The Melbourne House Dust Mite Study: Long-term efficacy of house dust mite reduction strategies

Richard Sporik, DM, MRCP,[a] David J. Hill, FRACP,[a] Philip J. Thompson, FRACP,[b] Geoffrey A. Stewart, PhD,[c] John B. Carlin, PhD,[d] Terry M. Nolan, PhD, FRACP, FAFPHM,[d] Andrew S. Kemp, PhD, FRACP,[e] and Clifford S. Hosking, MD, FRACP, FRCPA[f]

Melbourne, Nedlands, Newcastle, Australia

Background: Asthma severity among mite-sensitized individuals appears to be related to the degree of mite allergen exposure.

Objectives: The objective of this study was to assess the long-term effectiveness of mite avoidance measures in the homes of asthmatic children in Melbourne, Australia.

Methods: The concentration of house dust mite allergen (Der p 1) was measured on the child's mattress surfaces and bedroom floors in 85 homes on 10 occasions over a 16-month period. After the first three visits, all mattresses were covered with a semipermeable encasement, and carpeted bedroom floors were randomly allocated to regular applications of a placebo or an "anti-mite" shampoo.

Results: The concentration of Der p 1 recovered was initially high in the carpeted bedrooms ($n = 66$) (41.1, 95% confidence interval, 30.7 to 55.0 μg Der p 1 per gm) and mattresses ($n = 85$) (39.6, 27.2 to 57.7). During the initial observation period the concentration of mite allergen fell in the treatment, placebo, and parental control groups. During the seven treatment periods, no differences were seen between the Der p 1 concentrations in the groups using the "anti-mite" shampoo, placebo shampoo, or the parental control group (e.g., at visit 4; 12.6, 8.2 to 19.5; 14.8, 8.6 to 25.1; and 12.0, 8.1 to 17.7 μg/gm, respectively). In contrast, the concentration of Der p 1 in samples from uncarpeted floors and mattress encasements was low (4.1, 2.1 to 8.0 μg/gm and 4.2, 2.6 to 6.5 μg/gm, respectively) and insufficient dust for analysis was frequently obtained from these sites.

Conclusions: There was no additional benefit from the use of an "anti-mite" shampoo. The absence of carpets and the use of mattress encasements was a useful long-term strategy for mite allergen avoidance. (J Allergy Clin Immunol 1998;101: 451-6.)

From [a]the Department of Allergy, Royal Children's Hospital; [b]the University Department of Medicine, QEII Medical Centre, Nedlands; [c]the Department of Microbiology, University of West Australia; [d]Clinical Epidemiology and Biostatistics Unit, Royal Children's Hospital; [e]the Department of Clinical Immunology, Royal Children's Hospital; and [f]John Hunter's Children's Hospital, Newcastle.

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Reprint requests: David J. Hill, FRACP, Department of Allergy, Royal Children's Hospital, 151 Flemington Road, Melbourne, Victoria 3051 Australia.

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The majority of house dust mite–sensitized children and young adults demonstrate improvement in their asthma or eczema when moved to a totally mite-free environment.[1-4] However, these studies have usually been carried out at altitude or in hospitals, and attempts to replicate these effects in the homes of asthmatic patients have produced variable benefits.[5] Measurements of the major house dust allergen Der p 1 in patients' homes provides a useful measure of the likely benefits of intervention measures. Concentration thresholds for sensitization (> 2μg/gm) and disease exacerbation (>10 μg/gm of fine dust) have been proposed.[6] We have previously studied Der p 1 concentrations in the homes of 87 asthmatic children living in Melbourne over a 5-month period and found low concentrations in uncarpeted homes and on encased mattresses.[7] We now report the results of a randomized controlled trial of an anti-mite shampoo (Allerite) and an uncontrolled longitudinal study on the effect of mattress encasements in 85 of these homes over an additional 11-month period.

METHODS

One hundred seven children with moderately severe asthma under the care of a single physician were initially enrolled. All were house dust mite–sensitized (skin wheal ≥6 mm, extract *Dermatophagoides pteronyssinus*; Holister Stier, Spokane, Wash.) and all were on long-term preventative inhaled medication at the time of enrollment. The study was approved by the Ethics Committee of the Royal Children's Hospital, and informed consent was obtained.

