

Prevalence of the group 1 *Dermatophagoides* allergens Der p 1 and Der f 1 in homes with no dogs, healthy dogs and *Dermatophagoides*-sensitized atopic dogs in Liverpool

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Abstract *Dermatophagoides farinae* is a frequent allergen in canine atopic dermatitis despite its reported scarcity in the UK, and the aim of this study was to determine whether dogs were uniquely exposed to this species. Der f 1 and Der p 1 in dust collected from living room carpets, bedroom carpets and dog beds of 13 houses with no dogs, 13 with healthy dogs, and 16 with *Dermatophagoides*-sensitized atopic dogs were quantified by ELISA. Der p 1 levels ($\mu\text{g g}^{-1}$ house dust) were significantly higher than Der f 1 in living rooms (Der p 1 median = 1.9, 95% CI = 2.05–6.32, $n = 42$; Der f 1 median = 0.07, 95% CI = 0.01–0.06, $n = 42$), bedrooms (Der p 1 median = 4.35, SD = 5.52; Der f 1 median = 0.01, 95% CI = 0.001–0.1, $n = 42$) and dog beds (Der p 1 median = 1.04, 95% CI = 1.4–8.1, $n = 29$; Der f 1 median = 0.008, 95% CI = 0.01–0.04, $n = 29$) ($P < 0.0001$). Living rooms in houses without dogs had significantly greater Der p 1 levels (median = 7.0, 95% CI = 3.53–15.8, $n = 13$) than houses with healthy (median = 1.19, 95% CI = 0.44–3.49, $n = 13$) or atopic dogs (median = 0.78, 95% CI = 0.63–2.42, $n = 16$) ($P = 0.0004$). Environmental flea control in living rooms and washing dog beds was associated with significantly reduced Der p 1 levels. This confirms that *D. pteronyssinus* is common but *D. farinae* is rare in the sampling area. Apparent sensitization to *D. farinae* is probably due to cross-reaction. A combination of environmental measures could reduce allergen exposure.

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease of humans and dogs, most commonly associated with a type 1 hypersensitivity against environmental allergens.^{1,2} The most frequent allergens implicated in the UK are the house dust mites *Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*.^{3,4} There is abundant evidence that they are important in the pathogenesis of canine AD: *Dermatophagoides* species mites are present in the environment and on the coats of atopic dogs;⁵ sensitization (as measured by allergen specific intradermal tests [IDTs], serology, basophil release tests and peripheral blood mononuclear proliferation assays) is frequent;^{3,4,6–9} cutaneous exposure elicits AD-like lesions;¹⁰ and specific immunotherapy¹¹ and allergen avoidance¹² ameliorate the clinical signs.

Previous studies related the geographical distribution of *Dermatophagoides* species to their preferred relative humidity levels. *D. farinae* dominates in drier, continental climates.¹³ *D. pteronyssinus*, in contrast, is more common in the UK's humid, maritime climate and *D. farinae* has been identified in very few homes.^{14,15}

Nevertheless, despite the reported rarity of *D. farinae* in the UK, sensitization to *D. farinae* in atopic dogs is at least as frequent as that to *D. pteronyssinus*. A study of IDT reactions in Edinburgh and London found that 40–50% of dogs reacted to *D. farinae* but only 10–20% reacted to *D. pteronyssinus*, and concluded that sensitization to *D. pteronyssinus* in the absence of sensitization to *D. farinae* was rare.³ A later study in Edinburgh found that up to 67% and up to 50% of atopic sera bound to western blots of *D. farinae* and *D. pteronyssinus*, respectively.⁴ IDT reactions to *D. farinae* are also more frequent than to *D. pteronyssinus* in France¹⁶ and Japan.¹⁷

These findings suggest that dogs may be particularly exposed to *D. farinae*. A study in Germany found that having a dog in the household correlated with environmental levels of the stable group 1 allergen Der f 1.¹⁸ Canine squames may therefore be a particularly good substrate for *D. farinae*, or dog-owning households may have microclimates that favour this species. Previous reports may have underestimated *D. farinae* numbers by concentrating on areas occupied by humans and not, for instance, dog beds. Research in an area of Brazil thought to have low numbers of *D. farinae*, furthermore, found equal levels of environmental Der f 1 and Der p 1.¹⁹ The authors speculated that seasonal population fluctuations could lead to an artificially low estimate of *D. farinae* numbers. The distribution of *Dermatophagoides* species also varies within small

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geographical areas. In Croatia, for instance, *D. pteronyssinus* is more common than *D. farinae* along the Mediterranean coast but the reverse is true inland.²⁰

The aim of this study therefore was to quantify the levels of environmental Der p 1 and Der f 1 in homes without dogs, with healthy dogs, and with atopic dogs to estimate and compare exposure to *D. pteronyssinus* and *D. farinae* in Liverpool.

