all 19 cases of 35%, 60% and 60%, within the range of median probabilities for all physicians, 20–70%. Furthermore, although none of these three individuals agreed with ≥ 5 of the remaining study physicians significantly as assessed by κ analysis, this was also true for two of the respiratory physicians. The extremes of the κ value range (-0.2 and 0.76) were also not determined by these three physicians, as these interactions were both between respiratory physicians.

Phase 2

After reassessing of each case in light of the new case summary, the median κ was essentially unchanged at 0.28 (range -0.05 to 0.75).

DISCUSSION

This study has documented current diagnostic practice between six centres assessing relatively large numbers of potential cases of occupational asthma. The cases were selected purposefully from a larger observational cohort, recruited into each of the centres over a 12-month period. The cases were not intended to be highly investigated textbook examples of occupational asthma, but a range of cases reflecting the practice of each study centre. Transcription of the information from the original case notes produced summaries that were variable in their content.

The primary outcome measure used during the probability assessment was the rating of likely occupational asthma between 0% and 100%. This format was chosen so that the ratings could be treated as continuous variables, or dichotomised to assess levels of agreement.

It was thought appropriate to include all 12 physicians in the final analysis, including the single specialist registrar and two legislators. All had contributed to the study design and their inclusion allowed assessment of a range of opinions. Although the opinions of these three raters differed in some respects, they were broadly similar in range and median to the respiratory physicians. Neither did the inclusion of these physicians increase the extremes of κ values noted for all physician-physician interactions.

Each case generated a wide interquartile range of probabilities, although certain cases seemed to generate particularly disparate ratings. Those cases rated highly seemed to have a combination of features, including bronchial responsiveness measures and OASYS-2 style PEF charts. Although there were relatively low numbers of cases for further group analysis, there did not seem to be an influence of the amount of information supplied to the physician, with equally wide ranges of opinion in cases with good data.

Agreement as assessed by non-parametric correlation was generally good, with 11 of the 12 physicians agreeing with ≥ 5 of their colleagues significantly. Agreement assessed by κ analysis was predictably less good.

Table 2 Phase one: probabilities of each case representing occupational asthma

Case	OA	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	Median for each physician	Upper limit of IQR	Lower limit of IQR
Physician																							
1		80	90	55	40	30	60	50	70	40	20	80	40	30	50	30	40	30	45	60	45	60	35
2		60	—	20	20	15	95	40	70	10	35	55	35	25	75	40	30	20	35	51.25	35	51.25	20
3		70	15	65	1	0	30	10	30	65	10	80	10	30	80	20	10	20	10	47.5	20	47.5	10
4		55	40	60	10	70	60	30	55	30	10	90	20	45	65	55	15	30	30	40	40	57.5	30
5		70	—	49	—	—	70	49	70	—	51	100	40	—	95	55	—	55	70	90	70	70	51
6		75	51	60	25	25	97	51	80	51	51	100	51	45	90	70	85	75	51	80	60	80	51
7		50	50	50	70	5	100	70	100	0.5	0.5	80	0.5	1	90	5	1	1	1	65	50	70	1
8		70	30	50	20	0	70	40	70	40	30	75	10	20	55	30	10	20	40	70	40	62.5	20
9		30	30	30	20	30	80	30	70	30	20	60	30	30	30	30	40	30	20	60	30	50	30
10		75	50	60	0	0	50	60	60	60	50	70	50	60	80	70	0	30	70	30	60	65	40
11		30	60	50	20	30	90	10	60	50	50	80	30	20	75	40	10	30	70	80	50	65	30
12		40	30	50	25	15	40	25	45	35	20	60	25	30	60	40	35	25	30	65	35	42.5	25
Median for each case		65	45	52.5	20	15	70	40	70	40	25	80	30	30	75	40	15	30	35	72.5			
Upper limit of IQR for each case		71.3	50.8	60	25	30	91.3	50.3	70	50.5	50	82.5	40	37.5	82.5	55	37.5	30	55.8				
Lower limit of IQR for each case		47.5	30	50	15	2.5	57.5	28.8	58.8	30	17.5	67.5	17.5	22.5	58.8	30	10	20	20	38.8			

IQR, interquartile range; OA, likely diagnosis of occupational asthma at first presentation, made by treating physician; —, not supplied.

