

The effect of anti-allergic mattress encasings on house dust mite-induced early- and late-airway reactions in asthmatic patients. A double-blind, placebo-controlled study

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Summary

Background Anti-allergic mattress encasing may provide clinical benefit in asthmatic patients. However, the effect of mattress encasings on allergen-specific parameters, such as bronchial reactions to house dust mite (HDM) challenge, is not clear.

Objective To investigate the effect of anti-allergic mattress encasings on allergen sensitivity in patients with moderate to severe asthma.

Methods Twenty-seven patients with asthma and HDM allergy were studied in a double-blind, placebo-controlled study. Concentrations of *Dermatophagoides pteronyssinus* (Der p 1) were measured in mattress dust before and after 1 year of treatment; bronchial histamine challenge, bronchial challenge with HDM and intradermal skin challenges with HDM were performed. The number of eosinophils in peripheral blood was assessed.

Results In the active group, but not in the placebo group, there was a significant reduction in Der p 1 concentration in the dust collected from the mattresses after 1 year of treatment compared to before. There was a significant difference between the groups with respect to HDM-induced early-reaction (ER) in the airways and the number of blood eosinophils, which reflected an increase in ER and eosinophils in the placebo group without significant change in the active group. No significant improvement in PC₂₀ histamine, late-reaction (LR) and skin tests was found in either groups.

Conclusion Our data suggest that encasings protect against a further increase in allergen sensitivity in asthmatic patients, so their use should be recommended.

Keywords mattress encasings, allergic asthma, house dust mite, bronchial allergen challenge

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Introduction

Over the last decades, the prevalence and incidence of asthma have increased despite improved medical treatment. One of the contributing factors for this increase is thought to be exposure to allergens. House dust mite (HDM) has been proven to be one of the most important allergens. An association exists between the level of HDM exposure and the risk to develop asthma [1–3], i.e. the higher the exposure, the higher the risk. Moreover, avoidance of HDM, as can be reached in the mountains or during hospitalization, can result in an improvement of asthma symptoms, peak flow values, non-specific hyper-responsiveness and the use of medication [4–6].

Allergen avoidance is the first step in the treatment of patients with allergic asthma. Several procedures have been developed

to limit the exposure to HDM. Most procedures are expensive, require active participation of the patients and have not been extensively studied in double-blind, placebo-controlled studies. One such allergen avoidance measure is the application of encasings. Mattresses, pillows and bedding often contain large amounts of HDM, resulting in high exposure for several hours during the night. The use of specific anti-allergic mattress encasings can result in a strong reduction of Der p 1 levels on top of the mattress [7–9], with subsequent clinical improvement and a reduction in histamine-induced bronchial responsiveness in asthmatic patients [7,9]. These studies, however, have been generally performed in relatively mild asthmatics. In addition, there are no data on allergen-specific parameters, such as bronchial reactions to HDM challenge.

In the present study the effect of anti-allergic encasings was studied on bronchial response upon challenge with allergen as well as with histamine in patients with moderate to severe asthma in a double-blind, placebo-controlled design. In addition, other allergic parameters were studied, such as skin responsiveness to HDM, peripheral blood eosinophils and specific immunoglobulin (Ig)E to HDM.

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Table 1. Clinical data of the patients

Patient	Age	Sex	FEV ₁ (% pred.)	PC ₂₀ hist. (mg/mL)	(Skin test*)	Rhinitis	Medication asthma	Medication rhinitis
<i>Placebo</i>								
1	21	M	97	1.76	2.1	–	B, SB	–
2	11	M	79	0.74	1.1	+	B, SB	–
3	19	F	111	1.00	1.4	+	SB	–
4	11	M	97	4.00	1.4	+	B, SB	–
5	29	M	81	0.33	1.2	–	F, SM	–
6	21	M	73	2.43	2.0	+	B, SB	B
7	27	F	70	0.90	1.2	+	B, SB	B
8	44	M	70	1.89	1.2	+	F, SM	F
9	17	F	138	1.78	1.9	+	–	B
10	40	M	64	1.13	1.6	+	B, SB	–
11	29	M	103	0.71	1.5	+	–	–
12	28	F	73	0.45	2.2	–	F, SB	–
13	12	M	106	4.00	1.6	+	B, SB	–
Mean	24		89	1.62	1.6			
<i>Active</i>								
1	25	F	99	1.19	1.5	+	B, SB	–
2	29	M	99	0.95	1.6	–	B	–
3	41	M	67	3.79	2.2	+	B, SB	–
4	24	F	86	2.00	1.8	+	B, SB	–
5	31	M	81	1.22	1.5	–	–	–
6	24	M	80	0.55	1.1	+	B, SB	B
7	15	M	86	0.65	1.7	–	F	–
8	22	F	104	2.67	1.4	+	B	–
9	51	F	70	0.45	1.2	+	B, SB	B
10	32	M	68	0.42	1.1	–	B, SM	–
11	36	F	103	0.35	1.8	+	SB	B
12	13	M	79	2.45	2.6	+	SB	–
13	42	M	75	0.42	1.2	–	B, SM	–
14	18	F	103	2.35	1.2	+	F, SM	F
Mean	29		86	1.39	1.6			

