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House Dust Mite Allergen Levels in A Singapore Hospital

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SUMMARY **House dust mite allergens constitute one of the most important allergens in house dust. In this study, the levels of two common dust mite allergens, *Der p* I and *Der f* I, in a general hospital in Singapore were evaluated. Our results showed that these allergens were detected in 42/74 (or 57%) of the dust samples. *Der p* I was found to be the predominant allergen detected ($p < 0.001$). The allergen levels were, however, low with only 1/74 having a *Der p* I concentration above 2 $\mu g\ g^{-1}$ dust. None of the samples had *Der f* I concentrations above this level. Of the various niches studied (mattresses, pillows, sofas, carpets, blinds and floors), the blinds and floors had the lowest concentration of allergen ($p < 0.05$). These low levels in the hospital compared to homes were attributed to the vigorous cleaning schedule in the hospital, the use of plastic to encased mattresses and pillows, vinyl covered sofas and vinyl lined floors. These practices may be adopted in the home as a means to reduce mite allergen exposure.**

House dust mites (HDM), especially those of *Dermatophagoides pteronyssinus* and *Dermatophagoides fairnae*, constitute one of the most important inhalant allergens in the house dust.[1] Also, there exists a strong association between sensitisation to these allergens and atopic disorders such as asthma.[2] The warm and humid tropical climate of Singapore is a key factor in promoting mite growth.[3] It is not surprising that the major HDM allergens, *Der p* I and *Der f* I, have been found to be highly prevalent in the dust of Singapore homes, with allergen concentrations markedly above the reported sensitising levels of 2 ug per gram of fine dust in the majority of homes.[4,5] Consequently, sensitisation to these HDM allergens has been found to be present in the majority (90%) of our children with asthma.[6]

In this study, we extended our survey of HDM allergen levels[4,5,7] to niches within the hospital, in an attempt to obtain information on the levels of allergen exposure in the hospital environment, particularly of our inpatients. Furthermore, a comparison between hospital allergen levels with those of Singapore homes[5] would enable us to evaluate possible factors affecting the levels of these allergens in our environment. Comparisons made from this work would also compliment our studies on the control of HDM allergens in homes of atopic individuals.[8,9]

MATERIALS AND METHODS

Dust samples

A total of 74 dust samples were collected from the National University Hospital, of which 34 were obtained from various floor locations, 14 from hospital bed mattresses, 10 from sofas used by patients and visitors, and another 16 from various other niches within the hospital (pillows used by patients, carpets in the lobby and restaurant, and curtains). These samples were located in 2 non-air-conditioned wards, 1 air-conditioned ward, and

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air-conditioned outpatient clinic, doctors' offices, the hospital's lobby and restaurant, and ventilated corridors.

Collection of Dust Samples

House dust samples were collected using a Kirby Classic III (Kirby Co, USA) vacuum cleaner. The vacuum cleaner was adapted to collect the dust sample onto a filter paper (Whatman no. 3). The filter paper was supported by a dust trap located at the base of the cleaner attachment. To ensure uniformity in collection, each sample was obtained by vacuuming an area of 1 square meter for 2 minutes. The dust samples were stored at 4°C in a mini-grip lock bag until processed.

Allergen Extraction

Dust samples were sieved through a No. 45 mesh screen with 450 μm pores, (VWR No. 57332146) to remove large particles and fiber. The fine dust was then suspended in borate buffered saline (pH 8.0) containing 0.1% Tween 20 (BBS-T) in a proportion of 100 mg fine dust per 2 ml buffer. For samples containing less than 50 mg of fine dust, 1 ml BBS-T was added. The dust suspensions were mixed end over end in an orbital rotator (Multipurpose Rotator, Model 151, Scientific Laboratories, Inc, Bohemia, NY 11716) at 4°C overnight. The supernatant was then harvested by centrifugation, and frozen at -70°C until they were assayed for *Der p* 1 and *Der f* 1 allergens.

ELISA for *Der p* 1 and *Der f* 1 levels

A sandwich enzyme immunoassay was used as previously described.[10] Briefly, 96 well microtiter plates (Immunolon II, Dynatech, Cantilly, VA) were coated with 1 μg/well of monoclonal antibodies, 5H8 (*Der p* 1) or 6A8 (*Der f*1) (Chapman MD, Virginia, USA) in 50 mM carbonate - bicarbonate buffer, pH 9.6, overnight at 4°C. After blocking with 1% bovine serum albumin PBS-T, allergen samples were added. Standard curves were constructed using reference *D.pteronyssinus* or *D.farinae* extracts (Champman MD, Viginia, USA). The second antibody, biotinylated 4C1, followed by streptavidin-peroxidase (Sigma) were added after appropriate periods of incubation. Colour development was achieved with 1 mM ABTS in 70 mM citrate phosphate buffer, pH 4.2. The reaction was stopped by adding 0.1 ml 2 mM sodium azide, and absorbance read at 414 mm.

