



GUIDELINES UPDATE

Standards of care for occupational asthma: an update

David Fishwick,¹ Christopher Michael Barber,¹ Lisa M Bradshaw,¹ Jon G Ayres,² Richard Barraclough,³ Sherwood Burge,⁴ Jonathan M Corne,⁵ Paul Cullinan,⁶ Timothy Laszlo Frank,⁷ David Hendrick,⁸ Jennifer Hoyle,⁹ Andrew D Curran,¹⁰ Robert Niven,³ Tony Pickering,³ Peter Reid,¹¹ Alastair Robertson,⁴ Chris Stenton,⁸ Christopher J Warburton,¹² Paul J Nicholson¹³

► An additional appendix is published online only. To view this file please visit the journal online (<http://thorax.bmj.com/content/67/3.toc>).

¹Centre for Workplace Health, Sheffield, UK

²University of Birmingham, Birmingham, UK

³North West Lung Centre, Manchester, UK

⁴Heartlands Hospital, Birmingham, UK

⁵Queen's Medical Centre, Nottingham, UK

⁶Royal Brompton Hospital, London, UK

⁷General Practice Research Unit, Manchester, UK

⁸Royal Victoria Infirmary, Newcastle upon Tyne, UK

⁹North Manchester General Hospital, UK

¹⁰Centre for Workplace Health, Buxton, UK

¹¹Western General Hospital, Edinburgh, UK

¹²Aintree Chest Centre, University Hospital Aintree, Liverpool, UK

¹³British Occupational Health Research Foundation, Society of Occupational Medicine and Faculty of Occupational Medicine, London, UK

Correspondence to

Professor D Fishwick, Centre for Workplace Health, Respiratory Function Unit, A Floor, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK; d.fishwick@sheffield.ac.uk

Received 6 July 2011

Accepted 18 November 2011

Published Online First

9 December 2011

ABSTRACT

Background The British Thoracic Society (BTS) Standards of Care (SoC) Committee produced a standard of care for occupational asthma (OA) in 2008, based on a systematic evidence review performed in 2004 by the British Occupational Health Research Foundation (BOHRF).

Methods BOHRF updated the evidence base from 2004–2009 in 2010.

Results This article summarises the changes in evidence and is aimed at physicians, nurses and other healthcare professionals in primary and secondary care, occupational health and public health and at employers, workers and their health, safety and other representatives.

Conclusions Various recommendations and evidence ratings have changed in the management of asthma that may have an occupational cause.

BACKGROUND

The British Thoracic Society (BTS) Standards of Care (SoC) Committee produced a standard of care for occupational asthma (OA) in 2008,¹ based on a systematic evidence review performed in 2004 by the British Occupational Health Research Foundation (BOHRF).² BOHRF updated the evidence base from 2004 to 2009 in 2010.³

This article summarises the changes in evidence and is aimed at physicians, nurses and other healthcare professionals in primary and secondary care, occupational health and public health, and at employers, workers and their health, safety and other representatives.

STANDARD OF CARE UPDATE

It is not intended, nor should it be taken to imply, that these amendments to the SoC override existing legal obligations, for example the Health and Safety at Work Act 1974, the Management of Health and Safety at Work Regulations 1999, the Equality Act 2010, the Control of Substances Hazardous to Health Regulations 2002 and other relevant legislation.

General comments

The recent evidence supports the estimate that occupational factors account for one in six cases of adult asthma. The range of incidence for cases of OA has been upwardly revised to between 12 and 300 cases/million workers/year. Under-identification of OA persists.⁴

All the following statements are referenced to the 2010 BOHRF review,³ as details of the evidence review update, full references to statements in this document, an audit tool and case management can be found in the associated online supplement.

Prevention and health surveillance

Further evidence supports the role of health surveillance for identifying OA at an earlier stage, although screening questionnaires have significant false-negative response rates. Developing a workplace culture that supports workers to report symptoms accurately is key, as is workers' knowledge of a plan of action were they to report various work-related respiratory complaints.

Forced expiratory volume in 1 s (FEV₁) and forced vital capacity (FVC) measured to screen for OA are likely to detect few cases that would not otherwise be detected by respiratory questionnaire alone.

Education

All workers exposed to asthmagens should be warned about relevant symptoms potentially attributable to exposure, how agents in the workplace can affect health, and how best to avoid problems. Workers should be informed what to do, and in particular to whom they should report, if they develop relevant symptoms, particularly if these occur between health surveillance visits.

