

Effect of mattress encasings on atopic dermatitis outcome measures in a double-blind, placebo-controlled study: The Dutch Mite Avoidance Study

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Background: House dust mite (HDM) allergen might induce and maintain atopic dermatitis (AD). Reduction of allergen load by applying encasings might improve the clinical symptoms of AD.

Objective: We sought to investigate, in a randomized, double-blind, placebo-controlled study, whether reducing HDM allergen levels by using mattress, duvet, and pillow encasings for 12 months will result in improvement in AD symptoms.

Methods: Patients with AD (8-50 years old and allergic to HDM), having a Leicester sign score (a dermatitis score) of at least 1% extent and a severity score of 6 points or greater, were randomly allocated to an active (n = 45) or a placebo allergen-avoidance group (n = 41). Avoidance measures consisted of applying HDM-impermeable encasings for mattresses, pillows, and duvets for the active treatment group and cotton encasings for the placebo group. Effect on allergen concentrations (Der p 1 and Der p 1 plus Der f 1), Leicester sign score extent and severity, visual analogue scale scores for itching and sleeplessness, intradermal test results, atopy patch test results, total serum IgE levels, anti-Der p 1-specific IgE levels, and total blood eosinophil counts were studied.

Results: The active encasings reduced the Der p 1 allergen concentration in the mattress after 12 months with a factor 2.1 ($P = .007$) and the Der p 1 plus Der f 1 allergen concentration with a factor of 2.5 ($P = .005$); no significant change in allergen concentrations in mattresses was seen in the placebo group. Although the decrease in allergen load was significant, no differences in treatment-induced changes were seen between the placebo and active groups.

Conclusions: Use of HDM-impermeable encasings resulted in a significant decrease in Der p 1 and Der p 1 plus Der f 1 allergen concentrations. However, this reduction in allergen load did not result in significant changes in clinical parameters between the groups. Reduction of allergens in other environ-

ments (work, school, and outdoors) might be equally important in improving symptoms of AD. (*J Allergy Clin Immunol* 2002;110:500-6.)

Key words: Atopic dermatitis; mattress encasing; randomized, double-blind, placebo-controlled study; house dust mite; allergen avoidance; Leicester sign score

Atopic dermatitis (AD) is a chronically relapsing dermatitis with typical morphology and distribution. Hanifin and Rajka¹ defined major and minor diagnostic criteria for AD in 1980, and these criteria were generally accepted. House dust mite (HDM) allergen can play an important role in inducing and maintaining AD, either by means of penetration of HDM allergen through a damaged stratum corneum²⁻⁵ or by crossing the respiratory barrier.⁶⁻⁸ The atopy patch test (APT) is a clinical model to demonstrate induction of AD by means of epicutaneous contact with allergens. This model was described by Mitchell et al⁹ for the first time and further developed by Bruijnzeel-Koomen et al.¹⁰ Eczematous lesions can be induced by applying purified HDM allergen over 48 hours,¹⁰ which penetrates the skin and binds to surface-bound antigen-specific IgE on Langerhans cells.^{11,12} These cells presented the allergens to T cells, resulting in sequential activation of initial T_H2-type and late-phase T_H0/T_H1-type cells resembling lesional skin.^{13,14} The route through which it is activated by allergens must be further elucidated, but studies with APTs show that the skin can directly be challenged with HDM, resulting in AD-like reactions, such as erythema, papules, and vesicles.²⁻⁵ On the other hand, a worsening of skin symptoms can also be induced after bronchial inhalation of HDM allergen. One open trial study showed an increase in the Costa score 24 hours after allergen challenge,^{7,8} and a randomized, double-blind, placebo-controlled study showed exacerbation and newly developing AD lesions 1.5 to 17 hours after challenge.⁶

Several studies showed a beneficial effect of HDM avoidance measures on the improvement of atopic diseases. Most studies focused on allergic asthma and less on allergic rhinitis and AD.¹⁵ Roberts¹⁶ and August¹⁷ reported improvement of clinical symptoms in patients with AD after the combination of use of plastic encasings, removal of carpets, and vacuum cleaning. Tan et

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TABLE I. Inclusion and exclusion criteria

