

Effects of furniture polish on release of cat allergen-laden dust from wood surfaces

Abstract The purpose of this study was to evaluate the effects of furniture polish (FP) on the release of cat allergen (Fel d 1)-laden dust from wood surfaces and decay of Fel d 1 in dust over time. About 2 g of sieved (150 μm screen) allergen-laden dust was introduced as an aerosol into an experimental chamber by a Pitt-3 generator and allowed to settle onto two finished wood surfaces pre-treated with either distilled water (DW) or FP. After 24 h, each surface was vacuumed into separate plastic cassettes loaded with 37 mm diameter, 0.4 μm pore, polycarbonate filters. The recovered dust was weighed, extracted in phosphate-buffered saline, and assayed for Fel d 1 content using a two-site monoclonal antibody ELISA. After vacuuming, the remaining dust on the wood surfaces was wiped up with a water-moistened swab. The dusts were extracted from the swabs and assayed by ELISA for Fel d 1. More Fel d 1 was recovered by vacuuming from DW-pre-treated surfaces than from FP pre-treated surfaces (100% vs. $69 \pm 66\%$). On the contrary, more residual Fel d 1 ($>99.9\%$) on the vacuumed surfaces was recovered from FP than from DW pre-treated surfaces by wet swabs. The concentration of Fel d 1 in dust did not change significantly at room temperature over 80 days. In conclusion, FP on wood surfaces makes dust stick to the surface, which likely reduces the release of allergen-laden dust from the wood surface.

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Key words: Cat allergen; Fel d 1; Aerosolization; Airborne allergen; Furniture polish; Surface; Dust.

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Received for review 14 April 2004. Accepted for publication 10 August 2004.
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Practical Implications

Re-aerosolization of cat allergen-laden dusts from wood surfaces (e.g. wood floor or furniture) significantly increases the concentration of airborne cat allergens. Our study indicates that exposure to airborne cat allergens might be reduced by surface treatment with commercially available FP because cat allergen-laden dusts stuck more tightly to the wood surface treated with FP.

Introduction

Dust is well recognized as a reservoir for agents of human disease (Sporik et al., 1990; Tovey et al., 1981; Woodcock and Custovic, 1998). In fact, dust is considered the primary source of exposure to allergens that may be contributing to an increasing number of cases and of deaths from asthma (Carrer et al., 2001; Huss et al., 2001). Dust mites and fungi actually reproduce in dust, while other allergen-laden particles settle from air (e.g. dog and cat effluents) or are excreted into dust (cockroach fecal material and body parts) (Arruda et al., 2001). In addition to allergens, bacteria and viruses settle on surfaces and are transmitted by re-aerosolization, resulting in infection. Correlations between settled dust and airborne allergen particles and sensitization of people to these allergens have been well documented (de Blay et al., 1991; Luczynska et al., 1990; Mollet et al., 1997).

Most studies that have documented the importance of dust as a reservoir for allergens have focused on carpet and floor dust (Platts-Mills et al., 2000a). However, it is intuitively obvious that, at least for the settled allergen-laden particles, dust on either wood surfaces or upholstered furniture may be as, or more, important as a source for exposure when re-aerosolization occurs. In fact, in a previous study (Custovic et al., 1998), the highest concentration of Fel d 1 in houses was found on upholstered furniture. Dust on smooth surfaces is easily released and suspended in air, perhaps with only limited air movement, but certainly during various human or animal activities. Approaches to limiting the accumulation of dust in houses are routinely recommended by physicians as a means of controlling the symptoms of some allergies (Expert-Panel-Report-II, 1997; Perzanowski et al., 1997; Ruzicka et al., 1991). Even in the cleanest houses, however, surfaces do become dusty, and this dust likely

contributes to symptoms in sensitive individuals. Surface disinfection is frequently recommended in areas where infectious agents are likely to have settled. However, the nature of the surface associated with re-aerosolization is not often considered.