Educational programmes should be aimed at employers and healthcare professionals, including nurses and doctors (based in industry, primary and secondary care), occupational hygienists, and workers.

Diagnostic process

Health practitioners who suspect a worker of having OA should make an early referral to a physician with expertise in OA.

All those involved in the potential identification of OA have an obligation to minimise delays.

Medical history

The latest evidence supports the importance of nasal symptoms in addition to asthma symptoms. Specifically, rhino-conjunctivitis may precede or coincide with the onset of OA, and the risk of OA development is highest in the year following the onset of rhino-conjunctivitis.

Occupational history

Hairdressers have been added to the list of workers with OA most commonly reported to schemes.

Data from recent population studies also identify that cooks, healthcare workers, woodworkers and mechanics are also at greater risk of reporting asthma. The most frequently reported agents causing OA have been expanded to include adhesives, metals, resins in addition to isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes and wood dust.

The full list of most commonly reported agents, workers and jobs from population-based studies with elevated OA risks are given in table 2 in the online appendix.

Investigations

Lung function

All suspected cases of OA should undergo FEV₁ and FVC measurement according to agreed criteria, and the results compared with a predicted value and previous results if available.

Pre-shift to post-shift changes in lung function have high specificity but only low sensitivity for OA. If these changes are present, they may support a diagnosis, but they are frequently absent in people subsequently confirmed to have OA. It is recommended that pre-shift and post-shift FEV₁ changes are only used in conjunction with other diagnostic approaches.

There is a considerable evidence base for the use of serial peak expiratory flow (PEF) measurements to investigate workers when OA is suspected. With appropriate training and explanation, it is possible to achieve high-quality recordings in these workers. While these tests may be susceptible to falsification and transcription errors, they offer the best and easiest first-line approach to assessing physiological response to asthmagens at work. High-quality recordings can be obtained for over 70% of patients.

Serial PEF should be recorded at least four times a day for at least three continuous weeks. Recordings for shorter durations are of lower diagnostic value. It is best to aim for readings every 2 h, so that practically at least four good measures per day will be achieved. Suitable record forms can be downloaded from <http://www.occupationalasthma.com> or <http://www.scottish-shield.org>. Ideally, inhaled steroids should be withheld until the series is completed or required doses kept constant and as low as possible.

If the person is currently not exposed at work, serial PEF can be measured during a 2-week 'run in' period, followed by a return to work. All recordings should be entered onto a computer for analysis using suitable software. Computer-based analyses of PEF may be helpful in the diagnosis of OA. At least one software program calculates a work-effect index from discriminant analysis based on pattern recognition. Such analysis allows charts to be graded positive, equivocal or negative for a diagnosis of OA. A positive chart has a quoted sensitivity of approximately 75% and a specificity of 95% for a diagnosis of OA, although these estimates are quality dependent, and pooled estimates suggest 64% sensitivity (95% CI 43% to 80%) and a specificity of 77% (95% CI 67% to 85%).⁵ It is important to note that PEF charts do not confirm a specific cause, nor do they distinguish OA from work-aggravated asthma. Any problems with interpretation of serial PEF charts should be referred to a specialist centre.

Immunological testing

New evidence supports skin prick and serological testing as sensitive for detecting specific IgE caused by most high molecular weight agents, but these tests are not specific for diagnosing asthma or OA. Skin prick and serological tests are less sensitive for detecting specific IgE and OA caused by low

molecular weight agents and while specificity may be higher they are not specific for diagnosing OA.

Non-specific bronchial responsiveness

While assessment of non-specific bronchial responsiveness is a useful diagnostic investigation, single and serial measures have only moderate specificity and sensitivity for the validation of OA.

Specific broncho-provocation testing

Comments relating to specific broncho-provocation testing remain unchanged. These tests should be performed only in specialised (tertiary) centres. A positive test identifies the cause of OA, provided exposures received are equivalent to those in the workplace. Negative tests do not necessarily exclude OA, as the challenge may not adequately reproduce exposures at work. Exposure received during SBPT should be measured if practical.

Alternatively, workplace challenge may be used (a variation of serial PEF or serial FEV₁ measurements). This usually involves frequent monitoring of FEV₁ or PEF on multiple days of work, during and between periods of exposure to the suspected agents. One way of approaching this type of challenge is to take data from non-exposed days to calculate a mean and 95% CI of the 'expected' FEV₁ at each time point. These are compared with FEV₁ values measured on exposed days.

