

Changes in mite allergen *Der p I* in house dust following spraying with a tannic acid/acaricide solution

E. R. TOVEY, G. B. MARKS, M. MATTHEWS, W. F. GREEN and ANN WOOLCOCK

Department of Medicine, University of Sydney, DO6, Sydney, NSW 2006, Australia

Summary

The beds, carpets and furnishings in 15 houses were sprayed with a solution containing tannic acid and an acaricide in an attempt to reduce allergen concentrations. Dust was collected from these sites for 4 weeks following spraying and the mite allergen *Der p I* concentration was measured and compared with baseline concentrations. In a subgroup of houses, counts of live mites and estimates of aeroallergen were also made. Four untreated houses were monitored over the same period. In dust samples collected 3 days after spraying, the mean concentrations of *Der p I* in beds, carpets and furniture were 23%, 28% and 26% of the pretreatment levels. All these reductions were significant compared to untreated controls. Samples collected 4 weeks after treatment were not significantly different to baseline for each group. After the initial reduction, the rate of increase in allergen concentration was significantly greater in the spray-only beds than in the beds which had been both sprayed and fitted with occlusive covers. Both aeroallergen and live mites continued to be detected in houses after treatment with the spray. These studies suggest that such sprays are only temporarily effective when applied at the manufacturer's recommended volumes and additional approaches are required to control the bulk of allergens in houses.

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Introduction

There is increasing evidence that in genetically susceptible individuals exposure to *Pyroglyphidae* mite allergens in houses may be an important risk factor for asthma [1–6] and that reducing such exposure may reduce symptoms [7,8]. Exposure to mite allergens has been quantitatively associated with increased basophil sensitivity [1], an increased risk of sensitization [1–3] and increased symptoms of asthma [2–6]. While the most direct support for the benefits of allergen avoidance comes from experiments where mite-allergic asthmatics were confined to 'allergen-free' hospital rooms [7], 12 of 25 trials of allergen-avoidance measures in houses have also demonstrated some clinical benefit (see [8] for review). The extent of allergen reduction in most of these trials was not quantified, nor was reduction exercised throughout the house. It is likely that in the trials showing no benefit total exposure to allergens was not reduced sufficiently.

Comprehensive reductions in domestic allergens are difficult to achieve and maintain. Mite allergens readily become airborne during dust disturbance accompanying domestic activity and can become ubiquitously distributed throughout houses [9], increasing the numbers of sites where control must be exercised. Further, the concentration of mite allergens in many houses is 10–50 times what may be considered a 'safe' level (perhaps <0.5 µg/g). There is as yet insufficient evidence that the large reductions in allergen exposure required to achieve this level can be achieved by the generally recommended methods of dust control such as vacuum cleaning, carpet removal and the washing of bedding. Indeed in many houses, achieving a sufficient reduction of allergens may even require substantial additional changes to the way people build, furnish and live. New approaches to allergen control are clearly welcome.

The discovery by Green [10] that the treatment of house dust with a solution of tannic acid dramatically reduces the allergenicity of the dust provided the basis for a new method of chemically treating houses to remove aller-

Correspondence: Dr E. R. Tovey, Department of Medicine, University of Sydney, DO6, Sydney, NSW 2006, Australia.

gens. An initial laboratory-based trial of a commercial spray containing tannic acid combined with an acaricide [11], demonstrated that allergens were decreased by 1000-fold in the dust from bedding and carpets and that mites were killed within 30 min. This product offered considerable potential for simple, effective control of mite and other allergens in houses.

We used this product to treat 15 houses in the manner recommended by the manufacturer. Using an immunoassay for *Der p* I, we assessed changes in allergen concentration in dust from beds, carpets and furnishings over a 4-week period following spraying. As a control four untreated houses were monitored over the same period.

