

Natural exposure and serum antibodies to house dust mite of mite-allergic children with asthma in Atlanta

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*Pyroglyphid mites in house dust are important allergens associated with asthma in Europe, but comparable studies of house dust mites in the homes of patients with asthma have not been done in the United States. We examined the distribution of mites and mite allergen in the houses of 20 mite-sensitive children with asthma in Atlanta and measured IgE antibodies to mite allergens in their sera. One or more dust samples from bedding, bedroom floor, television room floor, or television room furniture from 17/20 houses contained >10,000 ng of antigen P₁ equivalent per gram of fine dust; amounts ranged from 280 to 230,400 ng/gm. Allergen levels were higher in dust samples from furniture and bedding than from floors. Dust samples obtained from houses in June to September had more mites and mite allergen than those houses sampled in March to April; relative humidity in the room also was higher in June to September. Mite numbers and allergen in floor and furniture samples were correlated with relative humidity in the room and were high when relative humidity was >50%; antigen P₁ equivalent was >10,000 ng/gm in 21/39 such samples. *Dermatophagoides pteronyssinus* was present in all houses and dominant in 11/20. *D. farinae* was found in 17 houses and was dominant in six. All children studied had high IgE antibody with either *D. farinae* or *D. pteronyssinus* RAST: 16 of the 20 children also had IgE to antigen P₁. It is likely that the IgE antibody responses in these 20 children with asthma were a direct result of exposure to high levels of mite allergen. We speculate that the mite sensitivity of these children might play a causative role in their asthma. These results also suggest that extracts of both *D. farinae* and *D. pteronyssinus* should be used for skin testing and immunotherapy in the United States. (J ALLERGY CLIN IMMUNOL 76: 782-8, 1985.)*

In Europe and many other parts of the world, pyroglyphid mites in house dust have been demonstrated to be important allergens associated with asthma.¹⁻⁶ Examination of samples of dust from houses in Europe has demonstrated large numbers of mites in bedding, rugs, and furniture, and mite numbers have been demonstrated to be highly correlated with *D. pteronyssinus* allergen content as measured by RIA.⁷⁻¹⁰ *D. pteronyssinus* probably is the most widely distributed of the species of house dust mites and certainly has been the most thoroughly studied species.¹¹⁻¹⁵ In the past it

Abbreviations used

RIA: Radioimmunoassay
NIBSC: National Institute of Biological Standards and Control

has been suggested that *D. farinae* is the most abundant species in the United States; only *D. farinae* extract is marketed in the United States for allergen skin testing and immunotherapy.¹⁶ However, there have been several reports of high levels of *D. pteronyssinus* in houses in the United States,^{10, 16-20} and our preliminary sampling in the Southeast and on the West Coast found both *Dermatophagoides* species present in allergic patients' houses.²¹ These two mites cross-react as judged by skin testing and RAST assays.^{11, 22-23} Furthermore, the major allergens produced by these two mites have been demonstrated to have both species specific and cross-reacting determinants.^{24, 25} Recently, we have demonstrated that al-

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TABLE I. Mite numbers and allergen in dust samples

Sample site	Geometric mean (Range)		
	Dust (mg)	Antigen P ₁ equivalent (ng/gm of dust)	Mite bodies* (per gm of dust)
Bedding	614 (78 to 4659)	11,385 (980 to 155,200)	100 (9 to 650)
Bedroom floor	250 (23 to 967)	5786 (280 to 84,240)	195 (9 to 4920)
Television room floor	(501) (151 to 1586)	7982 (340 to 230,000)	165 (9 to 3080)
Television room furniture	830 (95 to 3171)	11,327 (740 to 48,760)	155 (10 to 3270)

*Bodies are 90% dead.

lergen derived from either *D. pteronyssinus* or *D. farinae* can be measured in house dust by use of an RIA specific for the cross-reacting determinants on antigen P₁.²⁴

It was our experience that positive immediate skin tests to *D. farinae* extracts were common in children with asthma in Atlanta, Ga. The present study of 20 mite-sensitive children with asthma documents both the quantities of mites and mite allergen in dust samples from their houses and their serum mite-specific IgE antibodies. The results demonstrated that these children are exposed to high levels of mite antigen in their houses and have high levels of serum IgE antibodies presumably in response to this exposure. These levels of both allergen and serum antibodies are at least as high as those reported previously from London, England.^{8, 22, 25-27} Furthermore, in keeping with previous articles on mite growth,²⁸⁻³¹ our results demonstrate a striking correlation between mite allergen in carpets and furniture and relative humidity. High levels of allergen occur when the relative humidity in the room is >50%.