House dust samples were collected between July 1991 and October 1992. Ten home visits were undertaken for house dust collection: July (Winter), Visit 1; August/September (Winter/Spring), Visit 2; November (Spring), Visit 3; December, Visit 4; February (Summer), Visit 5; April (Autumn), Visit 6; June (Winter), Visit 7; July (Winter), Visit 8; September (Spring), Visit 9; and October (Spring), Visit 10. Each set of visits was completed within a 4-week period. Dust was collected from a predefined area of the child's bedroom floor at all visits and from either the child's mattress or mattress encasement at Visits 1 through 8 and 10. In an attempt to control for confounding domestic factors, dust was also collected from the parents' bedroom floor and mattress at these visits. Parents were asked not to clean the mattresses or floors 1 week before

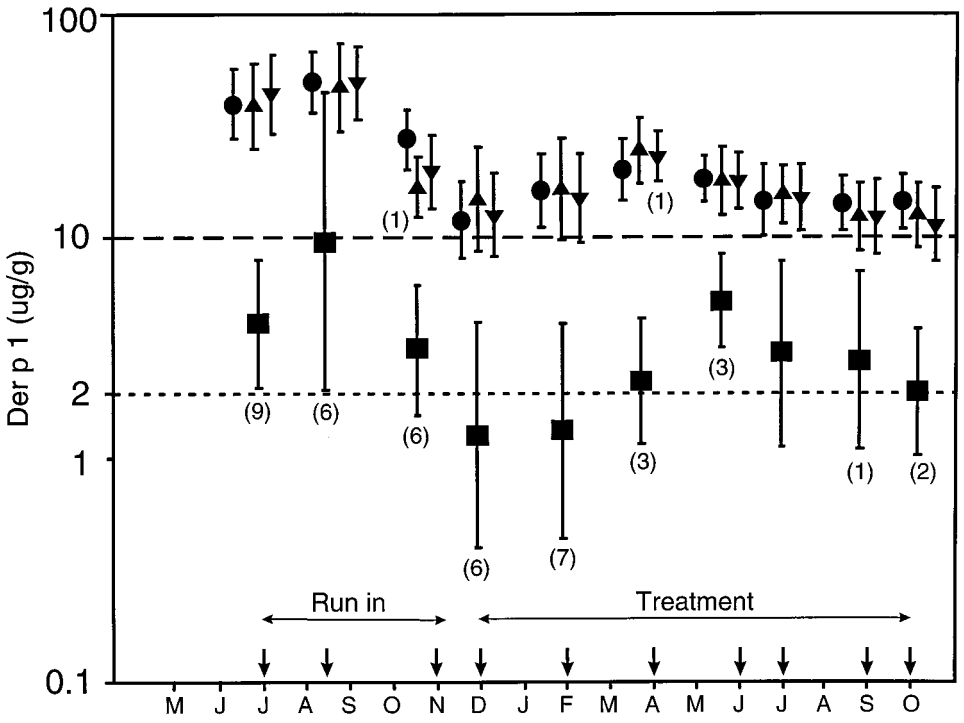

FIG. 1. Concentration of house dust mite allergen (micrograms per gram Der p 1) recovered from bedroom floors (geometric mean and 95% confidence intervals). *Circles,* samples obtained from parents' bedroom (n = 85); *squares,* children's bedroom without a carpet (n = 19); *triangles,* children's bedrooms with carpet in placebo group (n = 33); *inverted triangles,* children's bedrooms with carpet in treatment group (n = 33). Figures in parentheses show number of samples from children's bedroom floors in which insufficient dust (<10 mg) was obtained for analysis. Reference lines at 2 and 10 μg/gm represent provisional threshold concentrations associated with development of sensitization and exacerbations of symptoms, respectively. *Vertical arrows* indicate when samples were collected.

sampling and were encouraged not to clean the house more than twice per week. Mattress surfaces were sampled for 4 minutes, and a 1 m^2 specimen of floor adjacent to the bed was sampled for 2 minutes with a Nilfisk GS90 (700 Watts) vacuum cleaner by using the manufacturer's upholstery nozzle including a plastic insert containing the dust collection bag (Hoover Dustette, Melbourne, Australia). All collected samples were sealed in plastic bags and stored at 4° C until assayed.