MATERIALS AND METHODS

Sample populations

Atopic dermatitis was diagnosed by the Dermatology Service of The University of Liverpool Faculty of Veterinary Science on the basis of a compatible history and clinical signs (a chronic, perennial pruritus that was responsive to glucocorticoids but was not, or only partially, responsive to antimicrobial and antiparasitic treatment) and exclusion of other causes of pruritus.⁴ All atopic dogs, furthermore, had positive IDT reactions to *D. pteronyssinus* and *D. farinae* (Greer Laboratories, Lenoir, NC, USA; 0.001 w/v diluted in phosphate buffered saline/0.4% phenol) carried out under standard conditions.⁴ Briefly, the positive (0.1% histamine phosphate) and negative control (phosphate buffered saline with 0.4% phenol) sites were arbitrarily assigned scores of 4 and 0, respectively. Each test site was assigned a score in comparison to these sites. Test sites scoring 2 or more were considered positive.

Healthy dogs had no history or clinical signs consistent with an inflammatory dermatosis at the time of sampling. Homes without dogs were defined as having had no mammalian or avian pet present for the duration of the current occupancy (greater than 12 months in all cases). None of these households had any direct connection to The University of Liverpool Faculty of Veterinary Science.

No specific allergen avoidance measures were in place and no other terrestrial animals apart from dogs were present in any household. Households were selected from those that were known to the authors, that only had dogs, were willing to be sampled and that fulfilled the entry criteria.

Sample collection

Dust samples were collected from 13 houses with no dog, 13 houses with a healthy dog and 16 houses with an atopic dog. All the samples were collected between February and April in 2004. Dust was collected from living room carpets, bedroom carpets and dog beds by vacuuming four, evenly spaced 21 × 30 cm (i.e. A4) areas in each room for 30 s each using a Mitest® filter (Indoor Biotechnology, Cardiff, UK) according to the manufacturer's instructions. For smaller sites (e.g. dog beds) appropriate 21 × 30 cm areas were vacuumed for 1–2 min to give the same total vacuum time.

A standard questionnaire was used to collect data about the carpet (length of pile, age as stated by the owner and last time washed [with a mechanical wet and

dry or steam cleaner]), age of house as stated by the owner, use of central heating, number of human and animal occupants, the use of environmental ectoparasitocides, age (as stated by the owner) of the dog's bed and the last time it was washed, and the power of the different vacuum cleaners used to collect the samples. The length of carpet pile was categorized as short (< 1 cm), medium (1–2 cm) and long (> 2 cm).

Allergen extraction

One hundred mg of fine dust direct from the collection device was mixed end over end for 2 h with 2 mL 0.05% tween 20 in phosphate buffered saline at pH 7.4 (PBST) at room temperature. Samples less than 100 mg were incubated with the appropriate volume of PBST to give a 50 mg mL⁻¹ solution. Samples with < 30 mg were discarded. The solutions were then centrifuged at 550 g at 4 °C for 20 min and the supernatant stored at -20 °C.

