

Effect of mattress and pillow encasings on children with asthma and house dust mite allergy

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Background: House dust mite (HDM) allergy is a frequent cause of allergic asthma in children. Reduction of exposure seems to be the most logical way to treat these patients.

Objective: Our aim was to investigate whether mattress and pillow encasings resulted in an effective long-term control of HDM allergen levels, thereby reducing the need for asthma medication in children with asthma and HDM allergy.

Methods: In a prospective, double-blind, placebo-controlled study 60 children (age range, 6-15 years) with asthma and HDM allergy were randomized to active (allergy control) or placebo mattress and pillow encasings. After a 2-week baseline period, follow-up was performed every 3 months for 1 year. During the entire study period, the dose of inhaled steroids was tapered off to the lowest effective dose according to well-defined criteria.

Results: Fifty-two patients completed the trial, and 5 were excluded, leaving data from 47 children (26 in the active treatment group and 21 in the placebo group) for analysis. A significant perennial reduction in HDM allergen concentrations was seen only for the active treatment group. Also, a significant decrease in the dose of inhaled steroids (mean, 408 to 227 µg/d; $P < .001$) was found for the active treatment group only, with significant differences between groups after 9 and 12 months. After 1 year, the dose of inhaled steroids was reduced by at least 50% in significantly more children in the active treatment group than in the placebo group (73% vs 24%, $P < .01$).

Conclusion: Encasing of mattresses and pillows resulted in a significant long-term reduction in HDM allergen concentrations in mattresses and in the need for inhaled steroids in children with asthma and HDM allergy. (*J Allergy Clin Immunol* 2003;111:169-76.)

Key words: Mattress encasing, house dust mite allergy, childhood asthma, inhaled steroids

Abbreviations used

HDM: House dust mite
SPT: Skin prick test

In childhood, asthma is the most common chronic disease, with house dust mite (HDM) allergy being the most frequent cause of allergic asthma.¹⁻³ Previous studies have shown a relationship between the exposure to HDM allergens, especially in mattresses, and the development of HDM allergy.⁴⁻⁶ Moreover, a relationship between the concentration of HDM allergens and asthma symptoms and severity has been shown also.^{4,7,8} Several attempts have been made to reduce the amount of HDMs in mattresses. Polyurethane-covered mattress and bedding encasings are allergen impermeable but are permeable to perspiration. Previous studies have shown a significant reduction in HDM allergen levels in mattresses covered with polyurethane encasings.⁹⁻¹⁷ Also, a significant reduction in bronchial hyperresponsiveness has been shown in patients using polyurethane mattress encasings.^{10,12} However, these studies include small numbers of patients, a short follow-up period, and only a small degree of evaluation of changes in severity of the asthma symptoms and asthma medication.

We therefore aimed at investigating whether polyurethane mattress and pillow encasings resulted in effective long-term control of HDM allergen levels, thereby reducing the need for asthma medication in children with asthma and HDM allergy.

METHODS

Population

A total of 60 children (age range, 5-15 years) with asthma and documented allergy to HDMs from the Departments of Pediatrics at Sønderborg, Kolding, and Viborg Hospitals, Denmark, were included.

The inclusion criteria were all of the following: doctor-diagnosed asthma; a positive skin prick test (SPT) response to HDM (*Der-matophagoides pteronyssinus*); a positive bronchial provocation test result with HDM allergen extract (*D pteronyssinus*; PC₂₀ less than 100,000 SQU); and a total HDM concentration (Der p 1, Der f 1, and Der m 1) of ≥ 2000 ng/g dust from the child's mattress.

Exclusion criteria were previous treatment of the patient, parents, or siblings with mattress encasing; clinical relevant allergies other than HDM allergy (eg, pollen allergy and cat allergy if the child was exposed to a cat at home); previous immunotherapy; and other concomitant diseases or medications that might influence the symptoms.