Furniture polish (FP) treatment of surfaces on which dust collects could help to control dust allergen exposure by (1) facilitating the destruction of allergens or (2) causing dust to stick more tightly and thus reducing re-aerosolization. If treatment with FP were to reduce exposure by either of these mechanisms, significant improvement in symptoms of allergic diseases could result. We therefore tested the effect of commercially available FP on release of cat allergen (Fel d 1)-laden dust from wood surface. In addition, although the decay of Fel d 1 in dust has not been well characterized, we measured the decay of Fel d 1 in dust at room temperature over time.

Methods

Preparation of experimental settings

House dust was obtained from vacuum cleaner bags used in an apartment housing three cats in the Boston area. The collected dust was sieved through a 150- μ m screen and stored at room temperature for further use.

The experimental chamber was made of stainless steel (61 cm $\times$ 46 cm $\times$ 46 cm), and equipped with a Pitt-3 dust generator that was used for generating dry dust aerosols (Figure 1). The Pitt-3 dust generator aerosolizes dust by vibrating latex rubber dams, and this aerosolized dust is transferred to the experimental chamber by airflow passing from the Pitt-3 dust generator to the experimental chamber (Weyel et al., 1984). The Pitt-3 dust generator is reported to produce dust clouds in the range of 2–30 mg/m³, with a mass median particle diameter of 2.5–3 μ m and a geometric standard deviation of 1.5–1.8 μ m. A mixing fan in the experimental chamber ran throughout the process of aerosolization to ensure uniform distribution of aerosols in the environmental chamber. Dust on the surface in the experimental chamber was uniformly distributed.

Unfinished wood (61 cm $\times$ 46 cm) was obtained from a lumber house near the Boston area, cut vertically in half (60 cm $\times$ 23 cm), sanded gently with

fine grit sandpaper, and then painted twice with nitrocellulose.

Several FPs were purchased from a local shop in Boston, and one with typical ingredients was tested in our experiment. The ingredients of the selected FP were isobutene (1–3%), butane (1–3%), propane (1–3%), isoparaffinic hydrocarbon solvent (3–7%), silicone fluid (15–25%), and water (65–80%).

Measurement of allergen concentration in dust

Two-site monoclonal antibody ELISA assays for Fel d 1 were performed as described by the suppliers (Indoor Biotechnologies, Clywd, UK) to measure the allergen's concentration in dust (Chapman et al., 1988). The microtiter plates were immediately placed in a Vmax microplate reader (Molecular Devices Corp, Menlo Park, CA, USA) and absorbance at 405 nm was recorded every 15 s for a total of 5 min. The plate reader agitated the microtiter plate for 5 s between each measurement in order to mix the substrate properly. The results were expressed as the rate response instead of the end point response, as described in a previous study (Chew et al., 1999). Due to the reported interference of some chemicals with ELISA assays (Chew et al., 1999; Dybendal et al., 1990; Woodfolk et al., 1995), the effect of FP on the ELISA assay was examined before the effect of FP on releases from surfaces was tested. The presence of FP was determined to have no significant inhibitory effect on the assay at concentrations of 10% or less.

Experimental procedure

Half of the finished wood was pre-treated with FP; the other half was pre-treated with distilled water (DW) as a control. Both pieces were placed in the experimental chamber. Approximately 2 g of sieved allergen-laden dust was aerosolized by the Pitt-3 dust generator at 5 psig, and the aerosols generated were transferred into the experimental chamber as described above. The amount of aerosolized dust was determined to simulate the typical amount of dust on a house floor in an inner city. Data on the typical amount of dust accumulating on surfaces was obtained from the Boston inner city asthma study (Chew et al., 1998). After the aerosolized dust had completely settled on both surfaces (approximately 24 h), we recovered the settled dust by vacuuming, using open-faced three-part plastic filter cassettes with 37 mm nucleopore polycarbonate membranes at a rate of 1 cfm. The dusts collected on the polycarbonate membranes were weighed, suspended in elution buffer, and assayed by two-site mAb ELISA techniques to measure the concentration of Fel d 1. These experiments were run seven times. The dusts remaining on the vacuumed surfaces were removed by wet swab. Dusts on the swabs were extracted in 2 ml