Quantification of Der f 1 and Der p 1

An enzyme-linked immunosorbent assay (ELISA) was used to measure the concentrations of Der p 1 and Der f 1. Polystyrene microtitre wells (Immulon II®; Thermo Electron Bioscience Technologies, Basingstoke, UK) were coated with 200 ng/well anti-Der p 1 (mAb 10B9) or anti-Der f 1 (mAb 6A8) (Indoor Biotechnology, Cardiff, UK) and incubated at 4 °C for 16 h. Each well was blocked for 1 h at room temperature with 100 µL 1% bovine serum albumin (BSA) in PBST. One hundred µL of 10-fold dilutions from 250 to 0.5 ng mL⁻¹ of purified reference solutions of either Der f 1 or Der p 1 (Indoor Biotechnology) were added to the appropriate wells. One hundred µL of the test samples were added at 1/20 and 1/40 dilutions. Negative controls with no allergen were included and all wells were in duplicate. The plates were incubated for 1 h at room temperature before adding 100 µL of a 1/1000 dilution of biotinylated antigroup 1 antibody (mAbs 5H8 for Der p 1 and 4C1 for Der f 1; Indoor Biotechnology) to each well and incubating for a further hour at room temperature. One hundred µL of 250 ng mL⁻¹ streptavidin-peroxidase (Sigma, Poole, UK) was added to each well and the plates incubated at room temperature for 1 h. The assays were developed by adding 100 µL tetramethylbenzidine (TMB; Sigma) 0.1 mg mL⁻¹ in citrate buffer pH 4.0 with 0.006% hydrogen peroxide to each well for 30 min at room temperature and then stopping the reaction with 100 µL 1 M sulphuric acid. The plates were washed three times with PBST between each step. All reagents were diluted in PBST/1% BSA. A mixture of samples from each group and different rooms were included in each assay. Previous studies found no cross-reaction between the anti-Der p 1 and anti-Der f 1 reagents (Fig. 1).

Optical density was read at 450 nm (Multiskan EX, Thermo Electron Bioscience Technologies). The reference solutions were used to construct a standard curve for each plate (Fig. 1). Regression equations were used to calculate the µg allergen g⁻¹ house dust for each

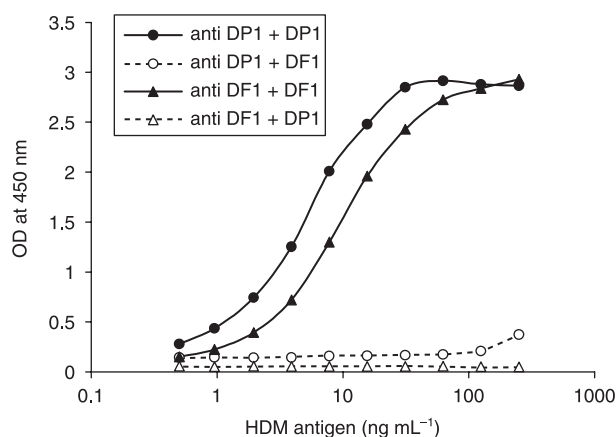


Figure 1. Standard curves generated by incubating dilutions of the purified Der p 1 and Der f 1 reference solutions with both the anti-Der p 1 and anti-Der f 1 reagents. The homologous reactions are represented by solid points and lines; the heterogeneous reactions by open points and dashed lines.

sample (Instat®; Graphpad Corp, San Diego, CA, USA).

Data analysis

Testing for Gaussian distribution before analysis (Kolmogorov-Smirnov test for normality; Instat®, Graphpad Corp) revealed a mix of normal and non-normal data sets. Nonparametric tests were therefore used. Kruskal-Wallis tests with Dunn's post-tests were used to compare the mean levels of allergen between homes without dogs, homes with healthy dogs and homes with atopic dogs for each room, and the amount of Der p 1 collected from short, medium or long carpets. Mann-Whitney tests were used to compare Der p 1 and Der f 1 levels in dog beds used by healthy and atopic dogs, and the effect of washing or environmental flea control on Der p 1 levels in carpets and dog beds. Wilcoxon matched pairs tests were used to compare the levels of Der p 1 and Der f 1 in matched rooms. Spearman rank correlations were used to analyse any association between the level of allergen collected and the power of the vacuum cleaner, age of the carpet and house, and number of occupants. Significance was set at $P < 0.05$ corrected for ties.

RESULTS

Der p 1 and Der f 1 levels in living rooms, bedrooms and dog beds

There were significantly higher levels of Der p 1 compared to Der f 1 in living rooms, bedrooms and dog beds (Fig. 2); $P < 0.0001$. Der f 1 levels were generally 100–1000 times lower than Der p 1 and close to the lower limit of detection.