Inclusion criteria	Exclusion criteria
1 AD	1 Pets at home and positive skin test response (index ≥ 0.7), RAST result (≥ 0.7), or both for pet allergen
2 HDM RAST ≥ 0.7 , skin test index ≥ 0.7 , or both	2 Pregnant or lactating
3 Der p 1 or Der f 1 concentration of ≥ 200 ng/g dust in the dust sample of the mattress	3 Daily use of oral steroids
4 No encasings or willing to remove them for the period of the study	4 Daily use of cyclosporine

Abbreviations used

AD: Atopic dermatitis
APT: Atopy patch test
HDM: House dust mite
IDT: Intradermal test
LSS: Leicester sign score
VAS: Visual analogue scale

al¹⁸ performed a double-blind controlled trial for a duration of 6 months on a study population of 60 patients with AD in 1993 and 1994. Both placebo and active treatment significantly reduced the Der p 1 concentration after 1 month, and the effect lasted for 6 months. Although both study groups showed significant reductions in the severity and area affected by AD, the change in the AD severity score was significantly higher in the active group than in the placebo group.

In this study we tested the hypothesis that mattress, duvet, and pillow encasings applied for 1 year would decrease the Der p 1 load, resulting in a long-lasting improvement of AD, as measured with the Leicester sign score (LSS).

METHODS

Subjects

Patients with AD (age range, 8–50 years) were recruited from the dermatology, allergology, and pulmonary outpatient clinics at the University Hospital in Utrecht, Rotterdam, and Groningen (the Netherlands), the SAL (a physician laboratory foundation in Utrecht and Groningen), and from members of the Dutch population, who contacted the departments after reading an article about the avoidance project in the media.

Patients who were included had a specific IgE level of at least 0.7 kU/L or greater for Der p 1 or a positive skin prick test response (index ≥ 0.7 ; index is calculated by dividing the mean diameter of the wheal produced by HDM allergen by the mean diameter of the wheal produced by histamine) for Der p 1 and AD, according to the criteria of Hanifin and Rajka.¹ The specific inclusion and exclusion criteria are shown in Table I.

Antihistamines and class 2 topical steroids were allowed between the test periods.

Study design

The study was a randomized, double-blind, placebo-controlled trial of 12 months' duration, with the active treatment consisting of the Goretex bedding system (HAL). The placebo group was given cotton encasings (HAL). The encasings were applied to the mattress, duvet, and pillows on all beds in the patient's bedroom.

The placebo encasings used in this study had an unloaded pore width of 0.1×0.08 mm. Applying a force of 200 N changed the pore width to 0.1×0.1 mm. The allergen barrier of the placebo encasing was 15% compared with the 98% barrier provided by the

active encasings. Patients contacted our clinics at 3 time points: the starting point (T0), after 4 months (T4), and after 12 months (T12).

Dust sampling

Dust samples were collected by a blinded study nurse using a vacuum cleaner (Rowenta RS 005 Dymbo, 1200 W) containing a 20- μ m filter paper in a filter chamber (ALK) over 5 minutes from a 1-m² area. Dust was sampled before treatment, at 4 months, and at 12 months from the mattress and floor of the patient's bedroom and from the living room. Dust samples were weighed and stored at -18°C until extracted. Der p 1 was measured by using a competitive RIA, as described for grass pollen allergen.¹⁹ For this assay, 50 μ L of a dilution of the dust extract was incubated at room temperature with 50 μ L of a 1:2500 dilution of a rabbit anti-*Dermatophagoides pteronyssinus* antiserum. After 2 hours, 1 ng of iodine 125-labeled affinity-purified Der p 1 and 0.5 mg of sepharose-coupled protein A was added. The final reaction volume was 400 μ L. After overnight incubation on a mixer, sepharose-bound radioactivity was measured. The results were compared with an in-house reference calibrated against the World Health Organization reference, assuming that 1 IU equals 0.125 ng. The lower limit of detection of this assay is 0.5 ng/mL.

Outcome measures

The primary outcome was the clinical skin score. The secondary outcomes were the early allergic responses to the intradermal test (IDT) with Der p 1, the APT with Der p 1, and the visual analogue scale (VAS) scores on itching and sleeplessness; total serum IgE levels; specific serum IgE levels against Der p 1; and total blood eosinophil counts.

