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Correlations between levels of mite and cat allergens in settled and airborne dust

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*Thirty homes in Rochester, Minn., 20 of which housed cats, were studied to compare cat and mite allergen concentrations in airborne and settled dust. With Air Sentinels in the bedroom and living room for airborne collections, and a Sample Vac for collections from living room carpet and bedroom mattress, immunochemical quantifications of each were made with various radiometric assays with polyclonal and monoclonal antibodies. The most significant correlations were found between airborne mite in the bedroom and living room ($p < 0.001$) and airborne mite in the bedroom and dust from the bedroom mattress ($p < 0.001$). Most houses had specific epitopes of both *Dermatophagoides pteronyssinus* and *D. farinae*, but *D. farinae* was present in greater amounts. In seven houses we found $>10 \mu\text{g}$ of group I mite allergen per gram of settled dust, indicating that some houses in Minnesota have concentrations of mite allergens high enough to cause allergic disease, even in the winter. Clinical interpretation of these data on air levels is hampered by uncertainty as to whether symptoms are more closely related to average steady-state exposure, which we measured, or to brief heavy concentrations. Further epidemiologic studies are needed to define the concentration of mite and cat allergens in the air that are risk factors for allergic disease. The concentration of cat allergen correlated with mite allergen in the air, but not in settled dust, presumably reflecting the fact that both are influenced by ventilation rate. (J ALLERGY CLIN IMMUNOL 1989;83:776-83.)*

It has long been assumed that mite allergens in settled house dust are similar to allergens in the air, and the amount of allergen a patient breathes, a function of the concentration in the air, correlates with the

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Abbreviations used

- Der p I*: Group I allergen from *Dermatophagoides pteronyssinus* (Dp)
Der f I: Group I allergen from *D. farinae* (Df)
Der p I Allergens in whole mite extract containing stated amount of *Der p I* measured by an antibody that cross-reacts with *Der f I*
Fel d I: Major allergen from cat *Felis domesticus*
 MAb: Monoclonal antibody

amount in settled dust.¹ This hypothesis has not yet been tested directly. Therefore, we measured the concentration of mite and cat allergens in the air of the bedroom and living room and in the dust collected from the mattress and living room carpet. As expected, we did find a correlation between airborne allergen concentrations in the two rooms, between mite allergen in the air and dust in each room, and between cat and mite allergens in the room air. The correlations were, however, only partial. Concentration of a pollutant, including allergens, is determined by the ratio between the rate of production and the rate of removal. Ventilation of the dwelling and other removal factors, particularly the settling characteristics of the particles, are important in determining the concentration of allergens in indoor air.

MATERIAL AND METHODS

Houses

Many individuals who volunteered their homes were allergic to house dust mite, and the households cannot necessarily be considered to represent a random cross-section of houses in the community. Basic information was collected about each house, including its age, heating system, air purification, humidification, pets, number of occupants, and frequency of house cleaning. Houses were excluded that had waterbeds, carpeting less than 6 months old, or mattresses less than 12 months old. Twenty houses were chosen because they contained at least one cat. All houses but one had forced-air heating systems. None was equipped with electronic or high-efficiency particle-filter devices. Measured humidity was quite uniform between extremes of 30% and 50%. Participants were instructed not to clean the house during the sampling period.

Dust collection

The sampling took place during two separate visits to each home during November and December of 1986 and January 1987. On the first visit, high-volume air samplers* operating at 200 L/min were placed in the living room and bedroom and operated for 24 hours. This instrument collects >99% dust particles >0.1 µg onto a polytetrafluoroethylene filter. The next day a 2 m² area was vacuumed in the living room in front of the sofa for 3 minutes at approximately 600 L/min. This specially designed vacuum† consisted of a dust-collecting chamber made of polyvinyl chloride. Dust was retained in the chamber with the same polytetrafluoroethylene filter medium used in the air sampler. The nozzle end of the vacuum was a standard 4 inch flared vacuum cleaner attachment. The vacuuming was then repeated on the bedroom mattress (also 2 m²) for 3 minutes. The attachments were cleaned between living room and bedroom vacuuming.

