

Influence of mite exposure on symptoms of mite-sensitive patients with asthma

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We selected 22 patients who reported exacerbations of asthma symptoms or rhinitis in only one particular dwelling and were free of symptoms in another. These aggravations were observed several times, suggesting house dust-induced asthma. The 22 patients (12 women and 10 men; mean age, 25.4 ± 9.7 years) were monosensitized to *Dermatophagoides pteronyssinus*; they had a positive intradermal test result to *D. pteronyssinus* after injection of 0.04 ml of a 10^{-5} mg/ml *D. pteronyssinus* extract (Institut Pasteur, Paris, France) and a Phadebas Radio Allergo Sorbent Test (RAST, Pharmacia AB, Uppsala, Sweden) for *D. pteronyssinus* score of equal to or greater than 3. For each patient, two house dust samples were obtained. The first one was from the dwelling in which they were free of symptoms, and the second one was from the dwelling in which they had symptoms of asthma or rhinitis. The 44 house dust samples were collected by the patients, who vacuumed their mattresses for 2 minutes per square meter. Dust bags were stored at 4° C in sealed plastic bags. Semiquantitative guanine determination was performed with the Acarex-test (Allergopharma, Hamburg, Germany). According to the manufacturer, results are semiquantitatively expressed in four classes, for which the guanine content is indicated as 0, +, ++, or ++++. A Wilcoxon signed-rank test was performed to determine whether there was a significant difference between the Acarex-test results from the dwellings in which patients were free

of symptoms and those in which patients had symptoms.

RESULTS

Results are summarized in Fig. 1. There is a significant difference between the Acarex-test classes determined for house dust samples from the dwellings in which patients were free of symptoms and the dwellings in which they experienced symptoms ($p < 0.001$). In the houses in which the patients had symptoms, Acarex-test classes were equal to or greater than ++ in 19 of 22 patients. In contrast, in dwellings where patients had no symptoms, Acarex test classes were equal to or greater than ++ in 5 of 22 patients. The highest Acarex-test values (i.e., class +++) were found in 9 of the 22 homes in which patients had symptoms and never in homes in which the same patients were free of symptoms.

DISCUSSION

In this study we selected *D. pteronyssinus*-sensitized patients for which the diagnosis of house dust mite allergy was mainly based on a worsening of symptoms in a given home in which they stayed only occasionally and on symptom improvement when the patients were away from these dwellings (i.e., the homes in which patients were free of symptoms were usually the major dwellings). Such clear evidence for the provocative role of the indoor environment in triggering asthma symptoms is not a common feature of house dust mite respiratory allergy. Mite exposure levels can be assessed by different methods: mite counts, assays of mite allergens, and guanine measurements. Using a commercial kit (i.e., Acarex-test) we were able to show¹ that an Acarex-test class 0 indicates *Der p 1* + *Der f 1* values from 0.125 to 11.5 µg/gm (mean, 1.7 µg/gm; SEM, 3.03 µg/gm); class + from 0.03 to 25.5 µg/gm (mean, 5.5 µg/gm; SEM: 0.5 µg/gm); class ++ from 3.9 to 102.3 µg/gm (mean, 35.2 µg/gm; SEM: 2.68 µg/gm) and class +++ from 24.1 to 481 µg/gm (mean, 131.3 µg/gm; SEM: 20.4 µg/gm). Moreover, we deter-

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Supported by a grant from the Ministère de l'Équipement, du Logement et de l'Aménagement du Territoire, n° 88-34-03 du 25 juillet 1988.

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J ALLERGY CLIN IMMUNOL 1994;93:136-8

