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RAST WITH HUMAN DANDER ALLERGEN IN ATOPIC DERMATITIS

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Abstract. Using highly purified allergens from skin flakes of the human scalp and from house dust, it was demonstrated that IgE antibodies against the former can be detected only in cases of severe atopic dermatitis (AD), but not in asthma (without eczema) and not in hay fever or rhinitis patients. The RAST scores for human dander correlated well with total IgE levels. Also, RAST scores for human dander allergen correlated very well with those for house dust. This could not have been due to allergenic cross-contamination, as the house dust RAST can be strongly positive in asthma and rhinitis patients, whereas the human dander RAST is always negative. Haemolytic complement consumption by both allergens, in contrast, correlated well in all these diseases. The results indicate a dissociation of basic mechanisms in asthma versus AD.

Key words: RAST: Human dander allergen; Atopic dermatitis

The allergen in skin flakes of the human scalp has long been associated with the atopic condition, most particularly with atopic dermatitis (AD) (7, 8, 9, 10, 11). In such patients, and in subjects with bronchial asthma, immediate-type skin reactions with aqueous human dander extracts are observed very frequently (7, 9, 11, 12, 14). Several authors have reported the successful transfer of this reactivity by way of the Prausnitz-Küstner reaction (8, 9, 11). Although this suggests mediation by reaginic antibody, no reports are available so far on the existence of IgE antibody against human dander components. In an unspecified group of sera of allergic patients, Brighton & Topping (5) reported very low RAST-scores with an extract of skin (not from the scalp). The present paper records our experience with human dander and house dust RAST in patients with atopic dermatitis, as opposed to bronchial asthma or other allergic disease.

MATERIALS AND METHODS

Patients and sera. A group of well-registered patients of the Dermatology Ward, with widespread AD of lung duration, was recalled for investigation. Patients with bronchial asthma, hay fever or vasomotor rhinitis were selected from subjects attending the Out-patient Department of Clinical Allergy. Venous blood samples were allowed to clot in glass and the sera were stored at −70°C until use.

Allergen and IgE-determination. House dust allergen was extracted from a pool of dust and purified to the stage of fraction E, as described elsewhere (1). Human dander allergen was obtained from acetone-washed human scalp scales and purified to the fraction E stage by a previously published schedule (1). The purified allergens were coupled to cellulose discs with cyanogen bromide *ad modum* Ceska, Eriksson & Varga (6). Radiolabelled anti-(Dε2)IgE antibody was purchased from Pharmacia Diagnostics, Uppsala. Total IgE was determined by RIST (Pharmacia reagents).

RESULTS

In previous communications (1, 3), we have reaffirmed the well-documented observation of a close correlation in the incidence of positive immediate-type skin reactions to house dust and human dander in atopic patients in general (7, 8, 9, 13, 14). Despite this association of reactivities in unselected groups of atopic subjects, no such correlation was found between house dust and human dander RASTs in the blood sera (3). The cause for this becomes evident on reviewing the results summarized in Figs. 1 and 2.

Fig. 1 shows that the human dander RAST was not found positive in patients with asthma or rhinitis uncomplicated by eczema; in such patients, only the house dust RAST was positive. Positive human dander RASTs were obtained only in cases of (severe) AD, as shown in Fig. 2. The data in Fig. 2 also demonstrate a close correlation in AD between the RASTs for house dust and human dander allergen.

In AD in adult patients, the probability of a positive human dander RAST increased with total IgE levels. As depicted in Fig. 3, there was a good correlation in this respect between RAST and RIST values, as has been reported before for RIST and house dust RAST (4).

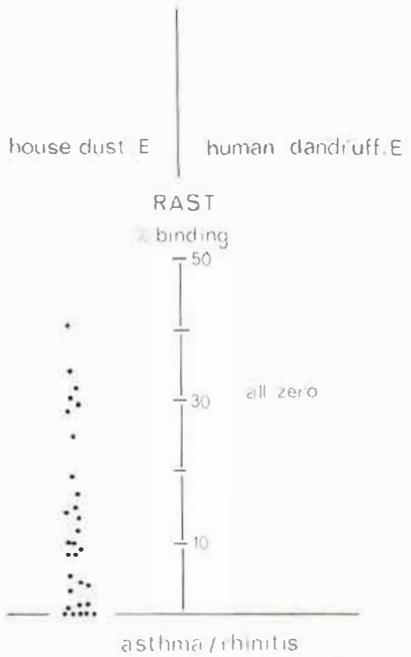

Fig. 1. RAST with purified house dust and human dander allergen in a group ($n=30$) of atopic patients with bronchial asthma or vasomotor rhinitis and with positive skin reactions to both allergens. RAST results expressed in % radioactivity bound to the discs from a standard incubation dose.