Other tests

Newer techniques are available to investigate potential cases of OA. The role of fractional exhaled nitric oxide measurements in the diagnosis of OA has not been established. A normal value does not exclude a diagnosis of OA.

With regard to sputum eosinophilia for the diagnosis of OA, such measurements may be helpful in the diagnosis of OA, although the absence of sputum eosinophilia does not exclude a diagnosis of OA.

Management

Medical management

The pharmacological management of OA does not differ from the management of asthma that is not work related (summarised by the BTS at <http://www.brit-thoracic.org.uk/>). Once a diagnosis of OA is confirmed, the patient should be advised (preferably verbally and in writing) that the prognosis is improved by early and complete removal from exposure.

Symptoms and functional impairment associated with OA may persist for many years after avoidance of further exposure to the causative agent. Evidence supports the view that OA may become a chronic condition, similar to non-OA, and may require similar prolonged medical management.

Patients with confirmed or possible OA should be followed up at a specialist centre while risks of continuing exposure to allergen remain. The recommended follow-up is every 3 months for 1 year, and then every 6 months thereafter.

Patients with confirmed OA who have left work, or who have no ongoing asthmagen exposure risk, should be followed up for a minimum of 3 years at a specialist centre.

Communicating with the workplace is useful, but requires the patient's written consent. Patients should be informed of the possible adverse health effects of continuing exposure to themselves and to co-workers should they not permit necessary workplace investigations.

Acknowledgements The authors would like to acknowledge the British Occupational Health Research Foundation, and the assistance of Professor Sir Anthony Newman Taylor.

Competing interests None.

Contributors All authors significantly contributed to the formulation of the standard of care given the recent evidence-based review, and all were involved in writing and developing the document.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. **Fishwick D**, Barber CM, Bradshaw LM, *et al*; British Thoracic Society Standards of Care Subcommittee Guidelines on Occupational Asthma. Standards of care for occupational asthma. *Thorax* 2008;**63**:240–50.

2. **Nicholson PJ**, Cullinan P, Newman Taylor AJ, *et al*. Evidence based guidelines for the prevention, identification, and management of occupational asthma. *Occup Environ Med* 2005;**62**:290–9.
3. **Occupational Asthma—Identification, Management and Prevention: Evidence Based Review and Guidelines**. British Occupational Health Research Foundation, 2010. ISBN 978-0-9564979-1-8. <http://www.bohrf.org.uk/downloads/OccupationalAsthmaEvidenceReview-Mar2010.pdf> (accessed 20 Sep 2011).
4. **Orriols R**, Costa R, Albanell M, *et al*. Reported occupational respiratory diseases in Catalonia. *Occup Environ Med* 2006;**63**:255–60.
5. **Tarlo SM**, Balmes J, Balkissoon R, *et al*. Diagnosis and management of work-related asthma: American College of Chest Physicians Consensus Statement. *Chest* 2008; **134**(Suppl 3):1S–41.

Pulmonary puzzles

ANSWER

From the question on page 273

Lymphomatoid granulomatosis (LYG) grade 1 was diagnosed. LYG is a rare T-cell rich, Epstein-Barr virus (EBV)-associated, B-cell lymphoproliferative disorder. Lung involvement is the predominant feature but extrapulmonary manifestations occur, especially skin and nervous system involvement such as optic neuritis.^{1 2} Systemic symptoms and lymphopenia are also described. Lung Involvement in LYG may be unilateral or bilateral and includes interstitial and alveolar compartments of the lung. The most common radiographic findings are multiple nodular opacities and masses with poorly defined margins distributed along the peribronchovascular bundle or interlobular septa.³ Reticular opacities associated with nodules, mediastinal lymphadenopathy and large conglomerate masses with air bronchograms are also observed.³

Histologic and immunohistochemical features are crucial for LYG diagnosis: mixed mononuclear cell infiltrate containing CD20-positive large B lymphocytes and numerous CD3-positive small lymphocytes, vascular infiltration, necrosis, and evidence of EBV infection.¹ Positivity for EBV infection in histological specimens ranges from 57 to 100% and therefore its absence does not rule out the diagnosis in proper clinical, radiological and histological scenarios, as was the case for our patient.^{1 4}

Grade 1 LYG contains only a few large B cells whereas numerous large B cells are present in grade 3 disease, which means worse prognosis. Current knowledge about LYG tends to categorise grades 2 and 3 as lymphomas, considering the

frequent presence of monomorphous foci of atypical B cells and the clinical behaviour.^{1 5}

The treatment of LYG is not well established. There has been a tendency to treat higher grade LYG with chemotherapy regimens for lymphomas but the treatment for grade 1 LYG is less clear.¹ There are some reports of spontaneous resolution and treatment success with interferon alfa-2b, corticosteroids, cyclophosphamide, and bone marrow transplant. Our patient was treated with corticosteroids, cyclophosphamide and rituximab with complete remission of lung lesions. The optic neuritis also remitted but the patient retained significant visual impairment. The patient was placed on prednisone and oral cyclophosphamide with slow tapering in the following 18 months. There have been no recrudescences in the last 3 years of follow-up.

