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The Atopic March

Sir,

With great interest, we read the paper of S.J. Khan et al who conducted a systematic review of twin and siblings studies to examine the allergic phenotypes and early-life environment factors that form the so-called atopic march.¹ The authors concluded that genetics play a bigger role in predisposing eczema to hay fever and eczema to asthma than environmental factors and that the link between eczema and asthma and hay fever is independent of shared early-life environmental factors. In fact, two main (major) phenotypes of atopic dermatitis (AD) can easily be distinguished, already in little children, by mean of a prick skin testing to the main food and inhalant allergens or by a serum IgE determination: an IgE-associated type and a non-IgE-associated type (also called the "intrinsic type") of AD.²⁻⁷ The latter type (former called "intrinsic" type of AD) is characterized by eczematous skin lesions in typical localization similar to the IgE-associated subgroup, but is not associated with a specific sensitization either in the skin tests or in RAST/CAP to common environmental allergens.^{3,4} Two long-term observations in a group of young children with AD seen at the age of 2-4 years, who were re-evaluated about 9 years later, showed that 46% respectively 68% of the children still suffered from AD, 43% resp. 45% from bronchial asthma, and 41% from allergic rhinitis.^{8,9} (The first percentage refer to the study of Novembre et al⁸ the second to our study⁹). However, the risk of developing asthma or allergic rhinitis, it means of an atopic march, was much higher in IgE-associated AD (51% resp. 60%) than in nonallergic AD (0% resp. 14%). For the long-term prognosis of AD and the risk of an atopic march, it is therefore important to know that the risk of the development of an IgE-mediated respiratory disease is much lower in the nonallergic AD.

CONFLICTS OF INTEREST

None to declare from both authors.

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