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Domestic allergens in public places III: house dust mite, cat, dog and cockroach allergens in British hospitals

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Summary

Background Exposure and sensitization to indoor allergens is a major cause of asthma.
Objectives This study investigated the levels of house dust mite, cat, dog and cockroach allergens in the dust and air in hospitals and the effects of regular vacuum cleaning on allergen levels in hospital chairs.
Methods Der p 1, Fel d 1, Can f 1 and Bla g 2 were measured in the dust collected by vacuuming upholstered chairs and a 1 m^2 area of carpet and mattress in 14 hospitals. Air samples were collected using an air sampler (flow rate 60 L/min) on 10 separate days for 4 h in the outpatient department in one of the hospitals during busy clinics when patients were waiting for their appointments. In addition, dust samples were collected on four occasions, at 4-weekly intervals, from 36 fabric covered chairs in the outpatient area of a busy chest clinic by vacuuming each chair for 2 min. During the intervening weeks, 18 of the chairs (active group) were each cleaned by vacuuming for 1 min, three times per week. Der p 1, Fel d 1, Can f 1 and Bla g 2 were assayed using monoclonal antibody-based ELISA.
Results In total, 83 carpets, 69 mattresses and 42 upholstered chairs were sampled. The levels of dust mite allergen Der p 1 and cockroach allergen Bla g 2 found in the hospital setting were low. High levels of Fel d 1 (GM 22.9 μg/g, range 4.5–58) and Can f 1 (GM 21.6 μg/g, range 4–63) were found in upholstered chairs. Airborne Can f 1 was detected on every occasion (range 0.12–0.56 ng/m^3), whilst detectable airborne Fel d 1 was found on 7 out of the 10 sampling days (range 0.09–0.22 ng/m^3). Der p 1 and Bla g 2 were below the detection limit in all airborne samples. Following repeated vacuuming the mean cat and dog allergen levels decreased significantly ($P < 0.001$) and were almost fivefold lower in the vacuumed chairs compared with the control group.
Conclusions Low levels of mite allergen are unlikely to be of any clinical significance to mite-sensitive asthmatic patients. However, upholstered chairs in hospitals constitute a significant reservoir of cat and dog allergen. Inhalation of airborne allergen in patients attending their hospital appointment may exacerbate asthma in those highly allergic to cats or dogs. These results question the wisdom of introducing soft furnishings and carpets into hospitals. Three-times weekly vacuuming significantly reduces allergen levels in upholstered chairs.

Keywords: allergens, cat, cockroach, dog, house dust mite, hospitals

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Introduction

The development of techniques for measuring the exposure to allergens has made possible a series of epidemiological studies in different parts of the world suggesting that

sensitization and exposure to indoor allergens may be a primary cause of asthma, particularly in children and young adults (reviewed in [1]).

Increasing asthma severity has a major impact on patients' lives and on health care costs. The severity of asthma may be related to allergen exposure [2,3]. A strong association has been found between sensitization and exposure to mite, cat and cockroach allergens and acute asthma in adults admitted to the hospital [4].

The First International Workshop on Mite Allergens and Asthma proposed a provisional threshold level of more than 2 μg Group 1 mite allergen/g dust that represent risk for sensitization and asthma, and a higher level of 10 μg Group 1 mite allergen/g dust was regarded as a risk for acute symptoms of asthma [5]. For other indoor allergens it is as yet difficult to establish the threshold levels and to distinguish the effect of exposure on sensitization from the effect on asthma symptoms. Nevertheless, the levels that could be considered significant have been proposed [4,6]. In some inner city areas of the USA both cat ownership and sensitization to cats is rare (the levels of Fel d 1 in the homes are low, usually < 1 μg/g of dust) [4]. Asthma symptoms in cat allergic patients often occur in the homes with cats (more than 95% of these contain > 8 μg Fel d 1/g of dust). Thus a provisional concentration that represents the risk for cat sensitization was proposed as > 1 μg of Fel d 1/g of dust, and a higher level of > 8 μg of Fel d 1/g of dust may be considered as level at which most cat allergic patients will experience symptoms [4]. Similarly, Ingram *et al.* indicated that for dog allergen 2 μg and 10 μg Can f 1/g of dust are the levels significant for sensitization and symptoms, respectively [6].

