

House dust mite exposure in asthmatic and healthy children: the difference is carpeting

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Aim – To determine whether house dust mite (HDM) exposure in living rooms and bedrooms is higher in asthmatic children than in those of age and sex matched healthy children, living in the same area. **Methods** – Types of floor-coverings were recorded and dust samples were collected by vacuum cleaning the total area of living rooms and bedrooms; *Der p I* and *Der p II* per gram fine dust concentrations were assessed. Twenty-five asthmatic children (RAST HDM $\geq$ class 3, age 6–12 years) and 25 healthy children participated in the study. **Results** – The frequency of cleaning and prevalence of smooth floor-coverings in bedrooms of asthmatic children were significantly higher. There were no differences in living rooms in this respect. The amount of fine dust/m² floor space was significantly lower in bedrooms of asthmatic children. Concentrations of HDM were low and no differences in *Der p I* and *Der p II* concentrations were observed between the two groups (asthmatic children: *Der p I* living room: 1.1 (0.04 – 59.4 μ g/g), bedroom: 0.5 (below detection – 19.3 μ g/g); nonasthmatic children: *Der p I* living room: 1.4 (below detection – 27.5 μ g/g), bedroom: 0.9 (below detection – 68.8 μ g/g). Smooth floor coverings contained significantly less fine dust, *Der p I*, and *Der p II* than carpeted floors. **Conclusions** – Low HDM concentrations are a general finding in Dutch dwellings in the present generation of children.

We observed a higher cleaning frequency, and more smooth floor coverings in bedrooms of asthmatic children than of healthy children, yet HDM concentrations were not significantly different. The latter can be explained by the observation that only 40% of the asthmatic children had smooth floor coverings in their bedrooms. Smooth floor coverings contain less fine dust and lower concentrations of *Der p I* and *Der p II* than carpeted floors.

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House dust mites (HDM) are the most important allergens for inducing asthmatic symptoms in allergic adults and children (1–6). Earlier studies have shown that exposure to allergens in newly diagnosed asthmatic adults is higher than in randomly selected controls (7, 8). Recently Sporik et al. (5) suggested that, in addition to genetic factors, exposure to HDM allergens in early childhood plays an important role in the subsequent development of asthma. Therefore, reduction of environmental HDM concentrations appears to be of clinical and prognostic relevance in the management of allergic asthma in children.

In a recent study we have found low concentrations of *Der p I* and *Der p II* in floor dust of living rooms and bedrooms of asthmatic children (9). These HDM levels were remarkably lower than previously reported in studies from other countries with

a similar climate (5, 8, 10). The lower concentrations of *Der p I* and *Der p II* in our study may have been the result of either advice given in former years to reduce allergen exposure or a general lower concentration of HDM in houses of this generation of children. We therefore investigated whether we could find a difference in HDM concentrations in living rooms and bedrooms between allergic asthmatic children and age and sex matched healthy children. We compared types of carpeting, cleaning frequencies, age of the houses, humidity, and amounts of fine dust and HDM concentrations in floor dust.

Patients and methods

We studied 25 allergic asthmatic and 25 healthy children, all attending primary school. The 25 con-

secutively selected asthmatic children visited the outpatient clinic for paediatric pulmonary diseases at our University Hospital. Routine standardized information with regard to reduction of environmental allergens was given previously by pediatricians and specially trained nurses at our outpatient clinic. This advice was given during the first visit and focused on HDM reduction in bedrooms.

Age and gender were similar between patients and controls (age: asthmatic children 9.1 ± 1.4 years, healthy controls 9.2 ± 2.1 years; gender (male/female): asthmatic children 14/11, healthy controls 15/10). Children with asthma were allergic to HDM, defined as a RAST $\geq$ class 3 and had increased airway responsiveness, defined as a histamine provocation concentration (PC_{20} histamine) less than or equal to 16 mg/ml (2-min inhalation) (11). No Acarosan®, mattress coverings or dehumidifiers were used in the investigated houses. All patients used inhaled corticosteroids, twice daily 200 or 400 µg, and short-acting β_2 -adrenergic drugs when needed. Short-acting β_2 -adrenergic drugs were withheld 8 h before the lung function measurements. A healthy classmate living in the same area of the city was asked to participate in the study as a control. Controls never had respiratory complaints such as wheezing, dyspnea, or cough and were never diagnosed as allergic and/or asthmatic. In addition their first-degree relatives did not suffer from either asthma or any other allergic disease. All healthy children had normal total IgE values (12) and normal spirometric lung function values. Two children in the control group had increased RAST values (HDM 67, grass 8.5, dog 0.7 PRU/ml, and grass 1.3 and dog 16.6 PRU/ml) but were asymptomatic, and were therefore included in the study. Analysis with and without these two controls showed no differences. Four children (4/25 = 16%) refused venous puncture and spirometry. The study was approved by the Medical Ethics Committee of our hospital.

