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# The prevention of occupational asthma

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**ABSTRACT: There is sufficient understanding of the causation of occupational asthma for preventive action to be appropriate. To date, attempts appear to have been largely unsuccessful and this appears to be largely due to nonscientific/technical obstacles. These include the fragmented nature of the disease, its low public and industrial profile, and its comparative rarity in single workplaces. Nonetheless the disease has high individual and societal costs.**

**Prevention strategies should be concentrated on workplace-exposure controls, accompanied by intense educational and managerial improvements. Methods of secondary prevention appear to be successful but require considerable refinement. Screening (out) of potential new employees is inefficient and likely to remain so; and in any case is beset by difficult ethical and legal issues.**

**There are only a handful of published studies reporting evaluations of preventive programmes. None is entirely rigorous but each suggests that primary and secondary prevention are both feasible and highly effective. The evaluation of preventive strategies is difficult, not only because of the low incidence of the disease in individual workplaces but also because of the failure of many epidemiologists to engage in this work. Considerably more cooperation between scientists in the field, regulatory authorities and industry is required.**

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On the face of it, occupational asthma (OA) should be preventable. The aetiological understanding of the disease is arguably sufficient to devise and enact primary preventive strategies. Yet there is no evidence that the incidence of OA has decreased. The gap between aetiological understanding and prevention is dependent on several influences, many of which are beyond the realm of medical science (table 1).

These include the nature, severity and frequency of the disease in question, and the costs of preventive action or otherwise. In this case there are a number of immediate obstacles to successful prevention. OA is a fragmented disease, defined by a clinical outcome rather than by a particular agent or occupation. Its immediate causes are diverse and difficult for policy makers, industry and campaigners to target; fortunately, the bulk of disease can generally be attributed to a small number of commonly used and potent allergens. OA is very rarely lethal and frequently improves after exposure has ceased. Furthermore, it is clinically indistinct from a disease ("community asthma") that is very common in most industrialised countries. Thus its manifestations are neither unknown nor especially feared. For these reasons, among others, the disease has a low profile and has not received widespread public attention. VERMA *et al.* [1], for example, in describing how evidence is translated into preventive strategies for occupational disease, cited occupational (bakers′) asthma as notably unsuccessful.

The current authors′ focus is on classical "occupational asthma", representing a hypersensitive response to a sensitising agent inhaled at work. WAGNER and WEGMAN [2] have argued that this perspective, derived from clinical (and medicolegal) necessity, is too narrow and should be broadened to include pre-existing asthma, which is exacerbated by (nonsensitising) workplace exposures. Preventive efforts that embrace both kinds of asthma would have a higher impact in public health terms, but a more general application of this approach has been criticised on the grounds of unworkability [3]. Very little is known about the broader relationships between work and asthma and in particular about the chronic effects of irritant exposures. It might reasonably be argued that too wide a perspective might, at this stage, dilute the drive towards, and effectiveness of, preventive efforts.

Below, the approaches to the primary and secondary prevention of the disease and reasons why these may or may not be practicable, effective or desirable are discussed.

**Previous articles in this series: No. 1**: Vandenplas O, Malo J-L. Definitions and types of work-related asthma: a nosological approach. *Eur Respir J* 2003; 21: 706–712. **No. 2**: Moscato G, Malo J-L, Bernstein D. Diagnosing occupational asthma: how, how much, how far? *Eur Respir J* 2003; 21: 879–885. **No. 3**: Mapp CE. The role of genetic factors in occupational asthma. *Eur Respir J* 2003; 22: 173–178. **No. 4**: Sastre J, Vandenplas O, Park H-S. Pathogenesis of occupational asthma. *Eur Respir J* 2003; 22: 364–373. **No. 5**: Gautrin D, Newman-Taylor AJ, Nordman H, Malo J-L. Controversies in epidemiology of occupational asthma. *Eur Respir J* 2003; 22: 551–559. **No. 6**: Vandenplas O, Toren K, Blanc PD. Health and socioeconomic impact of work-related asthma. *Eur Respir J* 2003; 22: 689–697.