Statistical analysis was carried out using the Statistical Software SAS version 6.08 for Windows.[11] The non-parametric Kruskal-Wallis one-way analysis of variance by ranks test was used to compare between the median allergen levels of the various niches as the levels were not normally distributed and the variances were heterogenous. The Chi-square or the Fisher's Exact (2-Tailed) test was used to compare the frequencies and the Spearman's Rank Correlation was used to analyse the correlation between allergen levels.

RESULTS

Hospital furniture and cleaning practices

All the hospital mattresses (full-sized beds and baby cots) were made of latex and were completely encased in thick plastic covers. On the average, bed linens were changed daily. After each patient's discharge, the occupant's bed was stripped of sheets, and the plastic cover damp wiped. Bedside furniture was cleaned similarly. For long-term staying patients, thorough cleaning of furniture was carried out every two to four weeks. Hospital sofas were all vinyl plastic covered and damp wiped once a week.

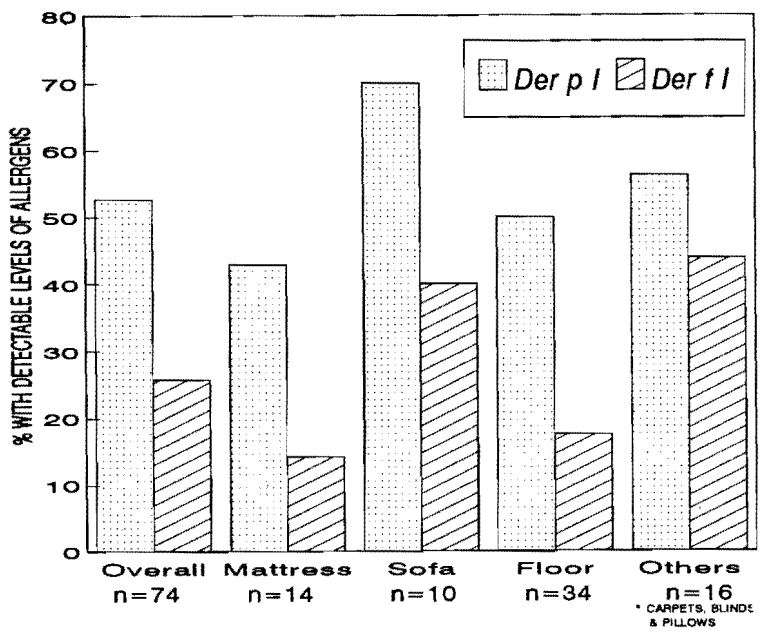

Fig. 1. Frequency of *Der p* I and *Der f* I allergens detected in 74 dust samples obtained from various locations of the hospital.

Hospital floors were vinyl-lined. The ward and lobby floors were swept and wet mopped twice a day, and daily for floors in clinics, corridors, and offices. Lobby carpets were vacuumed daily and shampooed once a week. The window blinds were made of canvas and cleaned once in 3 months.

Der p I and *Der f* I *levels*

Mite allergens were detected in 42/74 (57%) of the samples obtained. Overall, *Der p* I was more prevalent (39/74 or 53%) compared to *Der f* I (19/74 or 26%) ($p<0.001$). Fig. 1 shows the frequency of *Der p* I and *Der f* I allergens detected in dust samples obtained from the various niches surveyed in the hospital (Fig. 1).

For both allergens, the levels were generally lower on the floors and blinds (Table 1, Figs. 2A, B). Only one sample, obtained from a mattress, contained a *Der p* I level above the reported sensitising level of 2 μg g^{-1} dust (Fig. 2A). None of the samples had *Der f* I levels above the sensitising levels (Fig. 2B). When comparisons were made between air-conditioned and open wards, no differences were found between allergen levels in samples of comparable niches (mattresses, sofas, carpets, blinds) (data not shown). Allergen levels from the floor were, however, highest in the lobby and office areas compared to the floors of the wards and clinics ($p < 0.05$) (Figs. 3A, B).

There was no correlation observed between the levels of *Der p* I and *Der f* I in the samples obtained ($p > 0.05$).