Thorax 2012;**67**:280. doi:10.1136/thoraxjnl-2011-200573

REFERENCES

1. **Katzstein AL**, Duxtader E, Narendra S. Lymphomatoid granulomatosis: insights gained over 4 decades. *Am J Surg Pathol* 2010;**34**:e35–48.
2. **Myers JL**, Kurtin PJ, Katzstein AL, *et al*. Lymphomatoid granulomatosis. Evidence of immunophenotypic diversity and relationship to Epstein-Barr virus infection. *Am J Surg Pathol* 1995;**19**:1300–12.
3. **Lee JS**, Tuder R, Lynch DA. Lymphomatoid granulomatosis: radiologic features and pathologic correlations. *AJR Am J Roentgenol* 2000;**175**:1335–9.
4. **Nicholson AG**, Wotherspoon AC, Diss TC, *et al*. Lymphomatoid granulomatosis: evidence that some cases represent Epstein-Barr virus-associated B-cell lymphoma. *Histopathology* 1996;**d29**:317–24.
5. **Pittaluga S**, Wilson WH, Jaffe ES. Lymphomatoid granulomatosis. In: Swerdlow SH, Campo E, Harris NL, eds. *WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues*. Lyon: IARC, 2008:247–9.

ONLINE SUPPLEMENT

Appendix A

Evidence Review Update

EVIDENCE REVIEW UPDATE

BOHRF methodology is described in the 2004 and 2010 systematic reviews.[1,2] Studies addressing research questions were identified using MEDLINE and EMBASE, selected papers being critically appraised and graded using the modified Royal College of General Practitioners (RCGP) system and the Scottish Intercollegiate Guidelines Network (SIGN) system.

Updated evidence is discussed sequentially in the domains; (A) background, (B) prevention, (C) identification and evaluation of OA in a symptomatic worker and (D) management of the worker confirmed to have OA.

A. Background;

A1; BOHRF 2010 states "Occupational factors are estimated to account for 1 in 6 cases of adult asthma". This alters the original similar estimate of 15% of adult asthma, based on new evidence from Toren *et al* (2009).[3] The level of evidence for this statement is upgraded from ***SIGN 2++ to ***SIGN 1++.

A2; the incidence for cases of OA has been upwardly revised to between 12 and 300 cases/workers/year, based on new data, including from Bakerly *et al*,[4] Kogevinas *et al*,[5] McDonald *et al* [6] and Orriols *et al*. [7]

A3; The evidence rating for the statement "the incidence of OA identified by reporting schemes may be significantly underestimated" has increased to *SIGN 2+ from *SIGN 3, supported by data [7] identifying that the number of cases reported to a voluntary surveillance system were four fold that reported by the compulsory official system.

A4; most frequently reported agents causing OA are expanded to include adhesives, metals, resins in addition to isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes, and wood dust.

A5; hairdressers are added to the list of workers most commonly reported to reporting schemes with OA.

A6; added to the list of workers reported from population studies are cooks, healthcare workers, woodworkers and mechanics.

A12; the 2010 review states that rhinoconjunctivitis may precede or coincide with the onset of OA, now emphasising that rhinoconjunctivitis may not always precede the onset of OA.

A13; the statement "the risk of developing OA is highest in the year after the onset of occupational rhinitis" remains unchanged, but has a stronger evidence rating (***SIGN 2++) based on cohort and cross-sectional studies [8-10] not included earlier.

B. Prevention of OA;

B4; new data from a large study of workers with possible OA identified by health surveillance [11] further supports the usefulness of health surveillance for detecting OA early.

B6; added comment supports the earlier view that screening questionnaires used for case-finding generally underestimate the presence of OA. The strength of evidence is upgraded (**SIGN 2++), based on a study comparing results from a cohort of workers to a cross-sectional survey.[12] This noted significant disparity between possible OA identified by "in-

house" health surveillance and a research project in bakeries.

