

The allergenicity of feather bedding material

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Key words: feather allergy; house dust mite.

● IT HAS LONG been recognized that exposure to high levels of antigen is an important risk factor for the development and exacerbation of atopic asthma and other allergic disorders (1). For this reason allergen avoidance is advised for subjects with allergic disease and for children at risk of developing allergy (2). The principal indoor allergen is the house dust mite (3); several strategies have been suggested to reduce exposure to mite allergens in the bedroom, such as occlusive covers for mattresses, pillows and duvets (4).

Additionally, synthetic materials for pillows and duvets has been encouraged to avoid sensitization to feather allergens. However, a recent Finnish study has concluded that most positive skin test

reactions seen with feather extracts are due to mite allergens (5). Feathers used

Feather bedding material does not induce skin test response.

for production of bedding material are extensively washed before use and feather bedding appears less allergenic than synthetic alternatives (6). This is partly due to the outer covering associated with the two types. Synthetic fillings have lighter thinner covering material. This facilitates the entry of house dust mites into the filling. Allergic symptoms to bedding are most probably related to Der p 1 sensitivity rather than the filling.

We investigated the prevalence of sensitivity to feather bedding material in a dust mite sensitive population. Their previous exposure to feather bedding material was assessed during interview and compared with observed sensitivity. 105 patients with confirmed dust mite (Der p 1) sensitivity and a symptomatic history to dust were included in the study. A further 25

nonatopic subjects were included as a control group.

Each subject was skin prick tested against dust mite, grasses, cat and commercial chicken feather extract (Bayer) and against extracts of duck feather, goose feather and polyester filling material. A positive response was considered as equal to or greater than a 3-mm wheal difference from the negative control.

Protein concentrations from the two feather extracts were extremely low at 9 µg/g feathers for goose and 18.5 µg/g feathers for duck. This illustrates the efficiency of the washing process used for preparation of the bedding material.

All atopic subjects responded to the Der p 1 and some to cat and/or grasses. There were no positive reactions to any of the feather or synthetic bedding extracts or the commercial chicken feather skin test reagent used, confirming the low prevalence of feather allergy demonstrated by other workers (5). None of the nonatopic controls reacted to any of the skin test solutions apart from the histamine control.

Of the atopic subjects only 15% were using feather filled bedding material and 25% indicated previous use such but had discontinued use of such products following concern over possible allergenicity. Contact with feather bedding material more than two years previously was considered as no contact. There was no association between skin test reactivity and current or previous use of feather bedding.

In conclusion, the feather fillings used in this study do not have the potential to sensitize individuals to feather allergens in normal usage.

The nomenclature used in this study complies with that of the EAACI position paper (7).

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References

1. DREBORG S. Mite allergens. Collection, determination, expression of results, and risk levels for sensitisation and symptom induction. *Allergy* 1998;**53**:88–91.
2. CUSTOVIC A, SIMPSON BM, SIMPSON A, et al. Manchester Asthma and Allergy Study: low-allergen environment can be achieved and maintained during pregnancy and in early life. *J Allergy Clin Immunol* 2000;**105**:252–258.
3. CUSTOVIC A, SIMPSON A, WOODCOCK A. Importance of indoor allergens in the induction of allergy and elicitation of allergic disease. *Allergy* 1998;**53**:115–120.
4. VANLAAR CH, PEAT JK, MARKS GB, RIMMER J, TOVEY ER. Domestic control of house dust mite allergen in children's beds. *J Allergy Clin Immunol* 2000;**105**:1130–1133.
5. KILPIO K, MAKINEN-KILJUNEN S, HAAHTELA T, HANNUKSELA M. Allergy to feathers. *Allergy* 1998;**53**:159–164.
6. RAINS N, SIEBERS R, CRANE J, FITZHARRIS P. House dust mite allergen (Der p1) accumulation on new synthetic and feather pillows. *Clin Exp Allergy* 1999;**29**:182–185.
7. JOHANSSON SGO, O'B HOURIHANE J, BOUSQUET J, et al. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2001;**56**:813–824.

Unusual soy oil allergy

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Key words: elemental amino acid formula; non IgE-mediated allergy; soy oil.

● THOMAS WAS born uneventfully from a normal pregnancy and fed with milk formula. At one month of age the first signs of the IgE-associated subgroup (AD) of the atopic eczema/dermatitis syndrome (AEDS) (1) led to a prick test, which was positive to milk: 12 mm (codeine: 3 m). The pediatrician substituted soya milk but the AD worsened, and at five months of age the atopic dermatitis severity score

(SCORAD) was 45 (2). Prick tests to cow's milk and soya milk were negative, so diet was not modified till he reached 10 months of age. At this time AD was generalized and patch tests were carried out (3). Positivity to soya milk led to its withdrawal. Despite feeding with a casein extensive hydrolysate, the infant failed to improve: daily

rashes were accompanied by vomiting and diarrhoea. An amino acid formula (AAF)

IgE-mediated allergy to an elemental amino acid formula.

was prescribed. The digestive symptoms resolved but SCORAD remained at 34.