*, × times histamine reaction. B, beclomethasone or budesonide; F, fluticasone; SB, salbutamol; SM, salmeterol; FO, formoterol.

Methods

Patients

Twenty-seven atopic, non-smoking patients with asthma and HDM allergy (10 females and 17 males, age 11–51 years) participated in the study. The clinical data are presented in Table 1. Although most patients had moderate to severe asthma, three patients in each group did not regularly use inhaled steroids. The patients were selected for increased bronchial responsiveness to histamine inhalation (PC₂₀ < 4 mg/mL, 30 s inhalation of histamine phosphate solution), positive skin tests and/or elevated specific IgE to HDM allergen, early bronchial reaction to HDM inhalation and relevant HDM exposure on the mattress (> 1 µg Der p 1/g dust). All patients had FEV₁ values > 60% (predicted value). Pollen allergic patients were included and evaluated outside the pollen season. Patients did not have a history of respiratory tract infections in the previous six weeks or severe asthmatic attacks in the previous six months. Patients having a furred pet were only admitted when they had no pet allergy. None had received oral corticosteroids in the previous 6 months. All patients gave informed consent. The Medical Ethical Committee of Asthma Centre Heideheuvel, the Netherlands, approved the study.

Study design

The study was performed in a randomised placebo-controlled, double-blind, parallel-group design, comparing the effect of allergen-impermeable encasings on the mattresses, pillows and comforters with matching placebo encasings during one year. At the start of the study a trained respiratory nurse visited the houses of the patients in order to establish allergen avoidance measures already present. All patients included in the study had bedroom floors without carpets. Patients were instructed to wash their sheets each week at 60 °C. Apart from the mattress, pillow and comforter encasings no other allergen avoidance measures were taken. At the end of the study the nurse visited the houses again to verify that no other changes were made to the bedroom during the study. The patients were included continuously; the inclusion period lasted 2 years.

At the first visit patients underwent clinical evaluation. FEV₁ and vital capacity (VC) values were measured, skin tests were performed and a PC₂₀ histamine was assessed. Medication was withheld before the study period: inhaled steroids and sodium cromoglycate one week before the challenge, theophyllines, oral β₂-adrenergic drugs, long-acting inhaled β₂-adrenergic drugs and antihistamines 48 h before the tests and inhaled β₂-adrenergic drugs 6 hours before the tests. The patients continued

their regular medication after the tests. The patients were told not to change their medication during the intervention period and to record their medication use in an asthma diary.

Seventy-two hours after the bronchial histamine challenge the patients were admitted to the hospital for three consecutive days. On the first day, starting at 9.00 am, a challenge with a diluent control was performed. The second day, at 9.00 am, subjects underwent allergen challenge with HDM. Before the challenge blood sampling was performed for eosinophil counts. Spirometry was performed as on the first day. At the third day patients underwent a second histamine provocation.

Dust was collected from the mattresses of the patients for Der p 1 measurement before installing the encasings. After 1 year the same test procedure was repeated and dust was collected on top of the mattress cover.

Collection and extraction of house dust

Collection of dust Before, and at the end of the intervention, mattress dust was collected by a trained respiratory nurse using a vacuum cleaning apparatus (Philips type Vitall 377, 1300 watt, Eindhoven, the Netherlands). She vacuum cleaned the whole mattress during 2 min with a special filter device (Petersen-Bach A/S, Copenhagen, Denmark, mean pore size 5–6 µm, maximum pore size 10 µm). At the start of the study dust was collected directly from the mattress; at the end of the study dust was collected on top of the encasing, using always the same vacuum cleaner. The filters were stored in the freezer (–20 °C) until analysis at the end of the study.

