Recognition and surveillance of occupational asthma: a preventable illness with missed opportunities

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Occupational asthma is common, disabling and costly, and it is often difficult to diagnose. Incidence statistics are consequently unreliable, and there are formidable difficulties in recognizing and managing what should be a preventable illness. The opportunities have largely been missed. The author offers a personal view of what, ideally, should be done—recognizing that at present the ideal is not readily practical.

- Always consider the possibility of an occupational cause at the time adult-onset asthma is first recognized—the probability of this is of the order 9–15%.
- Do not prescribe treatment unless this possibility is remote or the asthma is life-threatening.
- If the possibility is not remote seek immediate advice from a specialized centre, without prescribing masking medication and without curtailing usual work practice.
- The specialized referral centre should place the accurate measurement of airway responsiveness at the centre of investigatory strategies.
- A return-to-work study, monitored by serial measurements of airway responsiveness and ventilatory function, provides adequate objective evidence for diagnosis in most cases.
- When a novel cause is suspected, specific inhalation provocation testing with the particular agent in the specialized centre is desirable.
- Regular competent surveillance is necessary in high-risk occupational environments; this should include environmental monitoring, the detection of relevant new symptoms, spirometry measurements, serum antibody studies (where available) and a robust protocol for managing inevitable failed attendances.

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Introduction

The editor has presented me with an unexpected, but welcome, opportunity. I am recalled from retirement to offer comments on a topic which captured much of my academic life—how to recognize occupational asthma more effectively and so lessen its burdens. I was not alone. I came to share with many colleagues a frustration that what has been discovered has not been exploited. Occupational asthma should be largely preventable, but so far we have largely missed the opportunities to prevent it and to lessen its physical and economic consequences. What follows is essentially a personal view focused more on recognition than surveillance, and not at all on management. It is necessarily focused on UK experience, but I believe the principles are global in nature.

Asthma arising in adult life very rarely resolves. Modern medication offers the possibility for control, but not cure, and the affected individual has asthma for the remaining of his/her life. There are two potential exceptions—asthma arising as an adverse reaction to certain drugs (e.g. beta blockers, non-steroidal anti-inflammatory agents) and asthma attributable to occupational agents. Prompt recognition of these causes and prompt removal from them, may lead to full and lasting resolution.1,2 This is a prize worth pursuing.

At an individual level, then, there is an opportunity to prevent persisting disability from asthma of occupational origin, providing it is recognized promptly and providing appropriate action is taken. That is, exposure ceases within, say, 6–12 months and at most 2 years of symptom onset (the sooner, the better). Occasionally a substantial reduction of exposure level is adequate, but more generally a new job in a different environment is necessary. A dilemma arises when the chances of alternative employment are slim and the asthma causes disablement of mild degree only. Since it may not resolve anyway, it is sometimes expedient and desirable to focus management on exposure control and medication while work continues under medical surveillance.

At a population level, there is the more obvious opportunity to modify the working environment as soon as an initial, ‘sentinel’, case is identified, and therefore prevent the development of asthma in other employees. The key point here is again prompt and unequivocal recognition, so that the exposure hazard is identified correctly and controlled or substituted as soon as possible.

Epidemiology—magnitude of the problem

In most industrially developed countries asthma is thought to be the commonest, currently occurring, respiratory disorder of occupational origin.3
Its estimated incidence in the UK varies as much as 10-fold according to how the diagnosis is made and how it is recorded, and ranges from about 350 to about 3500 per year.\textsuperscript{4–8} Data reported by respiratory and occupational physicians to the UK’s Surveillance of Work and Occupational Respiratory Disease (SWORD) project over the period 2002–2008 suggest an incidence at the lower end of this range—12.5/million employed/year.\textsuperscript{9} The incidence range reported among the employed from other industrially developed regions is particularly wide: 5/million in Massachusetts to 175/million in Finland.\textsuperscript{3} The cost to society in the USA has been estimated to be $1.6 billion annually,\textsuperscript{10} and in the UK to be more than £1.1 billion over a 10 year period.\textsuperscript{7} Most is borne by government (society’s tax payers) and affected individuals, rather than industry itself.

