

Allergy – a public health issue: when should governments act?

Epidemiologists have, in successive studies over the last two or three decades, demonstrated the progressively increasing health burden of all the allergic diseases but with particular emphasis on asthma, atopic dermatitis and allergic rhinitis. Latterly, attention has been directed at newer allergies such as peanut, latex and emerging novel foods. Given that the diseases pose a major economic burden on society, worldwide governmental attention must be focused on ways in which this veritable epidemic of allergic disease can be halted.

Unfortunately, answering the questions as to why the epidemic has occurred has proved very difficult. That there is a problem is not in doubt, but how the problem should be tackled is not so clear. However, as readers of the pages of *Pediatric Allergy and Immunology* will attest, this is not for want of trying. A vast number of groups worldwide are attempting to identify the key factors influencing the development of atopic disease and this has moved to investigations progressively earlier in life. There is little doubt that a number of factors linked with an affluent lifestyle can have an influence on the evolving immune responses of the fetus and infant, and, therefore, have an impact on risks for developing allergic disease. The 'hygiene' hypothesis has most recently gained the most credibility with conflicting views about the extent to which allergen load contributes, for good or for bad, to the emergence of allergy. Thus, while we await the results of controlled allergen avoidance studies commenced in fetal life, many people are beginning to believe that this will not provide definitive solutions to the problem and that some form of immune modulation may also be required.

Perhaps, therefore, in this rapidly changing field, it would be inappropriate for governments to take statutory action at this stage with regard to advising the general public on how to modify their lifestyle in order to reduce allergic disease. However, at least one piece of advice would seem entirely appropriate. Cigarette smoking is a health hazard at all ages and quite clearly has

very profound effects on the developing fetus. These effects extend beyond those of allergy, which raises the question of whether the time has come for all governments worldwide to take action to eliminate this major public health hazard. Such an act would certainly have an impact on the manifestations of allergic disease.

Unfortunately, making any further recommendations on modification of lifestyle might appear somewhat premature. However, the Department of Health in the UK has taken action, which I certainly believe is appropriate and reasonable, in the light of current knowledge of peanut allergy. Peanuts are not essential to the British diet but have been ingested in ever increasing quantities in the last two decades. Over this period, peanut allergy has become a prominent problem. There is no doubting the nutritional qualities of peanuts and for the nonatopic population, it is perfectly reasonable to recommend it as a good food. Nevertheless, to recommend it as appropriate for weaning foods or for inclusion in milk formulae would seem foolhardy. The Department of Health in the UK has taken the step of suggesting that, amongst atopic families, it would be sensible to avoid all peanut and peanut products (with the exception of purified peanut oils) during pregnancy and lactation with very late introduction of the products into the child's diet. We will at least be in a position to monitor this public health experiment by further assessments of peanut allergy prevalence in future years.

This, of course, raises the question of whether these recommendations should be extended to other foods, not to mention, whether we should also undertake intensive house mite avoidance measures and furred pet elimination. The Swedish Government have taken on this challenge by extensive public health education to persuade families to adopt such measures. It will, again, be very interesting to establish the outcome of this effort.

In the interim, governments and their advisers will never be winners. On the one hand, they may be criticized for taking premature action when evidence is incomplete. On the other hand, they

may well be accused of depriving the population of appropriate advice as information accumulates in the scientific literature. I believe that it is incumbent on health services to provide their populations with factual but comprehensible information as soon as it becomes available so that the general public can make informed decisions of their own. Once evidence becomes

incontrovertible then, of course, statutory action is required, which surely is the case in relation to environmental tobacco smoke, although perhaps not to allergens. I would be interested to hear the thoughts of others.

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Editor-in-Chief

In this issue

This issue is dominated by papers on peanut allergy, which has become a very prominent problem throughout North America and Europe. The uniqueness of this allergy lies in its capacity to generate acute severe and life-threatening reactions relatively more commonly than for most allergens. Furthermore, the extensive use of peanuts and peanut extracts in manufactured foods has led to accidental exposures occurring all too frequently with occasional tragic consequences. Two papers, one from Sweden and the other from South Africa, have attempted to identify when and how peanut exposure in early life might be generating the sensitivity. The Swedish study provides a little reassurance that perhaps the peanut oil content of vitamin supplements given to infants is not contributing significantly to the frequency of the problem, although there is a disturbing association with the use of such drops and a wider array of allergic symptoms on exposure to peanuts. This, in turn, may well be associated with more severe and life-threatening reactions. Thus the role of oils in sensitization is not yet totally exonerated. The South African group suggest that maternal consumption of peanuts during pregnancy may well contribute, which is reassuring to those of us who were involved with advising the Department of Health in the UK to recommend that atopic mothers should not eat peanuts during pregnancy or lactation.