It is possible that significant exposure to domestic allergens can occur outside homes. We have previously reported low levels of mite allergen Der p 1 in public places, but found high levels of cat allergen, Fel d 1, and dog allergen, Can f 1, in upholstered seats from public buildings, trains and buses [7,8]. This study investigated the concentrations of domestic allergens (mite-Der p 1, cat-Fel d 1, dog-Can f 1 and cockroach-Bla g 2) in British hospitals, and the effects of regular vacuum cleaning on allergen levels in hospital upholstered chairs.

Methods

House dust mite, cat, dog and cockroach allergen levels were measured in the dust collected from carpeted floors, mattresses and upholstered chairs in 14 hospitals. Of the participating hospitals, nine were situated in the North West of England, four in London and one in North Wales. Eight of them were National Health Service hospitals and six were private hospitals. All hospitals provided both adult and paediatric care. The type of the ward and age of the carpets, chairs and mattressses were recorded. All samples were collected between March and October 1995.

Dust samples were collected using a Medivac dust sampler (Medivac Plc, Wilmslow, UK) with air-flow rate 45 L/s through a 355 μm diameter mesh screen on to a 5 μm vinyl filter, thus enabling collection of fine dust samples. The filters were supported in a plastic dust trap located behind the cleaner attachment (nozzle). A $1m^2$ area of carpet and mattress (upper part) and the entire upholstered chair were sampled for 2 min. One hundred mg of fine dust was extracted with 2 mL borate-buffered saline with 0.1% Tween 20, pH 8.0. The dust was resuspended using a vortex mixer. Samples were then mixed end over end on an orbital rotator for 2 h at room temperature before being centrifuged for 20 min at 2500 RPM at 4 °C. Supernatants were removed and stored at -20 °C for future analysis of allergen content.

In addition, airborne allergens were measured in the outpatient department (OPD) in one of the participating hospitals. Air samples were collected using an air sampler (Rotheroe-Mitchell, London, UK; air flow rate 60 L/min) situated 1 m above the floor onto 37 mm Whatman GFA micro-glass fibre filter (pore size 0.3 μm). Each sampler had a flow meter indicator and a time counter. Flow rates were measured at commencement of the sampling, and then at 10 min, 30 min, 1 h, 2 h, 3 h and 4 h. The volume of the air sampled was calculated by multiplying a geometric mean of flow rate by the sampling time. Sampling was performed on 10 separate days for 4 h during busy clinics when patients were waiting for their appointments. Each filter was placed in a syringe and 1 ml of 1% bovine serum albumine in phosphate buffered saline with 0.1% Tween 20 was added. After the overnight extraction at 4 °C an extraction liquid was aspirated backwards and forwards several times through a three-way stop lock into a second syringe, then transferred into a test tube and centrifuged at 3000 rpm for 30 min at 4 °C [9,10]. The supernatants were removed with a pasteur pipette, coded, and stored at -20 °C.

ELISA measurement of indoor allergens

Can f 1 was measured by a two-site monoclonal antibody (mAb) ELISA using anti-Can f 1 MoAb 6E9 for allergen capture and polyclonal rabbit anti Can f 1 for detection [10]. Dust extracts were initially assayed at five-, 25- and 125-fold dilution for carpets and beds and at 100-, 500- and 2500-fold dilutions for chairs. The assay was quantitated using doubling dilutions of dog allergen standard (UVA 94/02) from 500 IU/mL to 1 IU/mL Can f 1. The UVA 94/02 (10000 IU Can f 1/mL) was substandardized against WHO/IUIS International Reference Preparation of dog hair and dander (NIBSC 84/685) which contains 100 000 IU/mL Can f 1. One International Unit is approximately 1 ng Can f 1 protein, and this value was used to calculate the results.

Table 1. Details of hospitals which participated in the study and the number of dust samples collected in each of them

Hospital	Type of ward	Carpet number (age)	Mattress number (age)	Chairs number (age)
1	Single rooms	5 (10 years)	5 **(4** years**)**	–
2	Single rooms	5 (4 months)	5 (2 years)	–
3	Single rooms	4 (5 years)	4 (5 years)	–
4	Single rooms	5 (2 years)	5 (2 years)	6 (4 years)
5	6-bed ward	10 (2 years)	10 (2 years)	6 (6 months)
6	Single rooms	8 (5 years)	8 (3 years)	–
7	20-bed ward	10 (10 years)	10 (4 years)	–
8	Single rooms	5 (9 years)	5 (5 years)	6 (9 years)
9	Single rooms	5 (3 years)	5 (3 years)	6 (6 years)
10	20-bed ward	10 (3 years)	10 (3 years)	6 (8 years)
11	20-bed ward	4 (4 years)	–	–
12	20-bed ward	5 (7 years)	–	5 (4 months)
13	Dayroom	5 (10 years)	–	5 (7 years)
14	Single room	2 (10 years)	2 (3 years)	2 (3 years)

