

Correspondence

Longitudinal changes in skin-prick test reactivity

I was intrigued to read Johnston *et al.*'s paper on changes in skin-prick test reactivity over a 2-year period in a population of schoolchildren with respiratory symptoms [1]. Many years ago, I investigated the variability of skin-test responses in two groups of children, one with cystic fibrosis and the other with allergic asthma [2]. Over a 6-month period, there was an increase in the number of cystic fibrosis children showing evidence of allergy from 58% to 69%, the major changes occurring in responses to *Aspergillus fumigatus*. In studying individual responses to seven common allergens amongst those that produced negative skin tests on first testing, there was a 10.5% gain at second testing. Amongst those positive at first testing, there was a 28% loss at the second test. In the group of allergic asthmatic children, there were inevitably none over 6 months who became non-atopic. Of the individual skin tests, 50% yielded positive reactions, of which there was a 15% loss of reactions after 6 months and, amongst those giving negative responses, there was a 9% increase. Most interestingly, there was a strong association between change in skin-test responsiveness and change in asthma control. Irrespective of whether the asthma improved due to treatment or spontaneously, this was associated with a loss of skin-test responses and worsening asthma was associated with acquisition of more positive reactions. At that time, I proposed that continuing allergen exposure and, therefore, continuing symptoms, maintained a mucosal defect which resulted in persistence of susceptibility and may even have enhanced new sensitization, whilst control of the mucosal inflammation would lead to re-establishment of normal mucosal defences and a tendency to lose sensitivities. My results might explain some of the differences between Johnston *et al.*'s observations and those of other studies discussed in the paper [3,4]. As the Johnston patients all had significant respiratory symptoms, it is likely that their airway mucosa were inflamed and, therefore, more liable to allow allergens to penetrate and sensitize.

Overall, the cystic fibrosis patients had a greater variability of skin-test response which suggests that the cause of the allergy was different and perhaps more related to a secondary mucosal abnormality due to infection. The question of whether cystic fibrosis heterozygosity contributes to the pool of atopy in the population has been addressed in the past, before the identification of the CF gene [5]. These studies must now be repeated using molecular genetics to identify heterozygotes. Such individuals may have a very different

underlying defect which might lead to greater variability of allergic skin-test responses than those with other putative atopy genes.

References

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Reply

We thank Professor Warner for his interest in our paper, and for his illuminating comments and interpretation. His suggestion that our findings in children of a higher rate of conversion from negative to positive skin-test results [1] than those reported in previous studies [2,3], might be explained by a high prevalence of respiratory symptoms, and therefore, presumably a damaged mucosal barrier, is highly plausible and echoes our own interpretations outlined in the discussion.

We have now had the opportunity to analyse the detailed respiratory symptoms and peak flow recordings (PEF) monitored for periods of one year from the dates of each skin-prick testing in 1987 and 1989. Interestingly, although all the children were recruited to these studies on

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