*Air Sentinel, Quan-Tec-Air, Inc., Minneapolis, Minn.

†Sample Vac, Quan-Tec-Air, Inc., Minneapolis, Minn.

MAB assays

Two-site assays for *Der p* I and *Der f* I were performed with radioiodinated 4C1 MAb, which reacts with epitope common to *Der p* I and *Der f* I according to the method of Chapman et al.² MAbs to *Der p* I (5H8) and *Der f* I (6A8) were absorbed (1 ng per well) to the surface of Immulon II microliter plates (Dynatech, Alexandria, Va.). Mite-reference allergens (*Dp* and *Df*) from Hollister-Stier Laboratories, Spokane, Wash., calibrated as 117 µg and 115 µg *Der p* I equivalents, respectively, were used in the assays. Fifty microliters of the mite standard at various concentrations were added to the wells along with 50 µl of diluting buffer.

Other wells contained the air- and dust-extract samples. After overnight incubation at room temperature, the wells were washed, and 30 ng of radioiodinated 4C1 MAb in 100 µl was added to each well. Overnight incubation and washing yielded bound 4C1 MAb proportional to the amount of bound *Der p* I and *Der f* I antigens.

Polyclonal antibody assays

Rabbit two-site assay. The samples were analyzed for mite concentration with a two-site radioimmunoassay. A rabbit antibody with high titer of precipitating antibody to many antigens in *Dp* (ALK, Copenhagen, Denmark) was used. To prepare affinity-purified antibody, 1 ml of this rabbit antiserum was incubated with solid phase *Dp* mite extract (ALK) linked to 1 gm of Sepharose 4B in a chromatography column. The column was washed free of unbound protein, and the antibody was eluted with 0.1 mol/L of glycine buffer, pH 2.7. The affinity-purified antibody was labeled with ¹²⁵I by chloramine-T. For the two-site assay, Immulon II wells were coated with 0.1 ml of the unfractionated rabbit serum diluted 1:1000 in 20 mmol/L of carbonate buffer, pH 9.2, and unoccupied protein binding sites on the wells were blocked with normal rabbit serum. After wells were washed, 0.1 ml of either the reference allergen dilution, air-filter eluates containing unknown amounts of allergen, or internal standards were added. The plates were incubated at 20°C for 18 hours and then washed. Then 0.1 ml of the affinity-purified radioiodinated antibody was added. After 4 hours of incubation, wells were washed and counted. The standard was a *Dp* extract prepared by Hollister-Stier Laboratories. This extract contained 7.2 IU³ and 117 mg/ml of antigen *Der p* I equivalent.⁴ In a cross-inhibition assay, a similar extract of *Df* from Hollister-Stier Laboratories was compared to the *Dp* extract. Inhibition curves (data not presented) were parallel, indicating that the rabbit antiserum binds primarily epitopes that are common to the two species and that the assay measures allergens contributed by both species. Group I allergens are under represented compared to the monoclonal and RAST-inhibition assays.

Dermatophagoides RAST inhibition. The RAST-inhibition procedure was carried out as described previously.⁵ Briefly, *Dp* extract from Hollister-Stier Laboratories was diluted in 200 mmol/L of bicarbonate buffer, pH 9.2,

TABLE I. Micrograms of *Dermatophagoides* allergens: *Der p* I or *Der f* I or *Der p* I equivalents