Materials and methods

Study design

Nineteen houses were divided into four treatment groups. In the maximal treatment group of five houses all carpets, beds and soft furnishings were treated with the tannic acid/acaricide spray and in addition all bedding (mattress, pillows and doona (duvet) or blankets) was encased in dust and mite-proof covers (Medisoft or Coolguard; Allersearch, Granville, NSW, Australia). In 10 houses the carpets, beds and furnishings were treated with the tannic acid/acaricide spray without additional measures. In five of these houses all rooms in the house were treated and in the remaining five houses only one bedroom and the lounge room were treated. Four houses which were not treated were also studied. In most houses two bedrooms were treated and/or sampled, but two houses had only one bedroom. In one house the leather furnishings were not sprayed, in one house the carpets were removed and in one house new bedding was introduced during the course of the trial. In total, nine beds received maximal treatment, 13 were treated with the spray only and 12 were untreated. Twenty-one beds used duvets and the remainder used blankets. Thirty-six floor areas were treated with the spray and 17 were not treated. Fifty-one floor areas were fully carpeted, five had only loose rugs which were regarded as the area for treatment and no houses had only hard floors. Thirteen items of soft furniture were treated and four items were sampled but not treated.

The spray used, Allersearch DMS, contains an acaricide and tannic acid as an allergen denaturing agent. The spray was applied using the hand-pump supplied with the 2.5 l pack at the recommended rate of 23 ml (~10 squeezes of the pump handle) per m² to bedding (to the top and sides of mattresses and to both sides of pillows, blankets and duvets), carpets and furnishings.

Dust samples were collected 2 weeks prior to treatment, just prior to treatment and at intervals of 3 days, 2 weeks and 4 weeks after treatment. A few follow-up samples could not be collected. Of the 104 samples required at each sampling period, 103 were collected at 3 days after spraying, 102 at 2 weeks and 84 at 4 weeks after spraying.

Sample collection

Dust samples were collected with a portable vacuum cleaner (Makita, model 4071D), modified to separately collect the coarse dust on an oblique wire mesh prefilter and the fine dust in conical pre-weighed bags of 25 µm pore nylon fabric with a surface area of 55 cm². Dust was collected by vacuuming a 1 m² area of carpet, soft furniture or bedding (mattress, pillows and bed coverings) for 1 min. Following collection, each bag was weighed and the weight of fine dust calculated. The dust was extracted in the bag using phosphate-buffered saline (PBS), pH 7.4, 0.05% Tween 20 buffer. The volume of solution used depended on the amount of dust in the bag: 2 ml for <0.1 g fine dust, 3 ml for >0.1 to 0.5 g and 5 ml for >0.5 g of dust. Extraction was performed under rotation for 30 min in a capped 10 ml tube, which had been modified to keep the bag in the top two-thirds of the tube during subsequent centrifugation at 3000 g for 10 min. The dust extract was removed from the centrifuge tube and was stored at -20°C until analysis.

Live mite counts

In five houses counts of live mites were made. Following the extraction of allergen, the fine dust was immediately emptied into a 25 ml tube and the live mites recovered by flotation in saturated NaCl with 30% sucrose [12]. Only the mobile live mites were used to evaluate changes in mite numbers because some mites may have been recently killed by the acaricide but not become desiccated.

Allergen assay

Mite allergen *Der p* I was assayed using a double monoclonal antibody ELISA, supplied by Associate Professor M. D. Chapman and Professor T. A. E. Platts-Mills (University of Virginia, Charlottesville, Virginia, U.S.A.) [13]. Extracts were diluted in 1% bovine-serum albumin (BSA) (Sigma Chemical Company, St Louis, Missouri, U.S.A.), PBS, 0.05% Tween and initially assayed in duplicate at 10- and 100-fold dilution. A standard curve and four allergen standards (two of mite culture extract prepared in the laboratory [14] and two *Der p* I standards supplied with the assay), were included on each microtitre plate. Plates were read on a BioRad

model 450 plate reader (BioRad, Richmond, California, U.S.A.) and calculations performed using Microplate Manager software (BioRad). For concentrations lying off the sloping portion of the standard curve (generally 5–70 ng/ml), the assays were repeated at an appropriate dilution, ranging from neat to 1:1600. Duplicate samples with a coefficient of variation >8% were rejected. The allergen concentration was expressed as μg of *Der p* I per g of fine dust and total allergen was calculated by multiplying allergen concentration by the weight of dust collected.