Der p 1 analysis

After sieving (0.35 mm), the amount of fine dust was weighed and a 10% (wt/vol) extraction in 0.01 mol/L NH_4HCO_3 buffer was performed by overnight rotation at 4 °C. The samples were centrifuged and the supernatants were used for the measurement of Der p 1 concentration. Der p 1 antigen was measured using an enzyme-linked immunosorbent assay (ELISA). Monoclonal antibodies (ALK, Hørsholm, Denmark) against Der p 1 were immobilized on a 96-well plate. The incubation with the dust extracts was followed by a second incubation step with a polyclonal antibody (horseradish-peroxidase). After adding 1,2 phenyldiamine HCL as substrate the absorption at 490 nm was measured using an ELISA reader.

Histamine challenge

Histamine phosphate solutions (doubling concentrations from 0.25–32 mg/mL) were administered through a De Vilbiss 646 nebulizer with a gauged output of 0.13 mg/mL. The nebulizer was mounted to a valve box containing an aerosol filter. The nebulization time was 30 s, during which the patient was instructed to breathe quietly. The test started with inhalation of a phosphate buffer aerosol. Prior to the inhalation three measurements of VC and FEV_1 were performed (Jaeger Masterscreen; Breda, the Netherlands). FEV_1 was measured after each doubling concentration. The PC_{20} histamine was derived by linear interpolation.

Bronchial allergen challenge.

Allergen solutions were prepared from stock solutions of *Dermatophagoides pteronyssinus* in phosphate buffer solution (PBS) supplemented with 0.03% human serum albumin and 0.5% phenol (SQ 503, ALK Benelux, Houten, the

Netherlands). Fivefold increasing concentrations of allergen solutions were prepared ranging from 80–10 000 BU/mL. The allergen solutions were administered through a De Vilbiss nebulizer mounted to a valve box containing an aerosol filter (output 0.13 mg/mL). The patients were instructed to breathe normally during 1 min for each dose. Increasing doses were given at 15-min intervals. The challenge procedure was terminated when the FEV_1 value fell more than 15% below the baseline value. After the last inhalation FEV_1 was recorded at 10-min intervals for the first hour, to determine the early asthmatic response and at 1-h intervals thereafter until 10 h after the last inhalation, to determine the late asthmatic response. FEV_1 values were corrected for diurnal variation, determined during the previous day.

The cumulative provocation dose of allergen necessary to induce a fall in FEV_1 value > 20% from baseline (PD_{20} allergen) was calculated. This PD_{20} allergen is based on the observed maximal fall of FEV_1 and the delivered dose of allergen, and characterizes the severity of the early and late asthmatic response [10,11].

Skin tests

Intradermal challenges (30 BU/mL, 0.03 mL) were performed on the back of the patients with a standardized HDM extract (ALK Benelux, SQ-503). The early response was scored as the weal diameter 15 min after challenge. Late responses were measured 6 h after challenge: indurations were determined according to a procedure suggested by Sokal [12]. The reactions on histamine and phosphate buffer diluent served as positive and negative controls.

Peripheral blood eosinophils

A blood sample was taken at 9.00 am at the start of the study and after 1 year.

Blood eosinophils were counted using Bürker-Türk counting chamber (Landsmeer, the Netherlands).

Mattress encasings

In the active group, mattresses, pillows and bedding were encased using ACb™ Allergy Control covers supplied by Cara C'air (Velsbroek, the Netherlands). The same company made the matched placebo covers. The encasings were placed in position by a research nurse and left *in situ* for 1 year. The patients were advised to use their normal sheets on top of the mattress, pillow and comforter encasings and use no other additional bedding.

Data analysis

Statistical analyses were performed with SPSS 9.0 standard version. Comparisons within groups (before and after intervention) were made with the Wilcoxon signed rank test (WSR). Mann–Whitney *U*-test was used for between group comparisons. *P*-values < 0.05 were considered significant. Data were log-transformed to obtain normal distribution, if necessary. The log-transformed data were analysed, using the sign test. These log-transformed values were expressed as mean value ± standard error of the mean (SEM).

Results

Demographic characteristics were similar between the two treatment groups (Table 1).