After an observational period (Visits 1, 2, and 3) the carpet in the children's bedrooms was randomly allocated to treatment with either an "anti-mite" shampoo (alcohol and glycol ether based; Allerite, Vax) or a placebo shampoo. The treatment used was masked from both the technician applying the shampoo and the participating families. The shampoo was applied after dust collection at each subsequent visit. All uncarpeted floors were also treated with the "anti-mite" shampoo. After the initial dust collection (Visit 1), if the child was already using a mattress encasement, this was removed. After an observational period (Visits 2 and 3), all the children's mattresses and pillows were covered with a new semipermeable encasement (made from high-density polyethylene fibers of Tyvek; DAC Covers, Allersearch). At Visit 10 (final) both the mattress encasement and the mattress were sampled. It was suggested that bed linen and mattress protectors should be washed weekly but that mattress encasements were not to be removed or washed but wiped with Allerite solution at each visit.

Dust samples were sieved, weighed, and extracted (100 mg per 2 ml) with borate-buffered saline containing 0.1% (vol/vol) Tween 20 and 250 U/ml of the serine protease inhibitor aprotonin (Bayer Pharmaceuticals, Sydney, Australia) for 2 hours at 4° C with rocking. When less than 100 mg of dust was obtained, samples were extracted with 0.1 ml of solution per 10 mg of dust. Samples weighing less than 10 mg were not analyzed. The samples were centrifuged, and supernatants were removed and analyzed for Der p 1 content by using a capture monoclonal antibody ELISA essentially as described.[8]

The concentration of mite allergen (micrograms Der p 1 per grams of fine dust) was compared between the children's and parents' bedroom floors and mattresses. Because a high proportion of children had mattress encasements at Visit 1, mattress dust obtained at Visit 3, when all encasements had been removed for 5 months, was used for baseline mattress comparisons. When there was insufficient dust for analysis (<10 mg), samples were excluded. Data were log transformed because of the heavily skewed distribution, and groups were summarized by geometric means and 95% confidence intervals. Parametric methods of comparison (Student's t test, paired and unpaired as appropriate) were used. To estimate the extent to which mite concentrations "tracked" or were serially correlated over time, an autoregressive model was fitted to data from households in which continuous sequences of measurements were available, adjusted for any differences caused by treatment interventions by using a generalized estimating equations method (Stata Statistical Software, Release 5.0; Stata Corporation, College Station, Tex.). In addition, to assess the effects of humidity, the mean monthly relative humidity and temperature

