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LETTERS TO THE EDITOR

Cohabiting with domestic mites

Dr IH Feather and colleagues have reviewed the central role of house dust mites in the aetiology of allergic asthma (January 1993;43:5–9). In my personal series of 744 consecutive asthmatic children aged 5–15 years compiled over the years 1985–91, positive skin prick test reactions to *Dermatophagoides pteronyssinus* (ALK, Copenhagen) were nevertheless found in only 180 subjects (24%) and more often among those from rural domiciles, large families, cramped dwellings, and blue collar social classes (table).

The mite population in homes is dependent on the supply of food—that is, human skin scales—and air humidity. Since there is generally no shortage of the former it is the humidity that is the decisive factor for mite survival. In many regions the circumstances are ideal for mite growth, and bedrooms in particular are universally infested with mites. In Scandinavia, however, the relative humidity indoors is low during the long cold season from September to May when the houses are heated continuously, and conditions are therefore unsuitable for mites. Modern energy saving with heat insulation and poor ventilation have caused a change in the traditional indoor climate and a rapid rise in mite concentrations and hypersensitivity to mites has resulted in Stockholm,[1 2] but in northern Scandinavia (for example, Finland) hypersensitivity to mites still remains more or less a social problem. Crowded living conditions provide a food supply for mites and an additional water vapour load which together provide conditions suitable for mite survival. In view of the limited efficiency of acaricides and vacuum cleaning, I agree with the opinion that the mite problem cannot be solved without a change of housing conditions in general.

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1 Nordvall SL, Eriksson M, Rylander E, Schwartz B. Sensitization of children in the Stockholm area to house dust mites. *Acta Paediatr Scand* 1988;77:716–20.
2 Wickman M, Nordvall SL, Pershagen G, Sundell J, Schwartz B. House dust mite sensitization in children and residential characteristics in a temperate region. *J Allergy Clin Immunol* 1991;88:89–95.

AUTHORS' REPLY We agree strongly with Dr Linna's observations that the microclimate, especially the indoor humidity, is the most critical determinant of house dust mite proliferation. Oulu is situated virtually on the Arctic circle and therefore represents a somewhat unusual climatic situation, perhaps analogous to the relatively mite free environments found at altitude in Europe. In the Oulu community, where hypersensitivity to mites is apparently lower than in more temperate regions, it would be interesting to know if the overall incidence of asthma is also lower than in these more temperate regions.

Dr Linna's observation on family size is also interesting in view of the observations of others that the risk of atopy changes with the relative age position of the child within the children of the family, and so it would also be interesting to see whether this accounted for the increased risk of sensitisation in the large families, or whether this was purely a further reflection of more crowded housing. Similarly, is the increased risk in the families of blue collar workers explained by the likelihood of more crowded housing in this group, as it is at some odds with other observations of a greater risk of atopy in the upper socioeconomic classes?

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Risk factors for house dust mite allergy

Factor	*Odds ratio*	*95% confidence limits*	*p*
Rural domicile	2·79	1·81 to 4·31	< 0·001
Large family*	2·61	1·76 to 3·87	< 0·001
Cramped dwelling**	2·35	1·53 to 3·60	< 0·001
Blue collar social class	1·45	1·01 to 2·09	< 0·05

*Four or more children. **Two or more persons per room.

Tuberculin positive children

We read with interest the article by Dr K Citron (October 1992;47:768–9) which raised a number of important points about mycobacterial infections in children and how to interpret a positive tuberculin reaction. There are, however, several aspects of this subject where confusion remains, and we have performed some studies that could add some information to this subject.

In Sweden we generally use the Mantoux technique with an intracutaneous injection of 2 tuberculin units PPD RT23 in accordance with the recommendation by WHO.[1] An induration of > 6 mm is designated as a positive tuberculin reaction. The general BCG vaccination of newborn children ceased in 1975. When healthy, non-BCG vaccinated Swedish born children with no history of tuberculosis were tuberculin tested, however, many were tuberculin positive. These unexpected findings stimulated some studies in which 7000 children were simultaneously or sequentially tested with tuberculin PPD RT23 and a sensitin (a tuberculin derived from atypical mycobacteria).[2] This study showed that 3% of the children had a tuberculin reaction >6 mm whereas up to one third of the children had an induration >6 mm to the sensitin. Those children who reacted with a tuberculin reaction >6 mm were examined by a physician and a chest radiograph was taken. None of these children had signs of tuberculosis. The results of these studies showed that children with moderate and large tuberculin reactions had still larger sensitin reactions indicating cross reaction caused by infection with atypical mycobacteria. There were, however, variations depending on age and geographical factors.

If chemoprophylaxis is considered in a child with a positive tuberculin reaction we recommend that a sensitin test is also performed. A sensitin reaction which clearly exceeds the tuberculin reaction indicates that the child is infected by atypical mycobacteria and that the positive tuberculin reaction is a cross reaction resulting from antigenic similarity between the tuberculin and sensitins used. If a known tuberculosis contact exists, however, the sensitin test is unnecessary.

In summary, infections by atypical mycobacteria are common in Swedish children and a similar situation might exist in other countries. In the Netherlands, for example, there has been a significant increase in non-specific tuberculin sensitivity during the last two decades.[3] The tuberculin test is of limited value in Sweden at present because of cross reactions with atypical mycobacteria, and may be of limited value in other countries with a low prevalence of tuberculosis.

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1 The WHO standard tuberculin test. *WHO/TB/Techn.Guide* 22 February 1963.
2 Lind A, Larsson LO, Bentzon MW, Magnusson M, Olofson J, Sjögren I, *et al.* Sensitivity to sensitins and tuberculin in Swedish children I. A study of schoolchildren in an urban area. *Tubercle* 1991; 72:29–36.
3 Bleiker MA, Misljenovic O, Styblo K. Study into the risk of tuberculous infection in schoolchildren in the city of Delft (the Netherlands) in the period 1966–85. *Selected Papers* 1987;23:19–24.

AUTHOR'S REPLY I thank Drs Larsson, Skoogh and Lind for pointing out that in Sweden children may be tuberculous positive as a result of sensitisation to atypical (non-tuberculous) mycobacteria.

In Britain surveys of schoolchildren have shown a greater prevalence of sensitivity to avian than to human PPD.[1] The proportion of unvaccinated tuberculin positive children who are Heaf grade 1 and 2 reactors has increased over the years and these weak reactions are likely to be due to sensitisation to non-tuberculous mycobacteria. This might account for the fact that the risk of tuberculin positive schoolchildren developing tuberculosis has greatly diminished during the last two decades in Britain.[2] The Department of Health has provided guidelines for the management of unvaccinated schoolchildren found to be tuberculin positive on routine testing.[3] Heaf grade 1 reactors are offered BCG. Grade 2 reactors need no follow up since their tuberculosis risk is very low. Grade 3 and 4 reactors have an appreciable risk of tuberculosis which has not diminished over the years.[4 5] These children should be referred for investigation and supervision which may include chemoprophylaxis.

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