The surprisingly wide estimates of UK incidence reflect the particular problem of recognition. Reported cases may underestimate the true incidence by a considerable degree, but false-positive diagnoses are likely to complicate the epidemiological picture further. The magnitude of diagnostic error cannot be estimated with meaningful precision. An incorrect diagnosis of occupational asthma does, however, place a particularly harsh burden on the individual involved, who may well be denied gainful employment inappropriately for the remainder of his/her life.

Why is recognition so difficult? Occupational asthma probably accounts for 9–15\% of all emergent cases of asthma in adults,\textsuperscript{3,8,11–13} and so the probability of an occupational cause in a newly asthmatic adult is low unless the clinical features are particularly characteristic or there is recognized exposure in a potent occupational environment. Exacerbations are typically work related, but improvement away from the workplace may require many days and improvement may not be recognized even if there is a holiday period of 1–2 weeks. Lessened sleep disturbance during such periods may provide the most persuasive diagnostic feature in the medical history, but this is often distorted by treatment or compensation issues, and asthma of non-occupational origin may show similar variability.\textsuperscript{14} Occupational asthma most commonly arises within 6–24 months of exposure onset to the inducing agent, but the latency period can be a matter of years. The take-away message: while typical cases may be recognized readily, many cases are not typical.

It helps, of course, to have some idea of the most potent causal agents and the most commonly affected settings. Table 1 gives details of the most common causal agents in the UK at present, and Table 2 identifies the annual reported incidences by occupation and working environment.

The great majority of individuals affected by occupational asthma are unable to continue working in the particular environment responsible for their illness. If the asthma does not resolve, they have difficulty in
finding alternative employment. There may still be difficulty, even with full resolution, since the individual’s particular skills and experience may be suitable only for work environments which harbour other asthma inducers. Sadly, many affected individuals never work again, thereby posing considerable financial, social and psychological burdens on them and their families, as well as financial and social burdens on the state.\(^4\) Their skills are lost to the labour force. Because the latency period from exposure onset to symptom onset is short, the susceptible individuals most likely to develop occupational asthma do so at a relatively young age.

**Airway responsiveness and asthmatic reactions**

Airway responsiveness (or bronchial reactivity/hyper-reactivity) is a term used to describe the inherent tendency of the bronchi to constrict
in response to what may be a wide range of stimuli, both specific (i.e. allergenic, pharmacological or idiosyncratic) and non-specific (irritant). If the level of airway responsiveness is sufficiently high these stimuli become potential ‘triggers’ (provokers) of asthmatic reactions, but they do not necessarily make the airways more responsive. Only agents which increase airway responsiveness (asthma ‘inducers’) have the potential to cause the development of asthma in subjects whose airway responsiveness lies otherwise within the normal, non-asthmatic, range.

Thus, asthma inducers cause asthma, while asthma triggers provoke asthmatic reactions in subjects who are already asthmatic. In recognizing environmental agents of relevance to asthma, and occupational asthma, it is useful to quantify airway responsiveness as well as demonstrate asthmatic reactions. It is also useful to realize that most cases of occupational asthma (90–95%) are thought to arise through hypersensitivity mechanisms, the affected subject becoming allergic to a particular respiratory sensitizer. Such agents both induce asthma and trigger asthmatic reactions.

The remaining 5–10% of cases arise through toxic mechanisms, there being an acute, almost invariably accidental, exposure which produces immediate injury to the lung and airway mucosa. Survivors usually recover fully, but a few are left with asthma (the reactive airway dysfunction syndrome) or obliterative bronchiolitis. Asthma from this mechanistic route poses few diagnostic problems, and is prevented by strategies which minimize the risk of industrial accidents with toxic chemicals. It is not relevant to the topic of this article. The dilemmas, and missed opportunities, lie with occupational asthma from presumed hypersensitivity mechanisms.