We also have the second review from Trevor Bryant, our statistical adviser. In this article, he provides very sensible advice on the way in which data can be presented graphically. I would strongly advise all our authors to pay attention to the recommendations before submitting papers in the future.

Sticking with our theme of food allergy, the group from Toulouse (headed by Denise Moneret-Vautrin who, of course, has had a long-standing

interest in the early life origins of peanut allergy) has conducted a survey amongst their allergy clinic referrals to give a perspective on the range and manifestations of food hypersensitivity reactions. Not unexpectedly, atopic dermatitis is the main symptom in early childhood but over time other manifestations appear including the life-threatening reactions. Egg, peanut, cow's milk, mustard and cod between them account for nearly 80% of all the hypersensitivities. Advisers in the European Union would do well to pay attention to the figures highlighted in this study in generating regulations on food labelling. However, the data are based on selected clinic referrals rather than a whole population study.

We have a most interesting paper from Bonville and colleagues from Syracuse. Currently we have relatively few submissions from the USA and hope that this will herald many more. They have measured levels of chemokines generated in supernatants of virus-infected respiratory cell cultures. This demonstrated differences dependent on the virus involved. However, *in vivo*, at least from the upper respiratory tract, the profile was slightly different. The authors focus on the potential role of the chemokines in drawing eosinophils into the airways as a mechanism by which reactive airway disease might develop. Nevertheless, I would choose to also consider the role of neutrophils which, of course, are seen in significantly increased numbers, particularly in respiratory syncytial virus (RSV) infection, and may also contribute to the evolution of asthma.

There then follows a most interesting study from Sweden further investigating the effect of exposure to animal dander in schools. It is becoming clear that animal dander allergy is progressively more important than the house dust mite in causing ongoing problems because of the ubiquitous nature of the allergen. Despite relatively mild disease in the patients studied,

exposure to the allergen in schools resulted in increased bronchial hyper-responsiveness and changes in eosinophil numbers suggesting recruitment to the airways. There was an interesting trend to increases in both eosinophil and neutrophil activation products in the sputum which, again, raises the question as to whether neutrophils might also have an important role in asthma.

The paper from Austria has demonstrated significant histamine release occurring very early in children having subsequent systemic reactions after insect sting challenges. While this would ostensibly appear to be a useful objective marker of severity, unfortunately the rigours of the assay mean that results will only be available long after the reaction has occurred. It will, however, help the clinician to distinguish those patients who have panicked and over-breathed during the course of the reaction which might be mis-attributed to a systemic response.

The paper from Oslo in Norway provides some interesting insights into medical practice in that part of the world. There have been interesting swings in fashion. In the 1970s, the dogma was that asthma was under-diagnosed and that all wheezing should be called asthma and appropriately treated. We are now beginning to get back to the stage we were in the 1950s and 1960s of separating a group of young children with wheezy bronchitis from those with asthma. The differences in frequency of exercise-induced bronchoconstriction and atopy in groups so diagnosed vindicates the 'splitter' rather than 'lumper' approach. It is not clear, however, from the

paper to what extent the diagnostic attribution is a reflection of severity of disease as based on frequency of wheeze or identification of non-virus-based precipitants.

We finally have a short paper on the use of a multi-dose powder inhaler with measurements of peak inspiratory flow through the inhaler. It is imperative that such studies are performed whenever any new inhaler device is introduced onto the market to establish which age range of children is able to use the device effectively.

We also have an exchange of letters about latex allergy. Jennifer Worth raises the question as to whether the newborn baby's exposure to latex on the gloves of the midwives or obstetricians might be contributing to the dramatic appearance of latex allergy in the paediatric community. However, it is perhaps stretching credibility to suggest that this exposure might also have contributed to the overall increase of atopic diseases. The multi-centre allergy study (MAS) reported by Bodo Niggemann (1) in a previous issue of the journal suggests otherwise, but clearly only a carefully controlled prospective study would provide the final answer.

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References

1. NIGGEMANN B, KULI M, BERGMANN R, WAHN U. Development of latex allergy in children up to 5 years of age – a retrospective analysis of risk factors. *Pediatr Allergy Immunol* 1998; 9: 36–9.