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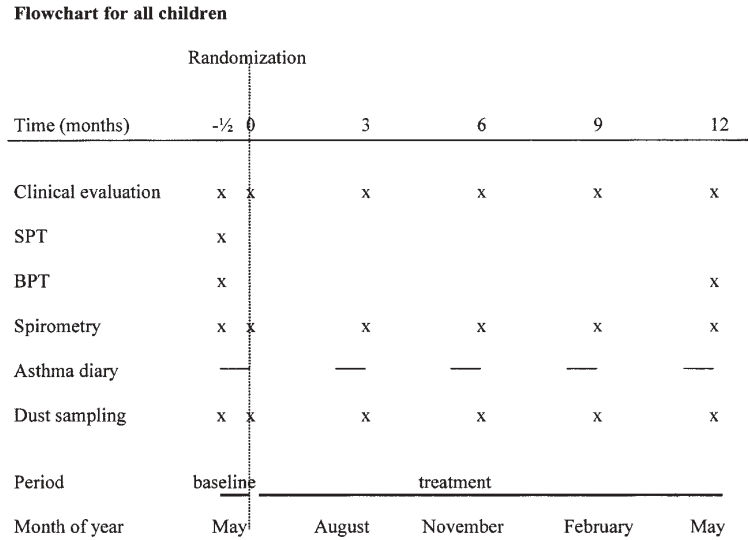


FIG 1. Flowchart. *BPT*, Bronchial provocation test; *asthma diary*, diary with registration of peak flow, symptoms, and asthma medication during a 2-week period.

During the study, the children were excluded if they changed the bed or mattress, moved to another room or residence, or did not take the medication as prescribed.

Design

The study is a prospective, double-blind, placebo-controlled, multicenter study with a 1-year follow-up period (Fig 1). After inclusion, the children were randomized to either an active treatment group ($n = 30$) that was provided mattress and pillow encasings coated with semipermeable polyurethane (Allergy Control) or a control group ($n = 30$) that received a placebo mattress and pillow covers. The placebo encasings were made especially for this purpose of a good quality of cotton to resemble the active treatment covers as much as possible.

The randomization was performed blockwise with 10 patients in each group by using a computer program after stratification according to age (2 groups: <10 years or ≥ 10 years of age), sex, initial HDM concentrations (2 groups: $<10,000$ ng/g or $\geq 10,000$ ng/g dust), and center. The Danish Allergy Association performed the randomization, and the code was not broken until all data were registered. All encasings were delivered directly from the manufacturer to all patients during May 1994 to avoid seasonal differences between the groups.

During the study, the encasings were to remain unwashed if possible, and changes in the child's mattress, bed, and bedroom, as well as changes in residence, including type of ventilation, were not allowed. It was recommended to wash the pillows and the blankets-duvets every 3 months after dust sampling and the sheets and pillow cases every 2 weeks.

A clinical evaluation with lung function measurement and adjustment of the pharmacologic treatment, as well as dust sampling from the child's mattress, were performed at inclusion, after baseline, and every 3 months during the 12-month treatment period. Symptom scores and peak flows were registered in an asthma diary for 2 weeks during the baseline period and before every follow-up visit during the treatment period. Bronchial provocation testing with HDM (*D pteronyssinus*) allergen extract was performed at inclusion and after the treatment period.

At inclusion and during the baseline and treatment periods, asthma medication was titrated to the lowest effective dose of inhaled

steroids and β_2 -agonists according to registration of symptoms, need for β_2 -agonists, and peak flow recorded in diaries, as well as lung function measurements determined by means of spirometry (FEV_1). At normal lung function, peak flow variability of less than 20%, and need for rescue medication less than twice per week, the dose of inhaled steroids was reduced to maintain control of symptoms, lung function, and use of rescue medication at the lowest effective dose.

The primary effect measures were changes in exposure to HDM allergens and the need for asthma medication, especially inhaled steroids. Secondary effect measures were changes in symptoms, peak flow and FEV_1 , and allergen-specific bronchial hyperresponsiveness, as measured by means of bronchial allergen challenge with HDM extract.

Dust samples were collected by means of vacuum cleaning the child's mattress with an ALK collection device for 5 minutes. The families' own vacuum cleaner was used, and the same cleaner was used for each child during the study period. Group 1 HDM allergens (Der p 1, Der f 1, and Der m 1) were measured by using species-specific ELISAs (environmental kit, ALK-Abelló A/S).

SPTs were performed with Soluprick SQ (ALK). The results were given as the mean wheal diameters, from which the mean diameters of the negative reference were subtracted. An SPT result of 3 mm or larger was regarded as positive.