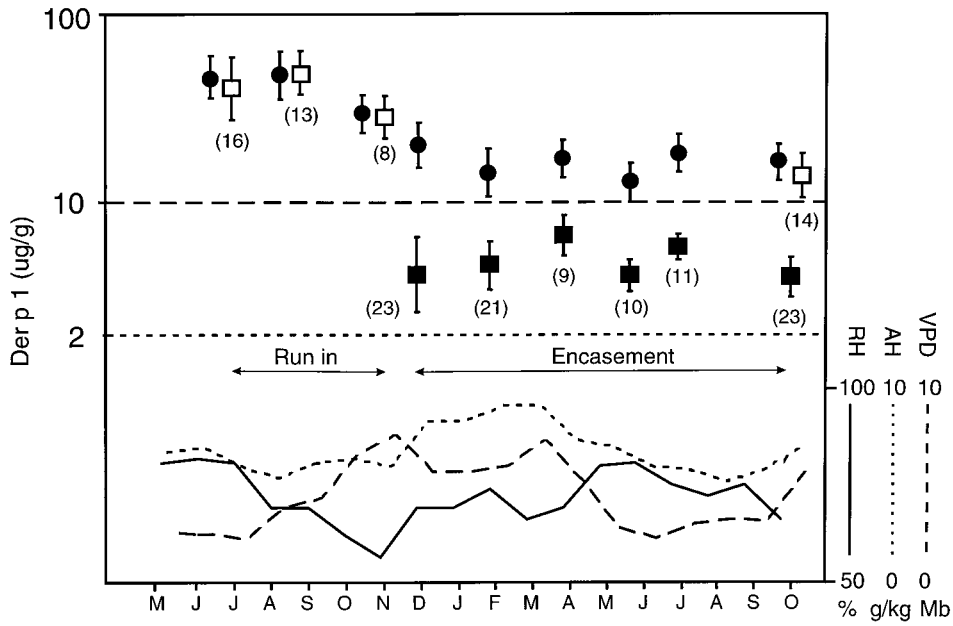

FIG. 2. Concentration of house dust mite allergen (micrograms per gram Der p 1) recovered from mattresses (geometric mean and 95% confidence intervals). *Filled circles*, samples obtained from parents' mattress (n = 85); *open squares,* samples obtained from child's mattress (n = 85); and *filled squares,* samples obtained from surface of child's mattress encasement (n = 85). Figures in parentheses show number of samples from children's mattresses in which insufficient dust (<10 mg) was obtained for analysis. Reference lines at 2 and 10 μg/gm represent provisional threshold concentrations associated with development of sensitization and exacerbations of symptoms, respectively. *Lower panel* represents relative humidity *(RH, solid line)*; vapor pressure deficit *(VPD, long dashes)*; and absolute humidity *(AH, short dashes)*.

(9 AM) were obtained from the Bureau of Meteorology in Melbourne. The specific vapor deficit,[9] an index of the drying power of air, was calculated (Saturation vapor pressure − [relative humidity × saturation vapor pressure/100]), and the absolute humidity was estimated with a psychrometric chart. No domestic humidity measurements were obtained. Correlations between humidity indices and allergen content were obtained with the geometric means of the allergen data values for each visit.

RESULTS

A total of 85 children completed the study. Nineteen children had no carpets in their bedrooms and 66 did, 33 of whom were allocated to the treatment arm. Twenty-two children had mattress encasements fitted at the initial visit. There was no difference in the geometric mean Der p 1 concentration of dust (Visit 1) from the homes of those children (n = 22) who did not complete the study and those who did.

Floors

The concentration of Der p 1 was high in all the initial samples except for those taken from uncarpeted floors. The mean Der p 1 concentrations (and 95% confidence intervals [CIs]) at Visit 1 of all the children's bedroom floor (carpeted and uncarpeted) and parent's bedroom floor samples were 32.0 μg/gm (95% CI, 23.4 to 43.8 μg/gm) and 39.5 μg/gm (95% CI, 27.6 to 56.5 μg/gm), respectively. A correlation was seen between samples from a child and their parents' floor (r = 0.64, p < 0.0001). The initial Der p 1 concentrations from carpeted children's bedroom floor samples were substantially higher, 41.1 μg/gm (95% CI, 30.7 to 55.0 μg/gm), than those recovered from uncarpeted children's floors, 4.1 μg/gm (95% CI, 2.1 to 8.0 μg/gm)(p < 0.0001). In addition, a large number of samples from uncarpeted floors had insufficient dust for analysis (Fig. 1).

During the observation period (Visits 1 through 3), the concentration of mite allergen fell in both the placebo and the treatment groups. During the seven treatment periods, no differences were seen between the Der p 1 concentrations in the groups using the "anti-mite" (e.g., at visit 4: 12.6, 8.2 to 19.5 μg/gm) or placebo shampoos (14.8, 8.6 to 25.1 μg/gm). Similar levels of mite allergen concentration were seen throughout the study in the untreated parents' carpets (e.g., at visit 4: 12.0, 8.1 to 17.7 μg/gm) (Fig. 1). The children with uncarpeted bedrooms continued to have consistently lower floor Der p 1 levels. Of all samples (177) obtained from uncarpeted floors, 24% were insufficient for analysis, 30% contained less than 2 μg/gm, and 63% contained less than 10 μg/gm Der p 1 compared with 0.3%, 6%, and 22%, respectively, of the 626 samples obtained from the carpeted bedroom floors.

Mattresses

The Der p 1 concentrations in the children's mattresses (Visit 3) were similar to those found in the