Aeroallergen measurement

Aeroallergen was estimated in six houses by collecting the airborne dust settling out in petri dishes over a period of 2 weeks. Plastic petri dishes (150 mm diameter) and the inner surface of their lids were pretreated with an aqueous solution of 2% teleostean gelatin (Sigma) for 15 min, then rinsed in distilled water and air-dried. This was done to minimize possible non-specific absorption of allergen to the collecting surface. To collect settling airborne dust, both the dish and the lid were exposed for 2 weeks in a location between 1 and 2 m above the floor level. Locations such as the top of shelves, cupboards and wardrobes were chosen. The airborne dust which settled in the dishes was eluted off with 1 ml of PBS, 0.05% Tween, 0.2% BSA, 0.1% sodium azide and the solution stored at 4°C until analysis for *Der p* I. Aeroallergen was expressed as nanograms of allergen per day.

Calculation of data

The distribution of allergen levels was positively skewed and a logarithmic transformation was used to normalize the distribution. All calculations were performed on transformed values. Geometric means (95% confidence limits) are reported.

The baseline allergen concentration for each item is the geometric mean of the two measurements performed prior to treatment. Comparison of baselines between treatment groups for each site was by one-way analysis of variance or *t*-test, as appropriate. Within-group estimates of the effect of treatment at each follow-up visit were expressed as mean (95% CI) percentage of baseline allergen remaining. This was calculated as the mean (95% CI) of the difference between the $\log_{10}$ allergen concentrations at follow-up and baseline. This value was antilogged and expressed as a percentage. Within-group estimates of the rate of increase of allergen concentration after the initial reduction were expressed as the fold increase (95% CI) between 3 days and 4 weeks after treatment. This was calculated in a manner analogous to that described above.

The reduction in allergen concentration at 3 days,

Table 1. Baseline concentrations of mite allergen *Der p* I/g fine dust in the beds of the three treatment groups and in the carpets and furnishings of the two treatment groups

	Baseline <i>Der p</i> I levels ($\mu\text{g/g}$ fine dust)		
	Spray and bedding covers	Spray only	Untreated
Beds			
<i>n</i>	9	13	12
Mean	27.7	13.2	38.1
95% CI	11.0, 68.2	7.0, 25.2	18.9, 76.9
Carpets			
<i>n</i>	n/a	36	17
Mean		14.5	22.2
95% CI		10.3, 20.3	10.6, 46.5
Furniture			
<i>n</i>	n/a	13	4
Mean		15.6	19.7
95% CI		8.8, 27.8	7.0, 55.6

expressed as the difference in $\log_{10}$ allergen concentrations between baseline and 3 days after treatment, was compared between treatment groups by analysis of variance. Analysis of covariance was used to test for the effect of baseline allergen concentration on this relation. A similar approach was used to compare the rate of increase in allergen concentration after the initial reduction. The significance tests were confirmed by non-parametric statistics (Kruskal–Wallis).

Mite counts and aeroallergen levels are expressed as a median and range.

A *P* value of <0.05 was regarded as significant. The data was collated on a SMART database (Innovative Software, Lenexa, Kansas, U.S.A.) and calculations were performed using the SAS statistical software (SAS Institute, Cary, North Carolina, U.S.A.).

Results

The mean concentrations ($\mu\text{g/g}$) of *Der p* I at baseline are shown in Table 1. There was no significant difference in allergen concentration of the dust between the treatment groups; in beds ($P=0.07$), carpets ($P=0.28$) or items of furniture ($P=0.60$).