B7; new evidence supports the statement that spirometry detects few cases of OA that would not otherwise be detected by respiratory questionnaire. The level of evidence was strengthened to **SIGN 2+ [from *SIGN 3] after re-evaluating the original study designs (cohort and cross-sectional studies).

C. Identification and evaluation of a case of OA in the worker presenting with respiratory symptoms;

C3; pre to post shift changes in lung function are again assigned a relatively low strength evidence for the statement that suggests that these tests have high specificity but only low sensitivity for a diagnosis of OA. Further data [13] are cited, assessing mean peak expiratory flow (PEF) changes across morning and day shifts and compared these between workers with OA confirmed using specific challenge testing and non-working asthma patients. Serial analysis using mean work-rest day PEF comparison had a sensitivity of 66.7% and a specificity of 100% for making a diagnosis of OA, whilst cross-shift changes in PEF in morning / day-shift workers had a poor sensitivity.

Substantial evidence supports the use of serial PEF measures as a useful investigation for occupational asthma. [14-20]

C4; the statement regarding the ability to obtain acceptable PEF readings is strengthened to ***SIGN 2++ [from **SIGN 3] adding "in specialist settings". Recent evidence suggests that specialist clinics are superior in obtaining good quality PEF data compared to "other health units". Sauni *et al* [21] assessed quality of diagnostic procedures, reviewing case notes of 150 patients referred to the Finnish Institute of Occupational Health with a suspected occupational cause of their asthma. Workplace measurements of serial PEF were performed in 51% of all cases; quality of measurements being "sufficient" in 52%. Serial PEF measurements were performed significantly ($p<0.01$) less often in other health clinics (23%) compared to occupational health (56%) or respiratory clinics (59%).

C5; the statement that the diagnostic performance of serial PEF measurements taken to investigate potential OA falls when fewer than 4 readings a day are taken is modified, adding "when records are shorter than three weeks in duration", based on a case series. The evidence rating remains unaltered.[22]

C6; minor alteration is made to the evidence statement regarding expert agreement when interpreting serial PEF, the new statement noting *SIGN 3 evidence for moderate agreement between experts.

C7; evidence relating to high sensitivity and specificity of serial PEF for a diagnosis of OA is strengthened to ***SIGN 1++, additional comment emphasising that these are quality dependent.

C8; this newly worded statement is assigned a reduced strength of evidence [*SIGN 3], noting "computer-based analyses of peak flow records may be helpful in the diagnosis of OA".

Assessment of non specific bronchial responsiveness (NSBR) may assist making a diagnosis of occupational asthma.[23]

C9; the section discussing single measurements of NSBR is expanded to include pooled estimates of sensitivity and specificity using specific bronchial provocation tests (SBP) as the reference standard.[24] Evidence statement C9 notes that a single measurement of non-specific reactivity has only moderate specificity and sensitivity for the validation of OA and is graded ***SIGN 1++.

C10; regarding temporal changes in NSBR, this statement notes: "changes in NSBR at and away from work alone have only moderate sensitivity and specificity for diagnosis (of OA) and

was downgraded to *SIGN 3 after re-evaluating studies, all being case series. The evidence that these measures are achievable remains unchanged (statement c11; *SIGN 3).

C12; A meta-analysis provided pooled estimates [24] of sensitivities and specificities of specific skin prick tests and serum specific IgE compared to SPBT. The two evidence statements are strengthened as follows; c12; both skin prick and serological tests are sensitive for detecting specific IgE and OA caused by most high molecular weight agents but are not specific for diagnosing asthma [*** SIGN1++] and; c13; overall, both skin prick and serological tests are less sensitive for detecting specific IgE and OA caused by low molecular weight agents and while specificity may be higher they are not specific for diagnosing asthma *** SIGN1++.

Specific challenge testing [25-27] and workplace challenge testing [28] remain important investigations for occupational asthma.

The 2010 review is expanded to include newer diagnostic modalities of exhaled nitric oxide (FeNO) and sputum eosinophilia, there being little evidence for these tests previously.

C15 states: "the role of FeNO measurements in the diagnosis of OA is not established" *SIGN 3.

C16 concludes: "In the clinical setting a normal FeNO does not exclude a diagnosis of OA", *SIGN3.

