
Decay of house-dust mite allergen Der f 1 at indoor climatic conditions

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Background: The decay of house-dust mite allergens is important for the outcome of avoidance measures for house-dust mite-allergic patients.

Objective: To quantify the stability of Der f 1 from mattress dust when exposed to domestic conditions.

Methods: Three samples of mattress dust were individually homogenized and divided into 64 subsamples. Mites were killed by freezing for 48 hours at -30°C . The subsamples were exposed in eight homes, three storerooms, and one greenhouse, where temperature and relative humidity were recorded. Der f 1 was determined in extracts of subsamples (enzyme-linked immunosorbent assay) at 0, 3, 12, and 24 months.

Results: In the three samples of mattress dust, the initial concentrations of Der f 1 (mean $\pm$ standard deviation; STD) were: 169 (12), 3.9 (0.4), and 31 (2.6) $\mu\text{g/g}$, respectively. Median half-life of Der f 1 in the mattress dust samples was 10 years in the exposure homes, 18 years in the store rooms, and 1.0 year in the greenhouse. No correlations among preserved Der f 1 and temperature, relative humidity, and absolute humidity in homes were found (Spearman rank correlation test).

Conclusion: Natural decay of Der f 1, with an estimated half-life of 10 years at housing conditions, has no practical consequence in reducing allergen exposure. Therefore, avoidance measures should include an active removal of the allergens.

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INTRODUCTION

House-dust mite (HDM) allergens in patients' environment play an important role in the development of nasal and bronchial hyperreactivity.¹ A substantial reduction in the allergen load can reduce the risk of sensitization,^{2–4} and symptoms can be alleviated in HDM-allergic patients.^{5–7}

The balance of supply and elimination of allergens results in the allergen load. If the HDMs are killed, the production of their allergens will discontinue, but dead mites, fecal, and cuticular fragments still contain HDM allergens.⁸ These allergens can be physically removed from the environment or they can lose their activity by degradation. Knowledge of the natural decay of the major HDM allergens is therefore an important parameter for the planning of avoidance measures and intervention strategies. Longtime studies of the decay of HDM allergens have been performed on natural dust,^{9,10} but in none of these was the dust exposed in dwellings.

The aim of the present study was to evaluate the decay of HDM allergens from mattress dust of HDM-allergic patients, when exposed to the climatic conditions of ordinary homes and climate controlled rooms for 2 years. Der f 1, the major

allergen from *Dermatophagoides farinae*, served as our model allergen.

METHODS

Material

Dust from three HDM-allergic patients' mattresses (6 to 10 years old) with high concentrations of Der f 1 was vacuumed for 1 hour to get sufficient material (sample size, 36 to 69 g). The dust then passed an 18-mesh metal sieve for removal of large particles. To homogenize the dust of each sample, we used a modified procedure designed for subdividing samples of coal.¹¹ The dust sample was spread out on baking-paper and the dust was shoveled from the edge toward the middle all the way around to make a pile in the center; the pile was then pressed down. The process was repeated six times. We used baking-paper as pad and shovel because it did not induce any visible static electricity.

Each of the three samples of mattress dust was divided with a spatula into 64 subsamples in 50-mL polypropylene test tubes. Weight of samples was noted before and after the exposure. Tightly woven textile, AllerGuard (Beirholms, Singapore, Republic of Singapore), was used to "seal" the openings of the tubes. This material is used for mattress- and duvet-covers, and according to the manufacturer, is permeable for humidity but retaining 99.7% of allergens 0.5- μm in size. All samples were frozen for 48 hours at -30°C to kill mites.

Exposure Sites

The tubes were mounted in eight homes and in four climate-controlled rooms. In all 16 series of tubes were placed, as

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series in the climate rooms were in duplicate. Every series comprised three subsamples of each of the three mattress dust samples.