The allergen levels 3 days after treatment compared with the baseline level for all sites in all treatment groups are shown in Fig. 1. There was a marked variability in response to treatment. The mean percentage of baseline allergen concentration remaining at 3 days, 2 weeks and

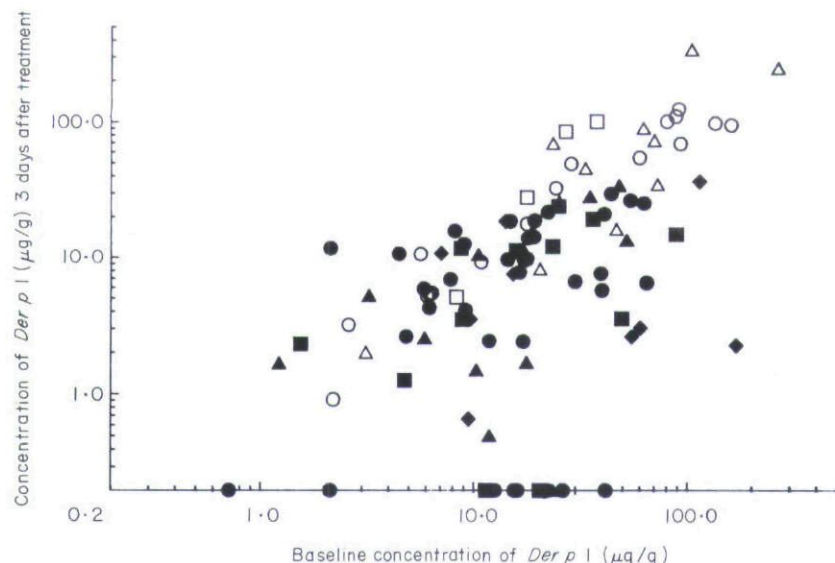


Fig. 1. The effect of treatment on allergen concentrations at 3 days after treatment compared to baseline (before treatment) allergen concentrations. Beds: (◆) occlusive covers and sprayed, (▲) sprayed only, (△) no treatment; carpets: (●) sprayed, (○) no treatment; furniture: (●) sprayed, (○) no treatment.

4 weeks after treatment within each treatment group are shown in Figs 2, 3 and 4, respectively. Because less dust was collected from the beds which had been fitted with occlusive covers, the reduction in total allergen collected was greater: mean (95% CI) for the three sampling periods being 8% (2%, 42%), 7% (2%, 29%) and 8% (2%, 32%) respectively.

There were significant reductions in allergen concentrations 3 days after treatment in the beds which were sprayed and had covers fitted and in the spray-only beds when either were compared to the untreated beds ($P=0.015$ and $P=0.019$ respectively). However, there

was no difference between the two treated groups of beds ($P=0.74$) at this time. In both carpets and furniture the allergen concentration in the treated groups were significantly reduced compared to the untreated groups 3 days after treatment ($P=0.005$ and $P=0.046$, respectively). These conclusions were not altered by adjustment for the baseline allergen concentrations.

The rate of increase over 4 weeks in allergen concentration at each site after the initial reduction due to treatment is shown in Table 2. The rate of increase in this period was significantly lower in the beds treated with spray and occlusive covers than in the beds treated with covers alone

