YEAR-ROUND HOUSEDUST MITE LEVELS ON THE HIGHVELD

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Objective. To determine the levels of the allergen DerP1, attributable to the house-dust mite (HDM) *Dermatophagoides pteronyssinus.*

Design. A four-season study conducted during 1994/95, sampling mattresses and carpets in the main bedrooms of suburban homes.

Setting. Thirty randomly selected homes in the Edenvale area, occupied by both black and white families living under similar socio-economic conditions in comparable environments.

Results. All homes tested positive for the allergen, and in 20% HDM levels exceeded levels recognised as 'safe' in terms of respiratory allergy, i.e. 2 µg/g of dust. Once mites were established in a home, they remained for months thereafter. The considerable seasonal variation recorded in HDM levels could not readily be explained.

Conclusion. The HDM is extremely sensitive to minimal variations in microclimate. Its year-round presence is of concern on the Highveld. Infestation levels below 2 µg/g of dust, until recently considered the critical point for sensitisation, may be significant triggers of symptoms.

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The house-dust mite (HDM), *Dermatophagoides pteronyssinus*, forms part of the natural ecosystem of the human habitat. This small creature, approximately 300 µm in length, feeds on human skin scales discarded by the body. The optimal environment for the mite is the bed, in and on the mattress, pillows and bedlinen. It is also found on carpets and soft furnishings. The mite faeces contain a protein that is known to be a major cause of respiratory allergy, particularly in humid and warm environments such as exist in coastal areas of KwaZulu-Natal.[1-4] Although the hot, dry conditions of the Highveld might appear to preclude HDM, the earlier work of Ordman[1] showed that HDMs were indeed present in Highveld homes, although frequently in lower numbers than at the coast.

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Two recent studies in Johannesburg[5,6] both detected about 25% reactivity to HDM in white allergy sufferers. The latest research conducted on the Highveld by Davis *et al.*[7] quantified the amount of mite allergen, DerP1, present in homes. Sixteen out of 30 of these homes were positive for DerP1, with the concentration of allergen greater than 2 µg/g of dust in 8 homes. (This was previously considered the critical level, beyond which sensitisation would occur. However, it is now believed that even lower levels are sensitising and may cause symptoms in susceptible subjects.) This study, and a subsequent one by the same team,[8] have been interesting in that they have probed what appears to be an anomalous situation.

South African clinicians are finding that rural blacks leading traditional lives, often in grassland biomes, rarely suffer from allergy. In contrast, once these people move to urban areas they frequently develop allergies.[9] For example, Van Niekerk *et al.*[10] found very low HDM allergy among Transkeian blacks, while none at all was found by Potter *et al.*[11] In contrast, the studies of Davis *et al.*[7] and Luyt *et al.*[8] conducted in Soweto, found that 45% of asthmatic children were sensitised to HDM. Simultaneously, they recorded only 23.4% HDM sensitivity in white asthmatic children in Johannesburg.

These studies pose two questions. First, why is the incidence of HDM allergy greater among black residents of Soweto than white residents of Johannesburg; and second, why is the incidence of HDM greater among urban blacks than rural blacks? The answer to the first question may partly lie in varying socio-economic conditions, but it seems obvious that changes in lifestyle associated with urbanisation induce an altered state in the immunological system.

The study presented here examines the incidence of HDM in the homes of blacks and whites living in the same neighbourhood under similar socio-economic conditions.

Materials and methods

The target area was suburban Edenvale, situated about 12 km east of Johannesburg. Over a 1-year period dust samples were taken in each of the four seasons to establish whether mite incidence was influenced by the weather. The first of the four sampling phases took place in the summer of 1994. During this initial phase 30 randomly selected homes of municipal workers in the Edenvale area were sampled. These homes were all situated in the same area within a radius of about 1 km, and were occupied by people of similar socio-economic status. Three of the 30 families were black. Black and white families were living under comparable conditions in identical brick and mortar housing. Furnishings and density of occupancy per home were similar, and cooking and heating were by means of electricity.

Where possible dust was collected from the main bedroom, using the Rainbow Vacuum Cleaner. Bed linen was removed and the mattress was sampled. An area of 1 m^2 on the mattress was vacuumed for 2 minutes. The dust sampled was placed in a plastic bag, sealed and labelled. Thereafter the carpet next to the bed was sampled, with 1 m^2 vacuumed for 1 minute. The dust sample was again placed in a plastic bag and labelled.