Recognition

Asthma is a common disease within the population at large, affecting 5–10% of adults to some degree, and there is often difficulty in distinguishing among incident cases the minority which are occupational in origin. The matter can be very challenging even for ‘experts’ with much experience. This is partly a consequence of inherent diagnostic difficulty, and partly a consequence of the lack of a standardized and practical diagnostic approach. While experience and skill in uncovering the history may be invaluable, such evidence is essentially subjective. A persuasive diagnosis requires objective evidence. A useful start is the demonstration of an IgE antibody response, confirming immunological hypersensitivity, but this may occur in the absence of asthma and assays are unavailable (or unreliable) for many inducing agents—e.g. most reactive chemicals of low molecular weight.
The gold standard in diagnosis comes from inhalation provocation tests with the causal agent.\textsuperscript{16,17} Less satisfactory methods involve monitoring ventilatory function (commonly by peak expiratory flow), or airway responsiveness, or both, to assess whether changes over time can be related to particular occupational exposures, or simply to periods at work. Respective specificities and sensitivities have been reviewed.\textsuperscript{17} I shall use illustrative cases to describe all three methods. In most ‘cases’ in real life, however, none of these objective methods is used. The diagnosis, with all its serious consequences, is ‘confirmed’ or ‘excluded’ largely through subjective guesswork.

This is a sorry state, though understandable in the absence of adequate numbers of fully equipped specialized referral centres.

\textit{Peak expiratory flow monitoring}

Hand-held peak flow meters provide a convenient method to assess ventilatory function over long periods. This has obvious benefit to the investigation of occupational asthma and their use has become widely popular. This is particularly so in the UK where OASYS (Occupational Asthma System) has evolved as a means of interpreting the data obtained.

Test subjects are asked to record measurements at approximately 2 hourly intervals (or whenever practical) over periods of 2–4 weeks, sometimes longer. This allows daily plots of the maximum, minimum and average values, and illustrates graphically whether ventilatory function follows a consistent pattern of decline on work days (or days with particular occupational exposure) compared with days away from work. The results may be analysed statistically, giving an overall score—work effect index—from 1 (negative) to 4 (strongly positive). A score of 2.51 or more indicates a significant work-related adverse effect, with reported specificity for occupational asthma of about 95%. Sensitivity is of the order 70%.\textsuperscript{18,19} Greater specificity can be obtained with a recently described method of statistical analysis.\textsuperscript{20}

\textit{Case study:} Figure 1 illustrates the case of a school science laboratory technician, who obtained PEF measurements on three to nine occasions daily over a 6 week period.\textsuperscript{21} In general there was improvement in PEF during each period away from work, and deterioration during each period at work. There were, however, anomalous days when PEF was unchanged at work or deteriorated away from work. It transpired that, unusually, the school had not been cleaned over a relevant weekend, and the technician’s asthma was later shown by specific inhalation provocation testing to be due to the residue on the floor of the material used to...
clean it. One anomalous day was thus explained. The score of 3.79 was in the strongly positive range.

The equipment required for PEF monitoring is inexpensive and readily available, and can be used repeatedly by the test subject himself/herself without supervision. Strongly positive results can be recognized easily from the plots (eyeballed) without sophisticated analysis, but the later is useful and should be used for its statistical authority. Facilities for OASYS analysis are available free online.22

The drawbacks to PEF monitoring lie with the unsupervised nature and crudity of the measurements. The technical quality underlying the submitted records cannot be scrutinized, unless the measurements are obtained with computerized recording devices that store flow-volume data by date and time. There may consequently be doubt as to whether maximum inspiratory and expiratory effort was uniformly applied, and whether all measurements were obtained at the recorded times.