Study design

The homes of the children were visited from March to May in the same year. Measurements in the houses of patients and their controls were performed on the same day.

Methods

The kind of carpeting, frequency of cleaning of living rooms and bedrooms, and the age of the houses were recorded on a checklist. Smooth floor-coverings were defined as hard floors that could be cleaned wet, while carpeted floor-coverings were defined as wall-to-wall carpeted floor-coverings ei-

ther from synthetic, wool, or cotton material.

All floor dust samples were obtained by the same technician, using a vacuum cleaner (Philips type T580, 1100 W). For every location a separate double-walled disposable paper bag was used (13). Dust was collected from the total area of the location in order to obtain a representative sample, vacuum cleaning time per square meter was according to the WHO international standards (14, 15). The total amount of fine dust of each sample was measured after filtering with a 355-µm aperture sieve. Each sample was analyzed for the amount of HDM, and the concentration was expressed in µg/g fine dust according to the WHO international standards (14, 15). The HDM allergens *Der p* I and *Der p* II were analyzed by sandwich immunoassays using monoclonal antibodies (16). A reference HDM extract was used, which was calibrated for the content of *Der p* I and *Der p* II by the Allergy Division of the Dutch Central Laboratory of the Blood Transfusion Service (Amsterdam), in serial dilutions for calculation of the allergen content of the HDM samples. The controlled samples in this reference laboratory resulted in comparable HDM concentrations. Air temperature and relative humidity were measured to obtain absolute humidity values (gram water vapor per kilogram of dry air) in each location.

Pulmonary function measurements

Spirometry (inspiratory slow vital capacity (VC) and forced expiratory value in one second (FEV_1)) was performed with a water-sealed spirometer (Lode BV, Groningen, The Netherlands). Peak expiratory flow (PEF) measurements were performed with a Wright peak flow meter. The best of three efforts was used for statistical analysis. Normal values from Zapletal et al. were used (17).

Statistical analysis

Statistical analysis was performed by SPSS/PC, version 4.0 (SPSS Inc, Chicago, IL, USA). Variables with a Gaussian distribution are presented as means $\pm$ standard deviations (SD). Variables with a non-Gaussian distribution (HDM allergen concentrations) are presented as medians with minimum and maximum values. Differences were calculated by paired t-tests and by nonparametric tests (Mann-Whitney U) as appropriate. Differences in discontinuous variables were calculated by chi-square tests. Correlations were calculated by Spearman's rank correlation coefficient. A p value of <0.05 was accepted as significant. When the amount of fine dust was less than 0.1 g per location, we used the 0.1 g values for calculations. When the allergen load was below detection concentration, we used the detection

concentration (0.01 µg/g for *Der p* I and *Der p* II) for calculations. HDM values below detection concentration were not overestimated due to nonparametric testing.

Results

Total IgE, specific IgE to HDM, and total number of eosinophils were significantly higher ($p < 0.05$) in the asthmatic children than the healthy children (total IgE: 359 (78–7699 IU/ml) versus 52 (4–674 IU/ml); specific IgE to HDM: 17.3 (2.1–180 PRU/ml) versus 0–67.0 PRU/ml); eosinophils: $5.0 \pm 3.4 \times 10^8$ /l versus $2.9 \pm 2.0 \times 10^8$ /l). Lung function values were similar in the two groups.