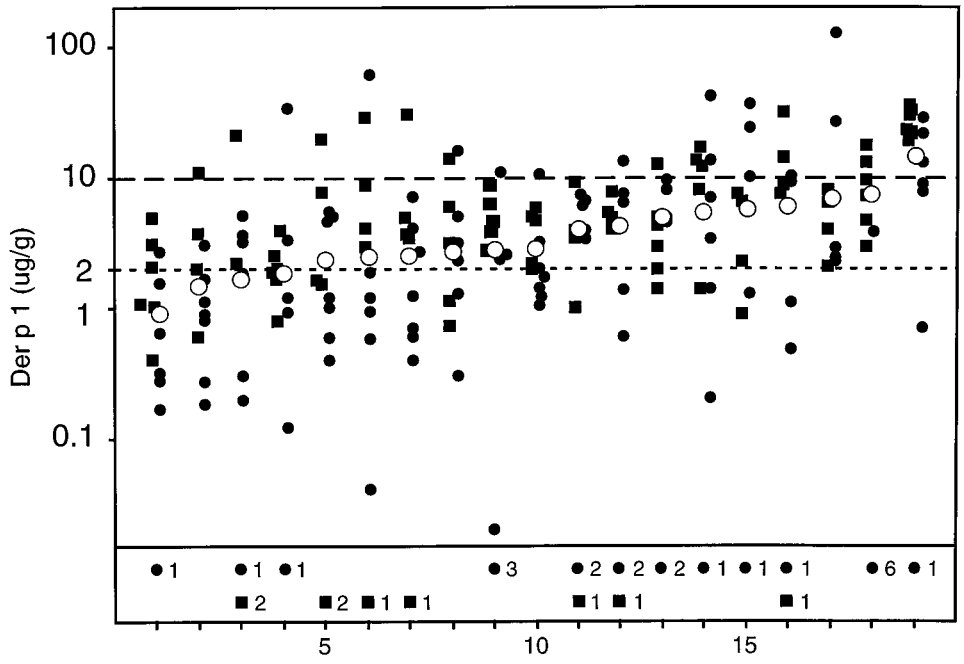

FIG. 3. Ranked concentration of mean house dust mite allergen (micrograms per gram Der p 1) from floor and mattress samples obtained from homes of 19 children with no carpets during period when mattress encasements were being used (Visits 4 through 10). *Closed circles,* samples collected from the floor; *closed squares,* samples collected from the mattress encasements; and *open circles,* the combined geometric mean concentration from each home. Reference lines at 2 and 10 μg/g represent provisional threshold concentrations associated with development of sensitization and exacerbations of symptoms, respectively. Number of samples with insufficient dust are indicated in lower panel; these were not included in analysis.

parents' mattresses: 28.3 μg/gm (95% CI, 22.0 to 36.6 μg/gm) and 29.2 μg/gm (95% CI, 23.2 to 36.6 μg/gm), respectively ($p = 0.87$) (Fig. 2). There was a correlation between Der p 1 levels in the child's and parents' mattresses ($r = 0.49, p < 0.0001$). The concentration of mite allergen from samples obtained from the child's or parents' mattress at Visit 3 was related to that found on their bedroom floors ($r = 0.22, p = 0.062$; $r = 0.41, p < 0.0001$, respectively).

The use of mattress encasements substantially lowered the concentration of Der p 1 recovered: 4.2 μg/gm (95% CI, 2.6 to 6.5 μg/gm) at Visit 4 compared with 28.4 μg/gm (95% CI, 22.0 to 36.6 μg/gm) at Visit 3. The median ratio (and range) of the Der p 1 concentration recovered from the child's mattress at Visit 3 and that recovered from their parent's mattress was 1.05 (range, 0.05 to 10). In contrast, the ratio of Der p 1 concentrations from the child's mattress encasement at Visit 10 and their parents' mattress was 0.36 (range, 0 to 4). When the mattress encasements were then removed, the Der p 1 concentration in dust obtained from the mattress itself had fallen relative to the levels 12 months previously: 13.8 μg/gm (95% CI, 10.5 to 18.1 μg/gm) compared with 28.4 μg/gm (95% CI, 22.0 to 36.6 μg/gm) ($p < 0.0001$). Of all samples (490) obtained from mattress encasements, 20% were insufficient for analysis, 12% contained less than 2 μg/gm Der p 1, and 64% contained less than 10 μg/gm compared with 16%, 2%, and 17% of the 326 samples recovered directly from the children's mattresses, or 3%, 7%, and 32% of the 735 samples recovered from the parents' mattresses.

In the bedrooms of the 19 children with no carpets during the period of using mattress encasements, none achieved year-round levels of less than 2 μg/gm and only two achieved levels less than 10 μg/gm. The overall group mean Der p 1 concentration was 3.5 μg/gm (95% CI, 2.5 to 4.8 μg/gm) during this period (Fig. 3).