A more fundamental issue relates to the non-specific nature of the results. A positive outcome suggests that asthma has worsened in the workplace, but this may be a consequence of exposure to non-specific irritants rather than a respiratory sensitizer. That is to say, any employee who happens to have asthma of non-occupational origin may experience worsening ventilatory function when exposed occupationally to irritant agents (even cold air), and as many as 25% have been reported to do so.14 Irritants may provoke asthmatic reactions but they do not cause asthma unless exposure concentrations reach ‘toxic’
levels. The point is well illustrated by a Canadian study in which PEF monitoring using OASYS-2 failed to distinguish work-exacerbated asthma from true occupational asthma. PEF monitoring therefore identifies asthma which worsens at work, but it is not a definitive test for occupational asthma.

**Airway responsiveness monitoring**

Monitoring airway responsiveness is more fundamental and so potentially more definitive. The critical issues are how readily can it be quantified, and how readily can significant increases (and decreases) be recognized? My colleagues and I have been particularly fortunate in Newcastle to quantify airway responsiveness with an in-house dosimeter. We use the widely accepted method of administering doubling cumulative doses of nebulized methacholine at 5 min intervals until a definite but safe asthmatic reaction has been demonstrated by measurements of FEV1, and we quantify airway responsiveness conventionally by the PD20 (provoking dose calculated by interpolation to produce a decrement of 20%).

Less conventionally, the dosimeter releases precisely controlled doses, and it is calibrated by true aerosol output, not the weight lost after nebulizer activation. A further, ‘clinical’, innovation is quantifying FEV1 as the mean of the best three of six measurements (M3/6). Because the FEV1 is particularly unstable immediately after bronchoconstrictor inhalation, M3/6 produces a less variable plot for calculating the PD20 than the more conventional lower of only two measurements (L1/2). The lower the PD20, the greater is the level of airway responsiveness. With the Newcastle method PD20 values <200 µg methacholine identify subjects with unequivocally active asthma and PD20 values >1000 µg identify subjects without active asthma. With PD20 values in the grey zone of 200–1000 µg, some subjects have evidence of mildly active asthma, others do not.

The precision of PD20 measurement is best quantified by the coefficient of repeatability, CR. The approximate lower 95% confidence limit for the second of a pair of measurements in the same subject is given by [first PD20/CR], while the approximate upper 95% confidence limit is given by [first PD20 × CR]. We found from a series of clinical studies, including epidemiological studies of a normal population and a working population at risk of asthma, that the CR varied between, approximately, 2 and 3. This indicates that changes in PD20 of less than 2-fold are not significant. PD20 increases of 3-fold or more indicate a significant decrease in airway responsiveness, while decreases to one-third or less indicate a significant increase in airway
responsiveness. PD20 changes of 2- to 3-fold are of borderline significance.\textsuperscript{23,27–31}

Case study: The patient worked in a chemical manufacturing plant.\textsuperscript{32} Particular suspicion fell on the biocide, isothiazolinone, because there had been published evidence that it has skin sensitizing properties and it became clear that he did have relevant exposure. When first assessed, during a period at work, he described mild asthmatic symptoms and had a PD20 of 115 \( \mu \text{g} \). A period away from work was arranged in collaboration with his employers. After 10 days the PD20 had increased 2.3-fold to 267 \( \mu \text{g} \), and after 18 days it had increased 9.6-fold to 1107 \( \mu \text{g} \). He then underwent a ‘return-to-work’ study after first recording PEF at hourly intervals from 07:00 to 23:00 h over 3 ‘control’ days. From these the mean values at each hour were plotted, and compared with similarly timed measurements over the first 3 days back to usual activities at work—Figure 2. PEF decline on Day 1 was suspicious of a late asthmatic reaction, while the declines on Days 2 and 3 were fully convincing. On the following day the PD20 had fallen 10-fold to 110 \( \mu \text{g} \), confirming that his return to work for just 3 days had produced a significant increase in airway responsiveness as well as typical late asthmatic reactions.