Table 1. – Factors influencing the prevention of occupational disease

| Influences | |
|---|---|
| Societal | Frequency of the disease |
| | Nature of the disease |
| | Perception of the disease |
| | Individual and societal costs of the disease |
| Technical | Strength of epidemiological or clinical evidence of cause/effect |
| | Identification of risk factors amenable to manipulation |
| | Availability of efficacious technical or organisational means of reducing important risk factors |
| | Availability of effective methods of secondary prevention |
| Business | Frequency of the disease |
| | Impact on consumers |
| | Public reputation |
| | Economic costs of the disease |
| | Efficiency and effectiveness of technical or organisational means of reducing important risk factors |
| | Effects on competitiveness |
| | Influence of employee or consumer organisations |

Incentives, both legal and economic, to industry are also explored. Last, a small number of successful examples of prevention are presented.

## Is it worth preventing occupational asthma?

It is widely accepted that ~10% of adult asthma is "attributable to occupational factors" [4]. The true figure may even be higher than this [5]. Asthma remains the most commonly reported occupational lung disease in most industrialised countries and long-standing surveillance schemes in Finland and the UK report no reductions in overall incidence over the past 10 yrs [6, 7]. Disease frequency alone, however, makes a poor case for prevention. While there is good evidence that the impact of OA at an individual level can be devastating there is very little evidence relating to its industrial or societal impacts [8]. A recent assessment of the financial costs of occupational asthma in the USA, in which a population-attributable risk of 15% was used, estimated that the national disease costs are $1.6 billion [9]. Equally useful would be targeted estimates of industry-specific costs, to include those of maintaining a clean environment, educating and training employees, and hiring and training new ones when controls fail. These are likely to be substantial. As far as the current authors are aware such estimates have not been published.

## Primary prevention

### *Pre-employment selection*

If the development of OA involves an important element of individual risk, then identification of susceptibility markers could conceivably be used in the selection of innately low-risk employees. Leaving aside its doubtful morality (or even legality), this approach appears to be highly inefficient. More broadly, a focus on the "susceptible host" may discourage efforts to reduce risks and prevent disease in populations [2]. Asthma, occupational or otherwise, is a complex disease, likely to have multiple genetically determined influences and currently it appears improbable that any single marker of predisposition, or even manageably small group of markers, will ever be sufficiently discriminatory to be useful. Age and sex appear to be unimportant, independent risk factors, although prognosis may be poorer in older persons [10]. The absence of "atopy" (variously defined) is used in several industries during the selection of new employees, but this is almost certainly a very inefficient approach. Some 30–40% of young adults in industrialised countries are now "atopic" in one way or another and their general exclusion dramatically reduces the pool of potential new employees. There is an increasing volume of published work that points towards human leukocyte antigen (HLA)-restricted susceptibility to several occupational agents [11], although this body of evidence is not internally consistent. No wholly sensitive or specific markers of individual (genetic) susceptibility have been identified.

As with most "competing causes" of disease, it may be that susceptibility has a more powerful effect at lower levels of environmental exposure [12]. Thus, their consideration may become more important as environmental controls improve. It is interesting to consider how a (genetic) screening marker might be used if a reasonably efficient one was available. The experience of berylliosis may prove instructive. Screening for Glu69 in HLA-DPB1, a marker of susceptibility with high sensitivity but low specificity, has recently been offered to some employees of the beryllium industry in the USA. The test is voluntary and the results are made known only to the employee, who is therefore free to act on the knowledge or otherwise; exclusion from employment is not the only available course of action. It remains to be seen whether this will result in a reduced incidence of beryllium sensitisation and to what extent the practice is acceptable.

It seems reasonable to apply some rules of exclusion at the stage of first employment. Persons with established OA from a particular agent ought not to be employed in a new job where there is further exposure to the same agent. More contentiously, it is not uncommon for prospective employers to decline offers to those with a history of OA to an agent which is entirely unrelated to any sensitiser they may encounter in the new job. There is no reliable evidence that such persons are, on account of their original OA, at increased risk of acquiring a second variant of the disease. Nonetheless this practice is a common cause of employment handicap to those who have acquired asthma at work.

More common still is the screening-out of potential employees with "community" asthma from jobs where there is a risk of exposure to sensitising agents, as advocated by some [13]. For those with severe or moderately severe asthma, this may reasonably be justified on the grounds that it would be unwise to put them at risk of developing an additional respiratory impairment. Such practice may fall foul of anti-discriminatory legislation, although in most countries this is yet to be tested in law. For those with mild asthma, or indeed those with a past history of the disease that is now quiescent, the value of screening is far less obvious; and will become increasingly so as the number of children who have at some stage acquired an asthmatic label rises. Nonetheless, there may be a role here for better education of those (with or without asthma) attending vocational schools and even those of school age considering a suitable career.