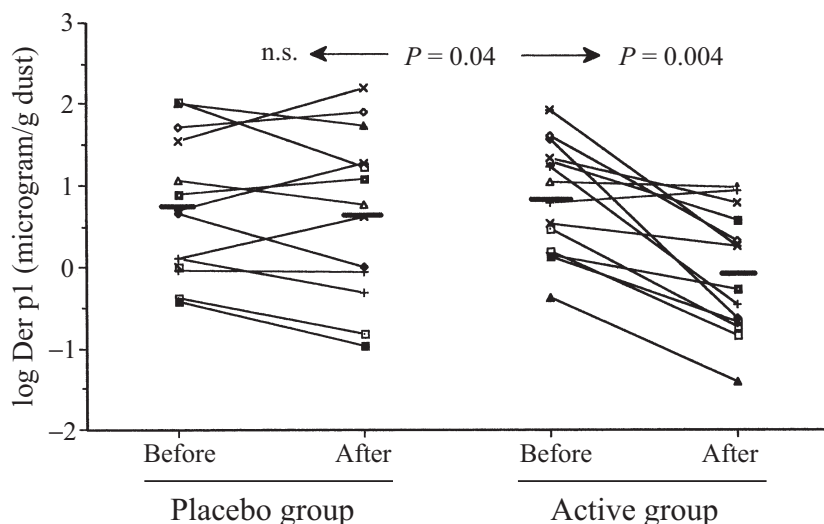


Fig. 1. Der p 1 concentrations on top of the mattress before and after 1 year of intervention: mean value (log data) of the groups is represented (Active: 0.85 and 0.06 μg Der p 1/g dust, respectively. Placebo: 0.77 and 0.66 μg Der p 1/g dust, respectively). — = mean value, n.s. = not significant.

There were no significant differences between the two groups with regard to Der p 1 concentrations, PC_{20} histamine, PD_{20} allergen, skin tests, blood eosinophils, skin tests to HDM and medication use. Analyses of the diary cards at 0, 4, 8, 12 months showed that there was no change in medication during the study in both groups.

Der p 1 concentrations on mattresses

Figure 1 shows geometric mean Der p 1 concentrations on the mattresses before and 12 months after the start of the study in both groups. Der p 1 concentrations on the mattresses were significantly lower in the active group after 1 year (16.2 ± 6 and $2.2 \pm 0.9 \mu\text{g}$ Der p 1/g fine dust, respectively; $P = 0.002$). In the placebo group, there was no significant reduction in Der p 1 (26.4 ± 11.7 and $27.0 \pm 12.8 \mu\text{g}$ Der p 1/g fine dust, respectively; $P = 0.58$). There was a significant difference in the treatment-induced change in Der p 1 concentration between the two groups ($P = 0.04$).

Early and late asthmatic responses

In several patients the allergen concentration required to induce a fall in FEV_1 of at least 20% differed during the study. Therefore, the early and late responses on HDM challenge were expressed as PD_{20} allergen.

PD_{20} early response Figure 2(a) shows the PD_{20} allergen of the early response before and 1 year after the intervention. All patients showed early response before treatment. In the active group, there was no significant change in PD_{20} allergen before and after 1 year ($750 \pm 175 \text{ BU/mL}$ and $611 \pm 157 \text{ BU/mL}$, respectively; $P = 0.79$). In the placebo group, the PD_{20} allergen after 1 year was significantly lower compared to before (PD_{20} $1580 \pm 646 \text{ BU/mL}$ and $473 \pm 161 \text{ BU/mL}$, respectively; $P = 0.003$), indicating a more sensitive response after 1 year. There was a significant difference in the treatment-induced change in PD_{20} allergen between the two groups ($P = 0.04$).

PD_{20} late response Figure 2(b) shows the PD_{20} allergen of the late response before and 1 year after the intervention.

Late-phase reactions could be demonstrated in ten patients in the placebo group and in nine patients in the active group.

There was no significant change in PD_{20} allergen during the year in the active group (PD_{20} $498 \pm 111 \text{ BU/mL}$ and $360 \pm 84 \text{ BU/mL}$, respectively; $P = 0.51$). In the placebo group there was a significant decrease in PD_{20} allergen after 1 year compared to before (PD_{20} $1103 \pm 385 \text{ BU/mL}$ and $394 \pm 144 \text{ BU/mL}$, respectively; $P = 0.002$). The treatment-induced change in PD_{20} allergen between the two groups was not significant ($P = 0.11$).

PC_{20} histamine

Mean log PC_{20} histamine in the active group was $0.24 \pm 0.31 \text{ mg/mL}$ at the start of the study and $0.32 \text{ mg/mL} \pm 0.29$ after 1 year, the difference being not significant ($P = 0.18$) (Fig. 3). In the placebo group, there was no significant change in PC_{20} histamine during one year (mean log PC_{20} histamine before $0.36 \text{ mg/mL} \pm 0.28$; after 1 year $0.39 \text{ mg/mL} \pm 0.28$; $P = 1.0$). The treatment-induced change in PC_{20} histamine between the two groups was not significant ($P = 0.77$).