Clinical assessment

Patients were evaluated at the start of the study and 4 and 12 months thereafter. We used the LSS for AD divided into a severity score and an extent score.^{18,20} The following variables were assessed.

Disease activity. Six clinical features (erythema, purulence, excoriation or crusting, dryness or scaling, cracking or fissuring, and lichenification) were graded at 6 defined body sites on a scale of 0 (none) to 3 (severe). This gives a total body disease activity score, the LSS severity score (maximum, 108).

Extent of disease. Assessment was made according to the rule of nines and resulted in the LSS extent score (maximum, 100%).

Sleep and itching. At each visit, the patients rated their symptoms of itching and loss of sleep during the preceding 2 weeks on a VAS (0–100 mm).^{21–23}

Skin tests

The IDT and skin prick test were performed at the flexor part of the lower arms with standardized HDM extract (30 BU/mL [2.94×10^{-2} μ g/mL Der p 1], 0.03 mL and 10,000 BU/mL [9.8μ g/mL Der p 1], respectively) as stated by the manufacturer (ALK-ABELLO). The early response was scored as the wheal diameter 15 minutes after challenge. The reactions with histamine phosphate solution and phosphate buffer diluent served as positive and negative controls, respectively.

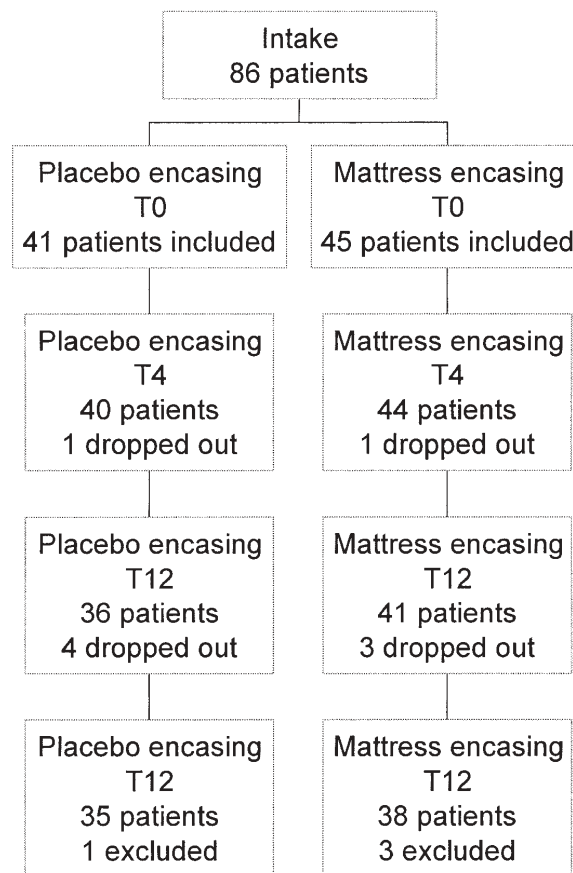


FIG 1. Inclusion, exclusion, and dropping out of patients who were randomly allocated among both groups.

Atopy patch test

The APT was performed as previously described.^{2,10,24} Clinically noninvolved skin of the back was stripped 10 times with adhesive tape, after which Der p 1 (0.1 mL aqueous extract, 10,000 AU/mL [4 µg Der p 1/mL and 15 µg Der p 2/mL], HAL) was applied with Leucotest patches (Beiersdorf AG). APT results were read after 24 hours. APT results were scored as follows: 0, no reaction present; 1+, erythema and induration present; 2+, erythema and papules present; and 3+, erythema, papules, and vesicles present.

Statistical analysis

All statistical tests were performed with SPSS 10.0 software as 2-tailed tests. The normal distribution of the variables was checked with the Kolmogorov-Smirnov test. The Wilcoxon matched-pairs signed-rank test and the paired-samples *t* test were used for analysis within groups, and the Mann-Whitney *U* test and the 2-sample *t* test were used for analysis between groups.