Bronchial provocation tests were performed through use of a reservoir method described by Matthys et al.¹⁸ This method involves the use of a high-quality nebulizer system (Pari Provacationest I) combined with a 10-L storage bag and allows for standardized pulmonary aerosol deposition at saturated ambient temperature and pressure conditions. The HDM extract used for the test was Aquagen SQ *Dermatophagoides pteronyssinus* (ALK, Denmark). First, a 0.9 mol/L NaCl solution was nebulized and inhaled, and then test solutions with 10, 32, 100, 320, 1000, 3200, 10,000, 32,000 and 100,000 SQU of HDM extract was nebulized and inhaled. FEV_1 was measured 3 times before exposure and immediately and 10 minutes after each inhalation; the highest value was recorded. The tests were stopped either after the highest concentration (100,000 SQU) or at the concentration causing $\geq 20\%$ decrease in FEV_1 in relation to baseline values. The PC_{20} was estimated by means of linear interpolation of the 2 last (log-transformed) concentrations tested. With this system (which is a closed system),

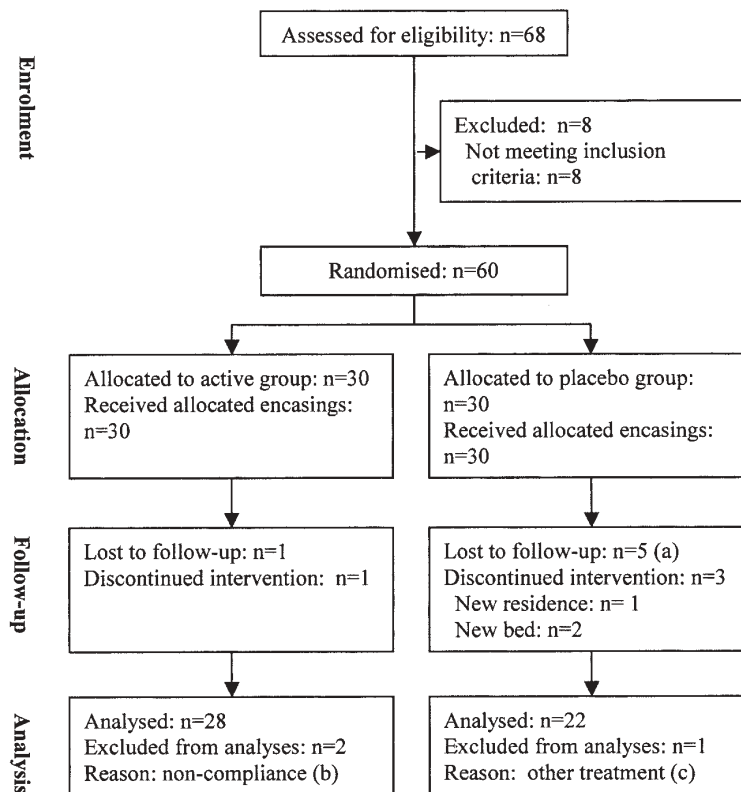


FIG 2. Flow diagram of the progress through the study. (a) Three subjects moved to another region, 2 before they received the encasings; (b) insufficient data concerning asthma medication and lack of dust samples; (c) concomitant treatment with unallowed medication (growth hormone).

PC₂₀ values equaled PD₂₀ values. The test result was positive if PC₂₀ was ≤100,000 SQU.

In each center the same device for measuring FEV₁ was used on all occasions.

Diaries for recording morning and evening peak expiratory flow symptoms (symptom scores of 0-3, with 0 being no symptoms and 3 the worst case) during night and day and the number of doses of β₂-agonist used on demand were recorded for 2 weeks before the visits occurring every 3 months.

Statistical analyses

A realistic sample size calculation was not possible because of the lack of relevant data on which to base a formal calculation. By using the existing literature with raw data on the effect of mattress encasing on HDM exposure, it was calculated that a case group of 23 patients would give a 99% chance to detect a difference in exposure to HDMs of 98% after 12 weeks.⁹ Because our follow-up period was 1 year and our primary effect measure was reduction in medication, we considered that inclusion of 60 patients, 30 in each group, which allowed for a dropout rate of 20%, was sufficient.

Statistical analysis was performed with SPSS 10.0 software. The distribution of HDM allergen concentrations and PC₂₀ values was normalized by using logarithmic transformation and univariate analysis of variance (ANOVA), and the Student *t* test of logarithmically transformed values was used for comparisons. Nonparametric analysis was used for comparison of change in doses of inhaled steroids. For comparison of bronchial hyperresponsiveness, a paired *t* test on logarithmically transformed values was used.