DISCUSSION

The HDM, *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae,* have been shown to be amongst the most important allergens causing asthma throughout the world.[13,14] This is evidenced by the presence of early and late phase res-

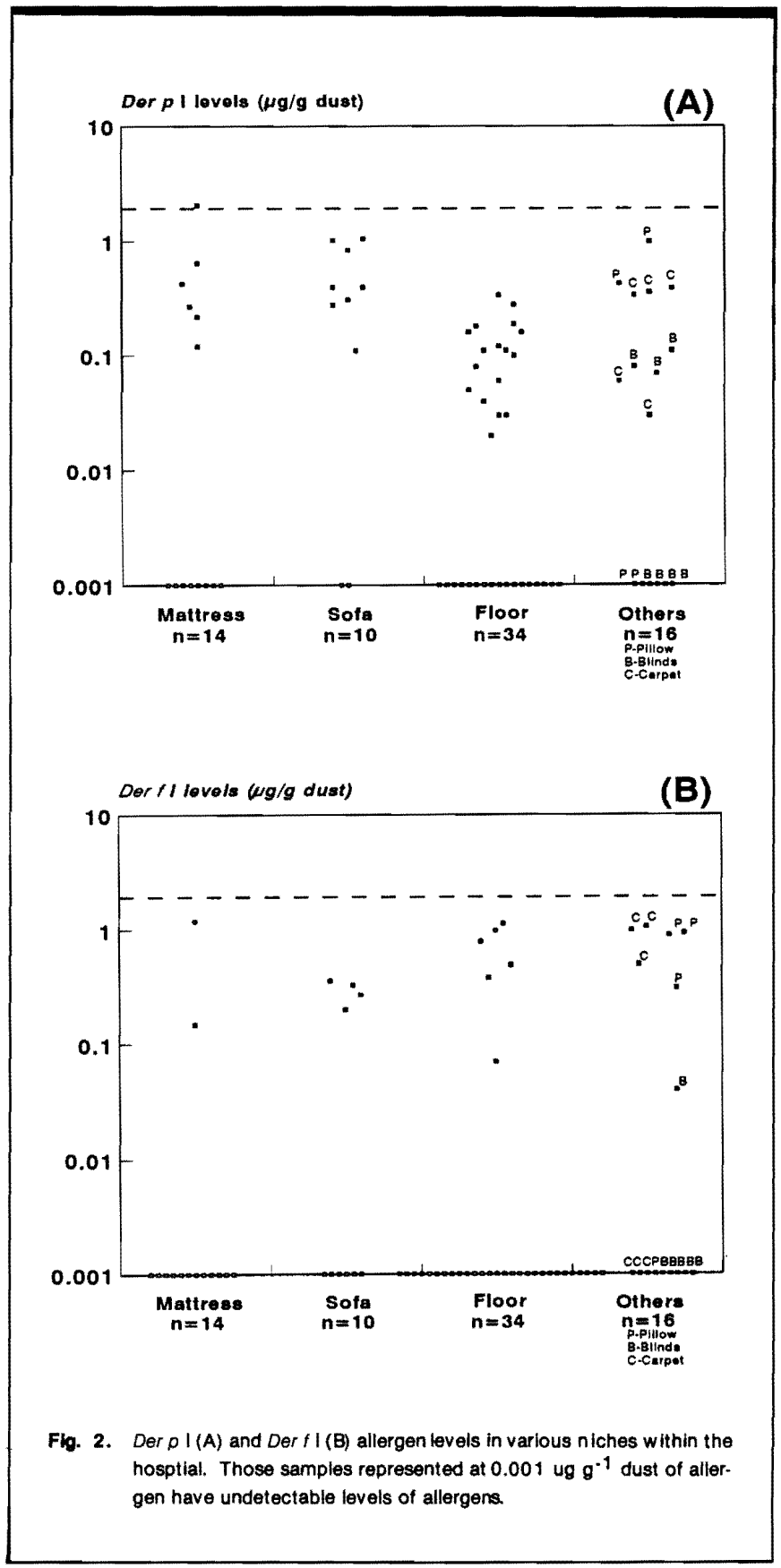

Fig. 2. *Der p* I (A) and *Der f* I (B) allergen levels in various niches within the hosptial. Those samples represented at 0.001 ug g^{-1} dust of allergen have undetectable levels of allergens.

ponses in the airways following exposure to these allergens,[15] and perhaps more importantly by the higher prevalence of asthma in communities where the mite allergens are prevalent[16] or development of asthma in a population following a change in lifestyle which encouraged mite growth.[17]

The development of monoclonal antibodies to the group I allergens of these mites have provided convenient and more direct means of assessment of mite allergen exposure.[18] In this study, we evaluated *Der p* I and *Der f* I levels in dust samples obtained from a general hospital in Singapore so as to obtain information on allergen exposure to our inpatients.