RESULTS

Eighty-six patients with AD were included in the study; 41 were randomly allocated to the placebo group, and 45 were allocated to the active treatment group. Two patients dropped out of the study before 4 months: one moved to another house, and the other found the study too stressful. Six other patients dropped out after 4 months and before 12 months: 2 became pregnant; 1 found the mattress encasing too hot, causing increased sweating and

resulting in an increase of AD symptoms; 1 was regularly using class 3 topical steroids; 1 moved to another house; and 1 had unknown reasons. Seventy-seven patients completed the study. Four patients were excluded because their dust samples were not complete (Fig 1). The general characteristics of the study population are presented in Table II. The patients were characterized on the basis of their allergic symptoms, such as AD, allergic rhinitis, and allergic asthma.

Dust outcome measures in the mattresses

In the active treatment group a significant decrease was seen in Der p 1 and Der p 1 plus Der f 1 concentrations between T0 and T12 and also for Der p 1 plus Der f 1 concentrations between T0 and T4 in the mattresses (Table III). The change in dust weight in the mattresses was not significant. The treatment-induced proportional change for Der p 1 plus Der f 1 concentrations was significantly higher in the active treatment group compared with that in the placebo group but not for Der p 1 alone (Fig 2). The proportional significant change of Der p 1 plus Der f 1 concentrations (log difference between T0 and T12) was chosen as a primary outcome measure for the effect of Goretex encasings, meaning that the difference in Der p 1 plus Der f 1 concentrations between T12 and T0 was significantly lower in the Goretex group compared with that in the placebo group in the between-group analysis.

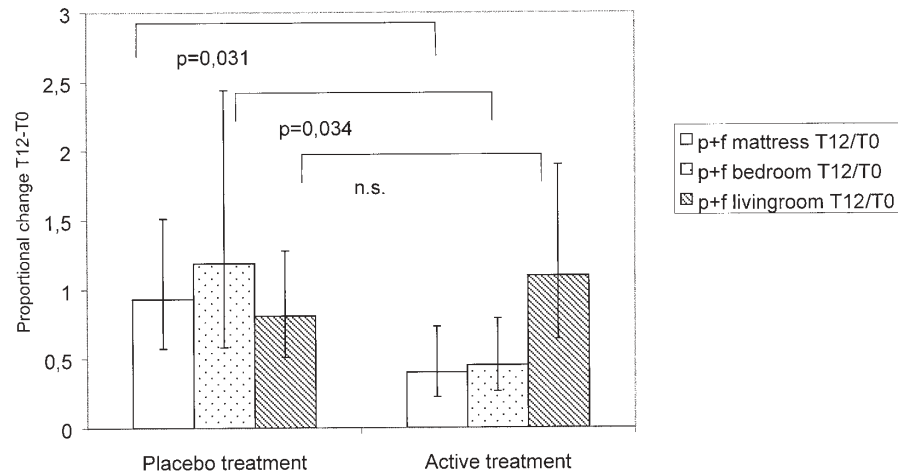


FIG 2. Der p 1 plus Der f 1 (p+f) levels at 12 months in proportion to those at the starting point.

Dust outcome measures in the bedroom and living room

In the active treatment group a significant decrease in Der p 1 and Der p 1 plus Der f 1 concentrations between T0 and T12 was seen in the bedroom. At the starting point there was a significantly higher concentration of Der p 1 plus Der f 1 concentrations in the bedrooms of the placebo group compared with in the bedrooms of the active treatment group (Table III); this was not caused by a difference in floor covering between groups (Table IV). No significant change was seen in the dust weight in the bedroom and living room (data not shown). In the placebo group there was no significant change in Der p 1 and Der p 1 plus Der f 1 concentrations (Table III). The treatment-induced change for Der p 1 plus Der f 1 concentrations was also significant in the bedroom but not in the living room and not for Der p 1 alone (Fig 2).

Primary and secondary outcomes

The LSS severity and extent, VAS scores for itching, and specific anti-Der p 1 IgE levels in the active treatment group at T12 compared with those at T0 were significantly lower, and the anti-Der p 1 IgE levels in the placebo encasing group were, at T12, significantly lower compared with those at the starting point. The VAS scores for sleeplessness, IDT results, APT results, total serum IgE levels, and total blood eosinophil counts did not significantly change. The change in LSS severity and extent and VAS scores for itching were not statistically different between the placebo and active encasing groups, as were the other secondary outcomes (Tables V and VI). Table IV shows the distribution of floor covering within our study group. Surprisingly, 40% of the patients in both groups with HDM allergy and AD have carpet covering in the bedroom, despite advice concerning measures to lower the domestic HDM levels, which most of them must have received in the past.