Bla g 2 was measured by a two-site monoclonal antibody based ELISA using anti Bla g 2 MoAb 7C11 for allergen capture and biotinylated 8F4 MoAb for detection. A control curve for Bla g 2 measurement was established using a reference *B. germanica* extract (UVA 93/04) which contained 30 Units/mL Bla g 2, and was diluted 1:6 to obtain 5 U/mL Bla g 2. Serial two-fold dilutions of the extract were used to generate curves from 5 U/mL down to 0.01 U/mL.

Der p 1 and Fel d 1 were assayed using a two-site immunometric ELISA as previously described [11,12]. The standard used to establish the control curve for Der p 1 assay (UVA 93/02) was considered to contain 2500 ng Der p 1/ml (relative to WHO/IUIS *D. pteronyssinus* standard NIBSC 82/518 which has been estimated to contain 12.5 μg Der p 1 per ampoule). UVA 91/01 standard for Fel d 1 contained 2 Units Fel d 1/mL (relative to CBER Cat E5 standard containing 9.7 Units/mL; 1Unit = 4 μg protein).

Air samples were assayed neat and at two-, four-, and eight-fold dilutions. For concentrations lying off the linear portion of the standard curve, the assays were repeated at an appropriate dilution.

The allergen data were found to follow a log-normal distribution; the results are thus reported as geometric means (GM). Allergen levels in different sites were compared using log-transformed data and the Student's *t*-test. Statistical significance was set at a conventional $< 5\%$ level.

Results

Dust samples were collected from the carpeted wards in all 14 hospitals, from beds in 11, and from upholstered chairs in outpatient departments in eight. Table 1 shows the type of ward, number of samples collected and age of carpets and upholstered chairs sampled. A total of 83 carpets, 69 mattresses and 42 upholstered chairs was sampled.

Samples with allergen levels below the lower level of detection of the assay (0.1 μg/g for Der p 1, 0.03 μg/g for Fel d 1 and 0.2 μg/g for Can f 1) were assigned values of 0.1 μg/g, 0.03 μg/g and 0.2 μg/g for Der p 1, Fel d 1 and Can f 1, respectively.

Figure 1 shows the concentrations of domestic allergens (GM and range) in the dust samples collected from different sampling sites. Der p 1, Fel d 1 and Can f 1 were significantly higher in upholstered chairs, than in carpets or mattresses ($P < 0.001$). There was a significant correlation between cat and dog allergen levels in upholstered chairs ($r = 0.52$, $P < 0.01$), but no correlation was found between Der p 1 and either Fel d 1 or Can f 1. Fel d 1 concentration > 8 μg/g was found in 36/42 chairs. Can f 1 was > 10 μg/g in 34/42 upholstered chairs. Der p 1 was < 10 μg/g in all samples collected. Cockroach allergen Bla g 2 was below the detection limit of the assay in all dust samples from hospitals. All mattresses, carpets and chairs were more than 1 year old. There was no correlation between the age of mattresses, carpets and chairs and allergen levels. No difference was found in any of the sampling sites between different hospitals.

The results of the air sampling with high volume samplers on ten separate days in the outpatient department in one of the hospitals are shown in Table 2. Airborne Can f 1 was detected on every occasion, whilst detectable airborne

Table 2. Airborne levels of cat and dog allergen in the outpatient area in one of the hospitals

Day	Can f 1 (ng/m^3)	Fel d 1 (ng/m^3)	Sample volume (m^3)
1	0.28	< 0.08	8.68
2	0.24	< 0.08	10.10
3	0.12	0.09	13.50
4	0.23	0.09	14.10
5	0.41	0.22	9.27
6	0.56	0.13	10.20
7	0.52	0.14	11.10
8	0.31	< 0.08	8.17
9	0.43	0.13	9.17
10	0.46	0.20	10.40

Fel d 1 was found on 7 out of 10 days. Der p 1 and Bla g 2 were below the detection limit in all airborne samples.