The eight homes of nonsmoking, nonallergic persons were two city apartments, four suburban houses, and two rural one-family houses. Three of the climate controlled rooms were storerooms at the Conservation Department of the National Museum of Denmark, Lyngby (1 kept relatively cold, 1 dry, and 1 humid) and 1 was a warm and humid greenhouse at the Botanical Garden, Copenhagen.

At initiation of the study (May 1999) 16 subsamples of each of the three mattress dust samples were extracted and analyzed. After 3, 12, and 24 months of exposure, a subsample of each of the three mattress-dust samples from the different homes and climate rooms was likewise extracted and analyzed.

Monitoring of Temperature and Humidity

In the eight homes the temperature and relative humidity was measured every 2 hours with an electronic thermometer and hygrometer coupled to a data-logger (Tinytag Ultra TGU-1500, Chichester, England). In the storerooms and the greenhouse thermo-hydrographic records were read weekly.

Processing of Subsamples

Individually weighed subsamples of mattress dust were extracted (0.125 M ammonium hydrogen carbonate + 0.1 M ϵ amino caproic acid + 0.1% sodium azide) and stored at -20°C until analyzed. The concentration of Der f 1 in the supernatant was evaluated in duplicate by enzyme-linked immunoadsorbent assay (ALK-Abelló, Hørsholm, Denmark).¹²

Evaluation of Der f 1 Half-Life ($T_{1/2}$)

If the concentration of Der f 1 decreases at a constant proportion, the logarithm of the concentration of Der f 1 will decrease proportional to time. We estimated the $T_{1/2}$ of Der f 1 from the slope of the regression line, where $\log(\text{Der f 1})$ at 0, 3, 12, and 24 months versus time was used individually for each mattress dust sample in each series. For time 0, however, we used results from all 16 subsamples from each mattress dust sample and assigned these to all sites of exposure.

Statistics

The statistical software package of SigmaStat 2.03 (SPSS, Chicago, IL), and Quattro Pro 8 (Corel, Ottawa, Canada) were used.

RESULTS

The mean concentrations (+ standard deviation) of Der f 1 at time 0 in the three mattress dust samples were 169 (12) $\mu\text{g/g}$, 3.9 (0.4) $\mu\text{g/g}$, and 31 (2.6) $\mu\text{g/g}$, respectively (Fig 1).

Median $T_{1/2}$ of Der f 1 in the mattress-dust samples was 10 years in the homes, 18 years in the store rooms, and 1.0 year in the greenhouse (Table 1).

No correlation was found between preserved Der f 1 (expressed as the 24th month result/mean of 0-months in %) and exposure mean, maximum respective 95% percentile temper-

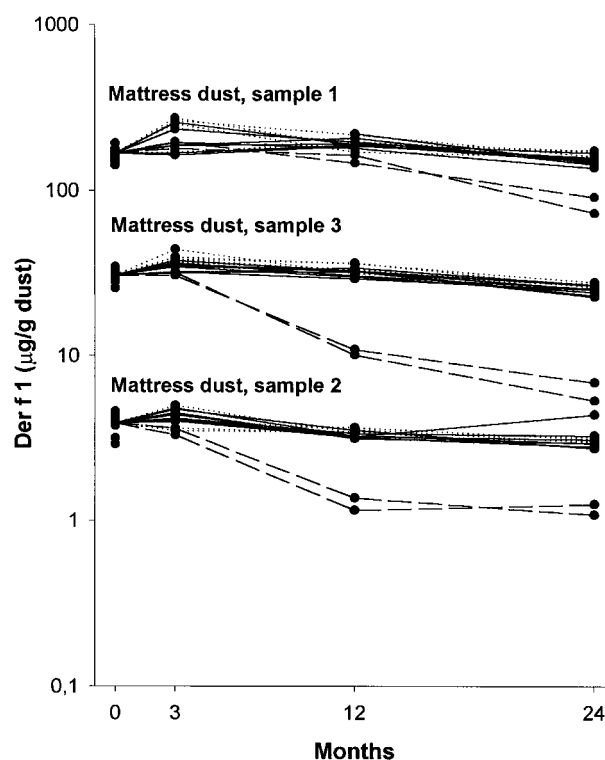


Figure 1. Concentration of Der f 1 at 0 and after 3, 12, and 24 months of exposure in homes, store rooms and greenhouse. Lines for each exposure series start at the initial (unexposed) mean concentration of Der f 1 in the three mattress dust samples. Homes:—; Storerooms, National Museum:.....; Greenhouse:---.

ature; mean relative humidity; or mean absolute humidity in homes (Spearman rank correlation test).