House No.	Air ($\mu\text{g [DA]} \times 10^{-6}/\text{m}^3$)							
	Living room				Bedroom			
	Monoclonal		Polyclonal		Monoclonal		Polyclonal	
	<i>Der p</i> I*	<i>Der f</i> I†	Rabbit‡	Human§	<i>Der p</i> I	<i>Der f</i> I	Rabbit	Human
1	ND	ND	10	ND	ND	ND	11	ND
2	ND	ND	9	ND	ND	ND	8	ND
3	ND	ND	15	ND	ND	ND	9	ND
4	ND	ND	17	ND	ND	ND	20	ND
5	ND	ND	11	ND	ND	ND	15	ND
6	ND	ND	10	ND	ND	ND	15	ND
7	ND	ND	12	ND	ND	ND	11	ND
8	ND	ND	12	ND	ND	ND	11	ND
9	ND	ND	13	ND	ND	ND	20	ND
10	ND	ND	26	ND	ND	ND	15	ND
11	18.5	27.4	14	496	18.5	21.6	16	605
12	22.0	31.4	33	737	13.9	29.6	29	565
13	19.6	17.2	97	724	13.6	20.6	39	684
14	22.4	42.9	33	559	16.4	29.6	25	723
15	53.2	78.2	164	1251	63.6	54.1	409	875
16	18.6	23.5	22	615	21.0	48.2	27	610
17	20.1	44.5	25	703	21.8	29.4	24	739
18	22.0	39.4	25	714	18.6	45.4	32	760
19	19.0	19.7	22	836	19.1	20.6	23	881
20	19.9	30.4	24	706	15.1	22.5	18	676
21	37.7	64.2	44	680	17.5	39.5	30	808
22	22.0	28.7	25	747	18.5	29.3	22	648
23	12.7	15.5	BD	769	13.4	19.9	23	730
24	BD	15.4	BD	755	BD	BD	BD	670
25	13.4	BD	21	713	15.2	15.3	28	678
26	30.4	69.7	92	625	28.8	36.2	48	527
27	16.4	24.5	20	731	BD	BD	51	705
28	26.7	48.5	77	684	20.2	40.8	48	626
29	BD	15.3	56	619	BD	BD	29	668
30	14.5	22.3	56	849	23.4	28.8	109	808
Mean $\pm$ SD	23 $\pm$ 9.7	35 $\pm$ 19.0	34 $\pm$ 34.7	726 $\pm$ 149.7	21 $\pm$ 11.6	31 $\pm$ 11.2	37 $\pm$ 58.6	699 $\pm$ 94.6
Detection limit	0.4	0.4	0.4	4.0	0.4	0.4	0.4	4.0

DA, *Dermatophagoides* allergens; ND, not done; BD, below detection.

**Der p* I monoclonal 4C1 assay.

†*Der f* I monoclonal 4C1 assay.

‡Rabbit *Dp-Der p* I equivalent/affinity-purified rabbit *Dp* assay.

§*Dp* RAST inhibition-*Der p* I equivalent assay.

and absorbed onto the Immulon plates and washed; 50 μl aliquots of the *Dp* mite standard or air and dust extracts, along with 50 μl aliquots of an undiluted human serum pool containing IgE to mite, were incubated overnight. After plates were washed, rabbit ^{125}I anti-IgE (20 ng per well) was added, and the plates were incubated overnight. The wells were washed again and counted. In this assay, too, the inhibition curves of reference preparations of the two mite species were parallel.

Fel d I assay. *Fel d* I (National Institutes of Health reference reagent) was absorbed to the surface of Immulon II wells. For the generation of the standard inhibition curve, the same reagent was added to washed wells at varied concentrations in 50 μl aliquots. Fifty microliters of undiluted human serum containing IgE antibodies to cat were added to the wells containing *Fel d* I or extracts of air or dust and incubated overnight. The wells were washed, and 100 μl of radioiodinated affinity-purified rabbit anti-IgE (20 ng)

Dust (μg [DA]/gm of dust)