Ethics

The study was carried out in accordance with the Declaration of Helsinki in its most recent version and good clinical practice. The study was approved by the Human Research Ethics Committee. All the parents were given oral and written information, and the parents provided informed consent before entering the study. Patients could be withdrawn or withdraw themselves from the study at any time.

RESULTS

A total of 60 patients were included and randomized, and as seen in Fig 2, data from 47 children, 26 actively treated patients and 21 control subjects, were included in the final analysis.

A total of 4 children in the active group and 3 children in the control group had box mattresses with a partial encasing, whereas all others had encasings covering the entire mattress and sealed with a zipper at one side.

In most cases (*n* = 27), the blankets-duvets and pillows were not washed at all during the study period. In the active group 8 of 26 and in the placebo group 12 of 21 families washed their blankets-duvets at least once during the treatment period. Only 2 encasings, both in the placebo group, were washed during the study.

The baseline dust samples were obtained 3 to 7 months before the start of the treatment period in 11 children (8 in

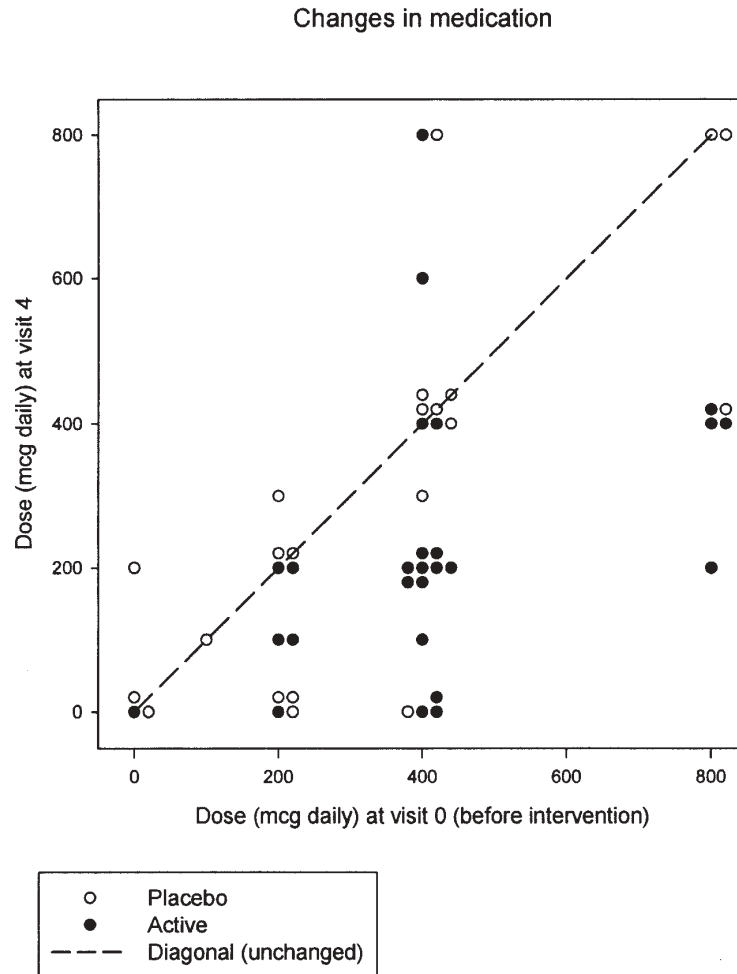


FIG 3. Change in need for inhaled steroids. Daily dose of inhaled steroid (in micrograms) by the end of the study (y-axis) as a function of initial dose (x-axis) in children in the active treatment (*filled circles*) and placebo (*open circles*) groups is shown. Results are expressed as individual data points.

the active treatment group and 3 in the placebo group). All other baseline measurements were performed during the last 2 weeks before the treatment period.

Exposure to HDM allergens

A great variation in mite concentrations was seen in both groups (Table I). By means of univariate ANOVA, including group and time (visits), a significant difference between the active treatment and placebo groups ($P = .014$), within groups during follow-up (between visits, $P < .001$), and between groups at different visits ($P = .038$) was found. As seen in Table I, repeated t tests of logarithmically transformed values showed a significant decrease in the concentrations of total HDM allergens in mattresses in the active group but not in the placebo group. The difference between groups was significant after 6 and 12 months. In the placebo group the highest HDM allergen concentrations were registered in November (ie, early winter).