The *Der p* I (53%) was found more prevalent than *Der f* I (26%) in the dust samples from the hospital, and this distribution is similar to that of the homes in Singapore.[4,5] This distribution of *Der p* I and *Der f* I allergens locally is comparable to most parts of the world,[18-24] with the exception of Ohio, USA where *Der f* I was found to be more prevalent.[25] The reason for this geographical distribution of *Der p* I and *Der f* I is uncertain, although the lower humidity in USA favours the growth of *D.farinae.*

Although there was a relatively high prevalence (57%) of these allergens in the hospital, only one out of the 74 samples had an allergen level above the reported sensitising level of 2 $\mu g\ g^{-1}$ dust[12] (Figs. 2A, B), indicating its presence in low levels in the hospital. In sharp contrast, allergen levels in homes were markedly higher, with 40% of homes having levels above the 2$\mu g\ g^{-1}$ dust threshold.[4,5] Similar findings have been reported in a study from the UK, showing lower mite numbers in the hospital compared to the home.[26]

These low allergen levels in the hospital may be attributed to the hospital's cleaning practices and type of furniture used. The use of

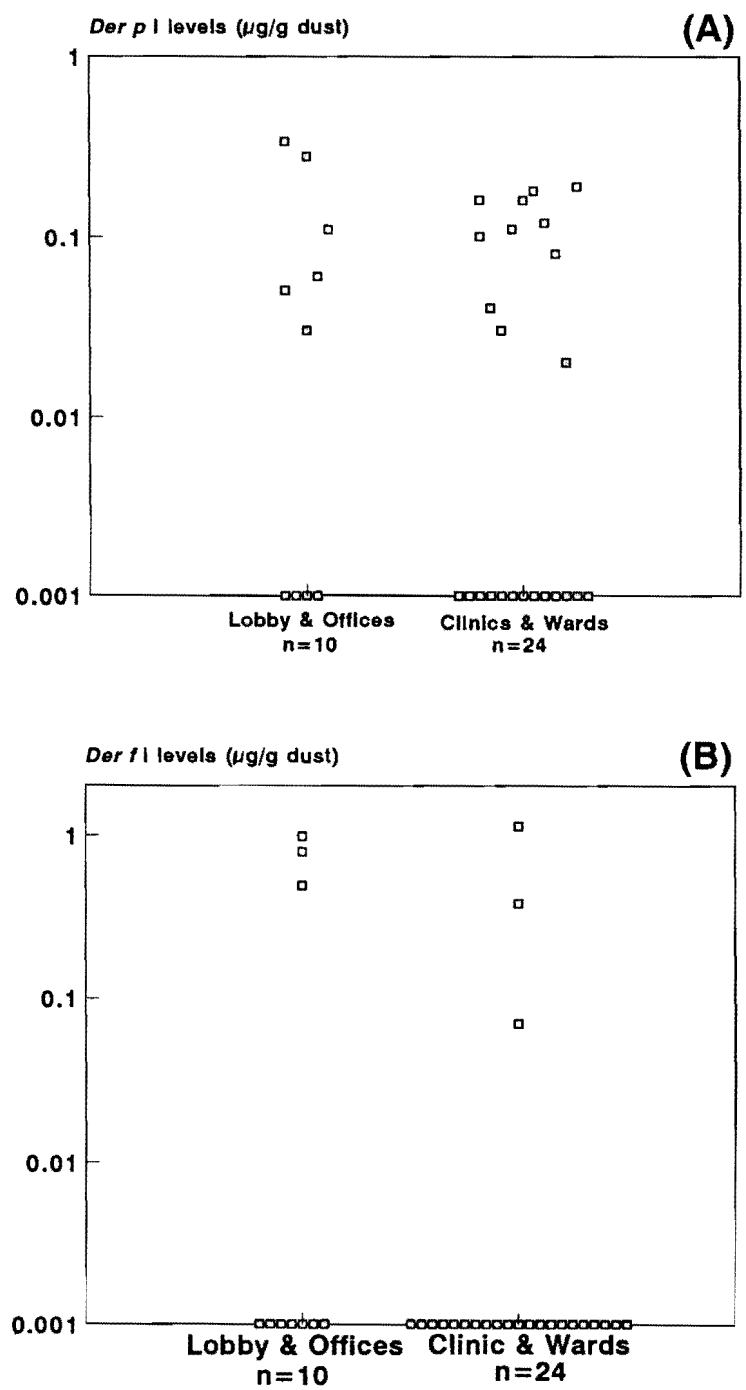

Fig. 3. *Der p* I (A) and *Der f* I (B) allergen levels in various floor locations within the hospital. Those samples represented at 0.001 $\mu g\ g^{-1}$ dust of allergen have undetectable levels of allergens.