Skin reactions

Early reactions Figure 4(a) shows the results of the early skin reactions after intradermal allergen challenge before and after 1 year in both groups. There was no significant change in skin reaction during 1 year in the active group ($18.5 \pm 1.3 \text{ mm}$ and $16.1 \pm 0.9 \text{ mm}$, respectively; $P = 0.12$). This was also the case in the placebo group ($18.7 \pm 1.8 \text{ mm}$ and $17.2 \pm 1.8 \text{ mm}$, respectively; $P = 0.58$). The treatment-induced change in early skin reaction was not significantly different between the two groups ($P = 0.66$).

Late reactions There was no significant change in skin reaction over 1 year follow-up both in the active group ($18.0 \pm 2.4 \text{ mm}$ and $13.5 \pm 2.2 \text{ mm}$, respectively; $P = 0.20$) and in the placebo group ($12.9 \pm 1.9 \text{ mm}$ and $13.6 \pm 1.2 \text{ mm}$, respectively; $P = 0.58$) (Fig. 4b). The treatment-induced change in early skin reaction was not significantly different ($P = 0.57$) between the two groups.

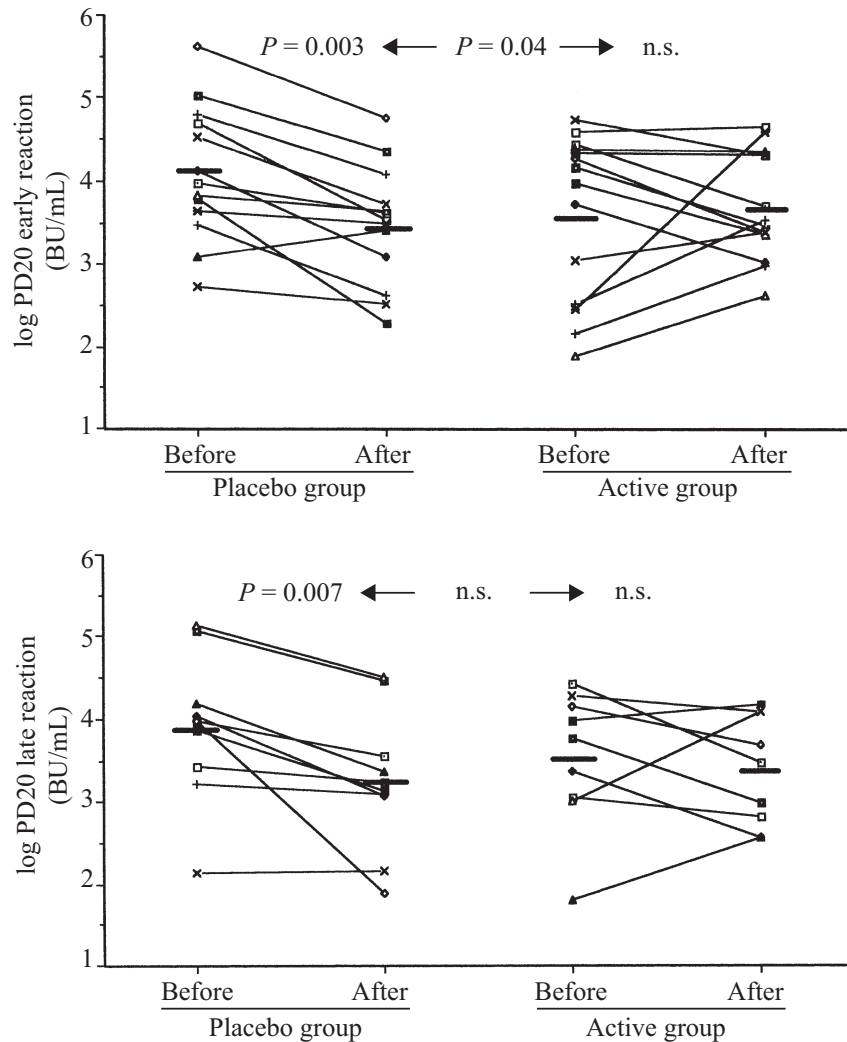


Fig. 2. (a) The effect of mattress encasings on PD₂₀ of early asthmatic response; mean value (log data) of the groups is represented (active: 3.62 and 3.69 μ g BU/mL, respectively. Placebo: 4.10 and 3.47 BU/mL, respectively). (b) The effect of mattress encasings on PD₂₀ of late asthmatic response; mean value (log data) of the groups is represented (active: 3.53 and 3.38 BU/mL, respectively. Placebo: 3.90 and 3.25 BU/mL, respectively). Only the results of patients who showed a late-phase response are represented. — = mean value, n.s. = not significant.