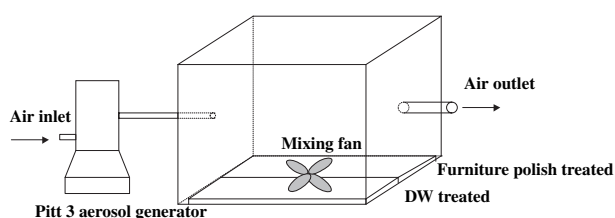


Fig. 1 Schematic diagram of the experimental aerosol chamber

phosphate-buffered saline, and then assayed by two-site mAb ELISA to measure the concentration of Fel d 1. The swab experiments were run three times.

Decay rate of cat allergen in dust

Collected and sieved dusts were stored at room temperature, and the Fel d 1 concentration in 0.1 g of dust was measured seven times over more than 80 days.

Data analysis

Data were manipulated using Excel and statistical analysis was performed using STATA (College Station, TX, USA).

Results

The data on dust and Fel d 1 recovered by vacuuming the surfaces are summarized in Table 1. Significantly more dust was recovered from the DW pre-treated surface (100%) than from the FP pre-treated surface (45% ± 22%) (Wilcoxon signed rank, *P* < 0.05). Fel d 1 concentrations in the dusts recovered were not significantly different from each other (33 ± 31 mU/mg in dust from the DW pre-treated surface vs. 45 ± 55 mU/mg in dust from the FP pre-treated surface). As a result, when the total amount of Fel d 1 recovered by vacuuming was estimated as a product of the weight of recovered dust (mg) and the Fel d 1 concentration (mU/mg), less Fel d 1 was recovered from a surface pre-treated with FP (69 ± 66%) than with DW (100%) (*P* = 0.03, Wilcoxon signed rank test).

Significant Fel d 1 was recovered by wet swab from the vacuumed FP-treated surfaces, whereas almost negligible Fel d 1 was recovered from the vacuumed DW-treated surfaces (*P* = 0.11, Wilcoxon sign rank test). More than 1000 times the amount of Fel d 1 was extracted by wet swab from the vacuumed FP pre-treated surfaces than from the DW pre-treated surfaces, which indicates that more dust and Fel d 1 stick to FP pre-treated surfaces than to DW pre-treated surfa-

Table 1 Amount of dust and Fel d 1 recovered by vacuuming from the wood surface pre-treated with either FP or DW (*n* = 7)

	DW	FP	<i>P</i> -value ^a
Recovered dust (mg)	144 ± 197	81 ± 116	
Relative percentage dust recoveries ^b	100	45 ± 22	<0.05
Fel d 1 concentration in recovered dust (mU/mg)	33 ± 31	44 ± 55	0.83
Relative percentage Fel d 1 recoveries ^b	100	69 ± 66	<0.05

^aWilcoxon signed rank test.

^bRelative percent dust recoveries: (the amount of dust (or Fel d 1) recovered from either DW or FP pre-treated wood)/(the amount of dust (or Fel d 1) recovered from DW pre-treated wood) × 100.

Table 2 Residual concentrations of Fel d 1 on the vacuumed wood surfaces pre-treated with either DW or FP, measured by wet swab sampling (*n* = 3)

	DW	FP	<i>P</i> -value ^a
Fel d 1 concentration (mU/ml)	0.2 ± 0.2	28 ± 22	
Relative percentage recoveries from each section	0.1 ± 0.09	99.9 ± 0.09b	0.11

^aWilcoxon signed rank test.

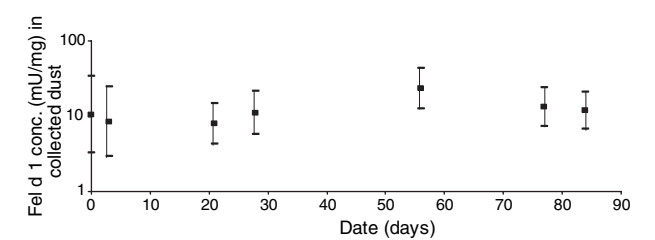


Fig. 2 Concentrations of Fel d 1 in sampled dust over time

ces. The results of wet swab sampling on the vacuumed surfaces are presented in Table 2.