Seasonal variation

There was a limited seasonal fluctuation of allergen concentrations with a peak in the winter (July) in samples from uncarpeted floors. This was 2 months after optimal ambient humidity conditions for house dust mite growth (relative humidity >70% and vapor pressure deficit at its lowest). There was some evidence of a correlation between the mean allergen content of the untreated parental bedroom carpets and mattresses and the relative humidity ($r = 0.62, p = 0.054$; $r = 0.62, p = 0.073$) and the vapor pressure deficit ($r = -0.61, p = 0.068$; $r = -0.50, p = 0.15$) but not with the absolute humidity ($r = -0.06, p = 0.88$; $r = -0.21, p = 0.59$) measured 2 months previously. The mite allergen concentrations tended to remain consistently high or low (tracked over time) within households: the estimated correlation between the mite concentrations at successive time points were 0.45 for the child's mattress (based

on 39 homes in which sufficient dust was collected at each visit to provide continuous sequences), 0.53 for the child's bedroom floor ($n = 61$), 0.52 for the parents' mattress ($n = 40$), and 0.65 for the parents' bedroom floor ($n = 60$).

DISCUSSION

The concentrations of house dust mite allergen obtained from the homes of mite-sensitized asthmatic children living in Melbourne were very high.[7] Current asthma treatment guidelines recommend, in addition to pharmacologic treatments, the identification and avoidance of aggravating factors such as house dust mite allergen.[10] The lowest concentrations of mite allergen in this study were found on uncarpeted floors, and, despite generally high levels of mite allergen in the child's mattress, substantial reductions were possible with the use of mattress encasements.[11] In addition, a large proportion of samples from uncarpeted floors and mattress encasements contained insufficient dust for analysis. Although these could not be included in the analysis, it is feasible that the reduction in allergen exposure may be greater than those reported. Both the active and placebo carpet treatments were associated with a fall in allergen concentration of approximately 60% from initial baseline. These changes were also seen in the untreated parents' bedroom. The reasons for this are unclear but may have been due to an increased diligence with cleaning during the study period. The study highlights the necessity of confirming in field trials the efficacy of "anti-mite" products[1, 12] and supports the recommendation of a recent expert panel that these agents should not be recommended routinely.[10]

A recent epidemiologic study[13] found that the risk of current asthma among house dust mite–sensitized children was small when mattress mite allergen exposure was below 7 μg/gm (similar to the previously proposed threshold of 10 μg/gm). Above this threshold, there was a quadrupling of risk for each doubling of mite allergen concentration. The levels found on mattress encasements and on uncarpeted floors were generally below the level associated with this increased risk of asthma and should be of clinical benefit.[13-17] The Der p 1 allergen concentrations were not, however, low enough to prevent sensitization,[18] suggesting that by using these methods, total mite allergen avoidance is not possible in every home in an environment highly favorable to mite growth.[5, 19]

Our avoidance regime might be improved by hot washing (>55° C) or treating the blankets and mattress encasements with acaricides each month.[20, 21] The house dust mite is critically dependent on the moisture content of air for hydration and survival.[9, 22] The finding that some homes had consistently high or low mite allergen concentrations may be explained by reduced ventilation and increased dampness. This was supported by the significant correlation between the allergen concentrations on floors and mattresses in the same house. However, because we were unable to collect data on indoor humidity, this remains speculative. The epidemiological consequence of our finding is that a single estimation of house dust mite allergen concentration may be a reasonably reliable way of estimating whether exposure has been high or low with regard to the community mean over the preceding year.

From this study, measures of relative humidity rather than absolute humidity would appear the better predictor of mite allergen concentration. To achieve total domestic mite avoidance, studies have suggested that depending on the local climate, reduction of indoor humidity by increasing ventilation[23, 24] or effective constant dehumidification[25] may be necessary. We are unable to say whether dehumidification would be effective or cost effective in Melbourne.

In conclusion, we have shown no additional benefit from the use of an "anti-mite" shampoo. Simple avoidance measures (the absence of carpets and mattress encasement) were associated with low concentrations of mite allergen. However, where conditions are ideal for mite growth, additional measures (e.g., hot washing of encasements and reduction in indoor humidity) may be required to keep levels consistently low.

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Correction

The following correction applies to the abstract by Seminario and Bochner entitled "Eosinophils (EOS) possess intracellular α-chain of the high affinity IgE receptor but little or no expression on their surface," which appeared in volume 101, p. S175, 1998, of The Journal.

The sentence that reads "Surface biotinylation of PB EOS followed by IP with mAb 22E7 and blotting with streptavidin again failed to detect γ-chain on the surface of EOS" should read "Surface biotinylation of PB EOS followed by IP with mAb 22E7 and blotting with streptavidin again failed to detect α-chain on the surface of EOS."