SUBJECTS AND METHODS

Patients

Twenty children (nine boys) who fulfilled the American Thoracic Society criteria for reversible obstructive airway disease (bronchial asthma)³² were selected from patients observed by the Allergy/Immunology and Pulmonary Disease Service at the Emory Clinic and Grady Memorial Hospital, Atlanta, Ga., on the basis of a history of positive skin test (wheal size >5 mm in diameter by puncture technique) to *D. farinae* extract 1:50 w/v (Hollister-Stier Laboratories, Spokane, Wash.) containing 27 µg/ml of antigen P₁ equivalent. Informed consent was obtained under a protocol approved by the Human Investigations Committee. Results of skin testing with other allergen extracts, including house dust (Hollister-Stier, 0.87 µg/ml antigen P₁ equivalent), were recorded.

Measurements of relative humidity

Relative humidity in rooms was calculated from wet and dry bulb temperature readings obtained at the time the dust sample was obtained by use of a psychrometer (Bendix model No. 566, Lewisburg, W. Va.). Outside relative humidity was obtained from local climatological data monthly summaries for Atlanta published by the National Oceanic and Atmospheric Administration, National Climatic Data Center, Asheville, N. C.

Sampling extraction and mite count on dust

Samples of dust were obtained and handled as described previously.⁸ Ten houses were visited in June to September 1983, and ten houses were visited in March to April 1984. Briefly, samples were collected onto a double layer of domestic tissue by use of a hand-held vacuum cleaner (Douglas Hand Vac Model L6701, Douglas Products, Walnut Ridge, Ark.) fitted with a filter holder (MS 681 SL, Camlab Ltd., Cambridge, U. K.). Tissue and dust samples were stored in sealed plastic bags at 4° C before extraction and mite counting. Collection of bedding samples by vacuuming pillows, bedclothes, and mattresses usually required 4 to 5 minutes. Floor dust from the bedroom and from the room where the television set was kept (usually living room or den) was collected from rugs, if they were present, or floor surface by vacuuming an area of approximately 1 m² for 2 minutes. Furniture in the television room also was sampled by vacuuming cushions, arms, and back for approximately 2 minutes. Samples were sieved through a 0.3 mm mesh screen to obtain fine dust. Mites were recovered from a 100 mg portion and identified under high-power microscopy. A separate 100 mg sample was extracted with 2 ml of borate-buffered saline, pH 8.0, for subsequent assay of antigen P₁ equivalent content.

Assay for antigen P₁ equivalent in dust extracts

Antigen P₁ equivalent was measured by a double antibody inhibition RIA with antibodies specifically directed against determinants common to the major allergen of *D. pteronyssinus* and *D. farinae* and ¹²⁵I-labeled pure antigen P₁.²³

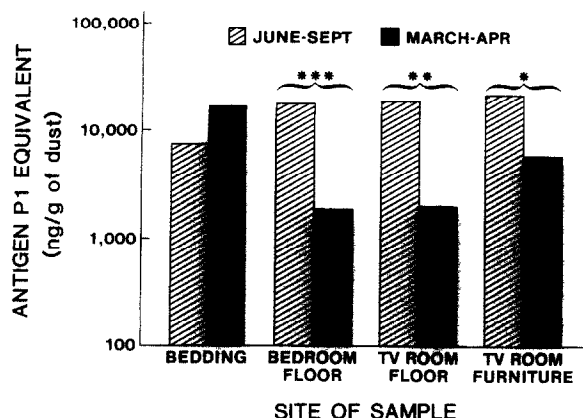


FIG. 1. Comparison of quantity of antigen P_1 equivalent in dust samples obtained during June to September with those obtained during March to April. Geometric mean values for sample sites are depicted. Quantities of antigen P_1 equivalent are displayed in nanograms per gram of fine dust. Statistically significant differences in quantities obtained in the two seasons are indicated. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