### *Preproduct screening*

For many years, there have been attempts to identify, prior to their widespread use, which newly introduced agents are likely to act as human respiratory sensitisers. This toxicological approach generally relies on animal models [14]. Using a mouse intranasal test, for example, the potencies of several protease and nonprotease enzymes in causing specific

immunoglobulin (Ig)G1 production varied 60-fold [15]. More broadly, the hazard evaluation of low molecular weight agents always includes an assessment of their dermal sensitisation potential. In most cases this is now done using an animal-based local lymph node assay, which tests the ability of topically applied chemical agents to induce proliferative lymphocyte responses in draining nodes [16]. Cytokine fingerprinting may offer a more specific index of respiratory sensitising potential [17–19]. To the extent that the potential of chemicals to cause immunological sensitisation depends on their mode of interaction with antigen-presenting cells and the subject's subsequent lymphocyte responses, and since antigen-presenting cells and lymphocytes are not confined to the skin, there is no *a priori* reason to assume that the dermal route of administration is critical in eliciting a sensitisation response. Arguably, chemicals that are positive in tests involving dermal application should be considered as sensitisers regardless of their mode of contact with the body, including through inhalation [20]. This is contentious, and further research into the relationship between dermal and respiratory sensitisation is required. Not only would this improve the identification of respiratory sensitisers, it might also assist in the development of more appropriate labelling and (primary) preventive measures at work. However, it is not yet established whether chemicals that are negative in skin sensitisation tests are thereby unlikely to cause respiratory sensitisation. Other means of premarketing product testing include clinical trials in human volunteers, such as that used to test an enzyme in a personal cleansing product [21].

### *Controlling exposure in the workplace*

In the case of high-molecular weight agents, there is sufficient evidence, gathered from analytical epidemiology, of an exposure-response gradient for occupational allergens. There are, however, important limitations as follows. 1) Much, although not all, of the evidence relates more strongly to occupational sensitisation (the development of specific IgE antibodies) than to asthma. The relationships between these two outcomes are poorly understood, although most would argue that the acquisition of IgE sensitisation to some agents is a strong predictor (if not precursor) of asthma. 2) The "shape" of the exposure-response relationship is unknown and there is, as yet, no human evidence which reliably allows the setting of indisputable no-effect thresholds [22]. 3) There is some evidence that high or continued exposures lead to immunological "tolerance" [23], although it is unclear whether these observations reflect survival pressures. 4) On the whole, it seems probable that the timing of high-intensity exposure is important. For instance, exposure/response relationships are more easily demonstrated when they consider the intensity of exposure at the time of onset of disease rather than at the time of study [23].

Given the evidence for a broad exposure/response relationship, primary preventive efforts should be concentrated in the control of workplace exposures. There are, however, few examples (see below) of situations where a reduction in exposure alone has been demonstrated to result in a reduced disease incidence. This is probably due more to a lack of research interest in this area than to a lack of efficacy of the approach. WEGMAN [24] suggests, in addition, an increasing but potentially damaging demand for "perfect" understanding of disease aetiology. Whilst laudable in principle, this may simply act (passively or otherwise) to defer timely preventive action and, moreover, is probably an unrealistic appeal. Much responsible public health action in other areas has been taken in the face of scientific uncertainty.

Methods of control generally follow standard occupational hygiene tenets and range from elimination or substitution, through enclosure and ventilation to work practices, personal protective equipment and appropriate monitoring, and administrative processes [25]. Elimination of an asthmagen from the workplace is unusual but occasionally practised. Enforcing suitable exposure levels to occupational asthmagens is difficult, because there are very few health-based legal standards. Those that exist are generally set on the basis of a substance's other hazardous properties (such as the irritant effects of isocyanates) or are of doubtful value (for example the current standard of 60 $ng \cdot m^{-3}$ for detergent subtilisins). The reasons for such omissions include: the lack of detailed evidence for most occupational sensitising agents concerning exposure-response relationships; the lack of consensus over whether there are or can be thresholds at which exposure(s) to sensitising agents induces no adverse health effects [26]; and the technical difficulties in measuring airborne allergens at the low intensities at which they probably exert their effects.