DISCUSSION

In this randomized, double-blind, placebo-controlled study of patients with AD, the effect of Goretex (n = 38)

TABLE II. General characteristics of the population

	Placebo	Active treatment
Age (y; $\bar{x}$, s)	26.2 $\pm$ 10.7 y	24.2 $\pm$ 10.36 y
AD (n)	2	6
AD + AR (n)	13	14
AD + AA (n)	3	2
AD + AA + AR (n)	17	16
Sex (M/F)	14:21	13:25
LSS severity (median [range])	14 (6-53)	17 (6-60)
LSS extent (%; median [range])	26 (2-87)	19.5 (3-88)
Total patients	35	38

AR, Allergic rhinitis; AA, allergic asthma.

and placebo encasings (n = 35) was studied. The LSS severity and extent score was used as the primary outcome measure. The secondary outcome measures were VAS scores for itching and sleeplessness, IDT and APT results with Der p 1, total serum IgE levels, specific IgE levels against Der p 1, and total blood eosinophil counts. Regarding the primary outcome measures, the decrease of LSS extent and severity at T12 compared with that at the starting point between the placebo and the Goretex encasings was not statistically significant. Goretex encasings did not result in an improvement of AD compared with that seen with placebo encasings. The treatment-induced change of secondary outcome measures between the groups did not improve either. In the scientific literature treatment effects of avoidance measures were either compared with baseline levels or compared between different treatment groups, and this resulted in different significant and nonsignificant outcomes. In our opinion, the differences between the beginning and final outcome variables of the placebo and active treatment groups should be compared. A significant improvement of clinical variables and decrease of HDM allergen levels in the treatment group does not imply that the treatment is causing the effect when the same improvement occurs in the placebo group. The study itself should last at least 12 months to avoid seasonal effects.

In 1996, Tan et al¹⁸ published a double-blind controlled trial including 60 patients with AD. The active HDM allergen avoidance treatment consisted of Goretex-covered mat-

TABLE III. Effect of placebo and active treatment on Der p 1 and Der p 1 plus Der f 1 allergen levels

Dust outcome measures	T0 placebo	T12 placebo	T0 active treatment	T4 active treatment	T12 active treatment
Mattress					
(geometric mean [95% CI])					
Der p 1	841 (494-1432)	679 (339-1360)	945 (555-1609)*	545 (198-1498)	446 (271-735)*
Der p 1 + Der f 1	3388 (1913-6000)	3749 (1921-7315)	4069 (2573-6437)†‡	1675 (938-2989)†	1319 (851-2046)‡
Bedroom					
(geometric mean [95% CI])					
Der p 1	542 (248-1076)	402 (203-798)	1171 (595-2302)§	499 (156-1598)	609 (335-1106)§
Der p 1 + Der f 1	1539 (771-3070)**	2220 (1235-3994)	4498 (2752-7352)¶**	1491 (624-3564)¶	2098 (1269-3469)¶
Living room					
(geometric mean [95% CI])					
Der p 1	979 (339-1360)#	298 (157-570)#	654 (344-1244)	494 (150-1632)	584 (326-1043)
Der p 1 + Der f 1	1114 (600-2067)	966 (509-1831)	1277 (676-2416)	1388 (648-2971)	1308 (735-2327)

Values are given as nanograms per gram of dust.

* $P = .007$, comparing T0 with T12 in the active treatment group (t test).

† $P = .009$, comparing T0 with T4 in the active treatment group (t test).

‡ $P = .005$, comparing T0 with T12 in the active treatment group (t test).

§ $P = .019$, comparing T0 with T12 in the active treatment group (t test).

¶ $P = .011$, comparing T0 with T4 in the active treatment group (t test).

¶ $P = .007$, comparing T0 with T12 in the active treatment group (t test).

$P = .045$, comparing T0 with T12 in the placebo group (t test).