Specific IgE

There was no significant change in the level of specific IgE after 1 year compared to before in the active group (54.1 ± 10.1 PRU/mL and 44.3 ± 10.4 PRU/mL, respectively; $P = 0.13$), neither in the placebo group (59.2 ± 112.6 PRU/mL and 53.3 ± 10.0 PRU/mL, respectively; $P = 0.20$).

The treatment-induced change in specific IgE was not significantly different ($P = 0.93$) between the two groups.

Peripheral blood eosinophils

Total blood eosinophil counts did not significantly change after 1 year in the active group ($382 \pm 47 \times 10^6$ L and $366 \pm 50 \times 10^6$ L, respectively; $P = 0.66$). In the placebo group total blood eosinophil counts were significantly higher after 1 year compared to before ($273 \pm 37 \times 10^6$ L and $436 \pm 69 \times 10^6$ L, respectively; $P = 0.03$). There was a significant difference in the treatment-induced change in number of blood eosinophils ($P < 0.05$) (Fig. 5).

Discussion

The present study was performed in order to investigate the effect of anti-allergic mattress encasings on allergen sensitivity in patients with moderate to severe asthma and HDM allergy. We found a significant reduction in Der p 1 concentrations in the dust collected from the mattresses in the active group, which was also significantly different from the placebo group. There was a significant difference in ER to allergen and number of peripheral blood eosinophils between the active and the placebo group. This difference reflected increases in ER and the number of eosinophils in the placebo group, whereas no change occurred in the treatment group. No significant differences between the groups occurred with respect to PC₂₀ histamine, late bronchial reaction, skin test responsiveness and specific IgE.

It has been demonstrated that higher exposure to HDM is associated with more severe asthma [13–16]. In an attempt to maximally reduce the HDM exposure at home, several

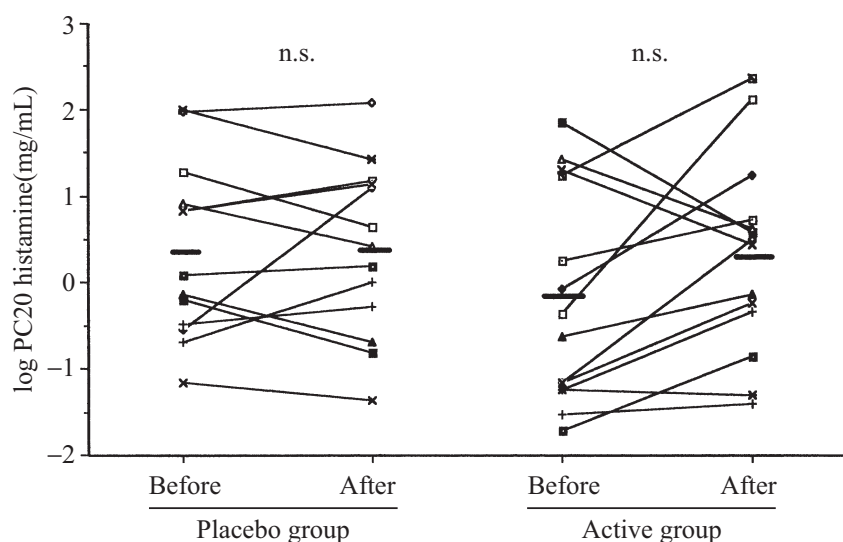


Fig. 3. PC₂₀ histamine before and after 1 year of intervention; mean value (log data) of the groups is represented (active: -0.36; 0.44 mg/mL, respectively. Placebo: 0.36; 0.39 mg/mL, respectively). — = mean value, n.s. = not significant.