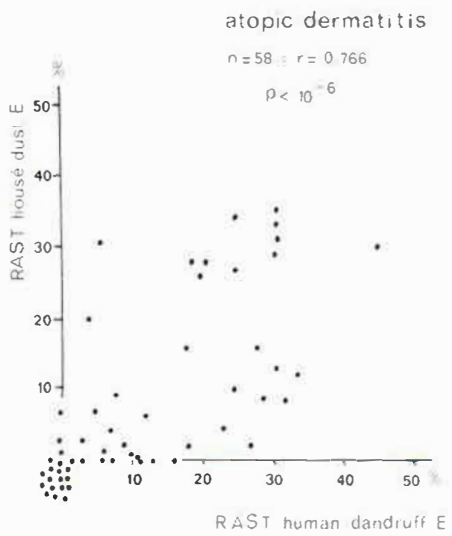

Fig. 2. Correlation of house dust and human dander RAST in the sera of patients ($n=58$) with atopic dermatitis. Spearman rank correlation coefficient $r=0.766$ ($P<10^{-6}$).

In the adult dermatitis group, very high RIST levels tended to be the rule ($n=66$, geometric mean IgE = 1 323 IU/ml), in association with positive human dander RASTs. In children with AD, below the age of 10, RIST values were lower ($n=12$, geom. mean IgE = 655 IU/ml) and the human dander RAST was frequently negative.

DISCUSSION

Immediate-type skin reactions to house dust and human dander allergen frequently run parallel (1, 3, 7, 8, 9, 13, 14). This is also true for the *in vitro* complement consumption with both allergens in various blood sera (3). In an unselected group of atopics, no parallelism was found in specific IgE antibody to both allergens (3). The present paper documents that this discrepancy is due to the fact that the RAST human dander is positive only in cases of AD, but not in bronchial asthma, hay fever or rhinitis. Figs. 1 and 2 clearly demonstrate that cross-contamination of the purified allergens had not occurred.

The data demonstrate a dissociation in the pattern of reactivity of the house dust and human dander allergens. The results of Young (13), Young & Bangma (14) and of French authors (7, 9) are relevant in this context. Despite parallel skin reactions to both allergens in asthma or atopic eczema,

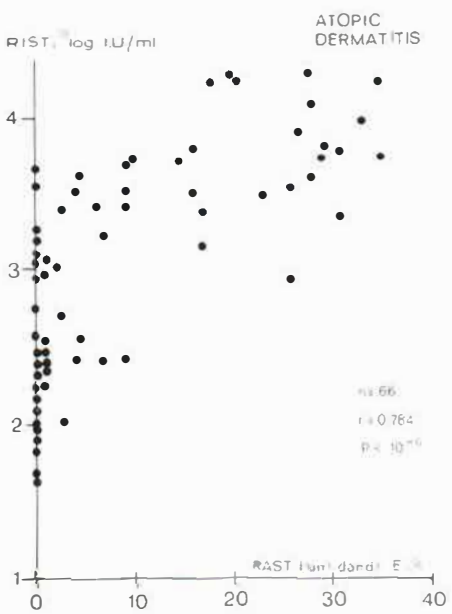

Fig. 3. Relationship between total IgE levels (RIST, in IU/ml) and human dander RAST scores in 66 patients with atopic dermatitis. Spearman rank correlation coefficient $r=0.784$ ($P<10^{-6}$).

inhalation-provocation tests with the dander allergen tended to be positive only in the eczema group, but were negative in the bronchial asthma patients. Current studies aim at further defining the precise role of the IgE antibody in these phenomena.

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DISCUSSION

Saurat (Paris). Q: Have you made electron microscopical studies of your starting material in preparing human dandruff allergens? Among others, you can have microbial products and yeasts in this material, so that the composition of such an antigen would be very broad.

A: I think the point is well taken, but we have no electron microscopic, not even microscopic studies of this. We take the pragmatic view that we use extracts as they are being used in practice by clinical allergists. I doubt, however, that this reactivity could be due to contamination to either moulds or bacteria, because we have been investigating a whole range of yeast extracts, etc., and none of these antigens has the extreme reactivity that the human dandruff antigen has. I cannot exclude the point, but I think it is very unlikely that it is contaminated by another antigen.

Zachariae (Aarhus). Q: What about mites?

A: We did not look for mites either, but we have investigated a whole series of commercial mite allergens and some we prepared ourselves, and none of these has the reactivity that the human dandruff antigen has.

Hanifin (Portland). Q: We discussed the possibility that staphylococcus may play a role in the etiology of atopic dermatitis. Have you done any qualitative studies with staphylococcal reactivity? I am also curious as to whether these patients react to human saliva.

A: I do not know of any studies where human saliva has been tested intracutaneously or otherwise in vivo. We have tested the reactivity of human saliva in vitro, but it is far from reaching the reactivity that one can solve with human dandruff. Human saliva does not react at all with complement. We have also investigated various extracts of *Staphylococcus aureus* and this does not run parallel with human dandruff extract. I think the reactivity of the human dandruff allergies has to do with decomposition reactions of the skin components themselves. Simon has proved that reactivity is really to be found in the scalp scales. We have recently investigated a preparation that was made of skin flakes not obtained from the scalp but this did not have any reactivity at all. I think that there is no evidence that this reactivity had to do with bacterial extract, with saliva, with yeast or moulds, but has to do with the decomposition products which arise in the human corneum itself.