It is probable that these obstacles will persist for the near future. Leaving aside the theoretical debates, there are far too few resources for detailed examination of the large number of occupational asthmagens. Legislative controls generally require, therefore, exposures to be kept to the minimum that is technically possible without undue financial, social or economic disruption. Where levels are set in such cases they are therefore "pragmatic" rather than "health based".

In so far as any occupational exposure standards are valuable, these approaches seem reasonable. Indeed there appears little alternative. Difficulties arise, however, over issues of compliance. In almost all cases compliance is easier for large firms than small [27] and especially when the former is engaged in large-scale manufacture of only a few products. Compliance may be perceived as inimical to productivity, at least in the short term. There is also serious concern over how meaningful many of these concepts are to those working in industry. Surveys of managers in UK chemical firms, most employing <10 workers, revealed an alarming level of misunderstanding or ignorance over legislated requirements concerning employees' exposure [28, 29]. Although most were taking steps to control workplace exposures, it was apparent that set exposure limits were of limited influence in their decision making. These findings prompted the UK Health and Safety Executive to their current efforts to devise simplified methods of setting and conveying occupational exposure limits.

Methods to enforce or encourage compliance can, broadly, be divided into the legal and the economic. Since legal approaches are backed by economic penalties and economic approaches must be sanctioned in law, the distinction is perhaps less than real. In the case of OA, a legal approach is generally taken whereby workplace control measures are legally enforceable and infringements penalised by fine or occasionally factory closure. These approaches are fairly slow, require extensive regulatory surveillance and have not been proven to be effective alone in reducing the incidence of OA. Sanctions directed against the primary manufacturers of sensitisers, and not against the users, may be more rapidly effective. At present, the latter are not liable if they have made full disclosure of their product's hazardous nature. Many would argue that extension of this responsibility would be both unfair and unworkable; it again appears to be untested. Outright bans on substance manufacture and use have not, as far as we are aware, been instituted, unlike for other hazardous substances, such as 2-naphthylamine in the UK rubber and dyestuff industries.

Economic measures are either punitive, the taxing of hazardous substances, or exhortatory, whereby financial incentives are provided by government to support the development

of safer alternatives. Both may be used in (phased) conjunction. In general, such approaches are more acceptable if there is limited use of the substance in question and if the costs of replacement are likely to be low. This is seldom the case for occupational sensitising agents. Insurance premiums are generally set in the knowledge of estimated or measured risk at a particular workplace, although a flat rate may be applied to smaller enterprises; this may be unwise if the risks are generically higher in such settings. In the environmental field, it is recognised that tough regulation encourages technological development and, in the long run, often leads to cost savings (Porter's paradox). Interestingly, compliance with general environmental regulations appears to be much higher than that for occupational health risks; this may reflect differences in public perceptions of the relative importance of the two types of hazards.

## Secondary prevention

By this we mean the early detection of OA on the grounds that action taken at this stage leads to a better prognosis. There is reasonable evidence that removal from exposure as soon as possible increases the chances of recovery, although it is not entirely clear that this is independent of age [10, 30]. More broadly, the detection of one case frequently leads to the detection of more cases in the same workplace.

Secondary prevention is practised through regular medical surveillance of employees. Its role in reducing morbidity from OA by early detection and early management, including avoiding further allergen exposure, has been studied in few settings. Even in these it is not clear which components are beneficial and in what form the programme should be delivered [31]. Medical surveillance is rarely used in isolation, but is usually introduced (either voluntarily or by legislation) when an increased risk of asthma in an industry or workplace has been demonstrated. Recognition also generally leads to improved occupational hygiene measures to reduce exposure, leading to a primary reduction in risk. Education of potentially exposed workers as to the risks of sensitisation and means of protection may also encourage symptomatic workers to seek advice from their healthcare providers. Consequently it becomes difficult to disentangle any effects specifically attributable to the components of medical surveillance from those arising as the result of accompanying interventions. There seems little doubt, however, that where surveillance is practised, it should be done so by qualified and competent personnel.

Surveillance programmes typically use a respiratory questionnaire, although these are neither standardised nor validated. Following first employment, surveillance is often conducted at annual or 6-monthly intervals. The timing may appropriately differ since there may be variations in the typical latencies of different sensitising agents [32]. However, it seems wise to maintain the most intensive surveillance for at least the first 2 yrs after the start of exposure [33, 34]. Since doubt remains over the latency associated with some sensitisers, in particular flour dust, most surveillance programmes are continued for much longer periods, in general annually and indefinitely. The present authors are not aware of any evidence that the detection of allergic symptoms outside the chest (nasal or eye symptoms, for example) is helpful in the prevention of OA; this could be valuably studied. Nonetheless, allergic rhinoconjunctivitis is indicative of sensitisation and is an outcome that itself should be prevented.

