

Mite elimination – clinical effect on eczema

Friedmann PS, Tan BB. Mite elimination – clinical effect on eczema. Allergy 1998; 53 (Suppl 48): 97–100. © Munksgaard 1998.

Allergic reactivity to house-dust mites (HDM) can be detected in patients with atopic eczema by prick and patch test challenge. To determine the clinical relevance of this reactivity, we performed a placebo-controlled, double-blind trial of anti-HDM measures. Active treatment comprised Gortex bags for all the bedding elements, a high-powered vacuum cleaner, and a spray containing benzyl alcohol and tannic acid to kill mites and denature allergens. Placebo treatment was light cotton bags, a cheap vacuum cleaner, and water spray. Forty-eight patients (28 active group) completed the trial, which lasted 6 months. Dust was sampled from the mattress surface and bedroom and living-room carpets before and at monthly intervals after institution of the measures. Dust was weighed and Der p 1 determined by ELISA (ALK). Patients were assessed for area and severity of eczema by a blinded observer. There was a highly significant reduction in bed surface dust – most beds yielded insufficient dust to extract and assay. Carpet Der p 1 levels were reduced to similar minimal levels by both active and placebo treatments (about 250 ng/m²). There were highly significant benefits on the eczema scores, the active treatment being greatly superior to placebo ($P < 0.0006$; analysis of covariance). In conclusion, Gortex bed bags were highly effective at containing dust within the bed. This was associated with clinical improvement in most patients with atopic eczema – the biggest improvements were seen in the most severely affected subjects. (The results of this study have been published [*Lancet* 1996;347:15–18].)

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Introduction

The atopic state is a genetically determined predisposition to develop allergic reactivity to environmental allergens. This susceptibility appears to be inherited in dominant fashion, and there is a 70% chance of children being atopic when both parents are atopic (1).

One of the main defining characteristics of atopic individuals is the capacity to form IgE-class antibodies in response to antigens that enter through mucosal routes (2). Immune dysregulation that favors IgE production appears to depend upon preferential activation of allergen-specific CD4⁺ helper T cells of the Th2 phenotype rather than of the Th1 type (3, 4).

Histologic examination of eczematous skin reveals an inflammatory infiltrate of mononuclear cells including CD4⁺ T cells similar to that seen in classical contact dermatitis. The questions that arise are, firstly, what is the nature of the allergic hypersensitivities in atopic eczema; secondly, what may be the provoking antigens; and, thirdly, are

these reactions of pathogenetic significance in eczema?

The nature of the allergic hypersensitivity in atopic dermatitis (AD)

Immediate-hypersensitivity reactions can be demonstrated in most subjects with atopic eczema, both on prick test challenge and also after intradermal inoculation of antigens. However, attempts to implicate immediate-hypersensitivity mechanisms in the eczema have been unsuccessful. Several studies have examined the presence of delayed-type hypersensitivity to a variety of antigens, including house-dust mites (HDM). Intradermal challenge elicits 48-h delayed-type responses, while epicutaneous patch tests elicit an eczematous response in a high proportion of subjects with atopic dermatitis (AD) (5–9). We have also performed such patch test challenges in subjects with AD: 26 adults and 24 children (mean age 8.9 years, range 5–13) with AD were studied when the eczema was quiescent. Thirteen non-

Table 1. Responses to challenge in patients with atopic eczema

Prick	Patch	Antigen			
		<i>D. pteronyssinus</i>	<i>D. farinae</i>	Cat	Grass
+	+	31/50	10/33	18/48	18/39
-	+	4/50	1/33	6/48	0/39
+	-	12/50	14/33	15/48	15/39
-	-	5/50	8/33	9/48	6/39

No positive responses were obtained in 13 nonatopic, healthy controls.

atopic healthy adults served as controls. Epicutaneous patch tests were applied in duplicate to the back on sites stripped with adhesive tape either seven or 15 times. Antigens included HDM (Aquagen, ALK) 2500 and 625 biologic units, and commercially available prick test solutions of *Dermatophagoides farinae*, cat, and mixed grasses (Bencard). Patches were applied for 48 h and read as for standard contact patch tests. Eczematous responses were obtained with at least one antigen in most atopic subjects (Table 1), but none were observed in nonatopic controls. Responses to HDM were dose related in terms of both number of tape strips and antigen concentration. Positive patch test responses to HDM appeared in 70%. There was a strong correlation between the presence of positive patch tests and positive immediate prick tests, but they were dissociable. Thus, occasional subjects gave positive patch tests but negative prick tests, and 25% gave positive prick tests and negative patch tests (Table 1).

