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Mite-sensitive asthma of childhood

Trial of avoidance measures

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Sarsfield, J. K., Gowland, G., Toy, R., and Norman, A. L. E. (1974). *Archives of Disease in Childhood*, **49**, 716. **Mite-sensitive asthma of childhood: trial of avoidance measures.** A trial of the effects of measures designed to reduce exposure to house-dust mites was performed on 14 children with mite-sensitive asthma. These measures reduced mite infestation of the beds and were associated with clinical improvement of asthmatic symptoms. Immunological studies revealed a tendency for levels of total serum IgE to fall during the course of the trial, but no such changes were apparent in the levels of anti-mite specific serum IgE. Possible reasons for this discrepancy are discussed.

The conclusion of the study is that the use of a plastic mattress cover and the other inexpensive and harmless avoidance measures described should be advised for all asthmatic children with evidence of mite-sensitivity before resort to drug prophylaxis or hyposensitization procedures.

Evidence has accumulated over the past decade that the house-dust mite, genus *Dermatophagoides*, is a world wide and potent allergenic component of house-dust (Voorhorst, Spieksma-Boezman, and Spieksma, 1964; Wharton, 1970). To flourish it requires warmth, humidity, and, in the case of the European species *D. pteronyssinus*, human skin scales as a food source. Hence, it is frequently found in situations associated with human habitation and human contact. The highest degrees of infestation have been reported in domestic beds and on upholstered furniture (Maunsell, Wraith, and Cunnington, 1968; Sarsfield, 1974). A recent detailed study charted its life cycle in its natural environment (Hughes and Maunsell, 1973). There is now ample evidence of sensitization to the allergenic components of this organism in asthmatic individuals (McAllen, Assem, and Maunsell, 1970) and its role in childhood asthma has been stressed (Sarsfield, 1974).

The most successful and direct treatment of allergic asthma is the removal of any identifiable offending allergen from the environment. Advice on how to reduce exposure to the house-dust mite allergen has appeared in published reports (Maunsell, Hughes, and Wraith, 1970; *Drugs and Therapeutics Bulletin*, 1972). As early as 1928, Dekker claimed symptomatic relief in his asthmatic patients who took measures to reduce exposure to 'bed-mites'. In spite of the long recognition of the presumed benefit of these avoidance measures, no trial of their usefulness has been reported. The present study was designed to subject them to trial in a group of mite-sensitive asthmatic children.

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Patients and methods

Patients. The trial was carried out on 14 children attending the asthma clinic of the University Department of Paediatrics at Seacroft Hospital, Leeds. Their ages ranged from 3 to 13 years (mean 6·9 years). These children had perennial asthma and were selected after initial clinical and immunological investigations had indicated that the prime offending allergen was *D. pteronyssinus*. None of these children was receiving any prophylactic therapy, nor had they been subjected to any course of hyposensitization.

The environmental assessment was performed by one of us (A.L.E.N.) in every case. The type, age, and heating system of the dwelling of each child was noted, and the degree of dampness assessed by inspection. Dust samples from the children's mattresses were collected and examined for mites as previously described (Sarsfield, 1974). Clinical progress was charted in daily

symptom diaries in which were recorded the frequency and severity of all asthmatic attacks. A 'points system' was devised: 1 point for a 'mild attack' (child mobile; symptoms transient and only a nuisance); 2 for a 'moderate attack' (child immobilized in bed or chair; drug treatment required); 3 for a 'severe attack' (serious or prolonged symptoms; medical attendance required). The total score was expressed in points per month. A retrospective assessment was also made from the past history using the same system.

Allergy assessment. Skin tests were performed by the prick method with commercial extracts of *D. pteronyssinus*, *D. farinae*, house-dust, grass pollens (B.2), and mould mix (A.13) (Bencard Ltd.). Nasal provocation tests with *D. farinae* allergen and the estimation of specific serum IgE to *D. pteronyssinus* were performed as previously described (Sarsfield, 1974).

Total serum IgE was measured by the radioactive single radial diffusion technique (Rowe, 1969). Results were expressed in units by reference to the Research Standard serum 68/341 (National Institute for Medical Research). Immunological investigations were performed on paired sera taken at the beginning and end of the trial, stored at −70 °C, and estimated in the same test run.

Avoidance measures. These were mainly directed at the child's bedroom. They were carefully explained in the clinic by the physician and supervised at home by the health visitor. It was stressed to the parents that the problem was no reflection on the cleanliness of the house or family. A printed sheet outlining the recommended measures was issued to them and this is reproduced below.