One serious difficulty with company-based occupational health surveillance is in the interpretation of questionnaire responses. It is feasible that workers in larger companies, where the possibilities of relocation with the same employer are higher, may be more willing to admit to work-related asthma symptoms than employees in small companies who are more likely to become unemployed if found to have occupational asthma. Thus, it might be anticipated that screening medical questionnaires have better compliance and sensitivity in larger corporate settings. What seems clear, in either case, is that each employee should know the exact consequences of responding to any health-related questionnaire.

The use of objective measures in medical surveillance should improve sensitivity where compliance with the questionnaire may be low. Conversely, where compliance is high, objective measures should improve specificity, particularly in workplaces where nonsensitising agents can cause upper airway irritant symptoms, which may result in high rates of respiratory symptoms. Most would agree, nevertheless, that in a setting where it is safest to maintain a low threshold for referring workers for full assessment, sensitivity is more important than specificity. Workplace spirometry is a traditional component of surveillance, but there is little data on its diagnostic performance. In a study of bakers who were fearful of job loss, spirometry in the surveillance programme did detect a few workers whose disease had not been revealed by questionnaire [35]. Conversely, in a relatively large diisocyanate-using company, where alternative jobs were available and employer-worker relations were good, the use of spirometry did not add to the questionnaire findings and most abnormal tests were due to technical factors or nonoccupational disease [36]. Regular spirometry, of course, may be useful for the detection of other disease in workplaces with respiratory risks.

Skin-prick testing (or the measurement of serum specific IgE) with a specific workplace allergen is feasible for some sensitisers, such as complex platinum salts and high molecular weight allergens, such as natural rubber latex, enzymes and flour. Asymptomatic skin sensitisation commonly occurs, as do work-related symptoms, due to other allergens or workplace irritants [37, 38], but the results interpreted with questionnaire responses and medical assessment, where indicated, can assist in appropriate decision making. In some cases, such as work with complex platinum salts or acid anhydrides, a positive skin test has a high predictive value for the development of OA and should lead to appropriate intervention with removal from further exposure [39]. Similarly, an effective medical surveillance programme relying mainly on questionnaires and skin testing has been developed in the detergent industry for enzyme-exposed workers [40]. Interestingly, the results of IgE measurements in this setting are used chiefly as an indication of successful exposure control [41].

## Are preventive programmes effective?

Formal techniques for evaluating the outcome of preventive measures have been exhaustively described by CHERRY [42]. In the case of OA, for which there are few meaningful "no adverse effect levels" for exposure, the ultimate objective must be to measure a change in disease (or sensitisation) incidence. This may be difficult where the baseline incidence is already low. Furthermore, practical obstacles lie in the difficulty of determining an adequate comparison group. These reasons alone probably explain why formal evaluation in the prevention of OA is virtually unknown; success is only likely to be found among very large industries, groups of similar industries or where the incidence is unusually high.

The former requires a considerable degree of co-operation or regulatory muscle; the latter, thankfully, is rare.

Crudely, the impact of preventive programmes may at least be surmised using data from external surveillance schemes. National schemes now exist in a number of countries and may provide a broad index of changing disease incidence, which, in turn, may be correlated in time with a preventive intervention. Doubts remain, however, over whether such schemes are sensitive enough to detect real change, particularly for asthma caused by individual agents. Furthermore, of course, such correlations must be interpreted with caution, as there is seldom any basis for comparison.

There are, undoubtedly, intense efforts being made in many industries to prevent OA. Few of these appear to have been evaluated systematically. Fewer still have reached publication, and none, as far as the current authors are aware, describe an unsuccessful intervention. Below a small number of evaluative reports are described. None of the studies has a sophisticated design, each relying on before/after comparisons with no other basis for reference. In each case the distinction between incident and prevalent cases is unclear, and in most there is uncertainty over denominators. These examples are not exhaustive; the authors are aware of others in the literature, but they involve either very small numbers [43], lack a control group [44] or describe what might be interpreted as tertiary preventive activities [39].