The important question is whether such allergic reactivity causes the production of the eczema. HDM are found in all homes and there is evidence that homes of patients with AD have higher levels than normal (10). Many workers have attempted to eradicate HDM and/or other antigens by the use of chemical acaricides which either kill mites or denature their antigens, barrier bedding systems, vacuum cleaning, and ventilation. A number of workers have examined clinical consequences of anti-HDM measures in asthma (11–13), but none of these studies monitored the effects on HDM-antigen (Der p 1) levels. Other workers looked at the effects of acaricides on mite-antigen load in isolation from the clinical setting (14, 15). The question of whether anti-HDM measures can benefit AD has received little attention and is the subject of conjecture and uncertainty. We have tested the hypothesis that the activity of AD is related to the domestic Der p 1 load; hence, a reduction in Der p 1 should result in relative improvement in AD. We examined a combination of anti-dust-mite measures that would be easily applicable in normal homes for effects on both Der

p 1 load and the clinical activity of AD, in a double-blind, placebo-controlled trial (16).

Patients with AD between the ages of 7 and 65 years were recruited. All patients gave at least one positive immediate response to prick test challenge with a range of aeroallergens (Bencard, UK). Pets were strictly forbidden.

Study design

Active treatment comprised the Goretex polyurethane bedding system (Intervent[®], WL Gore), benzyltannate complex spray (Allersearch DMS, Allersearch, Granville, NSW, Australia) for the carpets, and a high-filtration vacuum cleaner (Nilfisk, BSE, Suffolk, UK). The placebo group received light cotton bedcovers supplied by WL Gore. Water with a trace of alcohol was sprayed on the carpets in the same fashion as the active spray. A standard upright vacuum cleaner (Electrolux) with a poor filtration performance was used as the control cleaner. To standardize the treatment, a trained nurse applied the bedding system and performed the spraying in all households. The mattress, duvet, and pillows on all the beds in the patient's bedroom were covered. The bedroom and living-room carpets were sprayed at a rate of 50 ml/m² once every 3 months. The patients were instructed to vacuum the treated carpets daily and the rest of the house 2–3 times a week. Soft furnishing and toys were minimized in the bedrooms. The treatment period was 6 months long.

Dust sampling. Dust was sampled with a hand-held vacuum cleaner for 2 min from a 1 m² area, from the carpet by the head of the bed and in front of the settee in the living room, and the whole mattress surface, before treatment and at monthly intervals.

Dust analysis. Dust samples were weighed and extracted, and Der p 1 concentration was determined with a commercially available ELISA kit (ALK, Denmark).

Clinical assessment. One physician, who was "blinded" to the treatment groups, assessed all patients throughout the study. The surface area and severity of dermatitis were measured.

Results

Sixty patients (30 adults and 30 children) were randomly allocated to active or placebo treatment groups. Forty-eight patients (24 adults, 24 children)

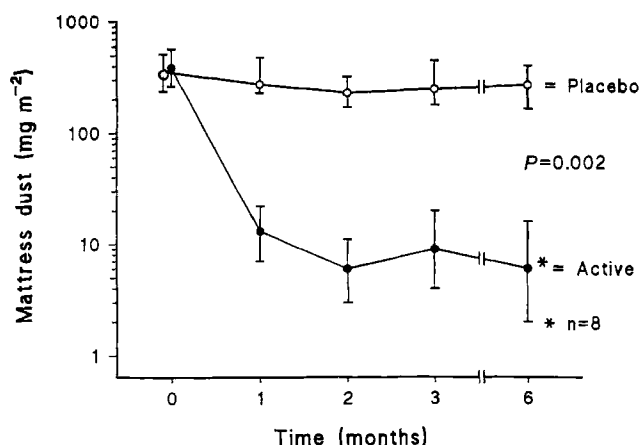


Fig. 1. Change in dust quantities (weight) on outside of bed bags after application of either Goretex or placebo bed encasements. *Not all beds were sampled, because at 3 months, sufficient dust for extraction and attempted assay of Der p 1 was found in only eight beds. Reproduced with permission from Tan et al. (16).

completed the 6 months; 28 patients in the active group and 20 in the placebo group.

Change in Der p 1 levels

Mattress dust. By 1 month, the dust load recovered from mattresses with Goretex covers had fallen by 98%, after which there was no further reduction; geometric mean at 3 months was 9 mg/m² (95% CI, 4–20 mg/m²) (Fig. 1). Because only 8/28 beds contained sufficient dust to extract and assay for Der p 1, dust weights are given; after 3 months, sampling was not performed on all beds. The placebo covers caused a small but significant reduction in the dust load (Fig. 1) which did not change significantly after 2 months.