Two evidence statements are included on the utility of sputum eosinophilia for the diagnosis of OA: both are graded *SIGN 3. These are C17 - the measurement of sputum eosinophils may be helpful in the diagnosis of OA and C18 - in the clinical setting the absence of sputum eosinophilia does not exclude a diagnosis of OA. Both statements were based on a recent studies including a meta analysis.[24]

D. Management principles for the worker confirmed to have OA

The inclusion of a meta-analysis [29] upgraded the evidence to *** SIGN 1++ for the statement D1; The symptoms and functional impairment of OA may persist for many years after avoidance of further exposure to the causative agent.

A new statement is included regarding the utility of reducing exposure; D7 [** SIGN 2+], although based solely on evidence related to exposure to natural rubber latex gloves.[30] Where clinical considerations permit, reduction of exposure may be a useful alternative associated with fewer socio-economic consequences to complete removal from exposure.

A new evidence statement D13, based in part on data from Fishwick *et al* [31] discusses referral delays, concluding: "lengthy diagnostic delay occurs for patients with OA" ** SIGN 3.

OCCUPATIONAL ASTHMA
Table 1
Summary of major BOHRF recommendations for health practitioners

<p>* SIGN 3; The positive predictive values of screening criteria are too poorly discriminating for screening out potentially susceptible individuals, particularly in the case of atopy where the trait is highly prevalent.</p> <p>** SIGN 2+; The likelihood of improvement or resolution of symptoms or of preventing deterioration is greater in workers who have little or no further exposure to the causative agent.</p> <p>** SIGN 2+; Occupational rhinitis and OA frequently occur as co-morbid conditions.</p> <p>** SIGN 2+; Rhinoconjunctivitis is more likely to appear before the onset of IgE associated OA.</p> <p>*** SIGN 2++; The risk of developing OA is highest in the year after the onset of occupational rhinitis.</p> <p>*** SIGN 1++; Occupational factors are estimated to account for about 1 in 6 cases of asthma in adults of working age, including new onset or recurrent disease.</p> <p>*** SIGN 2++; The workers most commonly reported to surveillance schemes of OA include animal handlers, bakers and pastry makers, chemical workers, food processing workers, hairdressers, paint sprayers, nurses and other health professionals, timber workers and welders.</p> <p>** SIGN 2+; The workers reported from population studies to be at increased risk of developing asthma include bakers, chemical workers, cleaners, cooks, electrical and electronic production workers, farm workers, food processors, forestry workers, healthcare workers, laboratory technicians, mechanics, metal workers, painters, plastics and rubber workers, storage workers, textile workers, waiters, welders and wood workers.</p> <p>*** SIGN 2++; The most frequently reported agents include isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes and wood dust.</p> <p>** SIGN 2+; In the clinical setting questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holiday have a high sensitivity, but relatively low specificity for the validation of OA.</p> <p>** SIGN 2+; In the clinical setting questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holiday have a high sensitivity, but relatively low specificity for the validation of OA.</p> <p>* SIGN 3; Free histories taken by experts have high sensitivity, but their specificity may be lower. These values may be affected by differences in language and populations.</p> <p>** SIGN 2+; Approximately one third of workers with OA are unemployed up to 6 years after diagnosis.</p> <p>** SIGN 2+; Workers with OA suffer financially.</p> <p>** SIGN 3; In specialist settings acceptable peak flow series can be obtained in around two thirds of those in whom a diagnosis of OA is being considered.</p> <p>* SIGN 3; The diagnostic performance of serial peak flow measurements falls when fewer than four readings a day are made and records are shorter than three weeks.</p> <p>** SIGN 3; There is high level of agreement between expert interpretations of serial peak flow records.</p> <p>** SIGN 3; Depending on the quality of the recorded series, the sensitivity and specificity of serial peak flow measurements are high in the diagnosis of OA.</p>
--

Table 2
Common agents and jobs related to OA

Source	Agent or job
Most commonly reported agents causing OA;	Isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes, adhesives, metals, resins and wood dust
Workers most commonly reported to surveillance schemes of OA include;	Animal handlers, bakers and pastry makers, chemical workers, food processing workers, hairdressers, paint sprayers, nurses and other health professionals, timber workers and welders
Workers reported from population studies to be at increased risk of developing asthma include;	Bakers, chemical workers, cleaners, cooks, electrical and electronic production workers, farm workers, food processors, forestry workers, healthcare workers, laboratory technicians, mechanics, metal workers, painters, plastics and rubber workers, storage workers, textile workers, waiters, welders and wood workers.