The total investigatory period to diagnosis in this particular case was of similar length to that for PEF monitoring, but the result was more definitive and the actual investigations required a few days only. A few, serial, PD20 measurements are less demanding of the test subject than multiple PEF measurements, but are more demanding of the

![Fig. 2 Serial hourly PEF measurements during the initial 3 days on returning to work after an absence of 18 days. Immediately prior to the return, PEF was recorded on 3 ‘control’ days and the mean values at each hour are shown.](https://academic.oup.com/bmb/article-abstract/95/1/175/270187)
supervising referral centre. Unfortunately the Newcastle equipment is not generally available, and regretfully our system has not yet been examined independently. Other methods (amongst the many) of airway responsiveness quantification may give satisfactory results, but the measurements may be importantly less precise, and so much greater change is necessary to achieve the same diagnostic significance.

**Specific inhalation provocation tests— the diagnostic gold standard**

When these tests are used diagnostic doubt is usually removed, but they are time-consuming (hence expensive), potentially hazardous, and available only in a few specialized centres. They are particularly useful when a novel agent has become a suspected, but hitherto unrecognized, cause of occupational asthma. This is illustrated by the following case study.33,34 The agent had the potential for consumer use in millions of homes on a global scale, and so there was a great need to be certain whether or not it is a respiratory sensitizer able to cause asthma.

**Case study:** A research and development laboratory had been investigating two detergent chemicals, one with low temperature bleach-activating properties (sodium iso-nonanoyl oxybenzene sulphonate, SINOS). Three staff members developed symptoms suspicious of asthma after other staff members had developed rhinitis, conjunctivitis and skin rashes of suspected occupational origin. Hospital-based inhalation provocation tests were requested to investigate whether either (or both) was responsible, and all three employees volunteered to participate. Neither chemical, nor any closely related chemical, had previously been recognized to cause asthma.

Environmental measurements identified the range of daily respirable exposure concentrations incurred during the normal course of work (all very low), thereby allowing estimates of daily cumulative exposure. To enhance safety, the inhalation challenge protocol began with a dose well short of the minimum estimate, and proceeded logarithmically with 3.2-fold increments (0.5 log_{10}) through one inhalation test per day. The planned doses, administered as aerosol solutions in saline from the Newcastle dosimeter, thus increased from 0.01 \( \mu \)g through the range of 0.032, 0.1, 0.32, \ldots \ 100 \( \mu \)g. ‘Dummy’ challenges were inserted into the protocol in a double-blind fashion using saline alone, neither the test subject nor the immediately supervising physician knowing the identity of the test substance on a given day. Three non-asthmatic and three asthmatic control subjects, all without occupational exposure to these agents, volunteered to undergo the same protocol, which necessarily proved to be very time consuming. No medication was used.

All three R&D staff members developed increases in airway responsiveness and mild asthmatic reactions during the course of the test series with SINOS, but none reacted to the other detergent agent, linear alkylbenzene sulphonate (LAS), or to the dummy challenges with
saline alone. None of the six control subjects had symptoms, increases in airway responsiveness, or significant decrements in ventilatory function, with either series of tests. Figure 3 illustrates the results of serial PD20 measurements in one of the R&D staff members. No significant change occurred in association with the LAS challenge series, but there was a more than 3-fold decrease in association with the SINOS series. Recovery was full after 2–3 weeks.

The extensive data from the three staff members and the six controls stimulated the subsequent development of two statistical approaches to evaluate changes in FEV1. Both proved to be much more sensitive than conventional methods. The first is illustrated in Figure 4. The shaded area in ‘A’ defines a summary measure (area decrement, AD) for FEV1 change 2–12 h after challenge with SINOS 32 μg. The columns in ‘B’ show the AD measurements on 3 control days and their 95% and 99% confidence intervals. Both are exceeded by the AD following SINOS 32 μg, indicating a significant late asthmatic reaction.

Figure 5 shows a lower 95% confidence boundary, calculated from the pooled variance of FEV1 measurements on 3 days without SINOS exposure. It therefore runs parallel to the means of the hourly measurements over these 3 days. If, as in this case, there is very little variability (the PD20 had returned to a non-asthmatic level prior to the test series), it is sometimes possible to show that a minor asthmatic reaction, causing few if any symptoms, is nevertheless statistically significant. Unnecessarily high challenge doses may thus be avoided, greatly diminishing risk.