### *Enzymes in the detergent powder industry*

Following very high incidence rates of OA and reports of a small number of cases in detergent users, granulated proteases (accompanied by more stringent engineering controls) were introduced in the 1970s. Two publications [45, 46], each sponsored by major manufacturing companies, describe dramatic reductions in the prevalence of OA following these interventions (fig. 1). Unfortunately, neither report incidence rates and it is not entirely clear how much of the falling prevalence might be due to factors such as the "survival" of unaffected employees. Nonetheless, the implication from each is that concerted (and successful) attempts to reduce workplace exposures can be highly effective in the primary prevention of occupational asthma.

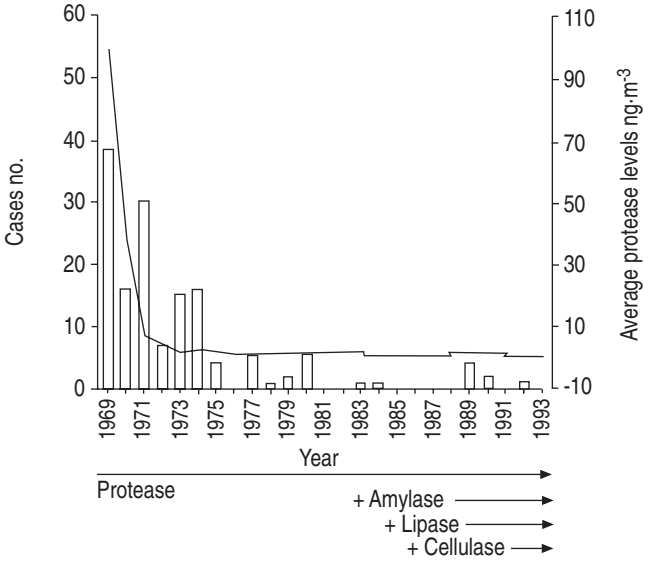


Fig. 1. – Occupational asthma, protease levels and enzyme use in the UK detergent industry. Reproduced from [45] with kind permission from the Society of Occupational Medicine.

Some 20 yrs later (fig. 1) other enzymes were introduced in a similar, encapsulated format and most biological washing powders now contain protease in combination with an amylase, cellulase or lipolase. Recently, a very large outbreak of OA in a single factory, with most cases sensitised to more than one enzyme type, was described [47]. The outbreak has not been fully explained but the rapid introduction of new enzyme types together with management failures to put industry guidelines into practice may have been important. An additional factor may have been the factory's obligation, through its position in the marketplace, to manufacture a large number of different detergent products in rapid succession. The lessons, perhaps, are that disease prevention may be contingent on forces beyond the immediate workplace environment; and that vigilance must be eternal, especially where new (potential) allergens are being introduced.

### *Diisocyanates*

An example of more structured evaluation is provided by the experience of OA due to diisocyanates. Legislation, introduced in l983 by the Ontario Ministry of Labour, required monitoring of diisocyanate levels to maintain 8-h concentrations <5 parts per billion (ppb) and short-term exposure levels <20 ppb. In addition, mandatory medical surveillance measures were introduced: a questionnaire and spirometry pre-employment, with repeated respiratory questionnaires every 6 months and spirometry at least on an annual basis. Workers with lower respiratory symptoms on questionnaire, or changes in spirometry, were required to have a medical assessment. There was no equivalent legislation to provide surveillance for other respiratory occupational sensitisers, although some nondiisocyanate-using firms (for example, those using enzymes) had their own programmes.

Throughout the evaluation period, diisocyanates remained the most commonly recognised cause of compensated occupational asthma (fig. 2). Indeed, there was an initial increase in annual compensation claims after introduction of the surveillance programme, consistent with increased case-finding [48]. This, however, was followed by reductions in the proportionate and actual numbers of accepted diisocyanate-induced asthma claims for the last few years in which data were reviewed. In addition, among workers in companies