Table 1. Allergen levels in dust samples of various locations in the hospital.

Location	n	*Der p* I ($\mu g\ g^{-1}$ dust)			*Der f* I ($\mu g\ g^{-1}$ dust)		
		positive@	Median	Range	Positive@	Median	Range
Overall	74	39	0.03	0-2.05	19	0	0-1.19
Mattress	14	6	0	0-2.05	2	0	0-1.19
Sofa	10	8	0.36	0-1.05	4	0	0-0.36
Floor	34	17	0.01	0-0.34	6	0	0-1.13
Others*	16	10	0.05	0-0.99	7	0	0-1.06

Positive@ - Number of samples with detectable levels of allergens
Others* - Pillows, blinds and carpets.

plastic covers to encase mattresses and pillows, vinyl covers instead of upholstered sofas, and vinyl lined floors in the hospital are important contributing factors. These measures are proven methods for mite avoidance in the home [27] In fact, mite avoidance studies that have not included the encasement of mattresses in plastic encasing and the removal of carpets have not been successful.[27] The frequent cleaning and changing of bed linen in the hospital are also likely to be important. On the other hand, the use of disinfectants for cleaning is unnecessary to keep mite levels down as this practice had been stopped in our hosptial for several years.

Although the overall allergen levels in the hospital were low, significant differences between sampling sites were noted. Lower levels of allergens were found in dust samples from the blinds and floor while those from mattresses and sofas have generally higher allergen levels (Figs. 2A, B). This result is comparative to other mite allergen surveys of homes, where allergen concentrations have been shown to be highest in the mattresses, sofas, and carpets.[4,28] However, the lower levels of allergens in the hospital carpets, as compared to those at homes where mite allergen levels could reach above $10\mu g\ g^{-1}$ fine dust,[4,5] could be attributed to the rigorous and regular vacuuming and shampooing regime of the carpets in the hospital.

Interestingly, the levels of allergen from floors of the lobby and offices were significantly higher than floor samples from other areas of the hospital (Figs. 3A, B), possibly indicating that the allergen may be brought in from the homes of visitors coming to the hospital. We, however, did not sample allergen levels in the clothing of visitors to confirm this.

The results of this study show that mite allergen levels can be controlled despite the conducive tropical climatic conditions of Singapore, and the presence of considerable human activity in the hospital. The type of furnishings and frequent cleaning measures adopted by the hospital appear to be efficient at controlling HDM allergen levels. These measures should therefore be enforced in the home.

ACKNOWLEDGEMENTS

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REFERENCES

1. Voorhorst R, Spieksma FThM, Varekamp H, Leupen MJ, Lyklema AW. The house dust mite *(Dermatophagoides pteronyssinus)* and the allergen it produces: identity with the house dust allergen. J Allergy 1967; 39 : 325.
2. Smith JM, Disney MA, Williams JP, Goels ZA. Clinical significance of skin reactions to mite extracts in children with asthma. Br Med J. 1969; 2 : 723-6.
3. Lee BW, Chew FT, Goh DYT, Lim SH, Tan HTW, Tan TK. House dust mite allergy-Its clinical relevance. J Singapore Paediatr Soc (In press).
4. Chew FT, Tan HTW, Tan TK, Lim SH, GanYY, GoH DYT, Siti Dahlia Bte MD, Lee BW. House dust mite allergens in Singapore homes. Allergy and Clin Immunol News 1994; Suppl 2 : 466.
5. Chew FT, Lee BW, Goh DYT, Lim SH, Quek CM, Tan HTW, Tan TK, Chan KL, Gan YY, Ho TM. House dust mite allergens in Singapore-1. Relationship with household cleaning and hygiene practices. In : The Proceedings of the Singapore Malaysia Congress of Medicine, 1994, pp. 65.