Effect of regular vacuum cleaning on cat and dog allergen levels in upholstered chairs

Following the finding of high levels of cat and dog allergens in the hospital upholstered chairs, we proceeded to investigate the effect of regular vacuum cleaning on Fel d 1 and Can f 1 levels. Fine dust samples were collected on four occasions, at 4-weekly intervals, from 36 fabric covered chairs in the outpatient area of a busy chest clinic by vacuuming each chair for 2 min. During the 12 intervening weeks, 18 of the chairs (active group) were cleaned by vacuuming for 1 min, three times per week, using a Nilfisk GM 210 vacuum cleaner with an inbuilt high efficiency particulate air (HEPA) filter, and 18 chairs served as controls.

At baseline there was no significant difference in allergen levels measured between the active and control chairs ($P > 0.1$). Following repeated vacuuming the mean Can f 1 and Fel d 1 decreased significantly in the active as compared with the control group (two factor analysis of variance with repeated measures; $P < 0.001$; Fig. 2). The difference between active and control chairs was even greater when the results were expressed as total allergen recovered (μg Can f 1 and Fel d 1 per chair). The total amount of allergen per chair (geometric mean-GM) was reduced by more than 95%:

1. Can f 1 level fell from GM 8.4 μg/chair (95% confidence interval-[CI], 6.2–11.4) to 0.5 (95% CI, 0.3–0.8), 0.2 (95% CI, 0.1–0.4) and 0.15 (95% CI, 0.1–0.2) μg/chair at 4, 8 and 12 weeks respectively. During the same period, allergen levels in the control group were unchanged, the total allergen recovered being from 5.3 μg/chair (95% CI, 3.5–8.1) at the beginning to 6.5 μg/chair (95% CI, 4.6–9.2) at the end of the study.

2. For Fel d 1, total allergen recovered decreased from 6.9 μg/chair (95% CI, 4.8–9.9) to 0.4 (95% CI, 0.2–0.6), 0.3 (95% CI, 0.2–0.4) and 0.3 (95% CI, 0.2–0.4) μg/chair at 4, 8 and 12 weeks respectively. Total allergen in the control chairs did not change significantly from 4.7 μg/chair (95% CI, 2.9–7.6) at the beginning to 6.9 μg/chair (95% CI, 5.4–8.7) at the end of the study.