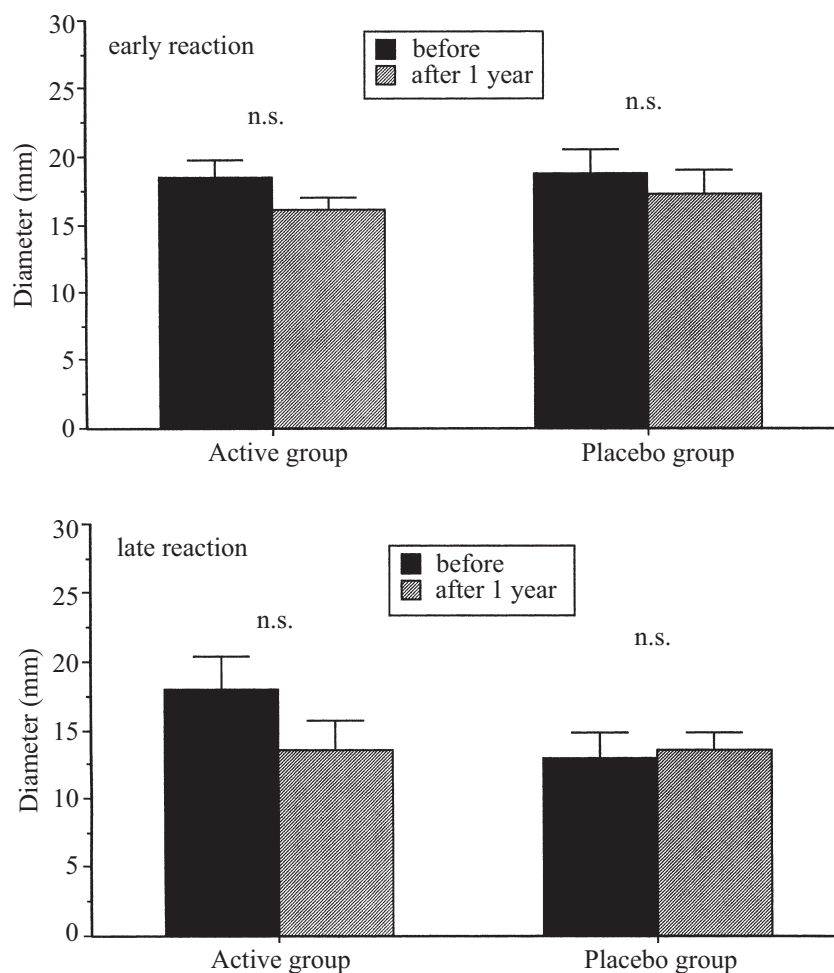


Fig. 4. Early (a) and late (b) skin reaction before and after 1 year of intervention (mm).

combinations of avoidance measures have been studied [6,17,18]. These studies are difficult to compare, because of the large differences in applied avoidance measures (often combination of chemical and physical methods), outcome parameters, study population and design.

In the Netherlands, mattresses and bedding are a major source of HDM and the use of anti-allergic encasings has become a standard procedure in the treatment of HDM-allergic asthmatic patients. Therefore, we have chosen to focus solely on the additional effect of mattress, pillow and bedding encasings,

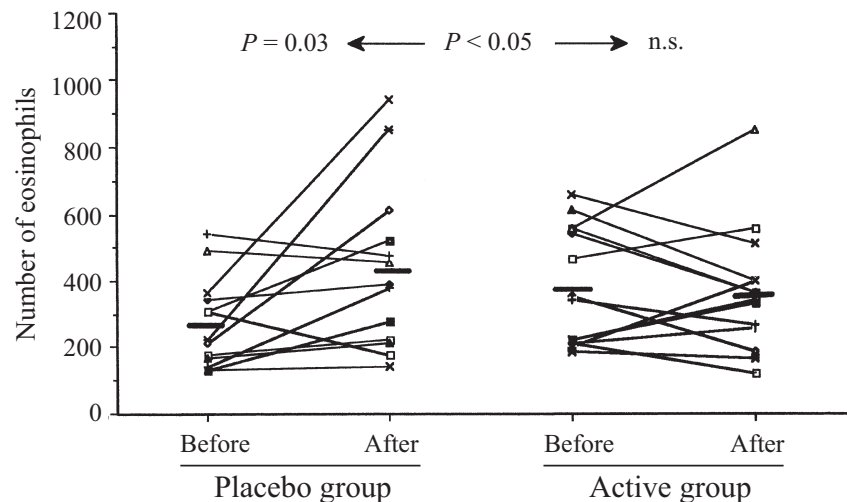


Fig. 5. The effect of mattress encasings on the number of peripheral blood eosinophils ($10^6/L$). — = mean value, n.s. = not significant.

leaving other allergen avoidance measures unchanged. In addition we have chosen to investigate a population of patients with severe bronchial hyper-responsiveness despite the use of inhaled bronchial corticosteroids.

In our study we found a significant reduction in Der p 1 concentrations on the mattresses in the active group after 1 year of intervention. The placebo encasings used in the study had a similar appearance as the active encasings, but did not protect against allergen exposure, as Der p 1 concentrations remained the same after 1 year. We have chosen 1 year of intervention to exclude seasonal variations in Der p 1 exposure, because seasonal variation in mite levels may easily exceed a factor of two or three when winters are relatively cold [15,19].