This assay can detect 0.3 ng/ml of antigen P_1 equivalent with an intra-assay variation of $\pm 6.4\%$. A control curve with use of a standard preparation of antigen P_1 was carried out in parallel with each assay; this standard, which contains 750 ng of antigen P_1 equivalent per milliliter, has been substandardized from the World Health Organization International Biologic Reference preparation obtained from the NIBSC, London, England (82/518).¹²

Assay for IgE and allergen-specific antibodies in serum samples

Total serum IgE was measured by inhibition RIA with rabbit anti-IgE (Calbiochem, Behring Corp., La Jolla, Calif.) and radiolabeled purified IgE myeloma (N.D.) protein kindly provided by Dr. S. G. O. Johansson.¹³ Serum IgE antimitic antibodies were measured by RAST as described previously.²² The assays were standardized relative to a reference pool of mite-allergic sera held at NIBSC (82/528).¹² *D. farinae* and *D. pteronyssinus* extracts were linked to CNBr-activated cellulose discs. Serum IgG and IgE antibodies to antigen P_1 were measured by use of radioimmuno-precipitation as described previously.¹³

RESULTS

The 20 children with asthma reported here ranged in ages from 2.2 to 15.3 years (mean = 9.8 years). Epicutaneous skin testing with other allergen extracts demonstrated the following positive tests (wheal >5 mm in diameter) in addition to *D. farinae*: one or more trees, positive in seven patients; one or more grasses, positive in 11 patients; ragweed, positive in three patients; other weeds, positive in one patient; molds, positive in seven patients; and feathers, positive in three patients. Eleven children were skin test positive to house dust; in none was the skin reaction

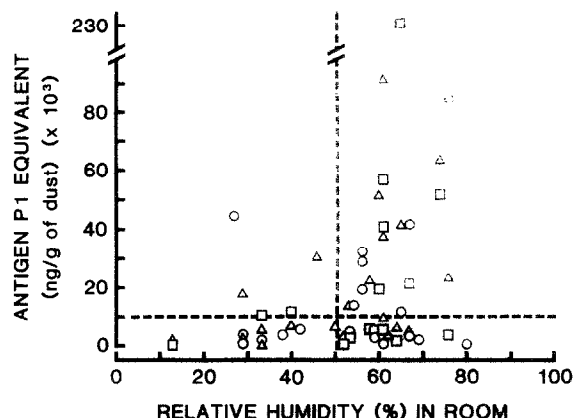


FIG. 2. Relationship between quantity of antigen P_1 equivalent in dust samples and relative humidity in the room when the sample was collected. Bedroom floor ($\circ$); television room floor ($\square$); television room furniture ($\triangle$).

to dust larger than that to *D. farinae*. The only positive skin test in four children was to *D. farinae* ($\pm$ dust).

Samples from bedding, floors, and furniture in the homes of these children with asthma were found to contain concentrations of antigen P_1 equivalent that ranged from 280 to 230,400 ng/gm of fine dust (Table I). We found >10,000 ng of antigen P_1 equivalent per gram in one or more samples from 17 of 20 houses. This level of allergen was present in 12/20 samples from bedding; 8/20 samples from bedroom floors; 8/16 samples from television room floors; and 10/20 samples from television room furniture. Dust samples from television room furniture contained the most antigen P_1 equivalent; samples from floors of bedroom and television rooms contained the least quantity of antigen P_1 equivalent. Samples from floors contained less antigen P_1 equivalent per mite than did samples from bedding or furniture. The number of mite bodies found was significantly correlated with antigen P_1 equivalent content of the dust ($r = 0.82$, $p < 0.001$). Neither the number of mites nor the total quantity of antigen P_1 equivalent was related with amount of dust in the sample.