Overall Case Management

Ideally, affected workers should be redeployed within the same workplace, in a non-exposed task, in order to minimise the consequences discussed below. In practice, this may not happen, and workers may remain exposed, or have their employment terminated. Where clinical considerations permit, reduction of exposure may be an acceptable alternative to complete removal from exposure, associated with fewer socio-economic consequences. Continued follow-up is essential in this situation to ensure symptoms and exposures are reduced and that lung function preserved. The use of software to assess individual (or group) FEV₁ decline with time (e.g. Spirola, TM, NIOSH, web address; <http://www.cdc.gov/niosh/topics/spirometry/spirola.html> last accessed 04.07.2011) is recommended.

Patients with OA should be told of the possibilities for compensation. Local context will alter regulations for compensation, e.g. in Great Britain they should be advised where appropriate about Industrial Injuries Disease Benefit and helped with this if necessary. More information can be found at; www.dwp.gov.uk/advisers/claimforms/ (last accessed 04.07.2011).

Patients should be advised about a potential civil claim where appropriate. In particular, patients should be told that if a civil claim is anticipated, a time limit might apply following the date of knowledge (the date the individual became aware that their asthma was occupational in origin) may apply beyond which it is not possible to commence a claim. This time period is usually 3 years in England. There are regional UK differences in Civil Law, and it is appropriate to tell patients to take advice from a personal injury lawyer, if they are contemplating legal action.

The Equality Act 2010 protects the rights of workers with various forms of disability. The legislation protects a disabled person thought to be at a "substantial disadvantage in comparison with persons who are not disabled". This means that employers have to make one or more reasonable adjustments for those satisfying the definition of disability by virtue of having asthma, whatever the cause.

Audit tool

The audit tool, against which clinical activity should be measured, is revised as follows. All patients with suspected OA should, as a minimum, have the following clearly documented in their health records.

By first visit

- Presence or absence of asthma prior to potentially harmful asthmagen exposure at work
- Presence or absence of work-related eye or nasal symptoms
- Presence or absence of work-related respiratory symptoms and their duration
- A full list of occupations held, their durations, and likely associated occupational exposures
- Current ongoing asthmagen exposure
- Whether other workers at the same workplace are affected
- FEV₁, FVC, and the degree of airflow limitation, compared to predicted values

By second visit

- If at work and appropriate; serial PEF measurements taken for at least 3 continuous weeks including rest days, with at least 4 good quality readings per day, analysed to assess work relatedness.
- If performed, the results of non specific bronchial responsiveness

- If exposed to allergen with appropriate specific IgE measure or skin prick test, the result of this test.

Once a diagnosis of OA is confirmed

- Letter to patient concerning advice about continuing employment
- Compensation advice (IIDB and civil action) where appropriate to the case

References

- 1 Nicholson PJ, Cullinan P, Newman Taylor AJ, Burge PS, Boyle C. Evidence based guidelines for the prevention, identification, and management of occupational asthma. *Occup Environ Med* 2005;**62**:290-299.
- 2 Occupational Asthma – identification, management and prevention: evidence based review and guidelines. British Occupational Health Research Foundation 2010. ISBN 978-0-9564979-1-8. Also found at ;
<http://www.bohrf.org.uk/downloads/OccupationalAsthmaEvidenceReview-Mar2010.pdf>. Last accessed 20.09.2011.
- 3 Toren K, Blanc P. Asthma caused by occupational exposures is common: A systematic analysis of estimates of the population-attributable fraction. *BMC Pulm Med* 2009;**9**:7.
- 4 Bakerly ND, Moore VC, Vellore AD *et al*. Fifteen-year trends in occupational asthma: data from the Shield surveillance scheme. *Occup Med (Lond)* 2008;**58**:169-174.
- 5 Kogevinas M, Zock J-P, Jarvis D *et al*. Exposure to substances in the workplace and new-onset asthma: an international prospective population-based study. *Lancet* 2007;**370**:336-341.
- 6 McDonald JC, Chen Y, Zekveld C *et al*. Incidence by occupation and industry of acute work related respiratory diseases in the UK, 1992-2001. *Occup Environ Med* 2005;**62**:836-842.

7 Orriols R, Costa R, Albanell M *et al.* Reported occupational respiratory diseases in Catalonia. *Occup Environ Med* 2006;**63**:255-260.

8 Gautrin D, Ghezze H, Infante-Rivard C *et al.* Natural history of sensitisation, symptoms and occupational diseases in apprentices exposed to laboratory animals. *Eur Respir J* 2001;**17**:904-908.