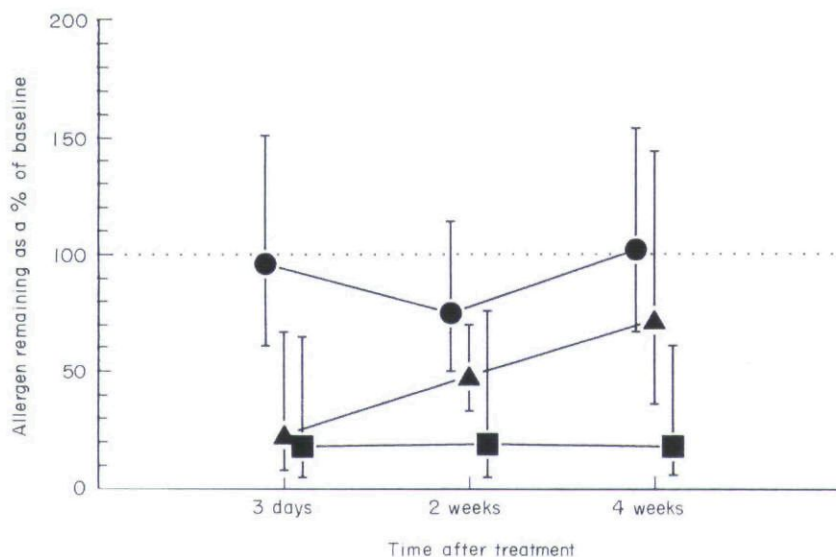
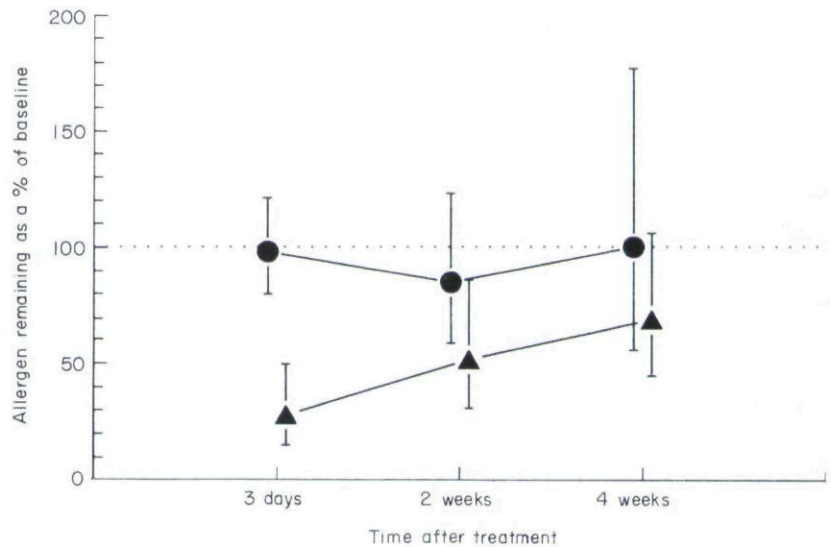


Fig. 2. The changes in mite allergen *Der p 1* in dust collected from beds following spraying with DMS. The allergen remaining as a percentage of baseline at 3 days, 2 weeks and 4 weeks after spraying is shown. The treatment groups were: ■—■ beds sprayed and occlusive covers fitted; ▲—▲ sprayed only; and ●—● unsprayed beds. The bars show the 95% confidence limits of the mean proportion of baseline allergen level.

Fig. 3. The changes in mite allergen *Der p* I in dust collected from carpets following spraying with DMS. The allergen remaining as a percentage of baseline at 3 days, 2 weeks and 4 weeks after spraying is shown. The treatment groups were: ▲—▲—▲ carpets sprayed with DMS; and ●—●—●, unsprayed carpets. The bars show the 95% confidence limits of the mean proportion of baseline allergen level.



($P=0.011$). The conclusion was not altered by adjustment for the initial allergen concentration after treatment.

The median numbers (range) of active live mites collected before and following spraying is shown in Table 3. This shows that active live mites continued to be found in most samples after treatment.

The median quantities (range) of airborne mite allergen collected per fortnight in the petri dishes in treated bedrooms and lounge rooms are shown in Table 4. Aeroallergen continued to be collected in all houses, although only the two lounge rooms with the highest levels showed marked reductions. One of these changed from 14.6 ng/day prior to spraying to 0.5 and then 3.98

after spraying and the other from 11.6 ng/day initially to 0.85 and 2.1 after spraying.