Dust samples obtained from houses in June to September had more mites and mite allergen than did those sampled in March to April. As depicted in Fig. 1 for antigen P_1 equivalent, this decline in allergen level in March to April was observed in samples from bedroom floor, television room floor, and television room furniture but not in samples from bedding. On the contrary, allergen in bedding was not different in March to April when allergen level was compared with the level in June to September. Similar differences were observed in number of mite bodies in samples collected in June to September compared to those collected in March to April.

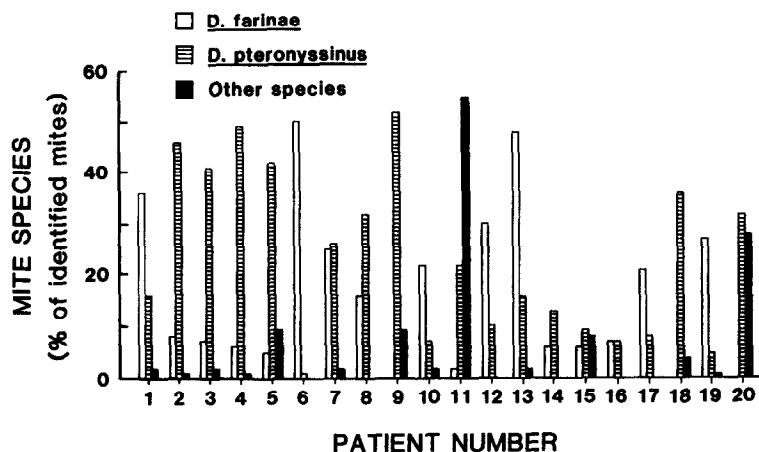


FIG. 3. Mite species in the houses of the 20 patients; species is depicted as percent of identified mites in all dust samples obtained from the house.

TABLE II. IgE and serum antibody measurements*

	Geometric mean	Range
Total IgE (U/ml)	577	96 to 6,142
<i>D. farinae</i> RAST (U/ml)	446	16 to 4,525
<i>D. pteronyssinus</i> RAST (U/ml)	563	35 to 10,400
IgE against antigen† P ₁ equivalent (U/ml)	61	8 to 1,247
IgG against antigen† P ₁ equivalent (U/ml)	608	16 to 12,500

*Values for RAST and antigen binding assays were obtained by reference to a standard antimitic human serum pool.¹² Values per ampule of the reference pool (NIBSC 82/528) were *D. farinae* RAST, 1530 U; *D. pteronyssinus* RAST, 2130 U; IgG anti-P₁, 394 U; and IgE anti-P₁, 136 U.

†Negative results assigned value = 1 less than lower limit of assay.

Relative humidity was much higher inside homes sampled during June to September ($65 \pm 6\%$) than in those homes sampled during March to April ($42 \pm 12\%$). Relative humidity recorded outside on the days that homes were sampled in June to September was high ($73 \pm 10\%$) but was not statistically different from that measured in March to April ($62 \pm 15\%$). Mite numbers and mite allergen levels in both floor samples and in television room furniture were correlated with relative humidity measured in the room at the time the samples were obtained but not to relative humidity outside. Last, it appeared that both mite numbers and mite allergen increased when the relative humidity in the room was $>50\%$ (Fig. 2).

D. pteronyssinus was the most common pyroglyphid species identified. It was present in all houses

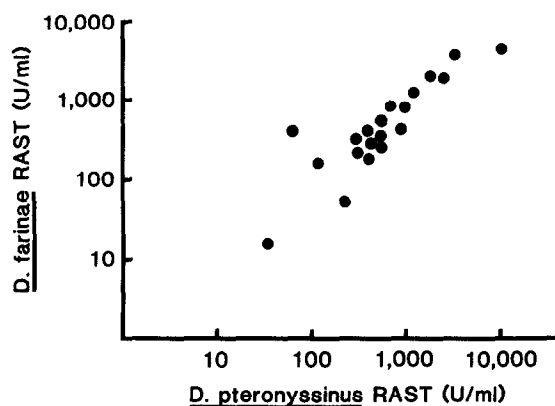


FIG. 4. Relationship between results of RAST by use of *D. pteronyssinus* or *D. farinae* ($r = 0.88$, $p < 0.001$).

and was the dominant species in 11 of 20 houses (Fig. 3). *D. farinae* was identified in 17 houses and was the dominant species in six. *Euroglyphus maynei* was dominant in one house that also contained both *D. pteronyssinus* and *D. farinae*. Low numbers of other species were observed in 13 homes; these species included *Cheyletus* sp and *Tarsonemus* sp, hypopal forms, and mites of the suborder gamasina.