Compared with baseline values, the median reduction in total HDM concentration remained stable in the active

treatment group between 81% and 89% in contrast to more varying reductions of between -1% and 70% in the placebo group.

No significant difference was seen in the mean amount of dust obtained between the active treatment and placebo groups, and mean values did not change during the study.

Medication

At inclusion, all but 4 of the children (1 in the active treatment group and 3 in the placebo group) were treated with inhaled steroids. Children treated with inhaled steroids used the same product (budesonide or fluticasone) during the entire study period. All children used short-acting β_2 -agonists as needed during the study period.

Fig 3 shows the relationship between the daily dose of inhaled steroids at the start and end of the treatment period. The daily dose of inhaled steroids was reduced by at least 100 $\mu\text{g}/\text{d}$ in most of the children (73% [19/26]) in the active treatment group, whereas this was the case for few children (29% [6/21]) in the placebo group. After 9 and 12 months, the dose of inhaled steroids was reduced

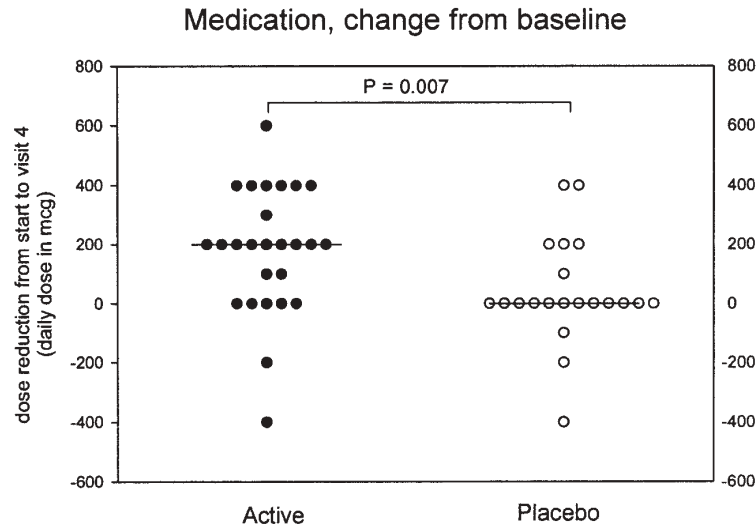


FIG 4. Change in need for inhaled steroids. Change from baseline in daily dose of inhaled steroid (in micrograms) by the end of the study in the active treatment (filled circles) and placebo (open circles) groups is shown. Results are expressed as individual data points, and *P* values for differences between medians are calculated by means of the Kruskal-Wallis test.

TABLE I. Values of total HDM (Der p 1, Der f 1, and Der m 1) concentrations in mattresses according to time and *P* values for differences compared with baseline and *P* values for differences between the active treatment and placebo groups

Time (mo)	Active treatment group (n = 26)			Placebo group (n = 21)			Active treatment vs placebo groups
	GM, ng/g dust	95% CI, ng/g dust	<i>P</i> value*	GM, ng/g dust	95% CI, ng/g dust	<i>P</i> value*	<i>P</i> value*
Baseline	15,604	9074-26,834		8791	3903-19,802		.251
3	2996	1702-5272	<.001	3307	1837-5953	.055	.818
6	3046	1788-5189	<.001	9923	5102-19,300	.820	.011
9	2636	1413-4919	<.001	5653	3357-9518	.204	.093
12	1456	787-2692	<.001	4311	2095-8870	.163	.032

GM, Geometric mean.

**t* Test of logarithmically transformed values.

by at least 50% in significantly more children in the active treatment group than in the placebo group, with the figures being 54% (14/26) versus 10% (2/21) after 9 months (*P* < .05) and 73% (19/26) versus 24% (5/21, *P* < .001) after 12 months. As seen in Fig 4, median change from baseline in the daily dose of inhaled steroids was 200 µg/d and 0 µg/d in the placebo group, with the difference being significant (*P* < .01).

Changes in the need for inhaled steroid according to time are shown in Table II. Only a few dosages (0, 100, 200, 300, 400, 600, and 800 µg/d) were used, and changes were performed stepwise between these dosages. Therefore median and range values provide little information, and we chose to present the data both as the median and mean of the daily dose of inhaled steroid. After the first 3 months, a successive change, mostly a reduction, in the dose of inhaled steroids occurred in the active treatment group, whereas only a few children in the placebo group succeeded in reducing the dose. The dose of inhaled steroids was reduced significantly in the

active treatment group only and significantly more in the active treatment group compared with in the placebo group. The decrease in the dose of inhaled steroids became significant after 9 and 12 months. The same was the case when considering only children with a need of at least 400 µg/d at baseline (data not shown).