Cleaning frequency of the living rooms were similar (asthmatic children: 3.5 (1–7) times per week, healthy children 2.5 (1–7) times per week). Cleaning frequency of the bedrooms was significantly higher in asthmatic children than healthy children (2.0 (1–7) times per week versus 1.0 (1–7) times per week, $p < 0.05$). The ages of the houses were similar (asthmatic children 20 (3–100 years), healthy children 20 (1–141 years)). No significant correlation was found between cleaning frequency or age of the buildings on one hand and the amount of fine dust or allergen concentrations of both locations of the asthmatic and healthy children on the other hand.

We observed comparable types of floor-coverings in living rooms of both groups of children (smooth floor-coverings in the asthmatic group: 9/25 = 36%; in the healthy group, 8/25 = 32%), but significantly more smooth floor-coverings in the bedrooms of the asthmatic children (10/25 = 40%) than in the bedrooms of the healthy children (3/25 = 12%). In 2 bedrooms (1 asthmatic and 1 healthy child) we collected less than 0.1 g fine dust. These samples were not analyzed for HDM.

Table 1. Amount of fine dust and type of floor-covering in living rooms and bedrooms

	AAC		NAC	
Living room				
All floor coverings	0.15	(0.01 – 1.81)*	0.11	(0.02 – 0.89)
Smooth	0.07	(0.01 – 0.27)	0.07	(0.03 – 0.29)
Carpet	0.08	(0.03 – 1.81)	0.16	(0.02 – 0.89)
Bedroom				
All floor coverings	0.03	(0.01 – 1.13) ¹	0.12	(0.01 – 1.93)
Smooth	0.02	(0.01 – 0.19)**	0.05	(0.02 – 0.12) ²
Carpet	0.14	(0.01 – 1.13)	0.13	(0.01 – 1.93)

AAC: allergic asthmatic children, NAC: nonasthmatic children. Values are expressed as median (minimum – maximum) value of gram fine dust/m² floor space in general or on type of floor-covering; significant differences by Mann-Whitney U test.

* $p < 0.05$ between living room and bedroom in AAC;

** $p < 0.05$ between types of carpeting in AAC;

1. $p = 0.08$ between AAC and NAC for all floor-coverings.

2. $p = 0.09$ between types of carpeting in bedroom NAC.

The amount of fine dust/m² floor space in the living rooms of the asthmatic group was significantly higher ($p < 0.05$) than in the bedrooms. We did not observe this difference in the group of healthy children (Table 1). In the bedrooms of the asthmatic children significantly less ($p < 0.05$) fine dust was found on smooth floor-coverings than on carpeted floor-coverings, this difference being almost significant in the control group ($p = 0.09$). Smooth floor-coverings (measured for all children) contained less dust per square meter floor space than carpeted floor-coverings in general, in the separate locations, and in both study groups (Table 2).

The *Der p* I and *Der p* II concentrations in floor dust (Fig. 1) were similar in living rooms and bedrooms, and no significant differences were found between the locations of the asthmatic and healthy children. In all locations some samples contained *Der p* I concentrations above international safety standards (14, 15): in the asthmatic group: 6 above 2 µg and 6 above 10 µg *Der p* I /g fine dust; in the control group, 7 above 2 µg and 6 above 10 µg *Der p* I /g fine dust.

Table 2 shows the relationship of type of floor-covering to HDM concentrations per location expressed in µg/g fine dust for *Der p* I and *Der p* II. Smooth floor-coverings contained significantly less *Der p* I than carpeted floor-coverings in general (measurements of all children), in the different loca-

Table 2. Amount of fine dust and house dust mite exposure in the locations by type of floor-covering