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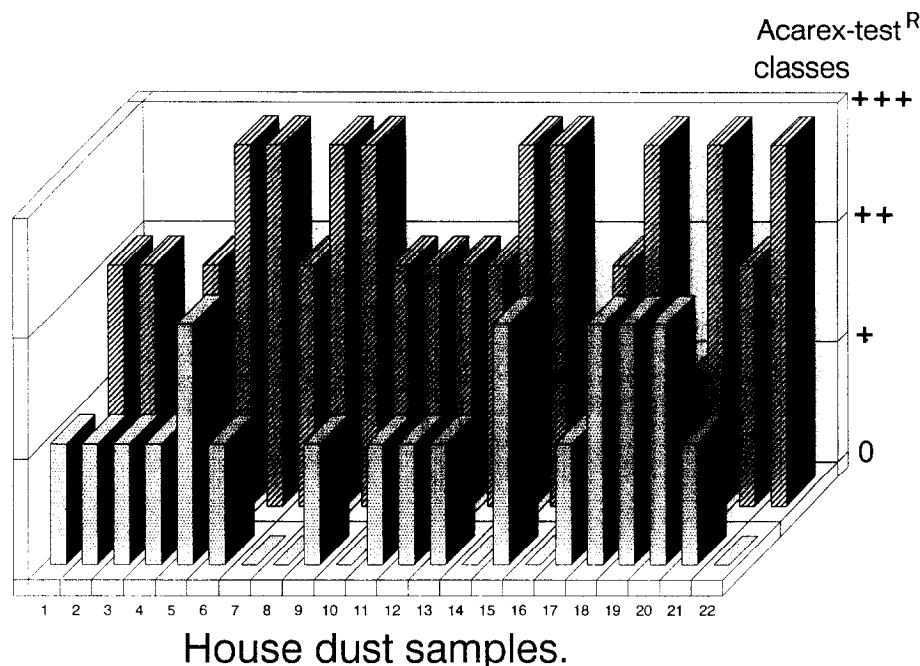


FIG. 1. Acarex-test classes in 44 house dust samples obtained from homes where 22 patients with *D. pteronyssinus* allergy had symptoms (striped bars) and from homes where these patients were free of symptoms (dotted bars).

mined the predictive value of Acarex-test classes: class 0 indicates an exposure level of 2 $\mu\text{g/gm}$ or less, class ++ a level of greater than 2 $\mu\text{g/gm}$ in all cases, and class +++ a level of greater than 10 $\mu\text{g/gm}$ in more than 80%. Class + does not have sufficient accuracy for predicting an exposure level. The results obtained in dwellings in which patients were free of symptoms are representative of varying sensitivities to *D. pteronyssinus* allergen levels in different patients observed under realistic conditions, because 11 patients tolerated an allergic load of house dust samples with Acarex class of equal to or greater than +, and five patients tolerated an Acarex class ++. The same intersubject variability has been shown in early asthmatic responses to *D. pteronyssinus* allergen bronchial challenge tests (i.e., 100-fold for provocative concentration causing a 20% decrease in forced expiratory volume in 1 second).² It also clearly appeared that in this group of patients the *Der p* I + *Der f* I levels associated with the onset of symptoms are almost always equal to or greater than 10 $\mu\text{g/gm}$ (i.e., Acarex classes ++ and +++).

Our study focused on the appearance of symptoms and not on the development of sensitization to mites. It differed from that by Sporik,³ who demonstrated that exposure to more than 10 μg

of mite allergens per gram of dust in early childhood was an important determinant of asthma development by the age of 11. This study is more closely related to those dealing with effects of environmental mite exposure on symptoms and medication requirements in mite-sensitive patients. Platts-Mills et al.⁴ studied houses of patients with mite allergy who had exacerbation of their asthma that required additional treatment; in 16 of 18 visits performed within 2 weeks of an exacerbation, in order to collect dust samples, they found an antigen P1 equivalent content of greater than 10 $\mu\text{g/gm}$. Vervloet et al.⁵ showed that among a group of 49 patients, those who required daily long-term treatment had a mean antigen P1 equivalent level of 10.8 $\mu\text{g/gm}$ dust. In our study the same patients were exposed to two different levels of mite allergens, each patient being his own control. Monosensitization minimized the possible role of other indoor allergens, whereas clear-cut reproducibility of the triggering of symptoms with changes in dwellings reduced the possible role of nonspecific triggers.

In conclusion, this study shows the importance of environmental mite allergen loads in the appearance of symptoms in a group of patients with mite allergy who complained of symptoms only in a given dwelling. It also provides evidence that

patients with positive *in vivo* and *in vitro* test results to *D. pteronyssinus* may not exhibit symptoms if they do not have sufficient exposure in their present environment. We have provided clear evidence of the provocative role of the indoor environment in triggering asthma symptoms. However, this clinical behavior is not always found, and one could suppose the development of asthma is also influenced by other factors such as nonspecific bronchial hyperreactivity.

We thank Miss F. Eckert for her excellent assistance and Miss B. Oesterlé for typing the manuscript.

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