9 Gross NJ. Allergy to laboratory animals: epidemiologic, clinical, and physiologic aspects, and a trial of cromolyn in its management. *J Allergy Clin Immunol* 1980;**66**:158-165.

10 Skjold T, Dahl R, Juhl B *et al.* The incidence of respiratory symptoms and sensitisation in baker apprentices. *Eur Respir J* 2008;**32**:452-459.

11 Mackie J. Effective health surveillance for occupational asthma in motor vehicle repair. *Occup Med (Lond)* 2008;**58**:551-555.

12 Brant A, Nightingale S, Berriman J *et al.* Supermarket baker's asthma: how accurate is routine health surveillance? *Occup Environ Med* 2005;**62**:395-399.

13 Park D, Moore VC, Burge CB *et al.* Serial PEF measurement is superior to cross-shift change in diagnosing occupational asthma. *Eur Respir J* 2009;**34**:574-578.

14 Zock JP, Brederode D, Heederik D. Between-And Within-Observer Agreement For Expert Judgment Of Peak Flow Graphs From A Working Population. *J Occup Environ Med* 1998;**40**:969-972.

15 Leroyer 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**:827-832.

16 Malo JL, Cartier A, Ghezzo H *et al.* Compliance With Peak Expiratory Flow Readings Affects The Within-And Between-Reader Reproducibility Of Interpretation Of Graphs In Subjects Investigated For Occupational Asthma. J Allergy Clin Immunol 1996;**98**:1132-1134.

17 Baldwin DR Gannon P, Bright P *et al.* Interpretation Of Occupational Peak Flow Records: Level Agreement Between Expert Clinicians And OASYS-2. Thorax 2002;**57**:860-864.

18 Huggins V, Anees W, Pantin CFA, Burge PS. Improving the quality of peak flow measurements for the diagnosis of occupational asthma. Occ Med 2005;**55**:385-388.

19 Anees W, Gannon PF, Huggins V, Pantin CFA, Burge PS. Effect of peak expiratory flow data quality on diagnostic sensitivity and specificity in occupational asthma. Eur Respir J 2004;**23**(5):730-734.

20 Gannon PF, Newton DT, Belcher J, Pantin CF, Burge PS. Development of OASYS-2: a system for the analysis of serial measurement of peak expiratory flow in workers with suspected occupational asthma. Thorax 1996;**51**(5):484-9.

21 Sauni R, Kauppi P, Helaskoski E *et al.* Audit of quality of diagnostic procedures for occupational asthma. Occup Med (Lond) 2009;**59**:230-236.

22 Anees W, Gannon PF, Huggins V *et al.* Effect of peak expiratory flow data quantity on diagnostic sensitivity and specificity in occupational asthma. *Eur Respir J* 2004;**23**:730-734.

23 Asthma in the workplace 2005. Bernstein IL, Chan-Yeung M, Malo JL, Bernstein DI. 2nd edition 1999. Marcel Dekker, Switzerland.

24 Beach J, Rowe BH, Blitz S *et al.* Diagnosis and management of work-related asthma. Evidence Report / Technology Assessment No. 129. Agency for Healthcare Research and Quality. Rockville, MD. 2005.

25 Moscato G, Dellabianca A, Vinci G *et al.* Toluene Di-isocyanate-Induced Asthma : Clinical Findings And Bronchial Responsiveness Studies In 113 Exposed Subjects With Work-Related Respiratory Symptoms. *J Occup Med* 1991;**33**:720-725.

26 Lin FJ, Chen H, Chan-Yeung M. New Method For An Occupational Dust Challenge Test. *Occup Environ Med* 1995;**52**:54-56.

27 Burge PS, O'Brien IM, Harries MG. Peak flow rate records in the diagnosis of occupational asthma due to isocyanates. *Thorax* 1979;**34**:317-323.

28 Stenton SC, Avery AJ, Walters EH, Hendrick DJ. Technical note: Statistical approaches to the identification of late asthmatic reactions. *Eur Respir J* 1994;**7**:806-12.

29 Rachiotis G, Savani R, Brant A *et al.* Outcome of occupational asthma after cessation of exposure: a systematic review. *Thorax* 2007;**62**:147-152.

30 NHS Plus / Royal College of Physicians. Latex allergy: occupational health aspects of management: a national guideline. Royal College of Physicians. London. 2008.

31 Fishwick D, Bradshaw L, Davies J *et al*. Are we failing workers with symptoms suggestive of occupational asthma? Prim Care Respir J 2007;**16**:304-310.