	"Smooth"	"Carpeted"
All locations		
	(n = 30)	(n = 70)
Dust, g/m ²	0.04 (0.01 – 0.29)	0.14 (0.01 – 1.93)*
<i>Der p</i> I	0.4 (n.d. – 33.0)	1.5 (n.d. – 68.8)*
<i>Der p</i> II	0.09 (n.d. – 13.3)	0.9 (n.d. – 59.7)*
All living rooms		
	(n = 17)	(n = 33)
Dust, g/m ²	0.07 (0.01 – 0.29)	0.16 (0.02 – 1.81)*
<i>Der p</i> I	0.3 (n.d. – 33.0)	1.6 (0.03 – 59.4)*
<i>Der p</i> II	0.09 (n.d. – 13.3)	0.6 (n.d. – 14.7)*
All bedrooms		
	(n = 13)	(n = 37)
Dust, g/m ²	0.02 (0.01 – 0.19)	0.13 (0.01 – 1.93)*
<i>Der p</i> I	0.4 (n.d. – 13.5)	1.1 (n.d. – 68.8)*
<i>Der p</i> II	0.09 (n.d. – 9.4)	1.0 (n.d. – 59.7)*
All AAC locations		
	(n = 19)	(n = 31)
Dust, g/m ²	0.03 (0.01 – 0.27)	0.14 (0.01 – 1.81)*
<i>Der p</i> I	0.4 (n.d. – 33.0)	1.1 (0.06 – 59.4)*
<i>Der p</i> II	0.10 (n.d. – 13.3)	0.5 (n.d. – 14.7)
All NAC locations		
	(n = 11)	(n = 39)
Dust, g/m ²	0.06 (0.17 – 0.29)	0.16 (0.01 – 1.93)*
<i>Der p</i> I	0.3 (n.d. – 1.7)	1.7 (n.d. – 68.8)*
<i>Der p</i> II	0.08 (0.01 – 4.1)	1.0 (n.d. – 59.7)*

Values are expressed as median (minimum – maximum) value; significant differences by Mann-Whitney U test.

AAC: allergic asthmatic children, NAC: nonasthmatic children. *Der p* I and *Der p* II are expressed as µg/g fine dust.

* $p < 0.05$ between carpeting.

1. $p = 0.06$ between carpeting.

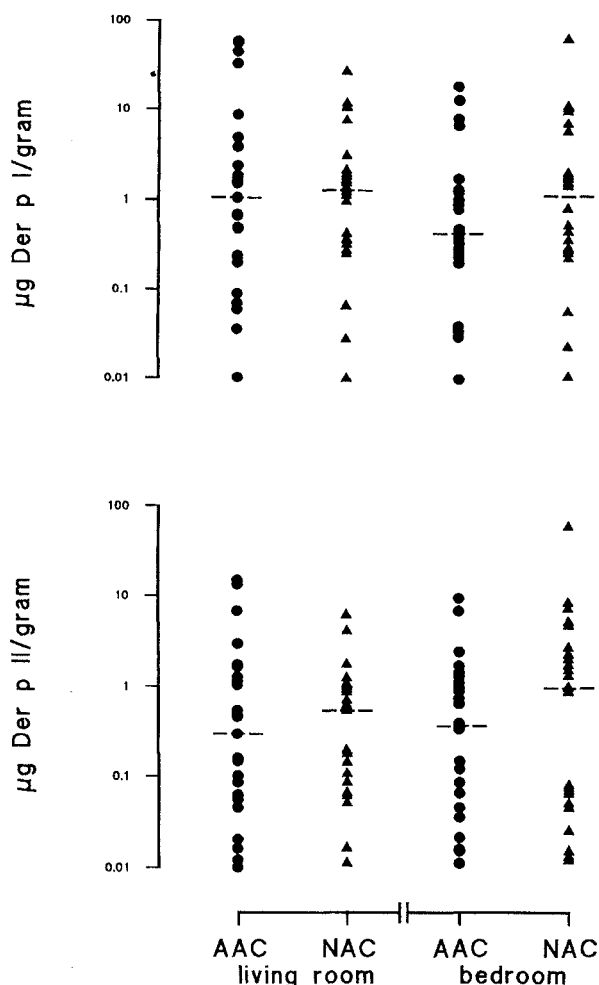


Fig 1. *Der p I* per gram fine dust ($\mu\text{g/g}$) (upper panel), and *Der p II* per gram fine dust ($\mu\text{g/g}$) (lower panel) in living rooms and bedrooms. Circles: values of the allergic asthmatic children (AAC), triangles: values of the nonallergic asthmatic children (NAC). ---: median value.

tions and in both study groups (Table 2). The same was true for *Der p II* in the living rooms of both groups and in the bedrooms of the healthy children, as well as for the levels of *Der p II* on smooth and carpeted floors in the bedrooms, and in all combined locations of the asthmatic children.

When *Der p I* and *Der p II* are expressed in $\mu\text{g}/\text{m}^2$ floor space, all comparisons in Table 2 showed significantly less allergen in favor of smooth floors ($p < 0.01$).