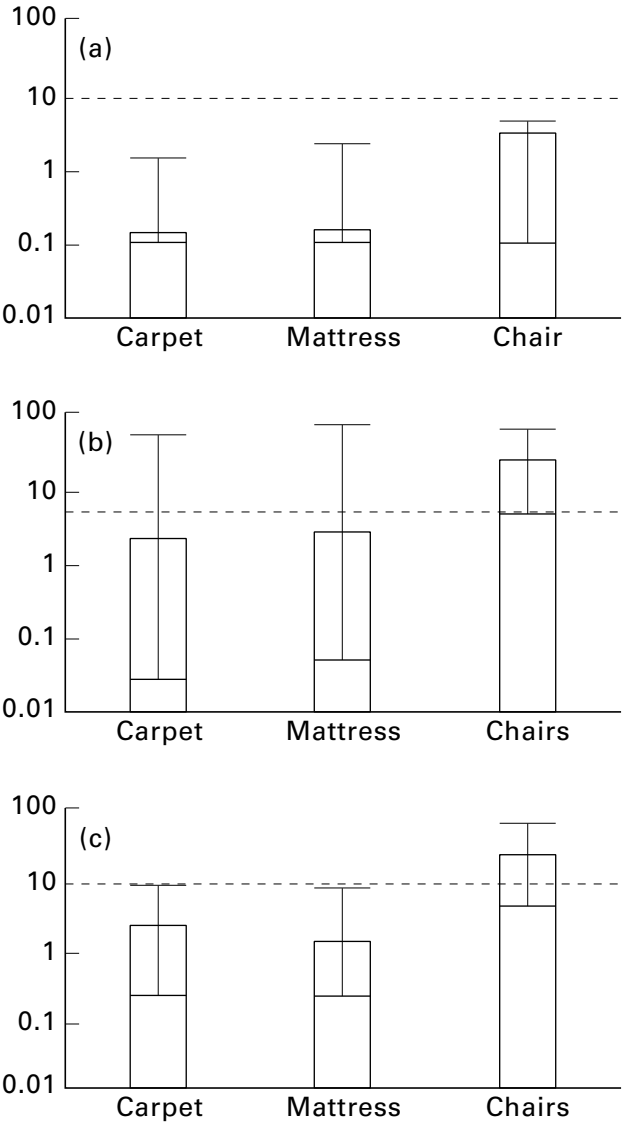

Fig. 1. Concentrations of domestic allergens in different sampling sites in 14 hospitals (Geometric mean and range). (a) Der p 1 (μg/g of dust); (b) Fel d 1 (μg/g of dust); (c) Can f 1 (μg/g of dust). The dotted lines represent the proposed threshold levels for acute asthma in sensitized asthmatic patients (10 μg/g for Der p 1 [9], 8 μg/g for Fel d 1 [4] and 10 μg/g for Can f 1 [6]).

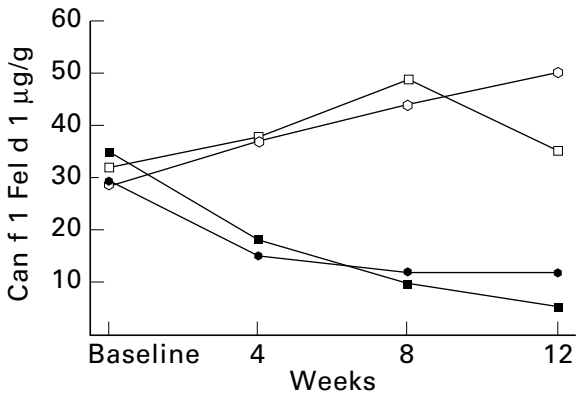

Fig. 2. The geometric means of cat (Fel d 1) and dog (Can f 1) allergen levels at baseline and after 4, 8 and 12 weeks of regular vacuum cleaning for 1 min, three times per week in active chairs ($n = 18$), and in control chairs ($n = 18$). The results are expressed as μg/g of fine dust. ■, Can f 1 (active); □, Can f 1 (control); ●, Fel d 1 (active); ○, Fel d 1 (control).

The observed difference between the groups were highly significant ($P < 0.001$; two-factor analysis of variance with repeated measures).

Discussion

Low levels of mite, cat, dog and cockroach allergens were found in carpeted wards and beds in 14 hospitals from different parts of the UK. Mite allergen levels were negligible and presented no health concern. None of the samples contained Der p 1 at a concentration $> 10\,\mu$g/g (threshold level regarded as a risk for acute attack of asthma in mite sensitive asthmatic patients) [5]. These findings are in agreement with previous studies [13,14]. In the current study, the low concentrations of mite allergen in hospitals were in sharp contrast to much higher levels found in domestic dwellings throughout the UK. A number of different factors that influence house dust mite population growth are likely to contribute to the low mite allergen levels in hospital dust. Mite survival depends on high indoor humidity. All hospitals that took part in the survey were mechanically ventilated without supplementary humidification. In addition, there are fewer sources of indoor-produced humidity in the hospitals as compared to domestic dwellings and indoor humidity in hospitals is considerably lower than in domestic households. Relative humidity levels which were measured in one of the hospitals were consistently below 45%, regarding as a minimum level required for mite growth. Housekeeping procedures may also influence the concentration of mite allergens, and all hospitals reported vacuum cleaning ward carpets on a daily basis. Furthermore, carpets in the hospitals participating in the survey were short pile and woven with man-made fibre which provides a less favourable microhabitat for mite survival [15]. The mattresses in hospital wards differ considerably from those used in homes. Whilst the majority of household mattresses are fabric covered, hospital mattresses are encased in impermeable vinyl covers. Furthermore, the bed linen is frequently changed and regularly hot-washed at water temperatures greater than 70 °C. The difference in mite allergen levels between domestic dwellings and hospitals indicates that even in geographic areas where climatic conditions facilitate mite survival, measures including encasing mattresses, regular hot washing of bedding, vigorous cleaning procedures, minimal mite microhabitats and low indoor humidity can contribute to low concentration of mite allergens within the indoor environment [14].