Der p 1 and Der f 1 levels in houses without dogs, with healthy dogs and with atopic dogs

Living rooms in houses with no dogs had significantly higher amounts of Der p 1 than houses with either

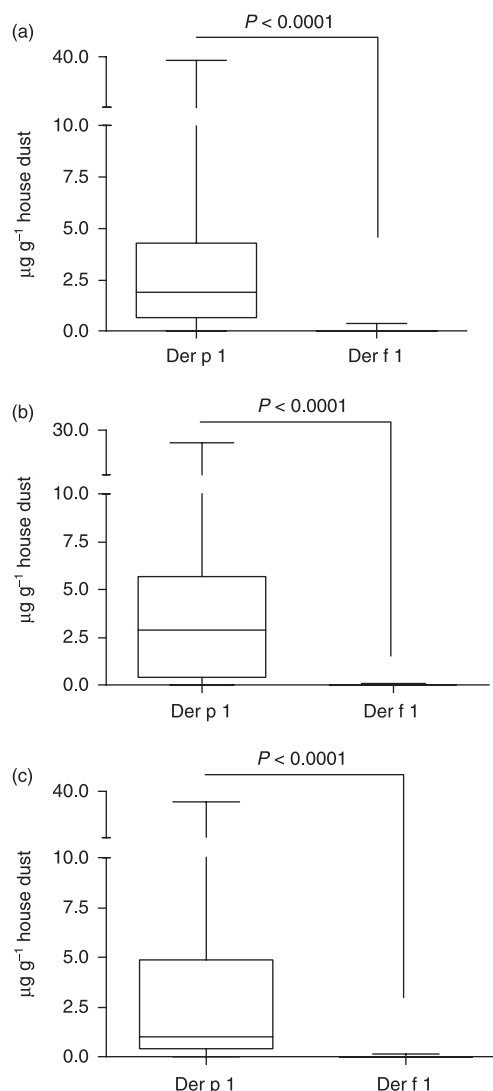


Figure 2. Levels of Der p 1 and Der f 1 ($\mu\text{g g}^{-1}$ house dust) in living rooms, bedrooms and dog beds (median, quartiles and range); (a) living rooms; (b) bedrooms; (c) dog beds.

healthy dogs or atopic dogs ($P = 0.0004$). There was no significant difference between the latter two groups. No significant differences were observed between levels of Der p 1 in bedrooms or dog beds of any group, or between levels of Der f 1 in the different rooms of any group (Table 1).

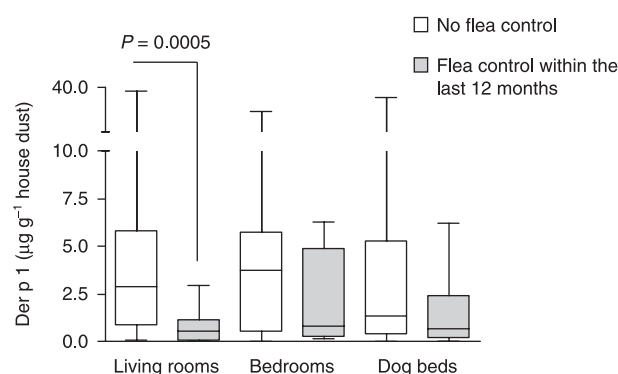
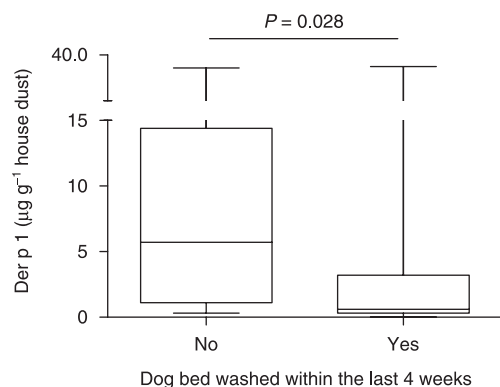
Der p 1 levels and environmental factors

Der p 1 levels were used to investigate factors that could affect the amount of allergen collected. Der f 1 levels were not analysed further in view of the very low levels detected.

The use of direct environmental flea control within the previous 12 months was associated with significantly reduced levels of Der p 1 collected from living rooms (Fig. 3); $P = 0.0005$. Der p 1 levels were also reduced following environmental flea control in dog beds and bedrooms, but the differences were not significant. The exact products used varied widely between and within households, although all contained a

Table 1. Levels of Der p 1 and Der f 1 ($\mu\text{g g}^{-1}$ house dust) in living rooms, bedrooms and dog beds in houses with no dogs, with healthy dogs and with atopic dogs