Some earlier studies using several different types of mattress encasings have also shown a reduction of Der p 1 exposure on top of the mattress [7–9,20–21]. Van der Heide et al. [9] showed the decrease in Der p 1 on the mattresses to be greater with mattress encasing than with acaricide treatment. In contrast, when combining impermeable covers over mattress, pillows and duvet with tannic acid/acaricidal spray on the carpets and furniture, Marks et al. found a 29% reduction in HDM allergen levels on the beds after two weeks but after 3 and 6 months the reduction in Der p 1 concentrations was not significant anymore [22]. The authors suggested that in a high HDM environment simple chemical treatment and encasement of bedding is not sufficient to cause a sustained beneficial reduction in allergen levels. Our study was performed in a high HDM environment as measured by Der p 1 concentrations on the mattress and showed a significant reduction in HDM. The difference between the studies is the presence of carpets on the floors in the study by Marks and the absence of carpets in our study. The above data combined suggest that encasings are effective in HDM reduction, provided that carpeting is avoided.

In the present study the significant reduction in Der p 1 exposure in the active group was not associated with significant effects on histamine-induced hyper-responsiveness. Other studies showed variable results with respect to effects on hyper-responsiveness [8,9,21–24]. This may be due to the duration of use and the effect of encasings; the study population duration seems not essential since both short-term (12 weeks–6 months) and long-term studies [9, 25] have shown beneficial effects.

The severity of asthma may have affected the outcome of the studies. Studies with a positive effect concerned patients with mild to moderate asthma [7]. For instance van der Heide et al. [9] included patients with a $PC_{20} < 32$ mg/mL (mean value 3.82 mg/mL), we included patients with severe hyper-responsiveness ($PC_{20} < 4$ mg/mL, mean value 1.39 mg/mL). Finally, mattress encasings reduce HDM exposure during the night, yet patients may still be exposed to relatively large amounts of HDM from other sources during the day. All studies so far have not measured these confounding factors. In addition, the severity of hyper-responsiveness is also determined by other factors such as other allergens [26–30], viruses and air pollution, which are not influenced by the avoidance measures. Especially in patients with more severe asthma the relative contribution of the night-time HDM exposure on the level of hyper-responsiveness may be limited.

We evaluated the effects of allergen avoidance on allergen-specific parameters in the airways, skin and peripheral blood. We did not find effects on early and late skin reaction to HDM nor on HDM-specific IgE in peripheral blood after 1 year of intervention. This is compatible with the data of Wickman et al. who also found no change in the degree of allergen sensitization assessed by skin prick tests or specific IgE antibodies against Der p 1 or Der f 1 after 18 months of HDM avoidance measures including mattress encasings [31]. Other studies also showed that the use of mattress encasings did not reduce total IgE, allergen-specific IgE or skin test sensitivity to HDM, despite the reduced HDM exposure [9,32].

Our study is, to our knowledge, the first to evaluate the effects of mattress encasings on allergen sensitivity in the airways. We could not demonstrate an increase in PD_{20} allergen, both in the early- and late-response after bronchial challenge with HDM, in the active group. In the placebo group, however, both ER and LR PD_{20} allergen, significantly decreased after 1 year compared to before, indicating an increase in allergen sensitivity. The difference between the groups was significant for the ER. This finding is difficult to explain. The increased allergen sensitivity can not be explained by an increase in allergen exposure or an increase in hyper-responsiveness, because these parameters did not change in the placebo group. Changes in PD_{20} cannot be explained by changes in PC_{20} histamine alone [33]. Furthermore,

the use of medication was unchanged in both study groups. The only parameter that changed during the study was Der p 1 exposure, which was significantly reduced on the beds of the patients in the active group. This might suggest that a reduction in Der p 1 exposure after the use of the anti-allergic mattress encasings may prevent a further deterioration in lower airway allergen sensitivity, as seen in the placebo group.

Another surprising finding was the significant increase in the number of peripheral blood eosinophils in the placebo group after 1 year which was not seen in the active group. Also this difference between the groups can only be explained by differences in allergen exposure. The significant increase in blood eosinophilia in the placebo group was associated with an increase in airway allergen sensitivity, but not with an increase in hyper-responsiveness. This latter finding is in contrast with other studies showing a positive correlation between blood eosinophilia and hyper-responsiveness [34], though in a cross-sectional way.

The present study showed that the use of anti-allergic mattress encasings in patients with moderate to severe asthma did not result in a reduction in hyper-responsiveness or airway allergen sensitivity, despite a significant reduction in Der p 1 exposure. It is possible that the limited efficacy is due to the severity of asthma in our study population. Since our data suggest that there is some protective effect against further increase in airway allergen sensitivity, the use of anti-allergic mattress encasings in this population should still be recommended.

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