In agreement with our findings in other public buildings, high levels of both cat and dog allergen were measured in hospital upholstered chairs, probably brought in on the clothing of cat and dog owners [7,8]. Measurable amounts of Fel d 1 were also found in the carpets of various types of public places in Michigan [16] and on floors in four schools in Sweden [17]. Higher levels of Fel d 1 were found on chairs than on floors in Swedish schools, probably as a result of children and teachers carrying it on their clothing [17]. Low concentrations of dog allergen were found in day nurseries in Marseilles, France [18]. Munir *et al.* have recently reported that curtains, mattresses, sofas and soft toys were the most important reservoirs for dog and cat allergens in Swedish day-care centres, and that they should be considered as a cause of cat or dog allergen exposure [19].

In the current study, the correlation between cat and dog allergen levels in upholstered chairs probably reflects a combination of frequent use and infrequent cleaning. Low levels of cat and dog allergen were recovered from either carpets which were vacuumed daily or covered mattresses, the only exception being high levels of Fel d 1 ($\sim 65\,\mu$g/g in mattress and $48\,\mu$g/g in carpet) found in one single room in one of the private hospitals.

It is interesting that we found no relationship between the age of chairs, carpets and mattresses and allergen load, which is unlike our findings in domestic dwellings where older carpets, sofas and mattresses contain significantly higher mite allergen levels [20]. This may reflect considerable differences in the indoor environment of average British homes where the conditions are usually suitable for mite population growth and Der p 1 accumulates over time, and hospitals where mite allergen levels seem to be universally low. Possible explanation for the universally high levels of pet allergens in the upholstered chairs in hospitals may be that as all chairs were older that 1 year, this may be sufficiently long period for the build-up of passively transferred allergen to plateau. This would also explain the

finding that allergen levels were very similar in different hospitals.

Concentrations of both Fel d 1 and Can f 1 in the upholstered chairs in hospitals were well above the proposed significant levels, and considerably higher than the levels found in the homes without pets in the same area [7,8,10]. Fel d 1 was $>8\,\mu$g/g in 86%, and Can f 1 was $>10\,\mu$g/g in 81% of the hospital chairs sampled. However, it is important to stress that the relevance of the proposed significant levels for pet allergens is as yet unclear (in particular the ones relating to asthma symptoms). It is likely that some very sensitive patients may have symptoms when exposed to very low dose of allergen, whilst in others the required dose for the same effect will be much higher. Both cat and dog airborne allergen were readily detectable in the outpatient waiting area. Although it is as yet impossible to assess the clinical significance of this finding, there is a possibility that inhalation of such airborne allergen is capable of exacerbating asthma in patients highly allergic to cats or dogs. The amount of cat and dog allergen found in dust from Swedish schools was considered to be high enough to probably cause perennial symptoms in most children with asthma who are sensitized to cats and dogs [17]. The levels that we report from British hospitals are much higher than those from Swedish schools, suggesting that we may need to find the way to reduce the exposure of patients attending the hospitals.

Our results clearly show that high levels of both cat and dog allergen can accumulate in hospital soft furnishing. Although all hospitals reported that carpets were vacuum cleaned daily, no regular cleaning policy was adopted for upholstered chairs. However, regular vacuum-cleaning of the upholstered chairs (three times per week, 1 min per chair) resulted in a significant reduction in cat and dog allergen levels, both when the results were expressed in μg allergen/g of fine dust and in total allergen recovered/chair. It would be interesting to speculate what would be the consequences of a more intensive vacuum-cleaning programme on an annual cleaning budget of a hospital. Vacuum-cleaning should be done with vacuum cleaners with built-in HEPA filters and double thickness vacuum cleaner bags, but not necessairly with special, 'allergy' cleaners [21,22]. All but one hospital in the present survey already used equipment that could be considered suitable. Depending on the number of upholstered chairs in individual hospitals, such a regime would add approximatelly 15 min/chair/month of a cleaning staff time, i.e. equivalent to one part-time cleaner for a hospital with 400 upholstered chairs.

In conclusion, upholstered chairs in hospitals may constitute a significant reservoir of cat and dog allergen. It is impossible to prevent cat and dog allergens being brought into hospital. However, thorough vacuum-cleaning not only of the floors, but of upholstered seats in particular, could reduce the build-up of Fel d 1 and Can f 1. A careful choice of vacuum cleaner is important, since those with single-thickness paper bags leak significant amount of cat allergen [21,22]. The current policy of introducing carpets and soft furnishings into clinical areas in hospitals should be reconsidered. If present, upholstered chairs in patient areas should be regularly vacuumed.

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