	Median (95% CI, n)	
	Der p 1	Der f 1
Living rooms		
No dogs	7 (3.53–15.8, 13)	0.07 (0.001–0.04, 13)
Healthy dogs	1.19 (0.44–3.49, 13)	0.07 (0.006–0.05, 13)
Atopic dogs	0.78 (0.63–2.42, 16)	0.07 (0.006–0.1, 16)
Bedrooms		
No dogs	2.76 (0.57–7.82, 13)	0.03 (0.01–0.05, 13)
Healthy dogs	3.99 (0.96–9.31, 13)	0.005 (0.006–0.04, 13)
Atopic dogs	2.89 (1.74–5.92, 16)	0.02 (0.01–0.05, 16)
Dog beds		
Healthy dogs	1.14 (0.3–5.35, 13)	0 (0.009–0.05, 13)
Atopic dogs	0.92 (0.34–12.27, 16)	0.03 (0.01–0.05, 16)

**Figure 3.** The effect on environmental flea control on Der p 1 levels; median, quartiles and range.**Figure 4.** The effect of washing on Der p 1 levels in dog beds; median, quartiles and range.

pyrethroid with or without an insect growth regulator, and analysis of an individual effect was not possible.

Der p 1 levels were also significantly reduced in dogs beds that had been washed within the previous 4 weeks compared to those that had not (Fig. 4); $P = 0.028$. The effect of washing on Der p 1 levels in carpets was not analysed as only two houses had had carpets cleaned within the previous 12 months.

There was significantly more Der p 1 collected in bedrooms with medium length ($n = 11$) carpets compared to long ($n = 12$) or short ($n = 19$) carpets ($P =$

0.035; data not presented) but no differences were seen between carpets of different length in living rooms (short $n = 20$, medium $n = 13$, long $n = 9$). There was no association between the amount of allergen collected and the power of vacuum cleaner used to collect the dust samples ($P = 0.75$), the age of the carpet ($P = 0.44$) or house ($P = 0.87$), and the number of human occupants ($P = 0.68$) ($n = 42$ in each case; data not presented). Only 3/13 homes with healthy dogs and 2/13 homes with atopic dogs had more than one dog. All the homes were two-story, detached or semidetached, brick-built and centrally heated with fitted carpets. None had active humidity control, although relative humidity was not measured. The bedrooms were on the first floor, and the living rooms and dog beds on the ground floor in each house. In all but two cases (one each in the healthy and atopic groups) dog beds were in the living room area.

DISCUSSION

These results demonstrate that, as estimated from group 1 allergen levels, significant populations of *D. pteronyssinus* are present in Liverpool homes but that *D. farinae* is very rare. These findings are consistent with the known predilection of *D. pteronyssinus* for humid, maritime climates and *D. farinae* for dryer, continental climates^{13,21} and the results from other UK medical studies.^{14,15} A recent study in Bristol, furthermore, found that *D. pteronyssinus* mites and Der p 1 were readily detected but that *D. farinae* and Der f 1 were rare in samples from the skin, hair and bedding of nonhospitalized pet dogs.²² A study in Columbus, Ohio, in contrast, reported that *D. farinae* was the most common and dominant house dust mite species in dog-owning households.⁵ These results suggest that *D. farinae* is uncommon in the UK and therefore of little relevance to British dogs. Both Liverpool and Bristol, however, are on the wetter, western side of the UK with mean annual rainfall of 756 mm and 869 mm, respectively, compared to 551 mm for Cambridge and 638 mm for Edinburgh (UK Meteorological Office; URL www.meto.gov.uk). These studies should therefore be repeated in the drier, eastern side of the UK, as regional differences in mite populations occur. In Croatia, for instance, *D. pteronyssinus* is more common than *D. farinae* along the more humid coast, but the reverse is true in the drier inland areas.²⁰ Indoor humidity was not measured in this study, although it is rare for British homes to have active humidity control.

This study found no differences in the levels of Der f 1 in houses with and without dogs, different rooms and dog beds. This contradicts an earlier study, which reported higher Der f 1 levels in houses with dogs.¹⁸ The authors speculated that canine squames may be a particularly good substrate for *D. farinae* or that dog-owning households may have microclimates that favour this species. This does not appear to be true in a *D. farinae* scarce environment, however, and any

relationships between dogs and different mite species require further study.