6. Lee BW, Teo J, Vellayapan K. Role of atopy in childhood asthma. J Singapore Paediatr Soc. 1989; 31 : 53-9.
7. Goh DYT, Chew FT, Lee BW, Lim SH, Tan HTW, Tan TK, Gan YY. House dust mite allergen levels in Singaproe schools. 7th Asean Pediatric Federation Conference, Bangkok, 1994.
8. Quek SC, Chong A, Connett GJ, Lee BW. Effects of an acaricide on asthmatic children with house dust mite allergy. Acta Paediatrica Japonica 1994; 36 : 1-4.
9. Chew FT, Lee BW, Goh DYT, Lim SH, Quek CM, Tan HTW, Tan TK, Chan KL, Gan YY, Ho TM. House dust mite allergens in Singapore-1. Effect of an acaricide on the concentration of house dust mite allergens. In : The Proceedings of the Singapore Malaysia Congress of Medicine, 1994, pp. 65.
10. Chapman MD, Heymann PW, Wilkins SR, Brown MB, Platts-Mills TAE. Monoclonal immunoassays for the major house dust mite *(Dermatophagoides)* allergens, *Der p* I and *Der f* I, and quantitative analysis of the allergen content of mite and house dust extracts. J Allergy Clin Immunol 1987; 80 : 184-94.
11. SAS Institute Inc. SAS/STAT user's guide, version 6, fourth edition, volumes 1 and 2, SAS Circle, Box 8000, Cary MB, 27512-80000.
12. Smith TF, Kelly LB, Heymann PW, Wilkins SR, Platts Mills TAE. Natural exposure and serum antibodies to house dust mite allergens in mite-allergic children with asthma in Atlanta. J Allergy Clin Immunol 1985; 76 : 782-8.
13. International Workshop Report. Dust mite allergens and asthma: A worldwide problem. Bull WHO 1988; 66 : 769-80.
14. Sarfield JK. Role of house dust mites in childhood asthma. Arch Dis Child 1974; 49 : 711-5.
15. Cockcroft DW. Mechanism of perenial allergic asthma. Lancet 1983; 2 : 253-6.
16. Charpin D, Birnbaum J, Haddi E, *et al.* Altitudes and allergy to house dust mites-A paradigm of the influence of environmental exposure on allergic sensitisation. Am Rev Resp Dis 1991; 143 : 983-6.
17. Dowse GK, Turner KJ, Steward GA, Alpers MP, Woolcock AJ. The association between *Dermatophagoides* mites and the increasing prevalence of asthma in village communities within the Papua New Guinea highlands. J Allergy Clin Immunol 1985; 75 : 75-83.
18. Kabasawa Y, Ishii A, Murata H, *et al.* Clinical significance of the house dust mite *(Dermatophagoides pteronyssinus)* in asthmatic children in Japan. Acta Allergol 1976; 31 : 442-52.
19. Murray AB, Zuk P. The seasonal variation in a population of house dust mites in a North American City. J Allergy Clin Immunol. 1979; 64 : 266-9.
20. Nadchatram M, Fournier G, Gordon BL, *et al.* House dust mites in Hawaii. Ann Allergy 1981; 46 : 197-9.
21. Gabriel M, Cunningham AM, Allan WG, *et al.* Mite allergy in Hong Kong. Clin Allergy 1982; 12 : 157-71.
22. Rueda LM. House dust mite (Acari) in Malaysia. Philipp Ent 1985; 6 : 428-34.
23. Chang YC, Hsieh KH. The study of house dust mites in Taiwan. Ann Allergy 1989; 62 : 101-6.
24. Malainual N, Chairat P, Chapman MD, Vichyanond P. House-dust mites distribution in Thailand. J Allergy Clin Immunol 1994; 93 (1 part 2) : 254.
25. Arlian LG, Bernstein IL, Gallagher JS. The prevalence of house dust mites, *Dermatophagoides spp,* and associated environmental conditions in homes in Ohio. J Allergy Clin Immunol 1982; 69 : 527-32.
26. Blythe ME, Ubaydi FAL, Williams JD, Morrison Smith J. Study of dust mite in three Birmingham hospitals. Br Med J.1985; 1 : 62-4.
27. Platts-Mills TAE, Chapman MD. Dust mites: Immunology, allergic disease, and environment control. J Allergy Clin Immunol 1987; 80 : 755-75.
28. Platts-Mills TAE, Hayde ML, Chapman MD, Susan RW. Seasonal variation in dust mite and grass-pollen allergens in dust from the houses of patients with asthma. J Allergy Clin Immunol 1987; 79 : 781-91.