'The following measures are designed to reduce exposure of your child to the type of dust which causes wheezing. Please follow them.

The bedroom

Essential measures

(1) Remove feather pillows and eiderdowns—replace with ones having a synthetic filling.

(2) Thoroughly vacuum the mattress, the pillows, and around the base of the bed or divan. Enclose the mattress top surface and sides in a plastic cover.*

(3) *Each day*—'damp-dust' the plastic mattress cover.

(4) *Each week*—change and wash pillow cases, sheets, and under-blanket: vacuum the bed base and around the covered mattress.

Desirable measures

(1) Woollen blankets should be replaced by nylon or 'terylene' quilts or by cotton cellular ones and be washed frequently.

(2) Have light washable curtains and wash frequently.

(3) Remove carpets and cover the floor with linoleum or vinyl.

(4) Use a vacuum cleaner with disposable paper bags.

Note: If your child shares a bedroom—ALL other beds in the room must be treated similarly.

Other rooms

Particular attention should be directed at removal of dust from upholstered furniture (which harbours the type of dust to which your child is sensitive). Vacuum clean this furniture at least twice a week, especially the head rest, arms, and edges of the seats.'

Design of trial.

(1) An initial assessment was made including skin and nasal tests, and a blood sample was taken. The parents's permission was obtained for the health visitor to call and assess the home environment. No mention was made of house-dust or mite sensitivity and no advice about dust avoidance was given. All patients were given a supply of salbutamol tablets or syrup, which was only to be taken in the event of an asthmatic attack. No prophylactic drug therapy (disodium cromoglycate, topical or oral corticosteroids) was prescribed. Diary cards were issued and the initial observation period started.

(2) Within the next week or so the health visitor made an environmental assessment and collected the first dust sample from the top of the child's mattress.

(3) After a minimal period of 6 weeks from the first clinic visit the problem of mite sensitivity was fully explained to the parents and the avoidance measures were recommended. When these were completed to our satisfaction, the second observation period began with continued recording of any symptoms in the management of treatment.

(4) During the second observation period a second dust sample was obtained from the covered mattress top surface, and near the end of the trial (December 1972) a second blood sample was obtained.

It proved impracticable to design this study as a blind trial. It was also impossible to carry out a cross-over trial because parents were so impressed by the benefits of dust-avoidance measures that they could not be persuaded to discontinue them. The results should therefore be interpreted with care.

Results

The results of initial assessment of the study group of the 14 asthmatic children are shown in Table I. It can be seen that all the children gave positive skin reactions to house-dust and house-dust mites, the 12 patients subjected to nasal provocation tests had positive results, and all had significant serum levels of specific IgE to *D. pteronyssinus*. These results are interpreted as good evidence of clinical hypersensitivity to the house-dust mite in all patients.

The results of environmental assessment and the mite counts of mattress dust samples are presented in Table II. It was found that the initial mite

*Obtainable from R. J. Morpeth (Contracts) Ltd., 39 Clayton Street, Newcastle upon Tyne NE1 5PN. Basic cost £1.

TABLE I

Results of initial allergy tests

Case no.	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Age (yr)	6	13	7	5	8	4	5	8	11	7	6	3	8	3
Skin tests (weal diameter in mm)														
D. farinae	5	7	5	4	5	7	5	5	5	4	5	4	5	4
D. pteronyssinus	5	10	7	5	5	9	6	5	15	4	5	4	5	4
House-dust	5	6	4	4	4	5	4	4	6	3	5	3	3	4
Pollens	0	2	2	0	3	1	1	3	1	0	0	2	4	2
Moulds	0	2	3	0	0	1	1	0	1	0	0	0	0	0
Nasal provocation test (*D. farinae*)	+	+	+	+	+	+	+	+	+	+	+	NT	+	NT
Specific serum IgE (*D. pteronyssinus*)	+	+	+	+	+	+	+	+	+	+	+	+	+	+

NT, not tested.

counts bore no relation to the dampness of the house. Some of the highest levels were found in satisfactorily heated and dry houses. In all cases it was possible to bring the level of mite infestation of the beds down to very low levels. Before avoidance, mite counts ranged from 8 to 186 (mean 80). After avoidance measures, counts fell to 0 to 8 (mean 2). This confirms the effectiveness of the avoidance measures advocated.