Patients had high levels of serum antibody by RAST with either *D. farinae* or *D. pteronyssinus* on the disc (Table II). Furthermore, there was a statistically significant correlation between results of *D. farinae* and *D. pteronyssinus* RAST ($r = 0.88$, $p < 0.001$) as depicted in Fig. 4. Most of the children (16/20) also had high levels of IgE antibody to antigen P₁ measured by radioimmunoprecipitation. As reported previously, IgE antibodies to antigen P₁ were, in each case, associated with IgG antibodies measured by a parallel technique.^{13, 22} However, four children had no detectable IgE or IgG antibody to antigen P₁ in spite of positive RAST results (Fig. 5).

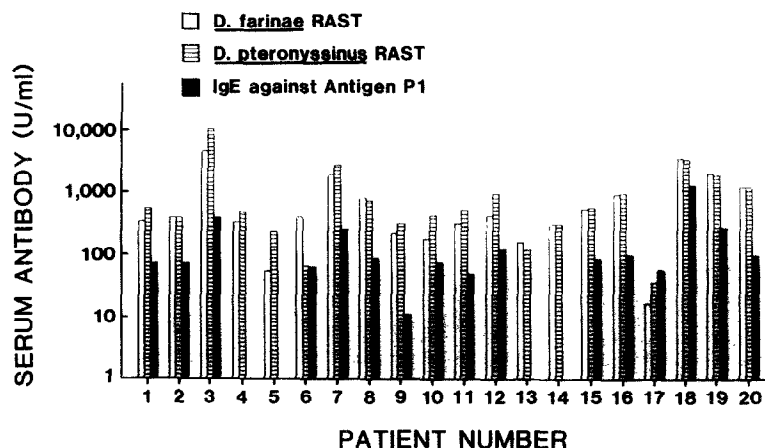


FIG. 5. Antimite antibodies in sera from the 20 patients. Results by use of *D. farinae* RAST and *D. pteronyssinus* RAST are compared with measurements of IgE against antigen P₁.

DISCUSSION

Our measurements of antigen P₁ equivalent in dust samples demonstrated that large amounts of mite allergen are present in houses in Atlanta. These levels are as high or higher than those levels reported previously from England^{8, 33} or from other areas of the United States.²¹ As has been demonstrated previously, total allergen content of dust samples was related to mite numbers rather than to the quantity of dust. We also found large variations in mite numbers, allergen content, and allergen per mite according to the site of sample. Amount of antigen P₁ equivalent per recovered mite body was increased in furniture and bedding when the amount was compared to that recovered from floors. Even in bedroom floor samples, only 10% of antigen P₁ equivalent content can be explained by the mite bodies themselves. Thus, these differences may reflect accumulation of mite debris, retention of mite bodies in fabric, or different growth characteristics for mites in the different sites. Our measurements of mite and mite allergen in furniture and carpets in television rooms leave little doubt that these rooms also contribute to allergen exposure. Therefore, mite avoidance measures should be directed to other rooms besides the bedroom. It should be noted that, although it has proved difficult to reduce mite levels in houses,^{34, 35} several articles have demonstrated improvement in asthmatic symptoms either by cleaning bedrooms^{36, 37} or by moving patients to "allergen-free" conditions.³⁸⁻⁴⁰ It may be that under very humid conditions, changes in the bedroom alone will not be adequate to improve clinical symptoms.