** $P = .011$, comparing T0 between the placebo and active treatment group (t test).

TABLE IV. Distribution of floor covering

	Placebo group (%)	Active treatment group (%)
Bedroom		
Smooth	53	58
Smooth + rug	0	3
Carpet	47	39
Living room		
Smooth	57	50
Smooth + rug	26	21
Carpet	17	29

tresses, benzyltannate spray, and a high-filtration vacuum cleaner used in bedrooms and living rooms for 6 months. The active group showed a drastic decrease in mattress-dust weight with time. Both placebo and active treatment significantly reduced the Der p 1 concentration in bedrooms and living rooms in 6 months. Although both study groups showed significant reductions in the severity and area affected by AD, the change in the AD severity score was significantly higher in the active group ($P = .008$) than in the placebo group. More recently, a new placebo-controlled trial of 12 months' duration was published.²⁵ The authors of the recent study included 40 patients, 11 of whom were unsensitized to HDM. They concluded that in their study the HDM exposure (2 patients in the placebo group and 6 patients in the active treatment group) and the eczema severity (18 patients in the placebo group and 22 patients in the active treatment group) was significantly reduced. Regarding the inclusion criteria and the number of patients used for analysis, the effect of mattress encasings is not as clear as the authors stated. Lack of improvement of AD in a double-blind, placebo-controlled study of HDM avoidance measures with only 10 patients with AD in 2 treatment groups was recently described.²⁶ The low power of this study could still account for the lack of a statistically significant difference between the 2 groups.

Lack of clinical efficacy might also be due to a low baseline level of clinical AD. In comparison with the data of Tan et al,¹⁸ the median score of severity seems almost in the same range (median score, 15-30), although the extent of AD in our study is somewhat less. Tan et al observed a decrease in the extent and severity of AD in control and verum groups. Because their patients were studied for 6 months, this could have been due to seasonal effects. For that reason, in our study patients were followed for 12 months.

Lack of effect in the present study might be due to a low baseline concentration of Der p 1, resulting in little change of Der p 1 after intervention. Some authors reported higher amounts of Der p 1 per gram of fine dust (geometric mean, 20-40 $\mu\text{g/g}$) and stated that it might be the mean level of Der p 1 allergen concentration for temperate climates without any intervention.²⁷⁻³² Others reported lower levels of Der p 1 (geometric mean, 400-10,000 ng/g) in temperate climates.³³⁻³⁷ However, our baseline data on Der p 1 in mattresses (841 and 945 ng/g dust for the placebo and active treatment groups, respectively) have the same mean concentration as earlier reported by Cloosterman et al,³⁸ and therefore this lower concentration of Der p 1 per gram of dust might be representative of the Dutch population. Some high-altitude studies reported lower geometric means for Der p 1 plus Der f 1 concentrations of 360 and 180 ng/g dust for Der p 1 in mattresses.^{39,40} There seems to be some room for improvement left in the active treatment group in this study (the median Der p 1 concentration in the mattress after 12 months' intervention in our study was 446 ng/g dust, and the Der p 1 plus Der f 1 concentration was 1319 ng/g dust), but it is doubtful whether mattress encasings alone can decrease the concentration of Der p 1 plus Der f 1 to this extent.

We tried to isolate the groups that would benefit most and divided the groups into low and high (less than and

TABLE V. Effect of placebo and active treatment on primary and secondary outcome measures

	T0 placebo	T12 placebo	T0 active treatment	T4 active treatment	T12 active treatment
LSS severity	14 (6-53)	15 (0-68)	17 (6-60)*†	10 (0-51)*	13 (0-55)†
LSS extent (%)	26 (2-86)	21 (0-75)	19.5 (3-88)‡	16.5 (0-99)	14.5 (0-87)‡
VAS score for itching (mm)	66.5 (0-100)	60 (0-100)	73.5 (0-100)§	67 (3-100)	63 (4-100)§
VAS score for sleeplessness (mm)	8.5 (0-69)	4 (0-83)	14 (0-100)	13 (0-87)	10 (0-90)
IDT result (mm; $\bar{x}$, s)	14 $\pm$ 6.8	13.5 $\pm$ 6.1	11.6 $\pm$ 6.3	12.3 $\pm$ 7.5	12.7 $\pm$ 7.6
APT result at 24 h	1 (0-3)	1 (0-2)	1 (0-2)	ND	1 (0-2)
Total serum IgE (kU/L)	721 (26-7400)	819 (22-18,400)	559 (63-25,200)	ND	421 (39-20,500)
Specific IgE (anti-Der p 1)	39 (0.3-90)	30.9 (0.2-90)	27.3 (0.2-90)	ND	25.8 (0.2-88.3)
Total blood eosinophils ($10^6/L$)	350 (10-2200)	295 (0.7-1640)	410 (20-1700)	ND	340 (50-1120)

Values are given as median (range) where shown.