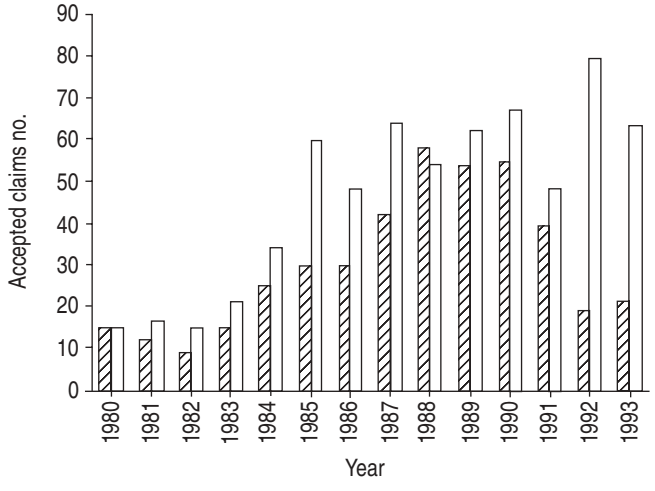


Fig. 2. – Accepted claims for diisocyanate-induced asthma (▨) and accepted claims from other causes of asthma (□), by year of onset, in Ontario. Reproduced from [48] with kind permission from BMJ publishing Group.

known to be in compliance with the programme, there was an earlier diagnosis of OA: a mean of 1.7 yrs after onset of symptoms compared to 2.7 yrs for workers without documented compliance. Similarly, there was a reduction in the mean time to diagnosis of diisocyanate asthma from 3 yrs to 2.1 yrs ($p=0.014$). Perhaps as a consequence, indices of asthma severity at the time of diagnosis suggested milder disease in those diagnosed in the second period of the study, both for the group accepted for diisocyanate asthma and the group accepted for other causes of OA.

When measured levels of diisocyanates were compared among diisocyanate-using companies who had compensated claims for OA with companies without accepted claims, the former were significantly more likely to have had a measured level of diisocyanates $\geqslant 0.005$ parts per million [49]. This is consistent with a previous report suggesting a low rate of sensitisation in a new plant engineered to minimise exposure levels [44].

These figures represent one of the first, large-scale attempts to measure the effectiveness of a prevention programme in OA. Although directed against a single agent, the intervention was aimed at a large number of firms of different sizes. The findings suggest that a combination of regulated exposure and medical surveillance can be successful in the (secondary) prevention of asthma at work; and even that the beneficial effects may spill over to other industries.

## *Laboratory animal allergy*

BOTHAM *et al.* [50], working in the occupational health unit of a large research establishment in the UK, studied a retrospectively assembled cohort of new employees working with laboratory animals. Those who entered employment between 1979 and 1982 were followed for 3 yrs; the cohorts starting in the subsequent 2 yrs were studied for 1–2 yrs. Figure 3 depicts the incidence rates of respiratory symptoms attributed to laboratory animal allergy in each of the annual cohorts. In 1981 a new code of practice for working with laboratory animals was introduced at the site, accompanied by a series of educational lectures designed to increase awareness of the laboratory animal allergies.

Although the design of this evaluation is fairly crude, "one-group pretest-posttest" in COOK and CAMPBELL′s nomenclature [51], it seems reasonable to attribute the improvements, in part at least, to the new preventive programme. The study is unusual in this field for its relatively careful design and analysis; it remains a useful example of how primary preventive measures may be evaluated with relative ease at a single site, albeit one with an apparently very high incidence of disease.

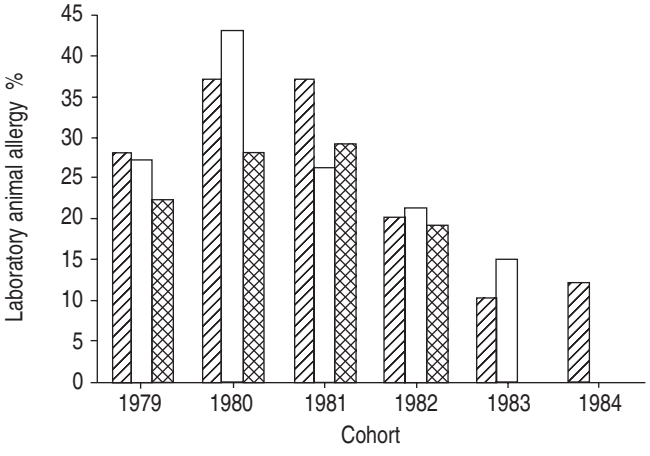


Fig. 3. – Incidence of laboratory animal allergy at a UK research site, 1979–1984. ▨: year 1; □: year 2; ■: year 3.