The symptom scores which reflect the frequency and severity of the asthmatic attacks are shown in Table III. In only two instances was the score obtained during the initial observation period lower than that derived retrospectively from the history. In 10 of 14 children it was higher, probably reflecting a more accurate assessment of the true handicap when daily symptom diaries were kept. The improvements in the children's asthmatic state were clinically dramatic and in every case there was clearly a drop in the symptom score (Table III). Nocturnal symptoms were especially relieved and 'trigger-factors' such as emotion and infection less often led into an asthmatic attack. Parents often noted that appetite was improved, but associated allergic rhinitis, if present, was unaffected.

Results of immunological tests on the paired sera were of interest. It might have been expected that levels of specific IgE to *D. pteronyssinus* would fall after reduction in exposure to this allergen. However, comparisons before and after avoidance measures, as expressed in final radioactive counts in the radioallergosorbent test, showed a rise in 8 cases and a fall in only 6 (Table IV). It seems probable that exposure to allergen was continuing in spite of a universal improvement in symptoms. In contrast to specific IgE levels, total IgE fell in 12 of 14 children. There was a rise in one patient and no detectable change in another (Table V).

TABLE II

Environmental factors and mite counts before and after avoidance

Case no.	House			Type of heating		Absolute mite count	
	Type*	Age (yr)	Damp	Living area	Bedroom	Before avoidance measures	After avoidance measures
1	T	50	No	Gas fire	Electric fire	82	0
2	D	2	No	Central	Central	88	0
3	T	80	Yes	Coal fire	None	12	5
4	S	10	No	Central	Central	64	0
5	S	10	No	Central	Central	64	4
6	S	40	Yes	Gas fire	Electric fire	170	0
7	S	10	No	Central	Central	105	2
8	T	60	No	Gas fire	Electric fire	42	2
9	T	70	No	Gas fire	None	186	1
10	S	40	Yes	Gas fire	Electric fire	40	1
11	S	10	No	Gas fire	Electric fire	8	8
12	T	70	Yes	Electric fire	Electric fire	23	5
13	S	20	No	Gas fire	Electric fire	15	1
14	T	70	Yes	Coal fire	None	120	4

*T, terraced; D, detached; S, semi-detached.

TABLE III

Effects of avoidance measures: frequency and severity of asthmatic attacks expressed as symptom scores per month

Case no.	Date first visit (mth/yr)	Date avoidance measures started (mth/yr)	Symptom score/mth		
			From history	During initial observation period	After avoidance measures taken
1	8/71	2/72	1·3	6·0	0·4
2	10/71	1/72	30	41	9
3	10/71	12/71	12·5	12·5	8·3
4	9/71	4/72	1·2	1·3	0·3
5	11/71	2/72	2·5	8·0	1·0
6	11/71	1/72	2·0	1·0	0·5
7	12/71	1/72	1·6	3·5	0·2
8	2/72	6/72	0·8	8·0	0·5
9	3/72	9/72	1·5	5·3	0·8
10	3/72	8/72	1·6	6·4	3·0
11	3/72	5/72	2·0	1·0	0·7
12	4/72	5/72	2·0	3·3	0·6
13	5/72	7/72	0·5	1·3	0
14	5/72	6/72	28·0	28·0	1·0

Discussion

The 14 asthmatic children in this trial were selected on the basis of good evidence that the house-dust mite was the principal offending allergen. The bedroom and bedding were the main centres of attention, with special emphasis on covering the mattress with a plastic cover. It was shown that the mite population on the mattress surface could be drastically reduced in practically all instances (Table II). Measures were also recommended to reduce exposure to 'mite-dust'.

Studies on asthmatic symptoms are notoriously difficult to control. Many factors apart from allergen exposure may influence symptoms. In this study the patient was used as his own control, employing symptom scores from the history and during an observation period of varying length. In only two instances was the score from the history greater than that of the observation period, underlining the need for careful recording of symptoms and providing evidence that premature establishment of avoidance measures probably did not occur to any great extent. The trial was fairly evenly spread over the whole year, reducing any possible seasonal influences. A health visitor assisted the mothers and checked that the recommended advice had been followed. After the introduction of dust-avoidance measures, reduction of symptoms was universally observed and in some cases dramatic relief was obtained (Table III). From these results it was concluded that measures aimed at the reduction of the mite population in the bedroom can

TABLE IV

Specific serum IgE to D. pteronyssinus *before and after dust avoidance measures*

Case no.	Time interval between samples (mth)	Specific IgE radioactivity (counts/½ min)		Percentage change
		Before avoidance	After avoidance	
1	11	664	575	−13
2	10	457	672	+45
3	10	1065	1186	+11
4	11	660	563	−16
5	9	1019	724	−26
6	5	930	799	−14
7	5	660	578	−12
8	8	432	580	+34
9	6	754	1010	+34
10	7	772	822	+6
11	5	562	614	+9
12	4	407	416	+2
13	4	468	523	+12
14	4	1026	944	−8