Antigenic content of the dust samples was determined using the method of Luczunska *et al.*[12] This analysis showed that every one of the 30 homes tested positive for HDM. However only 20% had concentrations of DerP1 above 2 µg/g of dust in either the mattress or the carpet sample, or both. The sampling protocol for the three subsequent phases concentrated on these 6 homes, in addition to which 4 of the remaining 'safe' homes were randomly added to the sample number, bringing the total to 10 for phases II - IV. The sampling procedure was identical in all phases. Phase II (autumn) occurred in April 1995, phase III (winter) in July 1995, and phase IV (spring) in October 1995.

Results

Table I shows results for phase I. The bedroom of home 22 was not carpeted. Table II shows results for subsequent phases: home 4 became vacated between phases I and II, and the

Table I. Phase I — summer DerP1 concentration (µg/g dust)

Home	Mattress	Carpet
1	1.17	1.40
2	0.45	0.16
3	1.13	0.75
4	21.30	0.91
5	4.33	0.26
6	1.23	0.68
7	1.04	1.31
8	0.15	0.71
9	0.12	0.70
10	0.09	0.13
11	9.99	6.33
12	0.22	0.09
13	0.15	0.28
14	0.36	0.56
15	0.09	0.50
16	0.09	0.05
17	4.03	0.72
18	1.09	0.51
19	5.61	1.75
20	0.40	0.18
21	0.36	0.11
22	0.01	nd
23	1.21	0.26
24	12.09	1.50
25	0.55	0.18
26	0.77	0.16
27	0.05	0.07
28	0.04	0.03
29	0.10	0.09
30	0.22	0.05

nd = no data.

Table II. Phases II, III and IV — autumn, winter and spring DerP1 concentrations (µg/g dust)

	Phase II		Phase III		Phase IV	
Home	Mattress	Carpet	Mattress	Carpet	Mattress	Carpet
4	nd	nd	nd	nd	nd	nd
5	3.84	0.10	4.76	0.15	0.56	0.08
11	5.28	9.35	nd	nd	1.53	1.24
17	4.74	0.43	4.67	3.20	1.45	1.35
19	10.88	14.89	10.35	7.98	1.58	1.73
23	17.10	108.37	18.53	31.22	1.96	0.66
24	0.65	2.91	0.51	0.56	0.36	0.72
26	0.81	0.10	0.84	0.07	0.91	0.03
28	0.14	0.12	0.12	0.04	0.31	0.14
29	0.71	0.10	1.64	0.11	0.09	0.04

nd = no data.

occupants of home 11 were away at the time of phase III sampling.

Discussion

Results showed a general trend, namely that once mites were established in a home they seemed to remain for months thereafter. However, the levels in both mattresses and carpets fluctuated considerably throughout the year, with no apparent consistency. For example, the mattresses in homes 5 and 17 showed similar levels from phase I through III, and then decreased noticeably in phase IV. In homes 11 and 19 there was almost a doubling in mattress HDM from phase I to II, but then a marked decrease between phases III and IV. The mattress in home 24 was heavily infested in phase I, but then became relatively 'safe' in subsequent phases.

Certain of these anomalous results can be explained, but not others. For example, in home 23 low levels were found in phase I, followed by exceptionally high mattress and carpet levels in phases II and III. There is no obvious explanation for this. However in this same home both carpet and mattress were replaced between phases III and IV, so that lower concentrations of HDM were found in phase IV. Homes 26 and 29 also replaced mattresses at this time, but in home 26 HDM levels were not significantly different between samplings. This may have been because of infested bedding. One possible explanation for the increased mattress levels from phases I to II and III in some homes was the use of excessive bedding, leading to higher bed temperatures and relative humidity, during colder months. However this is speculative and certainly does not explain decreases in other homes during the same time period.

This study compared homes of blacks and whites in similar circumstances and found no difference between the two racial groups, as was to be expected. Of the 3 black homes recorded, 1 had DerP1 levels exceeding 2 µg/g of dust. The conclusion is that the higher mite levels recorded in Soweto could be due to living conditions, as homes in Soweto are small and often overcrowded. Such conditions would also tend to increase relative humidity and temperature, however marginally. It seems clear, considering the variability of HDM within the sample homes, that mites are extremely sensitive to very small differences in the microclimates of individual homes. Of concern to clinicians on the Highveld is the fact that the HDM is without doubt a factor to be considered in the relatively dry atmosphere of the region. Their presence beyond the acceptable level in 20% of homes for most of the year (which was in fact an exceedingly dry year) probably indicates that during wet years HDM problems would be greater.

It is now emerging that even levels of HDM below 2 µg/g of dust may be significant triggers of sensitisation and symptoms. Further studies determining mite levels in relation to patient symptoms in this region are now necessary to resolve this debate.

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