Box 1 Case 16 as shown to the assessing physicians

Male

32 years old

Refrigeration company

Engineer, making refrigerators

Exposures: polyol, isocyanate, fibre glass, wax, dust

Has worked at this site since 1993. For the first 2–3 years he was a stores controller, handing out products through a hatch, no exposure to foam. Also worked on electrical sump assembly line with occasional exposure to soldering fumes.

He then worked for the last 2–3 years on a foam injection line as a metal fabricator making the outer casings of fridges/deep freezers, which entails assembling the outer casing of fridges on a jig using galvanised or stainless steel. Liquid isocyanate is piped from a central tank, unheated to a spray gun, which sprays isocyanate and polyol into a mould.

The above process takes place under an extracted hood. The operator wears an external, air-fed visor during the procedure and for 5 min thereafter. He also wears gloves and overalls. He first reported symptoms to his general practitioner as he has never seen an occupational health physician at work. He continues to work in the same job. He was referred to secondary care by a general practitioner.

Social history: never been a smoker

Treatment: not taking any current medication

Medical history: asthma in childhood, not problematic until the 1980s

Occupational history:

1987–91: Engineering, apprentice in machine tool setting

1991–2: Making die castings for car mirror assembly

1992–3: Industrial enamelling as a patrol inspector

1993–present day as above

Documented work-related symptoms

Wheezing: onset 1 November 2000

Cough: onset 1 November 2000 (worse at night)

Documented lung function:

6 September 2001: FEV₁ 3.78 (3.99); FVC 4.96 (4.73)30 November 2001: FEV₁ 3.58; FVC 4.7418 October 2002: FEV₁ 3.88; FVC 5.02

Documented bronchial responsiveness, 18 October 2002:

PD₂₀ >4800 µg methacholine, 14% fall in FEV₁, no symptoms

Documented specific bronchial challenge: not done

Documented immunological assessment: 6 September 2001

Skin tests: positive to house dust mite

22 October 2001

Specific IgE to isocyanates: TDI, MDI, HDI, all negative

Documented peak flow monitoring:

OASYS-2 plots:

11 June 2001–1 July 2001: Work Effect Index 1

7 May 2001–27 May 2001: Work Effect Index 2.5 (fig 1)

10 September 2001–4 November 2001: Work Effect Index 1.13

Chest x ray normal

Few studies have specifically addressed diagnostic reasoning in occupational asthma, and the potential reasons for differences in agreement.

A recent study has dealt with the agreement between expert assessments of serial PEF charts.⁷ Similar in design to this

Figure 1 Occupation Asthma SYstem (OASYS-2) plot for case 16: shaded areas represent periods of work. Standard OASYS-2 format plot of serial peak expiratory flow (PEF) shown on the vertical axis, with individual days shown on the horizontal axis. The bold stepwise line notes the average PEF taken for the day, the upper representing the maximum PEF recorded for the day, and the lower the minimum for the day.

study, various respiratory physicians were asked to rate OASYS-2 charts for a probability of occupational asthma. This study noted a median κ of agreement on occupational effect of 0.83 (range 0.56–0.94), and lower agreement on the diagnosis of asthma alone. One possible explanation cited was the speed with which physicians were asked to complete the task, although other smaller studies^{9 10} have found broadly similar results.

It is reasonable to assume, therefore, that some of the variations seen in our study relate to the already noted differences in serial PEF chart interpretation using an accepted technique⁷; only 10, however, of the 19 case summaries contained such data, and other sources of variability must also be important.

To our knowledge, no such assessments have been made of any other diagnostic procedure for occupational asthma (eg, immunological assessment or bronchial responsiveness), and in particular how a result determines the likely eventual diagnosis of occupational asthma being made.

Median scores for each physician varied widely, with some physicians commonly grading >50%, whereas others commonly graded <50%. These differences are difficult to explain, although contributing factors could include the type of training received, case mix seen, and working for claimants or defendants in medicolegal practice.