Living room				Bedroom			
Monoclonal		Polyclonal		Monoclonal		Polyclonal	
<i>Der p</i> I	<i>Der f</i> I	Rabbit	Human	<i>Der p</i> I	<i>Der f</i> I	Rabbit	Human
0.2	3.9	3.5	ND	0.3	21.6	3.3	ND
0.2	2.6	29.8	ND	0.5	12.5	2.3	ND
BD	2.6	33.0	ND	0.02	0.9	2.1	ND
0.1	4.4	1.3	0.7	0.9	34.2	273.0	BD
0.2	0.5	2.4	BD	BD	2.5	47.0	BD
1.6	11.7	7.4	7.9	3.7	19.8	35.0	BD
44	66.9	34.5	73.2	33	147.1	88.0	29.0
0.1	1.6	2.3	1.9	2.9	0.8	2.0	7.5
1.6	7.2	2.5	3.2	BD	9.2	12.8	3.0
BD	0.3	1.7	BD	BD	0.4	5.3	BD
0.1	0.1	9.4	BD	0.1	0.2	11.8	BD
BD	0.7	11.3	BD	BD	21.4	55.8	BD
1.0	0.1	255.0	8.8	0.9	2.8	61.0	6.0
BD	0.9	11.3	BD	BD	4.4	4.7	0.5
0.6	0.9	74.7	1.9	17.1	12.0	901.0	22.8
0.1	19.3	44.4	3.6	0.4	6.4	76.7	BD
BD	2.4	9.4	BD	0.1	1.4	20.3	2.1
BD	0.2	56.6	BD	0.1	2.1	20.6	0.5
BD	BD	3.4	0.6	0.1	2.6	15.6	0.7
BD	3.6	13.3	BD	0.03	4.3	4.6	0.5
BD	1.4	4.5	0.5	0.02	0.6	6.1	0.6
BD	0.7	2.3	BD	0.02	1.5	4.5	0.4
0.8	5.8	55.5	1.8	0.8	3.0	41.4	1.5
0.1	0.2	4.7	BD	BD	BD	7.7	BD
BD	0.1	5.9	BD	0.03	0.1	9.9	BD
0.2	2.3	19.2	0.7	1.2	3.0	50.0	1.8
BD	0.3	5.2	BD	BD	1.0	3.2	BD
0.03	0.6	8.5	BD	0.1	1.0	10.1	0.3
BD	0.1	1.0	0.5	BD	0.3	2.0	0.2
0.1	0.1	12.4	BD	1.5	1.0	52.2	4.1
3 ± 0.6		5 ± 12.6	24 ± 47.7	2.9 ± 7.6	11 ± 27.5	61 ± 166.9	5 ± 8.3
0.005		0.005	0.02	0.005	0.005	0.005	0.02

was added to the wells. The next day, the wells were washed and counted. The standard curve was constructed with the *Fel d* I reference reagent, and the results were expressed in *Fel d* I units.

Statistics

RAST inhibition and two-site assays were calculated by a regression equation with a Hewlett-Packard 9 E 45 P computer (Hewlett-Packard Co., Cupertino, Calif.). Spearman's correlation coefficients were calculated with a StatPac Gold program for the IBM PC (International Business Machines Corp., Armonk, N.Y.).

RESULTS

With the MAb assays, we found that most houses had the specific epitopes of group I allergens of both *Dp* and *Df* (Table I). Twenty-five of the 30 houses had $>1 \mu\text{g}/\text{gm}$ of group I allergen in either the carpet or mattress. In two houses, this was found only in the carpet, in 10, only in the mattress, and in 13, in both carpet and mattress. In no house was the elevation caused by *Dp* only. Elevation was caused by *Df* only in 16 houses, and in six houses, both species were elevated. Seven houses had at least one value $>10 \mu\text{g}/\text{gm}$, and in five of these houses, the elevation was

TABLE II. Correlation coefficients for mite concentration in different locations in the houses by different assays

	BR	LR	Air	Dust
	Air vs dust	Air vs dust	BR vs LR	BR vs LR
Monoclonal				
Der p I (5H8)	0.84*	0.18	0.84*	0.89*
Der f I (6A8)	0.37	0.06	0.69†	0.95*
Rabbit polyclonal	0.92*	0.47†	0.81*	0.23
RAST inhibition	0.47‡	0.10	0.63†	0.31

BR, bedroom; LR, living room.