None of the children were treated with systemic steroids during the study period.

There was a nonsignificant trend toward less use of β₂-agonist during the follow-up period, and no significant difference between the 2 groups was observed. The mean number of doses of β₂-agonist used during 2 weeks in the baseline period was 13 and 16 in the active treatment and placebo groups, respectively, whereas the corresponding figures by the end of the study were 5 and 9 doses.

Allergen-specific bronchial hyperresponsiveness and lung function

All provocations were performed in the summer. Table III shows a significant increase of PC₂₀ in both groups

TABLE II. Values of doses of inhaled steroids with 95% CI according to time and *P* values for reduction in dose compared with baseline within and between the active treatment and placebo groups

Time (mo)	Active treatment group (n = 26)				Placebo group (n = 21)				Dose reduction, active treatment vs placebo groups
	Median, μg^*	Mean, μg	95% CI, μg	Wilcoxon <i>P</i> value [†]	Median, μg^*	Mean, μg	95% CI, μg	Wilcoxon <i>P</i> value [†]	<i>P</i> value [‡]
Baseline	400	408	333-483	NA	400	329	227-431	NA	NS
3	400	396	323-369	.180	400	329	227-431	1.000	.199
6	400	358	280-436	.203	400	352	251-453	.457	.094
9	200	281	214-348	.011	400	333	216-450	.857	.031
12	200	227	154-300	.001	300	290	179-401	.307	.007

NA, Not applicable; NS, not significant.

*Range of 0 to 800 for all visits in both groups.

[†]The Friedman test was used for comparison within groups: active treatment group, $P < .001$; placebo group, $P = .257$. Secondly, the Wilcoxon test was used to compare baseline values with values at the 4 visits.

[‡]The Kruskal-Wallis test was used for comparisons between dose reductions in the 2 groups.

that was most pronounced in the active treatment group but without a significant difference between the groups.

In both the active treatment and the control group, morning and evening peak expiratory flow increased significantly from baseline to visits 3 and 4 after 9 and 12 months ($P < .01$), and FEV₁ increased significantly during the entire study period ($P < .01$ at every visit). No differences between the groups were registered.

Symptoms

No significant differences were registered in asthma symptom scores by day or night either over time or between the groups at any time. In the 2-week baseline period, the mean total daytime asthma symptom scores were 1.62 and 3.33, and the corresponding nighttime asthma symptom scores were 0.46 and 1.48 in the active treatment and placebo groups, respectively. After 12 months, the corresponding figures for daytime asthma scores were 1.73 and 2.57 and nighttime asthma symptom scores were 1.08 and 1.90 in the active treatment and placebo groups, respectively. The maximum score for daytime or nighttime symptoms was 3. No difference in exercise asthma symptoms was registered.

DISCUSSION

HDM allergy is the most common cause of allergic asthma in childhood. The relationship between exposure and asthma symptoms in sensitized individuals is complex, with some patients reacting to very low doses of allergen, whereas other patients tolerate rather high doses.^{4,19} Meanwhile, exposure to higher allergen levels usually is associated with more severe asthma in sensitized patients. Avoidance of HDM allergen exposure seems to be the most logical way to treat children with asthma and HDM allergy.

Many different avoidance measures for HDM allergens have been tested, but only a few have been subjected to controlled clinical trials.^{20,21} In most of the studies on HDM avoidance, a significant decrease in allergen exposure has not been achieved, the patients did not have documented HDM allergy, many had other clinically significant allergies, the study did not take into account the pharmacologic treatment, and the follow-up period was short. Despite this, some studies indicated an effect of HDM allergen-avoidance measures in children with asthma and HDM allergy.^{10,12,14}

In a recent meta-analysis²² it was concluded that methods aimed at reducing exposure to allergens from HDM seem to be ineffective and could not be recommended as prophylactic measures for asthmatic patients sensitive to mites. Meanwhile, this analysis included several studies with methods that were most unlikely to reduce mite allergen exposure. A reduction in HDM concentration was documented in only 6 of 22 studies included in the analyses, and the analyses did not include the most effective outcome variables.