Finally, the concentrations of Fel d 1 in dust did not significantly change over more than 80 days. The concentration of Fel d 1 ranged from 10.5 mU/mg (95% CI: 3.3–33.7 mU/mg) at time = 0 to 11.9 mU/mg (95% CI: 6.7–21.2 mU/mg) at time = 82 days. The Fel d 1 concentration over time is graphed in Figure 2.

Discussion

Allergen exposure due to re-aerosolization from surfaces can be significant, especially during periods of high activity and with allergen-laden dust inside the room. Cat allergen becomes airborne by minor disturbance and remains airborne for a much longer period of time than other allergens (de Blay et al., 1991). Our study demonstrates that allergen-laden dust sticks to a painted wood surface pre-treated with FP more strongly than to one pre-treated with DW. Stronger attachment of dust to the surface pre-treated with FP likely results in reduced dust re-aerosolization by room disturbances.

These characteristics are of particular importance for cat allergen because of its significant association with fine particles, resulting in a wide distribution and long airborne time (Wood et al., 1993). Two major sources of Fel d 1 in the house are the cat itself and furnishings (de Blay et al., 1991). Past studies reported that increased furnishings and low exchange rates play a significant role in increasing airborne cat allergen (de Blay et al., 1991; Luczynska et al., 1990). In the absence of direct shedding by a cat, cat allergen exposure is mostly generated by re-aerosolization of particles from reservoirs inside the room. Our study suggests that FP might help to reduce the concentration of, and possibly exposure to, airborne cat allergen by minimizing re-aerosolization from wood surfaces.

Hydrophobic particles, including most fungal spores, skin scales, and possibly other cat allergen-containing particles, may be bound more tightly to surfaces pre-treated with hydrophobic FP than to untreated (or DW pre-treated) surfaces. Ingredients such as isoparaffinic hydrocarbon solvent and silicone in FP may make dust stick to the surface stronger. We also found that concentrations of Fel d 1 in dust from FP pre-treated surfaces was similar to that from DW pre-treated surfaces. These results suggest that FP does not preferentially bind to dust containing cat allergen. However, as vacuuming at a rate of 1 cfm is considered very strong air disturbance and only removes 45% of dust from FP pre-treated wood, the dust-binding effect of FP is probably much more substantial during minimal or medium disturbances in the real world. In addition, we need to consider that the effect of FP is quite likely reduced in these experiments because the dust was heavily loaded onto the surfaces tested because of the limited working area.

Similar to a previous study (Wood et al., 1989), our study indicates that Fel d 1 is very resistant to decay in dust at room temperature for more than 80 days. The resistance to decay is commonly observed in other allergens as well (de Boer et al., 1995; Williams et al., 1999). In our study, the exposure of allergen-laden dust

to FP for 24 h did not increase the decay of Fel d 1. We did not examine the effect of FP over a prolonged period of time, which needs to be examined in the future.

There was significant variability in the amount of dust recovered by vacuuming in our study. This source of variability is most likely caused by different amounts of aerosolized dust generated by the Pitt 3 generator. This might be a problem if the effect of FP were influenced by the amount of loaded dust on surfaces. However, there was no significant association between percentage recovery and the amount of dust within the range of dust used in our experiments.

In conclusion, allergen avoidance should be recommended for not only managing symptomatic allergic individuals but also reducing the sensitized population (Platts-Mills et al., 2000b). The strategies for reducing allergen exposure include vacuum cleaning, air filtration, limiting upholstered furniture, and washing the cat. As a supplementary tool to these strategies, surface treatment with chemical agents such as FP can reduce airborne exposure by preventing re-aerosolization of allergen-laden dust. The attached dust on the surface can be wiped away during cleaning activity. In the future, field studies are needed to confirm our results.

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