One person reported a headache and one had marked nausea within 24 hr of spraying. No adverse reactions were reported by other people in these houses or by people in any of the other 13 houses where the spray was used.

Discussion

The treatment of beds, carpets and items of furniture with the spray containing tannic acid and an acaricide led to a moderate and temporary reduction in the concentration of mite allergen *Der p* I. As shown in Fig. 1, there was

Fig. 4. The changes in mite allergen *Der p* I in dust collected from soft furnishings following spraying with DMS. The allergen remaining as a percentage of baseline at 3 days, 2 weeks and 4 weeks after spraying is shown. The treatment groups were: ▲—▲—▲ furnishings sprayed; ●—●—● furnishings not sprayed. The bars show the 95% confidence limits of the mean proportion of baseline allergen level.

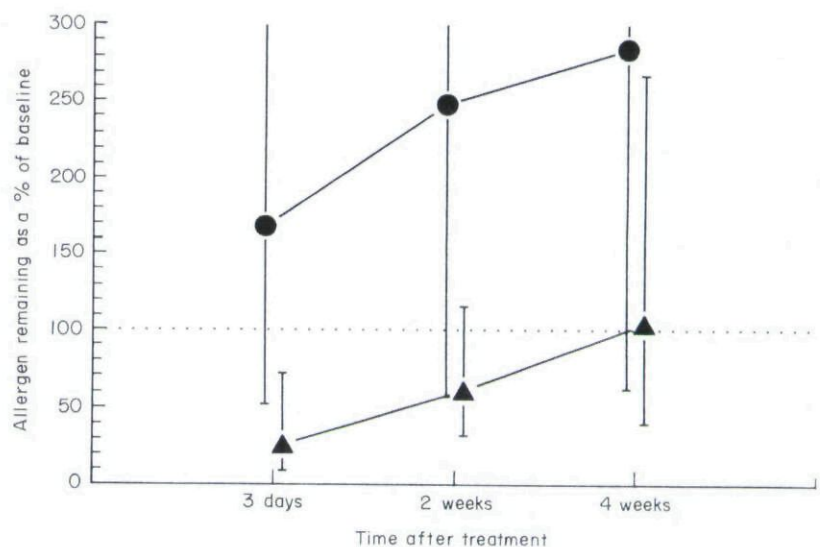


Table 2. Mean (95% CI) fold change in allergen concentration between 3 days and 4 weeks after treatment

	Mean	95% CI
Bed		
Spray and covers	1.00	0.48–2.05
Spray only	3.40	1.33–8.71
Carpets		
Treated	2.81	1.64–4.80
Furniture		
Treated	4.71	2.52–8.83

Table 3. Median number (range) of active live mites collected from items in a subgroup of houses which had been sprayed at the following collection times: just prior to spraying (day 0), and 3, 14 and 28 days after spraying

	Active live mites/item			
	Day 0	Day 3	Day 14	Day 28
Beds (n=7)	6 (0–28)	1.5 (0–12)	1 (0–12)	4 (1–20)
Carpets (n=12)	1.5 (0–386)	4 (0–376)	6 (0–88)	16 (0–436)
Furniture (n=4)	3.5 (0–138)	12 (8–62)	7 (0–80)	100 (4–528)

Table 4. Median quantity (ng) (range) of allergen *Der p* I per day collected in petri dishes in the 2 weeks of prior of treatment and in weeks 0–2 and 2–4 after treatment

	Period of the treatment (weeks)		
	–2 to 0	0 to +2	+2 to +4
Bedrooms (n=7)	1.06 (0.41–2.27)	0.84 (0.51–2.27)	0.86 (0.43–1.90)
Lounge rooms (n=5)	0.45 (0.30–14.6)	0.43 (0.23–0.85)	0.98 (0.20–3.98)

considerable variation in the change in allergen concentration. Factors responsible for this variation between similar sites were not investigated, although variation in the nature of the individual items, the degree of dust disturbance in the 3 days between spraying and sampling, and variations in both spray application and dust sampling could all have contributed. Overall, there was a mean reduction to about 20% of baseline 3 days after treatment followed by a gradual return to baseline over the subsequent 2 to 4 weeks. Greater initial reductions in allergen would probably have been observed if samples had been collected sooner than 3 days after spraying.