TABLE V

Total serum IgE levels before and after avoidance measures

Case no.	Total serum IgE (units/ml)*		Percentage change
	Before avoidance	After avoidance	
1	610	380	−38
2	600	400	−33
3	1100	900	−27
4	280	100	−64
5	860	420	−51
6	1600	900	−44
7	280	100	−64
8	540	300	−44
9	1400	1200	−14
10	2000	1300	−35
11	540	100	−81
12	100	100	0
13	300	600	+100
14	900	610	−33

*Values of ⩽100 units/ml are expressed as 100 units/ml.

be effective and that these result in symptomatic improvement in asthmatic children with proven mite sensitivity.

Great tact is required in some instances to 'put over' the concept of house-dust and mite allergy. The mother must not be allowed to think that dust is associated with uncleanliness or that she has failed in her role as housewife and mother. Supervision of the advised measures is recommended especially in the early stages: symptomatic improvement of the child usually engenders enthusiasm. The single most important step in avoidance procedures is the covering of the mattress with a plastic cover. The fine dust particles which carry the mite allergen probably cause the allergic wheezing. These are deeply ingrained in the mattress substance and only minimally removed by sweeping or simple vacuum cleaning. An impervious cover prevents them from rising and being inhaled.

It was expected that serum levels of specific IgE to *D. pteronyssinus* might fall after a period of successful avoidance. The expected fall only occurred in 6 patients. It was generally small, never exceeding 26%, and in 8 there was an actual rise (Table IV). Specific IgE levels to pollens have been measured by Berg and Johansson (1971) and they noticed rises during the pollen season with falling levels after the season. However, the situations are not strictly comparable as exposure to pollens is nil outside the appropriate season. With the mite allergen, perennial exposure will occur to some extent from many sources and this could be enough to maintain measurable levels of specific IgE in the serum. This would not detract from the beneficial clinical effect of avoidance of constant high levels of fine allergenic dust arising from the mattress and bedding.

The changes in total serum IgE levels (Table V) are characterized by a reduction in 12 of 14 of the paired sera. It seemed unlikely that these falls could be attributed to decreases in the specific anti-mite IgE component of the total levels. The longer storage time of the first serum samples, if significant, would be expected to have the reverse effect with lower values in these samples. Any effect from seasonal factors appears equally unlikely as sampling was fairly evenly spread over the whole year. The conclusion, therefore, remains that the avoidance measures and/or their results (fewer asthma attacks) were responsible for these falls. The absence of clinical symptoms *per se* has not been noted to correspond to falls in total or specific IgE levels. Indeed, Berg and Johansson (1971) presented evidence of the opposite effect. Their patients treated successfully with disodium cromoglycate for pollen sensitive asthma showed a greater rise in IgE during the pollen season when compared with an untreated group. This they attributed to a higher exposure in the clinically fit, active child to the sensitizing allergen. One could speculate that the avoidance measures in the present study remove other distinct and possibly more potent antigens than the house-dust mite. These may not be allergenic or contribute to the clinical symptoms of asthma but appear to stimulate a sizeable contribution to the levels of total circulating IgE. The measures employed will also reduce the irritant effects of house-dust which may allow easier ingress of antigens across mucosal surfaces. The dust may also possess the properties of an adjuvant to IgE production which would also be reduced.

Provocation tests and the presence of specific IgE, however, indicate frequent *in vivo* sensitization to the mite in allergic subjects. McAllen *et al.* (1970) calculated that to provoke a clinical attack of wheezing as little as 0·05g–1μg of allergen is needed, an amount of mite material weighing less than one whole live mite. Stenius *et al.* (1971) suggested that the levels of specific IgE against *Dermatophagoides* sp. were in a range comparable to those obtained against grass pollens in appropriately sensitive patients. This comparability of anti-mite IgE levels with an established allergen and the very small amounts required in bronchial provocation tests support the arguments in favour of its clinical relevance. The discrepancy observed between the changes in serum total and specific IgE levels therefore remains unexplained and further study of

the allergenic components of house-dust is indicated.

The main conclusion of this study is the necessity for a thorough search for offending allergens in the asthmatic child. If the house-dust mite is implicated then the simple, harmless, and inexpensive measures described should first be tried before resort to expensive and possibly harmful drugs or hyposensitization procedures of doubtful efficacy.

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