DISCUSSION

Knowledge of the decay of HDM allergens in the indoor environment provides information on how actively the HDM allergen reservoirs have to be removed from the homes of HDM-allergic patients. In this study we used homogenized mattress dust collected from the bedding of HDM-allergic patients with high concentrations of Der f 1, and exposed it for 2 years in ordinary homes and climate-controlled rooms.

Most studies on HDM allergen stability have been made on standardized HDM allergen extracts, produced for diagnostic purpose or for the use in HDM-specific immunotherapy.¹³⁻¹⁷ These studies show that HDM allergens lose substantial allergen activity when stored at room temperature. In such extracts the HDMs originated from cultures and were suspended in different solutions, a situation fundamentally different from what is found in the homes of the HDM-allergic patients. Attempt to evaluate the decay of Der p 1 and Der p 2 in house dust over years has been made in the laboratory, where Der p 1 and Der p 2 were found to be very stable, but no estimate of $T_{1/2}$ was given.^{9,10} We found an estimated median $T_{1/2}$ of 10 years for Der f 1 in mattress dust, and it was

Table 1. $T_{1/2}$ (Years) of Der f 1

	Sample 1	Sample 2	Sample 3	Median
Homes				
1	154	∞	11	
2	105	7.4	15	
3	220	4.5	7.7	
4	∞	5.7	20	
5	31	4.8	9.4	
6	10	4.1	6.2	
7	∞	4.9	6.8	
8	30	4.7	7.1	
Median	129	4.8	8.6	9.9
Storerooms				
1a	∞	6.9	133	
1b	73	7.0	11	
2a	∞	7.7	21	
2b	287	5.8	19	
3a	1241	4.3	16	
3b	∞	5.4	14	
Median	∞	6.3	18	18
Greenhouse				
1a	2.0	1.0	0.8	
1b	2.6	1.0	0.9	
Median	2.3	1.0	0.8	1.0

$T_{1/2}$ was calculated from the slope of the regression line (x: time; y: Der f 1 concentrations); if the slope was ≥ 0 , $T_{1/2}$ was classified as ∞ .

unaffected of the temperature and humidity in the dwellings where it was exposed. Enhanced decay, however, was observed in the samples exposed in a warm and humid greenhouse. The faster decay of allergens exposed in the greenhouse may be attributable to preexisting enzymes or molds, activated under these conditions. The HDM allergens of group 1, 3, 6, and 9 are proteolytic enzymes and might have been able to contribute to the degradation.^{18,19} Also, the exposure to light and noxious gases may be different in the greenhouse²⁰ compared with the homes.

The 3-month subsamples from the mattress dust showed a trend toward higher Der f 1 concentrations compared with the 0-month subsamples, but this only became significant for sample 1. Such a tendency could be caused by release of allergens from newly killed mites or could be a consequence of the interassay-variation (which is <10%).²¹ That the mites survive the freezing procedure is a less likely explanation.²² The allergen concentrations were not relevantly influenced by the negligible change in the weight of the dust sample during this study.

CONCLUSION

The $T_{1/2}$ of the HDM allergen Der f 1 from mattress dust, when exposed in dwellings, was estimated to be approximately 10 years. The natural decay at housing conditions is slow and seems not to be increased by realistic changes in the indoor climate. This natural decay of Der f 1 has no practical consequence in reducing allergen exposure.

Therefore, avoidance measures should include an active removal of the allergens.

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