When occlusive covers were fitted to the mattress, pillows and bedding there was a prolonged reduction in both the weight of dust collected and in its concentration of allergen. The approximately ninefold reductions in total allergen seen here are not as great as the approximately 100-fold reductions reported for vinyl covers by Owen *et al.* [15]. The continued detection of allergen in the beds suggests that allergen may accumulate quite rapidly from other sources. These may include aeroallergen sources, night clothes and from the placing of apparel, toys and similar items on beds.

The more rapid return to baseline in furnishings may occur because of the greater disturbance of the untreated dust in furnishings when they are used, the greater quantity of allergenic dust contained in furnishings and differences in the surfaces of furnishings reducing the efficiency of treatment.

Aeroallergen continued to be collected in all rooms following treatment despite temporary reductions in the allergen content of dust collected with a vacuum cleaner from sites within the room. This suggests that allergenic dust from ubiquitous sources continues to circulate in the air of these rooms. This method of measuring aeroallergens is likely to provide similar data to prolonged sample collection at low airflow rates onto horizontal filters of similar area because the majority of mite allergens are carried on large particles which settle rapidly from air. The method has the advantages that it is cheap, silent and many collections can be made simultaneously.

The most likely reason for the inability to gain adequate control of mite allergens in this study was the low rate of application of the spray (23 ml/m²) applied to all items. This is the rate recommended by the manufacturers of DMS and is greater than, or similar to, the rate used by other commercial tannic acid-based sprays. This application rate, which is equal to 2.3 µl/cm², is insufficient to penetrate further than 2–3 mm below the surface of carpet. The depth of the carpet pile, plus backing and underfelt, is commonly 10–15 mm. Similarly, the preferred bed covering in these houses was doonas (duvets) which may require a heavier rate of application than blankets.

Presumably allergenic dust from below the treated surface layer of carpets, bedding and furniture soon replaces the surface dust. The same reason also applies to the failure of the spray to adequately control live mites which continued to be found in most samples. Mites inhabit the subsurface layers of both carpets [16] and mattresses [17]. In the original studies, Green used 140 ml/m² on carpets and 46 ml/m² (both sides sprayed with 23 ml/m²) on blankets which resulted in a much greater reduction in mite numbers [11]. The effectiveness of higher application rates, such as those used in the original study, on different types of carpets and bedding needs to be further investigated.

The magnitude and time course of the allergen reduction is similar to reports elsewhere in abstracts. Price *et al.* [18] reported that 2 weeks after spraying with DMS, *Der p* I concentration in carpets and furniture were 50% and 15% respectively of pretreatment levels. Other studies have used similar volumes of a 3% solution of tannic acid. Miller *et al.* [19] reported nearly 100-fold reduction in the mite allergen, and in another study [20] a 20-fold reduction in cat allergen, both in samples collected 24 hr after spraying, but the latter reduction was only twofold (not significant) in samples collected one week later. Thompson *et al.* [21] reported significant reductions in *Der p* I allergen content of carpet dust up to 4 weeks after spraying.

The chemical treatment of house dust with an allergen denaturing-acaricide solution has the potential to significantly reduce the levels of domestic allergens. However, it would seem unlikely that any surface chemical treatment of items containing a large reservoir of allergen could offer long-term allergen control. For items such as pillows and mattresses, impermeable covers seem the preferred alternative. The future of such a chemical treatment to reduce allergens lies in defining its limitations and using its unique properties as a component of a multi-system approach to allergen control in the home.

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