Houses in Atlanta sampled in June to September demonstrated higher levels in carpets and furniture than in those sampled in March to April; this difference was not observed in samples from bedding. This

suggests that mite growth in carpets and furniture may be more sensitive to seasonal changes in relative humidity than mite growth in bedding. Mite and mite allergen content in samples from these sites were related to relative humidity in the room, and both appeared to increase appreciably when the relative humidity was >50% (Fig. 2). Our results are consistent with previous results demonstrating that mite numbers in houses^{28, 30} and mite growth in culture³¹ are markedly reduced by decreased relative humidity, i.e., <50%.

The significant quantities of mite and mite allergen in samples from Atlanta may be related to the humidity in Atlanta, since the monthly mean relative humidity is always >50%. We presume that the decrease in humidity and therefore mite numbers in March to April when compared to June to September was the result of heating homes during the winter. Thus, we would predict that the use of humidifiers in homes in the winter might increase mite numbers and contribute to symptoms of mite-sensitive patients. It may be that a decrease in mite number in bedding during the winter is prevented by supplemental humidity provided by the occupant.

At the present time there is no exact way of deciding whether or not a particular number of mites or allergen level is relevant to asthma. However, there have been several articles that relate a particular number of mites per gram of dust to symptoms of asthma. Korsgaard,³⁹ in Denmark, found that the levels in the houses of allergic patients with asthma were >300/gm; in London, levels around 200/gm were regarded as significant.^{8, 30} For allergen levels, it has been proposed that >10,000 ng of antigen P₁ (or antigen P₁ equivalent) per gram of dust should be regarded as a high level.^{8, 24} Certainly, allergic patients with asthma in London who moved from houses with >10,000 ng of

antigen P₁ per gram of dust into an allergen-free room, mean level 230 ng of antigen P₁ per gram, demonstrated highly significant improvement.^{38, 40} With the use of this criterion of >200 mites per gram or >10,000 ng of antigen P₁ equivalent per gram, the levels found in Atlanta would be regarded as high or very high. Similarly, when the levels of IgE antibody are compared with levels found previously in allergic patients in London or Hong Kong by use of the same assays, the levels found in these children in Atlanta are as high or higher.^{22, 26, 27} Comparison of results between Europe and the United States is possible because the assays are standardized relative to lyophilized reference preparations. For allergen assays, the standard is the new World Health Organization International Standard for *D. pteronyssinus* (NIBSC 82/518).¹² The results listed here for antigen P₁ equivalent can be compared directly with previous results for antigen P₁ produced by *D. pteronyssinus*. For antibody assays, the standard is a pool of mite-allergic sera established freeze-dried in London (NIBSC 82/528).¹² We believe that our results suggest that there is a direct relationship between mite-allergen exposure and IgE antibody responses in the 20 children with asthma reported here. We speculate that the mite sensitivity of these children plays a causative role in their asthma.

Our results also suggest that the use of mite extracts in the United States for skin testing and immunotherapy needs to be reconsidered. *D. pteronyssinus* rather than *D. farinae* was the most common pyroglyphid species in these Atlanta houses. These two mites, as well as *E. maynei*, produce a major allergen that is at least partially cross-reactive; there are also other "major" and "minor" allergens that are less well characterized. It should also be noted that one child's house contained *E. maynei* as the dominant mite species, with smaller numbers of *D. pteronyssinus* (see Fig. 3, patient No. 11). Four of the 20 children reported had no demonstrable IgE antibody to antigen P₁ but had positive skin tests and RAST to both *D. farinae* and *D. pteronyssinus*. Although there was a good correlation between RAST results for the two species, the results were not equal. These data taken together suggest that extract from either *D. farinae* or *D. pteronyssinus* by itself might not identify all mite-allergic patients. The degree of cross-reactivity between these mites appears to be as close as that occurring between the grasses, where it has been normal policy to use a mixture of grasses both for diagnosis and treatment of grass-pollen allergy. Therefore, we suggest that extracts of both *D. farinae* and *D. pteronyssinus* should be used to identify mite-allergic patients in the United States. Likewise, until the cross-reactivity between the two species is fully

evaluated, it would be better to use extracts of both species for immunotherapy. This would provide all "minor allergens" occurring in these two *Dermatophagoides* species.

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