In our prospective, double-blind, placebo-controlled randomized study, we only included children with doctor-diagnosed asthma and HDM allergy documented by means of bronchial challenge. Children with other clinically significant allergies were not included, and other mite-reducing measures were not allowed during the study period. In contrast to previous studies, the clinical efficacy was measured on the basis of medication-sparing effect, especially steroid-sparing effect, under the control of symptoms and lung function. Thus inhaled steroids were tapered off to the lowest effective dose before and during the study to avoid the possibility that pharmacologic overtreatment would mask a clinical effect of the avoidance measures. Our primary effect measures were the long-term effects of mattress encasings on HDM aller-

TABLE III. PC₂₀ values according to time and *P* values for differences compared with baseline

Time (mo)	Active treatment group (n = 25)			Placebo group (n = 21)			Active treatment vs placebo groups
	GM, SQU/mL	95% CI, SQU/mL	<i>P</i> value*	GM, SQU/mL	95% CI, SQU/mL	<i>P</i> value*	<i>P</i> value†
Baseline	1643	784-3447	—	2507	1090-5765	—	NS
12	6906	3628-13,146	.001	7813	4091-14,921	.023	NS

GM, Geometric mean; NS, not significant.

*Paired *t* test.

†Nonpaired *t* test.

gen exposure and the lowest effective dose of pharmacologic treatment, especially inhaled steroids. Because the children all were well treated at inclusion, we did not expect any effect on symptoms, lung function, and allergen-specific bronchial hyperresponsiveness.

The possible influence of seasonal variation of HDM concentrations was taken into account by a 1-year study with quarterly measurements of HDM allergens on mattresses.

The motivation of the children and their parents was high, and the compliance was good, except that they did not wash the blankets-duvets. The follow-up rate was high, and despite the fact that compliance is a very common problem in the treatment of asthma, only a few children were excluded for that reason.

We found a significant and stable perennial reduction of the concentration of HDM allergens in the active treatment group in contrast to the placebo group, where a possible seasonal variation was observed with a high level of HDM allergens during the winter period. Even in the active treatment group the variation in concentrations of HDM allergens was high. In the active treatment group one child with a partial encasing had no change in mite exposure, and in another a decrease from inclusion to baseline and a subsequent increase during the first 3 months of treatment was seen. Apart from these 2 patients, the HDM allergen concentration decreased by at least 89% in the active group, whereas it was 70% in the placebo group.

In our study many factors might contribute to the variation in HDM concentrations. The blankets-duvets were not washed as recommended, therefore creating a reservoir of HDM allergens, and although standardized as much as possible, the parents did the dust sampling with their own vacuum cleaners, which might influence the result. Also, the increased focus on the disease and HDM allergy also might have changed the cleaning habits of the families in both groups. The placebo encasings were of a very good quality of cotton and might also have had an effect as a barrier against HDM allergens.

We found a significant effect of the coated polyurethane mattress and pillow encasings on the need for inhaled steroids. Thus the dose of inhaled steroids was reduced by approximately 50% in the active treatment group, without worsening in symptoms, lung function, or need for rescue medication. The effect as regards reduction in the need for inhaled steroids was not significant until after 6 months, but we cannot exclude a delay caused by anxiety of the children and their parents, who

were afraid of symptom worsening. Despite a reduction in dose of inhaled steroids in the active treatment group, no significant difference between the groups in allergen-specific bronchial hyperresponsiveness was found. A significant increase in peak flow and lung function, as measured with FEV₁, was found in both groups, probably because of growth of the children and better compliance and treatment as a result of more focus on their treatment. As expected, no differences in lung function and symptoms were found between the 2 groups.

The dropouts were due to well-described reasons, but if dropouts were in part caused by insufficient symptom control, their distribution seems to strengthen our findings of a positive effect of mattress encasing. Most of the children without need for inhaled steroids were from the control group, but in all children the dose of inhaled steroids was titrated to the lowest effective dose both during the baseline and treatment periods, and we found the same steroid-sparing effect when the analyses were restricted to the children with a documented need for at least 400 µg/d inhaled steroids at inclusion. Thus it is not likely that the difference between the active treatment and placebo groups was due to skewness or overtreatment at inclusion in the active treatment group.

In conclusion, we found a significant and clinically relevant long-term effect of semipermeable polyurethane mattress and pillow encasings in children with documented asthma and HDM allergy, resulting in a reduced exposure to HDM allergens and a reduced need for inhaled steroids.

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