Tests of this nature are highly sophisticated, and the investigation described was unusually extensive and time consuming. Fortunately,
most cases of suspected occupational asthma do not require this level of investigation.

**Surveillance**

The COSHH (Control of Substances Hazardous to Health) regulations within the UK require employers to make assessments of their work environments in order to identify agents that are potentially hazardous and bring about appropriate measures of exposure control. If known
respiratory sensitizers are identified, there is a potential risk of occupational asthma, but the magnitude of this risk is difficult to quantify. Control and surveillance measures consequently lack standardization. The risk varies importantly according to exposure level and sensitizing potency of the agent, and susceptibility of exposed individuals. The latter cannot be quantified, and sporadic cases are inevitable even if exposure controls reduce the risk to what might be deemed an acceptable level. A key point is that there is no exposure level which entirely eliminates the idiosyncratic risk associated with hypersensitivity. This is poorly understood, and many employers and their advisers show inappropriate confidence in excluding the possibility of an occupational cause if asthma arises despite monitored exposure levels falling within regulatory limits.

With agents recognized to have high sensitizing potency (e.g. isocyanates, flour, platinum salts) the risk of asthma is generally considered to justify a surveillance programme. Most commonly, new employees complete a questionnaire, and proceed to work in potentially hazardous sites only if there is no reported evidence of pre-existing asthma—a procedure of questionable justification, which has not been shown to diminish the occupational asthma risk. Occasionally a medical examination is carried out also, most usefully with spirometry. Medical questionnaires are then re-administered at intervals, sometimes with spirometry and (if appropriate) IgE measurements, in the hope that emergent asthma or sensitization is identified promptly.

Fig. 5 Serial hourly FEV1 measurements associated with SINOS 0.1 μg challenge in one subject. The lower boundary represents the lower 95% confidence limit for the measurements obtained on 3 ‘control’ days without exposure. The boundary is parallel to the mean values because the variance was pooled. Excursions of at least 1 h indicate significant late asthmatic reactions.
Recommendations

There is no lack of recommendation for preventing occupational asthma, and the reader is referred to a number of authoritative sources. Despite these, the incidence appears to have diminished only a little over recent decades. This is largely because guidelines have not been followed. In addition the continual introduction of new reactive chemicals into industry has increased the risk by an unquantifiable degree, and so it is plausible that management and prevention strategies have been more successful than crude annual incidence statistics suggest.

My own recommendations do not conflict materially with those of learned professional bodies or governmental departments. They simply reflect personal preferences, with the UK most in mind.

Recognition

The diagnosis of occupational asthma is often difficult, and so possible cases are best referred to centres of special interest and experience. A number in Britain are well known to the British Thoracic Society and the Health & Safety Executive (Group of Occupational Respiratory Disease Specialists). Additional centres are needed to provide a comprehensive national service within the UK, as is appropriate funding, and there is an urgent need for an effective referral procedure which overcomes the practical and financial barriers of Primary Care Trusts and Health Region boundaries. In particular, physicians employed directly by industry
should be encouraged to refer potential cases directly. Mere advice to the relevant general practitioner that such a referral is indicated is not necessarily accepted, and will inevitably be associated with delay.

Delay is almost inevitable, and often critical, because of the understandable knee-jerk reaction of any physician faced with a new case of adult-onset asthma. Asthma treatment is prescribed, symptoms improve, and the relation with work is obscured. Instead, the physician who is first consulted should initially consider whether the asthma could be a consequence of occupational exposure (a 9–15% probability). Only if there is serious disability should treatment be offered before diagnostic doubt about an occupational cause, if there is any, is removed.