* $p < 0.001$.† $p < 0.01$.‡ $p < 0.05$.**TABLE III.** Correlation coefficients between the four mite assays by location in the houses

	Air		Dust	
Assays	LR	BR	Carpet	Mattress
1* vs 2†	0.85‡	0.78‡	0.44	0.30
1 vs 3§	0.73†	0.83‡	0.43	0.94‡
1 vs 4	0.49¶	0.34	0.69#	0.90‡
2 vs 3	0.65#	0.52¶	0.17	0.38
2 vs 4	0.29	0.13	0.20	0.19
3 vs 4	0.59#	0.23	0.88‡	0.84‡

LR, Living room; BR, bedroom.

*1, Der p I assay.

†2, Der f I assay.

‡ $p < 0.001$.

§3, rabbit two-site (Der p I equivalents).

||4, human RAST inhibition (Der p I equivalents).

¶ $p < 0.05$.# $p < 0.01$.

in both rooms. There was good correlation between the concentration of group I allergens in the air or dust of the bedrooms and living rooms of these houses but no correlation between the concentration in the air and the concentration in the dust, except for Der p I in the bedroom (see Table II).

With the rabbit polyclonal antibody assay, the micrograms of Der p I equivalents found in the bedroom and living room air ranged from undetectable ($<10 \times 10^{-6} \mu\text{g}/\text{m}^3$) to $409 \times 10^{-6} \mu\text{g}/\text{m}^3$ with a mean of $34 \times 10^{-6} \mu\text{g}/\text{m}^3$ and $38 \times 10^{-6} \mu\text{g}$, respectively (Table I). The micrograms of Der p I equivalents found in the settled dust in the living room and bedroom ranged from 1.0 to 901 $\mu\text{g}/\text{gm}$ with a mean of 24 and 61 $\mu\text{g}/\text{gm}$, respectively (Table I). RAST-inhibition (human IgE) assays of air samples expressed in Der p I equivalents were tenfold higher

than in the rabbit two-site assay. Conversely, two-site assays (rabbit antibody) of dust samples expressed in Der p I equivalents were about tenfold higher than in the human RAST-inhibition assay. In the individual houses, the amount of Der p I equivalents found in the living room air correlated with amounts found in the bedroom air, mite allergen in the living room air correlated with allergen in the carpet, and mite allergen in the bedroom air correlated with allergen in the mattress. The mite allergen in carpet correlated weakly with allergen in the mattress (Table III).

With the RAST-inhibition assay with human IgE antibody to mite, we found weak correlations between allergen concentrations of the air in the two rooms and between mattress dust and bedroom air (Table II).

There is quite good rank-order correlation between the various assays expressed as Der p I or Der f I equivalents (Table III), although the monoclonal assay for Der f I correlated poorly with the other assays, presumably because the standard for the other assays was Dp. The absolute values, however, differed among the assays and place of sampling. The rabbit polyclonal two-site assay elicited consistently fivefold to tenfold higher values than the other assays in settled dust, and the IgE RAST-inhibition assay elicited twentyfold higher values in airborne dust (Table I).

In the homes with cats, the amount of cat allergen (Fel d I) in the air ranged from $27 \times 10^{-6} \text{U}/\text{m}^3$ to $345 \text{U} \times 10^{-6}/\text{m}^3$ with a mean of $175 \times 10^{-6} \text{U}/\text{m}^3$ (Table IV). The concentration of Fel d I per gram of settled dust was higher in the carpet (2.5 U/gm) than in the mattress (0.9 U/gm), presumably because the mattress is usually covered by bedding. The concentration of Fel d I in the air of the bedroom correlated with the air in the living room ($r = 0.61$; $p < 0.01$). Cat-allergen concentration also correlated with mite-allergen concentration in the air but not with mite allergen in settled dust (Table V).

