

EDITORIAL

House dust mite control with Acaroson—an extreme test?

The development of effective methods for alleviating symptoms of house dust mite allergy, by tackling mites and their allergens in homes, has been the aim of research workers for almost two decades. Two major strategies have evolved; allergen avoidance/inactivation and mite population control by extermination.

The avoidance of mite allergen exposure is based on the use of mattress and bedding covers, thorough vacuum cleaning or replacement of carpets with hard floor coverings [1–3]. Rather than directly killing mites, these methods involve removing allergens and mites from the home, placing barriers between patients and major allergen sources and providing fewer habitats in which the mites can live and breed. Additionally, allergens can be denatured using tannic acid sprays [4] or by boil-washing [5]. Also, patients have been removed from their homes to allergen-free environments, such as hospitals [6].

Mite populations can be controlled by killing them with chemical acaricides [7,8] or via the physical effects of heating and/or drying with electric blankets [9], natural or mechanical ventilation [10] and freezing furnishings with liquid nitrogen [11]. Avoidance and mite control are not mutually exclusive procedures: for example, removal of the dead mites by vacuum cleaning is essential following use of acaricides or physical methods, and reduction of domestic humidity has been combined with barrier methods in several clinical trials [12,13]. However, no integration of the two strategies, that utilizes the best methods from each, has yet been tested in a controlled clinical trial.

A profusion of proprietary acaricides have been developed in recent years, with varying degrees of scientific assessment prior to their marketing. Some have never been tested outside the laboratory, let alone in rigorously-conducted controlled clinical trials. Acaroson, containing solidified benzyl benzoate, is exceptional in that it has been tested repeatedly in field and clinical trials and is by far the most well-researched product available [cf. 7,8,14 for many references].

In this issue of *Clinical and Experimental Allergy*, Kneist *et al.* report the second part of their placebo-controlled trial on mite control with Acaroson in homes of patients with mite-allergic rhinitis. The first part [14], addressing clinical efficacy, was the subject of a previous editorial in this Journal [15]. The present part deals primarily with effects of Acaroson treatment on mite populations and allergen levels via guanine scores.

The results appear to demonstrate that there were statistically significantly greater reductions in mite popu-

lation growth in mattresses, other upholstered furniture and on floors of 10 homes in which Acaroson treatment was used (twice in a year), compared with 10 homes in which placebo preparations were used. Additionally, by the end of the trial, slightly fewer floors and furnishings in the Acaroson treatment group had guanine scores above 1 (considered to be weakly-positive) than in the placebo group.

The study is unusual in that volunteers were selected from a group of patients with mite-allergic rhinitis, most of whom had already carried out allergen avoidance measures without accruing any clear clinical relief. According to Kneist *et al.* purportedly, they represent an extreme population, with intractable symptoms, and were chosen so that Acaroson could be given a severe test. But are these volunteers really a suitable group for giving Acaroson a rigorous assessment? In fact, only five patients performed avoidance measures in all bedrooms and the living-room as advised, and seven did not make any attempts at avoidance in their own bedrooms. The measures advocated were based on replacement of furnishings. Mattress covers do not appear to have been used. We are given no idea what effect these rather piecemeal efforts had on the mite populations or guanine levels (other than the removal of carpets was effective in reducing the values of these parameters) because no pre-avoidance baseline data is presented. It is hardly surprising that avoidance measures conferred no clinical benefit if the majority of patients did not fully comply.

The efficacy of an acaricide is simply a measure of how good it is at killing mites. Clinical improvement is not a direct measure of acaricidal efficacy, but a consequence of reduced allergen-exposure following removal by vacuum-cleaning of mites killed by the acaricide. If vacuum-cleaning is not done properly, large numbers of dead mites may remain in the dust; they gradually disintegrate and more allergen-bearing, aerosolizable particles are generated. Thus, in a clinical trial it is important to standardize allergen removal and control for its effects: this was not done in the study by Kneist *et al.*

Non-clinical evidence for the extreme nature of the volunteer group is presented: the microclimate in the homes was damp and highly conducive to mite population growth although, paradoxically, the authors admit that the homes did not necessarily contain large mite populations; only two had 'excessive' contamination (> 1300 mites/gram of dust). Surely, an extreme test for efficacy of an acaricide would be to use it in homes which all had heavy mite infestations and compare the numbers

of dead mites with those from the placebo-treatment homes. One would expect to see a large rise in numbers of dead mites in the first set of dust samples taken after acaricidal treatment, whereas this effect would be absent in the placebo group.

However, Kneist *et al.* have chosen to use an index of mite population growth, whereas an index of mortality may have been more suitable. There are two major problems associated with this choice. Firstly, live mites tend to be difficult to detach from fabrics by vacuum sampling, but dead mites are relatively easy to remove. Estimating live mite populations by vacuum sampling is not very accurate, especially when combined with a mite separation method of such low extraction efficiency as the one they used. Secondly, there is no immediate way of comparing their data with that in any previous trial with Acarosan: no-one else has used this index. Some idea of fold-changes ($\pm$ ranges) in live mite numbers can be extrapolated from their formula, using $N_0 = 1$. It may be useful to present these values here: floors placebo 1.235 (+42.5, -0.249), Acarosan 0.588 (+1.197, -0.088); mattresses placebo 1.139 (+2.411, -0.357), Acarosan 0.295 (+5.0, -0.002); furniture placebo 0.71 (+3.255, -0.482); Acarosan 0.21 (+3.25, -0.086). Plotted on a graph, these values appear somewhat less impressive than those in Fig. 2. We are given no explanation of the enormous range for the floors of the placebo-treated group, nor are we told in how many cases Acarosan failed to reduce the live mite populations.

The authors' economy with important data is unfortunate; it would have been useful to know the changes in numbers of total mites, as well as live and/or dead ones, on each sampling occasion in each home. This would allow a better appreciation of the variation between homes. Also, it would have been useful to know the actual guanine scores rather than the percentage of scores that fell to a value of 1 (Fig. 1). Why was this data not presented?

Intervention and dust-removal took place in the homes of both groups. Thus, natural fluctuations in mite population density and allergen levels were not controlled for. This is a shortcoming in a trial of 12 months duration. We have no way of knowing what proportion of the reductions in mite population growth and guanine scores was due to normal seasonal fluctuations.

In summary, I can only reiterate the conclusions of Godfrey [15], that many of these criticisms are minor when considered in isolation but, when combined, serve to weaken the study considerably. The results suggest that Acarosan is an efficient acaricide, but do not prove this. Nor can it be said that Acarosan was given an extreme test.

References

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