Specific Hyposensitization for Atopic Dermatitis: A New Era?

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To control the course of atopic dermatitis (AD) by a 'specific' approach is a natural wish of many patients and has been therefore a 'long-standing ambition of clinicians' [1]. Specific hyposensitization has been for long regarded as a potential tool but its results have convinced very few dermatologists; this is why specific hyposensitization is rarely advocated for the treatment of AD [2]. The WHO/ International Union of Immunological Societies working group report [3] even states that '... allergic immunotherapy ... is not indicated ... in dermatological diseases'.

For specific hyposensitization being acknowledged as an effective mode of therapy in some patients with AD, at least three prerequisites should be fulfilled.

(i) Inhalant (airborne) allergens (with which hyposensitization is usually performed in atopic patients with respiratory allergies) should be considered as significant factors in AD. There has been a long-standing debate on this but many now agree that, in the light of recent observations, airborne allergens such as house dust and pollens might play a role in perpetuating and/or exacerbating AD [reviewed in 4].

(ii) Accurate diagnostic methods should be available to objectively identify the particular airborne allergen implicated in individual patients. So far this is a difficult step. By combining careful case history, prick tests, patch tests with inhalant allergens [5] and radioallergosorbent tests (RAST), one can seldom reach more than a 'presumptive impression' of responsibility of a single allergen.

(iii) An effective mode of 'hyposensitization' should be available, which would induce significant improvement within not too long a period of time. So far, specific hyposensitization therapy, as used for respiratory allergies, does not, in most cases of AD, provide that type of result.

In this issue of *Dermatologica*, Leroy et al. [6] report on a novel approach of specific hyposensitization that induced an impressive improvement within 3–4 months in 10 patients with AD. The method used by Leroy et al. consists in injecting to the patient not the allergen alone but a mixture of the allergen plus the patient's own antibodies to this allergen, i.e. allergen-antibody complexes. Why was this approach chosen? Because allergen-antibody complexes appear as a valuable tool to suppress IgE antibody production; the same group, led by Jean-Marie R. Saint-Rémy, from the Université Catholique de Louvain, has recently shown that injection of such complexes in asthmatic patients resulted in a significant improvement of the symptoms, a decrease in specific antibodies to the allergen (Dermatophagoid.es pteronyssinus) [7] and an increase in the serum concentration of anti-idiotypic antibodies. (Anti-idiotypic antibodies are antibodies that react with the variable portion of immunoglobulins which is specific for a given antigen.) The rationale behind this procedure is that anti-idiotypic antibodies reduce the

production of the antiallergen antibodies (that are thought to induce allergic symptoms) via the process called by immuno-nologists 'idiotypic suppression'.

Whichever the mechanism of clinical improvement in AD is (Leroy et al. [6] not only discuss idiotypic suppression but also modulation of T cell function and functional inactivation of B cells through binding of antigen-antibody complexes), the therapeutic value appears such that anyone dealing with AD patients would think: 'When could I try this method?' The study presented by Leroy et al. is an open one, and although convincing, we should await the results of ongoing double-blind trials. There are also two practical problems to be solved before the technique is used in practice.

One is the patient selection: In their study Leroy et al. [6] have selected patients with positive prick tests and RAST to D. pteronyssinus and reasoned that this allergen is the principal airborne allergen in areas of temperate climate. This allergen was then chosen for preparing the allergen-antibody complexes. They clearly indicate that they might have, by chance, selected a small subgroup of patients with AD that responded to this novel therapy with this particular allergen. Reliable criteria for identi-

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fying both patients that could respond to injections of allergen-antibody complexes and the relevant allergen to be included in the complex remain to be established. AD patients have usually both positive prick tests and RAST to several airborne allergens.

The other practical problem is the preparation of the allergen-antibody complexes. Once the 'relevant' allergen is identified, one should prepare the complexes with the patient's own antibodies. These antibodies have been prepared in the study of Leroy et al. from each patient's serum by salt precipitation, gel filtration chromatography and specific immunoadsorption on a Sepharose column onto which the allergen had been insolubilized. Such a procedure implies that adequate laboratory facilities are available; these are not beyond the possibilities, even for private laboratories, because if the therapeutic value of the procedure is confirmed, commercially available kits will probably be developed.

Obviously, the technique proposed by Leroy et al. [6] would be difficult to use, and many efforts should be made before it may reach our practice. This field has been so disappointing in the past that even those who never prescribed 'specific hyposensitization' in AD (like the author of this Editorial) should consider that there might now be a 'hole in the wall'. The future will tell us if it was overenthusiastic to think in 1991 that a new era in the management of AD is opening.

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