No significant differences in humidity were found between living rooms and bedrooms of both groups of children. Values in the living rooms were 7.2 ± 0.9 g/kg and 7.7 ± 0.9 g/kg and in the bedrooms 6.2 ± 0.6 g/kg and 6.5 ± 1.1 g/kg, for the asthmatic and healthy children respectively. Absolute humidity in the bedrooms was significantly lower than in the living rooms in both groups. We did not find any significant correlation between absolute humidity and HDM allergen concentrations.

Discussion

We did not observe significant differences in *Der p I* and *Der p II* exposure between asthmatic and healthy children. As expected, smooth floors contained significantly lower *Der p I* and *Der p II* concentrations than carpeted floors.

The HDM exposure levels were low as compared to those reported from other countries with a similar climate (5, 8, 10), confirming our previous findings (9). In that report, we hypothesized that the observed lower exposure concentrations were the result of previously given routine advice with regard to preventive measures to reduce HDM exposure. The results of this study disprove this hypothesis.

Bedrooms of the asthmatics had significantly more smooth floor-coverings than the control group and therefore contained less dust. When all smooth floor-coverings were compared with all carpeted floors we indeed observed significantly lower HDM concentrations on smooth floor-coverings. Although we observed that bedrooms of asthmatic children were more frequently cleaned, we could not find a correlation between cleaning frequency and the amount of fine dust or allergen concentrations.

High concentrations of HDM may be an important risk factor for both development and severity of asthmatic symptoms in sensitized children (5, 18). Our observation indicates that parents, probably as a result of former advice, focus on adjusting the type of floor-covering in the bedrooms of their children. This is in line with findings in Denmark (19), where houses of nonallergic individuals contained significantly higher amounts of cat allergen than houses of allergic individuals.

Our study shows that smooth floor-coverings are associated with less dust and lower HDM concentrations. It is known that HDM flourish at higher temperature and humidity, suggesting that reduction of humidity is important in the battle against HDM. We observed no differences in absolute humidity between the houses of the two study groups, and especially not in the bedrooms. This does not imply that humidity is not important. Korsgaard (7) found a higher number of HDM and a higher absolute humidity in his asthmatic group than in the control group. Absolute humidity values in his and our study are comparable. Moreover, we could not confirm the observed correlation between humidity and allergen concentrations. A difference between his and our study is that we only measured humidity at one point of time. Furthermore, measurements in the Danish study were performed in November and December, while our measurements took place from March to May. It may well be that humidity differences may exist at other times of the year.

We did not observe significant differences in *Der*

p I and *Der p* II concentrations between asthmatic children and their controls in bedrooms and living rooms. According to the international standards for preventive measures to reduce HDM exposure, a concentration of 2 µg *Der p* I /g fine dust should be a risk factor for sensitisation, and a concentration of 10 µg *Der p* I /g fine dust a major risk factor for acute asthma in mite-allergic individuals. Although the median values of *Der p* I in our study were rather low, in both types of locations some sample concentrations (14/100 = 14%) were above 10 µg *Der p* I /g fine dust. Seventy-six percent of the allergic asthmatic children and maintenance medication of ICS were exposed to lower concentrations of *Der p* I than 2 µg/g fine dust in living room and/or bedroom. This indicates that lower concentrations of *Der p* I are also of importance to the clinical expression of the disease (20), and that the international recommendations should be revised. The low HDM concentrations from our pediatric population are in accordance with observations in houses of adults from our region (16), and a recent publication on HDM concentrations from more southern regions in The Netherlands (18). Despite the observed lower HDM concentrations, all Dutch studies report a large individual variation. It seems, therefore, worthwhile to verify individual HDM concentrations in these locations and relate them to allergen-reduction measures.

We conclude that the observed low HDM concentrations in Dutch dwellings are a general finding in this generation of children. We have observed a higher cleaning frequency, and a higher prevalence of smooth floor-coverings in bedrooms of asthmatic children than of healthy children, yet HDM concentrations were not significantly different. The latter can be explained by the observation that only 40% of the asthmatic children had smooth floor-coverings in their bedrooms. Finally we confirm previous data in that smooth floor-coverings contain less fine dust and lower concentrations of *Der p* I and *Der p* II.

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