This AAF contains a soy lipidic emulsion. Skin tests with separate components of AAF were carried out. Patch tests were positive to the soy oil emulsion only. An oral challenge to the soy oil emulsion (30 ml in 240 ml of water) induced a rash four hours later.

The AAF was stopped for three days. A partial regression of the inflammatory state was observed. Conversely, the reintroduction of 240 ml of AAF on the fourth and fifth days provoked a rash in the afternoon of day four, and exacerbation of the eczema on day five. SCORAD rose from 27 to 45.

Neocate Advance® that does not contain soy lipidic emulsion was substituted. A complete recovery was observed six weeks later.

Following IgE-dependent allergy to cow's milk, the child developed T cell mediated allergy to soy (1). In such cases, replacing cow's milk with extensive hydrolysates (EH) is advised (4). However, allergy to casein EH occurs in 8% of cases (5). Substitution with AAF is usually effective, and safety is excellent (6). A case of allergy to AAF was attributed to contamination (7).

The role of soy oil was suspected on the basis of the T cell-mediated allergy to soy. Indeed, allergens in soy lecithins and soy oil have been evidenced (8,9) and allergic reactions to soy lecithin have been observed (10).

AAFs are used specifically for infants presenting with severe CMA, often associated with multiple food intolerance or with allergy to hydrolysates. These infants can be considered to be the most atopic subjects, reacting to traces of allergens. It would seem advisable to

replace the soy oil by other vegetable lipids in AAFs.

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References

1. JOHANSSON SGO, O'B HOURIHANE J, BOUSQUET J, et al. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2001;**56**:813–824.
2. EUROPEAN TASK FORCE ON ATOPIC DERMATITIS. Severity scoring of atopic dermatitis: the SCORAD index. *Dermatology* 1993;**186**:23–31.
3. ISOLAURI E, TURJANMAA KE. Combined skin prick and patch testing enhances identification of food allergy in infants with atopic dermatitis. *J Allergy Clin Immunol* 1996;**97**:9–15.
4. HÖST A, KOLETZKO B, DREBORG S, MURARO A, WAHN U, AGGETT P, et al. Dietary products used in infants for treatment and prevention of food allergy. *Arch Dis Child* 1999;**81**:80–84.
5. DE BOISSIEU D, DUPONT C. Time course of allergy to extensively hydrolyzed cow's milk proteins in infants. *J Pediatr* 2000;**136**:119–120.
6. ISOLAURI E, SÜTAS Y, MÄKINEN-KILJUNEN S, OJA SS, ISOSOMPI R, TURJANMAA K. Efficacy and safety of hydrolyzed cow milk and amino-derived formulas in infants with cow milk allergy. *J Pediatr* 1995;**127**:550–557.
7. NILSSON C, OMAN H, HALLDEN G, LILJA G, LUNDBERG M, HÄRFÄST B. A case of allergy to cow's milk hydrolysate. *Allergy* 1999;**54**:1322–1326.
8. AWAZUHARA H, KAWAI H, BABA M, MATSUI T, KOMIYAMA A. Antigenicity of the proteins in soy lecithin and soy oil in soybean allergy. *Clin Exp Allergy* 1998;**28**:1559–1564.
9. ZITOUNI N, ERRAHALI Y, KANNY G, MOUTETE F, METCHE M, MONERET-VAUTRIN DA, et al. Soy allergens are detected in some edible soy oils. *J Allergy Clin Immunol* 2001; **107**:S188.
10. PALM M, MONERET-VAUTRIN DA, KANNY G, DENERY-PAPINI S, FRÉMONT S. Food allergy to egg and soy lecithins. *Allergy* 1999;**54**:116–117.

Natural course of AEDS

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Key words: asthma; atopic dermatitis; childhood; intrinsic type; natural course.

• WHAT WAS previously called atopic dermatitis (AD) is a common inflammatory skin disease that often develops in early childhood. Its natural course over the time varies considerably from case to case: the skin manifestation can disappear after infancy, childhood or puberty, but it can also persist in the adolescent and in the adult phase (1,2). Of children with AD 30–60% will at a later age develop respiratory diseases such as asthma bronchiale (AB) or allergic rhinitis (AR). Recently, the term 'atopic eczema/dermatitis syndrome' (AEDS) was proposed by the EAACI nomenclature task force (3) to underline the fact that AD is not one single disease but rather an

Children with nonallergic variety of allergic eczema/dermatitis syndrome rarely get asthma.

aggregation of several diseases with certain clinical characteristics in common (4). The nonallergic AEDS (former called 'intrinsic' type of AD) is characterized by eczematous skin lesions in typical localization similar to the IgE-associated subgroup of AEDS, but is not associated with a specific sensitization either in the skin tests nor in RAST/CAP to common environmental allergens (5).

In this study we performed clinical and allergological investigations: skin prick tests (SPT), total and specific IgE determinations against an inhalant (Sx1) and a food mix (Fx5)) in a group of 22 children with AEDS seen at the age of 2–4 years.

Eight years later the same children, now aged 10–12 years, were re-evaluated. In the follow-up we investigated the persistence of the skin disease after early childhood and the development of respiratory allergies. In particular, we were interested to know how many patients with the first diagnosis of nonallergic AEDS were still