Carpet dust. In all homes, the concentration of Der p 1 was higher in bedroom carpets than in living-room carpets (Table 2). In the bedroom carpets, Der p 1 concentration fell by 77% in the active group compared to 32% in the placebo group ($P=0.07$, unpaired t -test). In living-room carpets,

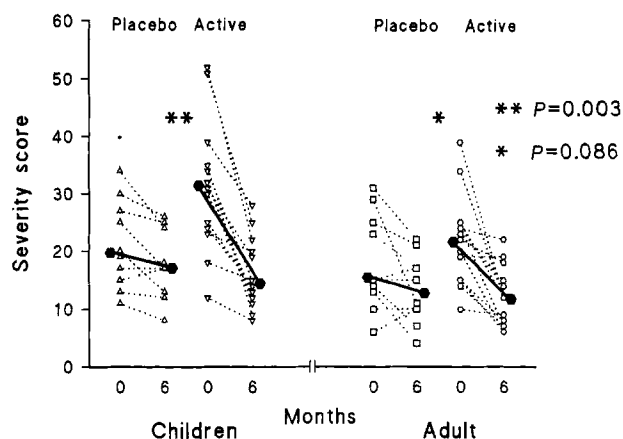


Fig. 2. Change in severity of atopic eczema after institution of anti-HDM measures. Adults and children are presented separately. Scores before and after 6 months for each individual are connected by dotted lines. Median scores for groups are connected by solid lines.

the Der p 1 levels fell by 52% and 16% in the active and placebo groups, respectively ($P=0.55$, unpaired t -test). However, in active- and placebo-treated carpets, the final Der p 1 concentration was about the same, confirming that total eradication of Der p 1 from carpets is virtually impossible.

Effects on atopic dermatitis

Both active and placebo groups showed significant improvements in the severity and area affected by eczema (Fig. 2). The group receiving active treatment showed a mean improvement in severity score of 12.6 units, while the placebo group improved by a mean of 4.2 units; the difference between the means was significant ($P=0.006$; analysis of covariance; Fig. 2). The area affected by eczema also improved in both groups, the mean difference in area affected was 10% (CI 3–17; $P=0.006$). To determine whether children and adults differed in their responses to dust-mite eradication measures, they were analysed separately, and children showed significantly greater clinical improvement (Fig. 2).

There were a number of important findings relating to the efficacy of these measures. Firstly, the Goretex bedding bags caused a major reduction in the quantities (weights) of dust in beds – and we could not retrieve sufficient dust (>50 mg) to determine the level of Der p 1 in 68% of beds. This reduction was maximal at 1 month and did not change with time. Treatment of bedroom and living-room carpets with the combination of a spray with acaricidal and denaturing properties and a high filtration vacuum cleaner reduced Der p 1 levels significantly. The placebo treatment – a standard vacuum cleaner with a poor performance in tests

Table 2. Effect of anti-dust mite measures on Der p 1 levels in carpets

	Der p 1 concentration ng/m ² Bedroom carpet	Geometric mean (range) Living-room carpet
Active treatment		
At baseline	3669 (16–42 014)	1321 (0–30 130)
At 6 months	249 (0–15 625)	271 (0–4270)
Placebo treatment		
At baseline	2161 (32–36 756)	913 (54–22 391)
At 6 months	225 (0–18 063)	271 (7–6726)

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reported by the Consumer Association of Britain – had a similar effect! From these data, it is not possible to draw conclusions regarding the relative contributions of the spray and the high-filtration vacuum cleaner. In any event, the final Der p 1 levels in the carpets, bedroom, and living room of both treatment groups were similar (Table 2), suggesting that more complete elimination of Der p 1 from carpets is virtually impossible. This may be particularly so in the presence of upholstered furniture, which forms an additional reservoir for HDM.

Overall, the patients receiving active treatment showed a highly significant clinical improvement compared with those in the placebo group. In some individuals, the amelioration was dramatic with a major improvement in life quality. It appeared that the greatest benefits were seen in children, but a formal study would be required to test for real differences between responses of adults and children (Such a study has been reported by Tan et al. [16].)

The hypothesis under test was that the clinical benefit derived from anti-HDM measures should be related to the reduction in Der p 1 concentration in some parts of the domestic environment. Indeed, the analysis of covariance suggested that a significant component of the treatment effect was related to the changes in dust and Der p 1 in mattresses and bedroom carpets. However, there were no simple correlations between either initial dust and eczema severity or final dust and eczema severity.

In conclusion, effective avoidance of contact with Der p 1 in beds is achievable with Goretex bedcovers, but there do not appear to be great differences between the effects of high-filtration and “normal” vacuum cleaners. Anti-dust-mite measures were associated with highly significant improvements in eczema severity. However, the heterogeneity of responses clearly indicates that for some individuals, other factors are important. Hence, methods are needed to identify those patients who will benefit most from these measures and to identify what the provoking factors are for those who do not.

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