ND, Not done.

* $P = .010$, comparing T0 with T4 in the active treatment group (Wilcoxon matched-pairs test).

† $P = .017$, comparing T0 with T12 in the active treatment group (Wilcoxon matched-pairs test).

‡ $P = .038$, comparing T0 with T12 in the active treatment group (Wilcoxon matched-pairs test).

§ $P = .045$, comparing T0 with T12 in the active treatment group (Wilcoxon matched-pairs test).

|| $P = .025$, comparing T0 with T12 in the placebo group (Wilcoxon matched-pairs test).

¶ $P = .013$, comparing T0 with T12 in the active treatment group (Wilcoxon matched-pairs test).

TABLE VI. Difference (T12-T0) of placebo and active treatment on primary and secondary outcome measures

	Placebo encasing group	Active treatment group
LSS severity	-2 (-22 to 28)	-4 (-26 to 29)
LSS extent (%)	-4 (-45 to 33)	-3.5 (-51 to 39)
VAS score for itching (mm)	-8.5 (-89 to 77)	-4 (-90 to 71)
VAS score for sleeplessness (mm)	0 (-56 to 83)	0 (-75 to 64)
IDT result (mm; $\bar{x}$, s)	-0.5 $\pm$ 4.8	0.73 $\pm$ 4.6
APT result at 24 h	0 (-1 to 1)	0 (-2 to 2)
Total serum IgE (kU/L)	-11 (-2200 to 16,330)	-7 (-4700 to 1400)
Specific IgE (anti-Der p 1)	-2.15 (-58.5 to 44.6)	-2.15 (-30.2 to 33.9)
Total blood eosinophils ($10^6/L$)	10 (-560 to 480)	-40 (-660 to 400)

Values are given as median (range) where shown.

*There were no significant treatment-induced changes between the 2 groups.

greater than geometric mean) Der p 1 exposure and low and high (less than and greater than median severity and extent) AD score. No significant difference in effect between these subgroups was seen (data not shown).

Another reason why the decrease in allergen exposure did not result in clinical improvement might be that the sleeping period in which the decrease in concentration of Der p 1 or Der p 1 plus Der f 1 in mattresses and bedrooms is experienced could be too short. Patients might still be exposed to higher HDM levels outside the bedroom. Also, other allergens inside and outside the domestic environment might have aggravated and maintained AD. Reduction of allergens in other environments (work, school, and outdoors) might be equally important to improvement of AD.

The risk of asthma in children sensitized to cats compared with that in children sensitized to mites was much lower in the regions with high mite allergen exposure but similar in regions with relatively low Der p 1 concentrations. Apparently, the presence of mite allergens in homes surpasses the effect of other allergens.⁴¹ The same mechanism might apply to patients with AD. For that reason, the highest treatment effect could be expected from decreasing the concentration of Der p 1.

A meta-analysis of HDM control measures in the management of asthma performed in 1998 led the authors to the conclusion that current chemical and physical methods aimed at reducing exposure to allergens from HDMs seemed to be ineffective and that these methods could not be recommended as prophylactic treatment for asthmatic patients who are sensitive to mites. It was even stated that factors tended to be associated with an overestimation of reported treatment effects.⁴² There are not many studies of patients with AD and the use of mattress encasings, and therefore a meta-analysis concerning the use of mattress encasings in this group is, at present, not possible.

Conclusively, the odds are evened. The randomized, double-blind, placebo-controlled study of Tan et al¹⁸ does show improvement of AD when the active treatment group is compared with the placebo treatment group. Our randomized, double-blind, placebo-controlled study shows no significant improvement of AD between the 2 groups. The usefulness of mattress encasings in reducing allergen load is established. However, relevant clinical benefit is doubtful.

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