It is interesting to note the apparent discrepancies

TABLE IV. CAT 1 (*Fe/d l*) units

Sample No.	Air		Dust	
	$U \times 10^6/m^3$		U/gm	
	LR	BR	LR	BR
1	BD	BD	BD	BD
2	BD	BD	1.70	BD
3	BD	BD	11.10	1.80
4	BD	BD	BD	BD
5	BD	BD	BD	BD
6	BD	BD	BD	BD
7	BD	BD	BD	BD
8	286	325	BD	BD
9	BD	BD	BD	BD
10	BD	BD	BD	BD
11	42	38	0.85	0.73
12	266	222	8.03	1.63
13	264	222	4.17	1.60
14	114	227	3.71	0.83
15	314	184	1.92	3.23
16	253	260	7.83	1.67
17	274	111	1.67	0.75
18	27	63	0.92	0.49
19	54	45	0.95	0.37
20	139	104	1.75	0.60
21	314	252	1.13	0.69
22	240	229	0.80	0.29
23	49	52	1.25	0.41
24	158	43	1.76	1.24
25	94	104	1.96	1.10
26	345	54	0.97	0.30
27	281	295	1.78	0.36
28	260	262	0.92	0.45
29	141	52	0.62	0.38
30	241	277	0.73	0.64
	$\bar{x} = 198 \pm 103$	$\bar{x} = 163 \pm 100$	$\bar{x} = 2.5 \pm 2.81$	$\bar{x} = 0.93 \pm 0.72$
Detection limit	2	2	0.2	0.2

LR, Living room; BR, bedroom; BD, below detection.

in home 3 and 8 with regard to the presence of cat allergen. Home 3 had allergen in the dust but undetectable in the air. Home 8 had allergen in the air but not in the dust. Two adults had lived in home 3 only 2 weeks, and unlike the other homes, this home had radiant hot-water heat. Without the activities of children and in the absence of forced-air heat, cat allergen in the carpet from the previous owner's pet apparently did not become airborne. The family in home 8 had acquired two new kittens just the week before, which explains the finding of allergen in the air but not dust.

DISCUSSION

Our finding that many houses contained both species of *Dermatophagoides* confirms results previously

published by others.^{1, 6, 7} In Minnesota the predominant species was *Df*. These data were collected in the winter when furnaces were operating and windows were closed. Ventilation at this time is primarily by air permeation. We do not have data on these specific houses, but studies of similar houses indicate a range of air exchange rates of 0.2 to 0.5 times per hour. Thus, considering that these houses had forced-air furnaces that recirculate and redistribute the air, it is not surprising that allergen concentrations in the air in the bedroom and living room were similar. The correlation of mite concentration in living room carpet and bedroom mattress dust suggests that similar factors, presumably mainly humidity, determine mite growth in the two rooms. It is necessary, however, to

TABLE V. Correlating coefficients between Cat (*Fel d* I) and mite (various assays) in the air in bedrooms and living rooms

Mite assay	BR	LR
Monoclonal		
Der p I	0.10	0.49*
Der f I	0.30	0.55†
Rabbit	0.26	0.60†
RAST	0.04	0.25

BR, Bedroom; LR, living room.

* $p < 0.05$.† $p < 0.01$.

discuss the reasons for discrepancies between the results with various mite assays and the reasons for discrepancies between air and settled dust.

Factors determining the concentration of any substance in the air can be expressed in a mass balance equation

$$C_{ss} = \frac{PQ_v C_o + S}{E_v Q_v + K + NE_d Q_d}$$

C_{ss} represents steady-state concentration, the numerator represents the rates of production, and the denominator, the rates of removal. $PQ_v C_o$ is the outdoor source term. P is penetration factor, Q_v equals ventilation rate, and C_o is outdoor concentration. S is indoor source substance-generation rate. It has two components, de novo generation into the air and re-suspension of settled allergen. $E_v Q_v$ is removal by ventilation. E_v is ventilation efficiency, the rate of exhaust concentration to concentration of occupied space divided by a mixing factor, and Q_v is ventilation rate. K is natural decay rate (settling). $NE_d Q_d$ is removal rate by air cleaning. N is contaminant removal efficiency, E_d is device ventilation efficiency, and Q_d is device airflow rate.