A similar study has been reported from a pharmaceutical research institution in the USA [52]. As above, there is some lack of clarity over the calculations of incidence used, but the findings suggest that laboratory animal allergy can be effectively prevented by a targeted programme; in this case including education and training, modification of work practices, engineering controls, the use of personal protective equipment and a standardised system of surveillance. The latter included yearly measurement of specific IgE antibodies to a variety of animal allergens encountered either at home or at work. A positive result to any of these had a low predictive value (30%) for self-reported symptoms; the comparative figure for a positive result to a workplace allergen was not reported. BOTHAM *et al.* [53] suggest that the presence of specific IgE to laboratory species is highly predictive of subsequent clinical allergy.

## *Natural rubber latex*

The annual number of allowed claims for latex-induced OA declined after the Ontario Workplace Safety and Insurance Board encouraged hospitals to use powder-free, low-protein or nonlatex gloves in 1996 [54]. In Germany, a widespread information campaign for hospital administrators was accompanied by a revision in the (compulsory) technical regulations for dangerous substances, which stated that only low-allergen, powder-free latex gloves should be used in healthcare settings [55]. These changes took place between 1997 and 1998. Data on the number of suspected cases of skin or respiratory latex allergy reported to a large insurance company, covering around half of the nation′s hospitals, were collected between 1996 and 2001 (fig. 4). The temporal relationship, with a 2-yr lag, between the decline in purchases of powdered gloves by acute care hospitals and the fall in the number of cases of OA was interpreted as evidence of success for the preventive programme. A decline in latex allergy has also been documented, although on a far smaller scale, in hospitals and dental schools where powdered latex gloves

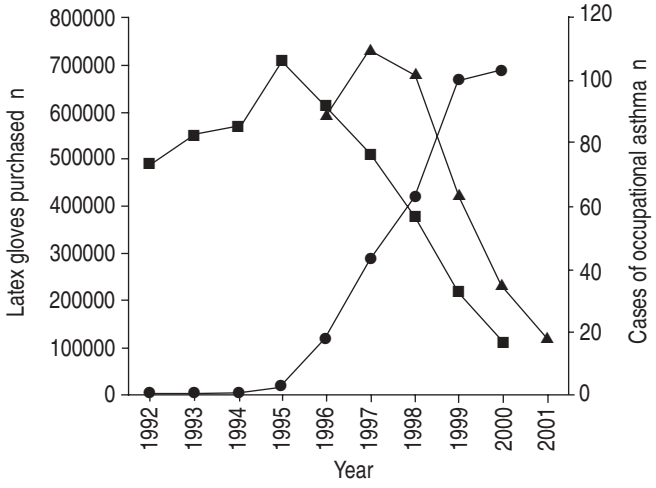


Fig. 4. – Reported cases of occupational asthma (latex) and gloves purchases, 1992–2001, Germany. ■: powdered gloves; ▲: powder-free gloves; ●: cases of occupational asthma. Reproduced from [55] with kind permission from MOSBY, Inc.

were substituted by low-protein or powder-free latex gloves [56, 57].

The story of latex allergy is instructive. There is increasing evidence, albeit largely anecdotal, that after only some 15 yrs, the epidemic of occupational allergy among healthcare workers is coming to an end. This has probably been achieved through the rapid understanding that glove dusting powder (and protein content) was directly related to the risk of sensitisation and, importantly, that each of these could be rectified with relative ease. It is tempting to suggest too that the very high profile of the disease, which was undoubtedly helpful in understanding its aetiology, was a result of its heavy impact on healthcare professionals.

## Conclusions

OA is an important industrial disease because it is not uncommon, is moderately disabling and is costly at both individual and societal levels. The present authors argue, however, that there is little evidence of sustained, successful prevention of the disease. This is not because its aetiology is not sufficiently well understood. Rather it has arisen because the disease is conceptually fragmented, has a low industrial and public profile and, although induced by myriad agents, is relatively uncommon within a single workplace; all of which conspire to make the disease difficult to target.

Primary preventive efforts should be concentrated on exposure reduction through improved dust controls accompanied by intense educational programmes within at-risk workforces. Pre-employment screening measures are doubtful ethically and legally, and are highly inefficient. This situation is unlikely to change in the near future, but the issue may become more intense as the prevalence of constitutional allergies in the community rises. Secondary prevention is almost certainly useful in reducing the impact of the disease, but current methods require considerable refinement.

There is always room for further and more detailed aetiological study of occupational but the authors suggest that there should be an increased focus on the application of epidemiology. Thus, decisions on disease control should be made in the light of existing knowledge and scientists in this area should engage in their structured evaluation. This will certainly require more extensive collaboration with regulatory bodies and industry than is presently the rule.

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