The consequences of missing or delaying a competent diagnosis of occupational asthma are serious, as are the consequences of diagnosing occupational asthma incorrectly, and I recommend an immediate phone call to a referral centre without the use of masking medication. Work should continue until airway responsiveness and IgE reactivity (for most high-molecular weight allergens and a few low-molecular weight allergens) can be measured, but this should be done within 1–2 weeks, when an urgent preliminary assessment is carried out in the referral centre. If an occupational cause for the asthma appears plausible, liaison with the employer becomes critical. Is there a recognized asthma-inducer within the workplace, is the patient likely to have exposure, and have other members of the workforce become affected? Depending on the outcome, there may be a need for investigation.

My preferred approach when an occupational cause of the asthma does merit investigation is to carry out a return-to-work study after a period of 2–3 weeks away from work. The great majority of subjects with occupational asthma will improve significantly after 2–3 weeks without exposure, and this should be demonstrable objectively by a second measurement of airway responsiveness. That the improvement is no coincidence is shown by a prompt increase in airway responsiveness as the subject returns to work, preferably with spirometric or PEF evidence that ventilatory function decreases in parallel. The case underlying Figure 2 provides a good example, but the statistical approaches of Figures 4 and/or 5 should be used additionally to identify unequivocal late asthmatic reactions. ‘Late’ asthmatic reactions (i.e. those commencing an hour or more after exposure onset and persisting for several hours) carry more diagnostic significance than non-specific immediate reactions because, unlike immediate reactions, they are closely associated with exposure to sensitizing agents and increases in airway responsiveness.
**Surveillance**

It may be difficult to determine whether surveillance is necessary. Advice is available in the UK from the Health & Safety Executive,\(^6\) professional organizations such as the British Thoracic Society\(^39\) and the Society of Occupational Medicine,\(^41\) as well as specialist referral centres.\(^40\) If potent sensitizers are used within a particular workplace, or cases of occupational asthma have already been identified there, particular justification is needed if surveillance is not used. If it is to be used, it needs to be efficient; it should involve the environment and the workforce.

All too frequently one hears from employees that when environmental measurements are due, usual work practices (even usual production) are discontinued in the hope that regulatory standards are met. This is, of course, misguided and counterproductive. Not only may it reinforce an unjustified sense of security (exposure levels seem well controlled), but it will distort apparent dose–response relations if occupational asthma occurs nevertheless. Exposure standards will appear inadequate, and the sensitizing potency of the particular asthma inducer will be exaggerated. This may lead to unnecessarily stringent and costly engineering adaptations to ensure adequate compliance with revised regulations.

For certain asthma inducers, particularly those of high molecular weight (e.g. flour/enzymes), there may be useful blood antibody tests that can be used at regular intervals to identify emerging sensitization within the workforce. Negative antibody tests throughout the workforce provide reassurance that exposure controls are adequate. Conversely, the development of antibodies provides a warning that exposure is not adequately controlled and that asthma may follow.

Clinical surveillance of the individual should include questionnaire enquiry about relevant symptoms, spirometry and (where relevant) specific IgE assay. The symptoms should include skin rashes, conjunctival upset and (particularly) nasal upset in addition to symptoms of asthma, since an excess prevalence of these associated disorders within the workforce commonly heralds the emergence of occupational asthma. There should also be an enquiry as to whether new medications have been prescribed. The individual may be surprisingly unaware of the significance of the GP prescribing a bronchodilator inhaler, and it may be that a beta blocker has been prescribed for asymptomatic hypertension.

A continuing absence of reported relevant symptoms coupled with normal (and unchanged) spirometry is very reassuring, despite the possibility of the questionnaire data being inaccurate. If one or other provides a hint of emergent asthma, the individual should be...
interviewed, and both medical history and spirometry should be checked competently. If a suspicion of emergent asthma remains, the referral centre should be consulted. If necessary, measurement of airway responsiveness may be needed; a level within the normal (non-asthmatic) range provides objective reassurance. A measurement of equivocal significance should, conversely, indicate a need for closer surveillance and a further measurement within 3–6 months. A measurement within the asthmatic range requires expert evaluation.

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References

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