Inasmuch as mites live out their life cycle in carpets, bedding, and furniture, it follows that suspension of settled dust is the primary source of airborne mite allergens. The factors determining mite-allergen concentration in the air will be rate of suspension from activity in the room divided by rates of ventilation and settling. Particles settle at different rates, dependent on their mean mass aerodynamic diameter. It is likely that dense 10 μ g of fecal pellets settle more rapidly than smaller, less dense particles from other mite sources, and indeed there are some data supporting this prediction. Assay of airborne concentrations of group I allergens demonstrated them associated primarily with large particles that settled rapidly,⁸ whereas allergens measured by RAST inhibition dis-

closed a considerable amount of mixed mite allergens in particles $<5 \mu$ g mean mass aerodynamic diameter and that such particles stayed suspended in the air for prolonged periods.⁹

The group I monoclonal assays measure preferentially fecal allergens.^{2, 10} Preliminary evidence suggests that our rabbit polyclonal antibody two-site assay measures allergens chiefly other than group I (data not presented). The IgE RAST appears to measure all mite allergens. Therefore, the variation in the amount of mite allergen measured by the different assays suggests that the mosaic of mite allergens in settled dust may be quite different than that in airborne dust because group I allergens settle faster. It is also possible that the proportions of the various mite allergens vary between houses as they do between mite-culture extracts.^{1, 11, 12} Further studies are needed to clarify these issues.¹³

With cat allergen, the situation is different. A major source is direct shedding from the animal itself; suspension from settled dust contributes also. The relative importance of the two sources is not known, but the lack of correlation between air and dust suggests that the cat itself is more important.

Although there have not yet been studies comparing the rate of shedding of allergen into the air by different cats, we have found that a single cat sheds about 5×10^{-4} units of *Fel d* I per minute. Cat allergen settles more slowly than mite allergen.⁹ Calculations substituting experimental values into the above equation indicate that this production rate is sufficient to account for the measured air concentrations of *Fel d* I. For example, assuming an average home volume equal to 300 m^3 and the outdoor source term ($PQ_v C_o$) and removal by air-cleaning devices ($NE_d Q_d$) are zero and that rate of settling and suppression are balanced out, the steady-state equation becomes

$$C_{ss} = 300 \times 10^{-4} \text{ U/hr} \times [300 \text{ m}^3/\text{hr}]^{-1}$$

or

$$1 \times 10^{-4} \text{ U/m}^3 \text{ for one change of air per hour}$$

or

$$C_{ss} = 300 \times 10^{-4} \text{ U/hr} \times [1500 \text{ m}^3/\text{hr}]^{-1}$$

or

$$2 \times 10^{-5} \text{ U/m}^3 \text{ for five changes of air per hour}$$

The variation in cat-allergen concentration between houses presumably depends chiefly on variations in ventilation. The variation in airborne levels of mite allergen depends both on variation in ventilation and on variation in mite-allergen production. To the extent that indoor humidity in the cold dry Minnesota winter is affected by ventilation, the variation in ventilation

also could be expected to affect mite-allergen production.

Interpretation of the clinical implications of these data is subject to the limitation that we measured the average steady-state concentration of allergen in the air during a 24-hour period. It is possible that symptoms are related more to brief, heavy concentrations than to average levels. Nevertheless, a concentration of group I allergens of 1 to 10 $\mu\text{g/gm}$ of settled dust corresponding roughly to >300 mites per gram has been proposed as a risk factor for developing asthma from mite allergy.^{1, 3, 14} If this is correct, two thirds of the houses we sampled contained sufficient concentrations to be of concern, and one quarter of these houses contained concentrations of *Der f I* $>10 \mu\text{g/gm}$. Wood et al.¹⁵ have recently reported similar values in settled house dust in Baltimore, Md.

A concentration of 1 to 10 μg of group I allergen per gram of settled dust corresponds roughly to a steady-state concentration of 20 to $50 \times 10^{-6} \mu\text{g/m}^3$ of group I allergens measured by the monoclonal assay, but 500 to $1000 \times 10^{-6} \mu\text{g/m}^3$ of *Der p I* equivalents measured by RAST inhibition. Correlation between mite-allergen concentrations in settled dust and in the air is not sufficiently strong enough that these guidelines for risk factors can be considered absolute. Further epidemiologic studies are needed to define the concentration of airborne allergens that are risk factors for allergic respiratory disease.

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