Although local,¹⁰ European¹¹ and international guidance^{12 13} on diagnosis exist, these are not consistent, and there is little help to the physician in weighting various relevant investigations, and their combination when making or excluding a

Main messages

- Making an accurate diagnosis of occupational asthma is important, and of equal importance is avoiding mislabelling workers without this condition.
- Although UK-based physicians with particular interest in occupational asthma broadly agree with one another about diagnosis when assessing paper summary cases, areas of disagreement exist.

diagnosis. The recent British Occupational Health Research Foundation guidelines¹⁴ include a compilation of the evidence pertaining to the diagnosis of occupational asthma; they place more reliance on serial measures of PEF than on single or sequential measures of airway responsiveness, although this point remains under debate. Although this research exercise is no substitute for personal history taking and patient contact, and the lack of such clinical contact and free discussion with the patient may explain a significant proportion of the variation seen, further work is needed to benchmark our domestic situation, and help inform a national standard of care for occupational asthma. Nationally approved evidence based guidance may lead in time to more similar practices between physicians assessing such cases.

Authors' affiliations

David Fishwick, Lisa Bradshaw, Mandy Henson, Chris Barber, Andrew D Curran, Centre for Workplace Health, Health and Safety Laboratory, Harpur Hill, Buxton, Derbyshire, UK

Chris Stenton, David Hendrick, Royal Victoria Infirmary, Newcastle upon Tyne, UK

Sherwood Burge, Occupational Lung Disease Unit, Birmingham Heartlands Hospital, Birmingham, UK

Rob Niven, Tony Pickering, North West Lung Centre, Wythenshawe Hospital, Manchester, UK

Chris Warburton, University Hospital, Aintree, Liverpool, UK

Trevor Rogers, Doncaster Royal Infirmary, Doncaster, South Yorkshire, UK

Roger Rawbone, Nerys Williams, Health and Safety Executive, UK

Paul Cullinan, Royal Brompton Hospital, London, UK

Jon Ayres, Department of Environmental and Occupational Medicine, University of Aberdeen, Aberdeen, UK

REFERENCES

- 1 **Blanc PD**, Toren K. How much adult asthma can be attributed to occupational factors? *Am J Med* 1999;**107**:580–7.
- 2 **Meredith S**, Nordman H. Occupational asthma: measures of frequency from four countries. *Thorax* 1996;**51**:435–40.
- 3 **American Thoracic Society Statement**. Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003;**167**:787–97.
- 4 **Montanaro A**. Prognosis of occupational asthma. *Ann Allergy Asthma Immunol* 1999;**83**(Pt 2):593–6.
- 5 **Baldwin DR**, Gannon P, Bright P, *et al*. Interpretation of occupational peak flow records: level of agreement between expert clinicians and OASYS-2-2. *Thorax* 2002;**57**:860–4.
- 6 **Gannon PF**, Newton DT, Belcher J, *et al*. Development of OASYS-2-2: a system for the analysis of serial measurement of peak expiratory flow in workers with suspected occupational asthma. *Thorax* 1996;**51**:484–9.
- 7 **Baldwin DR**, Gannon P, Bright P, *et al*. Interpretation of occupational peak flow records: level of agreement between expert clinicians and OASYS-2-2. *Thorax* 2002;**57**:860–4.
- 8 **Malo JL**, Cote J, Cartier A, *et al*. How many times per day should peak expiratory flow rates be assessed when investigating occupational asthma? *Thorax* 1993;**48**:1211–17.
- 9 **Lerooyer C**, Perfetti L, Trudeau C, *et al*. Comparison of serial monitoring of peak expiratory flow and FEV1 in the diagnosis of occupational asthma. *Am J Respir Crit Care Med* 1998;**158**:827–32.
- 10 **British Thoracic Society; Scottish Intercollegiate Guidelines Network**. British guideline on the management of asthma. *Thorax* 2003;**58**(Suppl 1):i1–94.
- 11 **Moscato G**, Godnic-Cvar J, Maestrelli P, *et al*. Statement on self-monitoring of peak expiratory flows in the investigation of occupational asthma. Subcommittee on Occupational Allergy of the European Academy of Allergy and Clinical

- Immunology. American Academy of Allergy and Clinical Immunology. European Respiratory Society. American College of Allergy, Asthma and Immunology. *Eur Respir J* 1995;**8**:1605–10.
- 12 **Chan-Yeung M.** Assessment of asthma in the workplace. ACCP consensus statement. American College of Chest Physicians. *Chest* 1995;**108**:1084–117.
- 13 **Tarlo SM,** Boulet LP, Cartier A, *et al.* Canadian Thoracic Society guidelines for occupational asthma. *Can Respir J* 1998;**5**:289–300.
- 14 **British Occupational Health Research Foundation.** *Guidelines for the prevention, identification and management of occupational asthma: evidence review and recommendations.* London: BOHRF, 2004.