Sensitization to dust mite allergens has been shown to be dose dependant in humans.²³ In our study, however, group 1 allergen levels were no higher in homes with atopic dogs than in those with healthy dogs. Thirty-eight per cent (15/39) of samples from homes with healthy dogs and 42% (20/48) of samples from homes with atopic dogs, furthermore, had Der p 1 levels above $2 \mu\text{g g}^{-1}$ house dust, which is the accepted threshold for sensitization in humans.²³ These results would not support a comparable theory that canine atopic dermatitis develops in response to a high environmental allergen load, and suggest that intrinsic factors are more important. In fact, our study found only one significant difference in allergen exposure: less Der p 1 was recovered from living rooms of houses with dogs (healthy or atopic) than from houses without dogs. This is possibly due to differences in household management as the living room is often a room that dogs share with their owners. This could necessitate more frequent and/or intense cleaning and vacuuming to remove hair and dirt, which could both reduce the amount of organic material to sustain dust mite populations and remove Der p 1 more efficiently.²⁴ Vacuum cleaner type has been associated with Der p 1 levels,²⁵ but the relative power of the different vacuum cleaners used in the study did not affect the results.

Der p 1 levels in dog beds were significantly reduced by washing within the previous month. It is likely that washing physically removes mites and Der p 1, preventing a build up of allergen over time.²⁵ Previous studies have recovered more group 1 allergens from dog beds,⁵ carpets and mattresses²⁵ over 1 year old although the effect of washing was not examined. Unfortunately, we could not determine the effect of washing carpets, as only two carpets had been cleaned within 12 months of the study. Unlike previous findings²⁵ the age of the carpet had little effect on the amount of Der p 1 recovered, although the fact that all but two carpets were more than 3 years old may have masked any differences.

The length of the carpet had little overall effect on the amount of Der p 1 detected, although this finding may reflect low statistical power in our study. We did, however, recover more allergen from medium length carpets in bedrooms, which is in line with previous work that reported that medium length carpets harboured most allergen.⁵ This could be because long carpets reduce the efficacy of vacuuming and therefore allergen collection. Other studies have shown a relationship between pile length and mite numbers, with hard flooring regarded as best for mite avoidance.^{26,27}

The use of direct environmental flea control preparations was also associated with reduced Der p 1 levels although this was only significant in living rooms. The exact products varied between households, but environmental flea control would be expected to reduce mite numbers, as commonly employed constituents

such as permethrin and methoprene are acaricidal.^{28,29} Acaricides have been used to reduce dust mite numbers and improve clinical signs in atopic dogs¹² but are unlikely to be effective alone as they do not generally affect residual allergen levels unless denaturing. More stringent cleaning combined with environmental flea control could explain why the reduction in Der p 1 levels was more marked in living rooms. Further studies are required but these results do suggest that direct intervention can have an impact on environmental allergen levels.

All the atopic dogs had positive IDTs to both *D. farinae* and *D. pteronyssinus* despite the lack of exposure to *D. farinae*. Other authors have also suggested that IgE responses in atopic dogs are specific to cross-reacting allergens present in both species.³⁰ There is generally 80–90% homology between *D. farinae* and *D. pteronyssinus* allergens^{31–34} and a cross-inhibition study with canine sera found that *D. farinae* and *D. pteronyssinus* allergens strongly cross-react.³⁵ The apparent importance of *D. farinae* in canine dermatology^{3,16,17} could therefore simply reflect the concentration of key allergens in widely used extracts. Further work to define and standardize the allergen content of *Dermatophagoides* extracts in different geographical areas is clearly needed.

This study estimated the prevalence of *D. pteronyssinus* and *D. farinae* by quantifying Der p 1 and Der f 1, as mite detection can be unreliable. Previous studies have only found mites in 22–30% of samples, speciation can be difficult and mite populations are subject to seasonal fluctuations.^{5,19,22} Group 1 allergens, in contrast, are abundant, stable and can be quantified using specific reagents. Der p 1 and Der f 1 are, respectively, 222 and 223 amino-acid, 25 kDa, papain-like cysteine proteases.³⁴ They are vulnerable to heat, pH and reduction³⁶ but are nevertheless stable in house dust, with an estimated half-life of 10 years under normal household conditions.³⁷ Group 1 allergens are predominantly associated with mite faecal pellets.^{31,38} *D. pteronyssinus* produces approximately 20 faecal pellets, comprising up to 10% Der p 1 (equivalent to $0.1 \text{ ng pellet}^{-1}$), per day.^{39,40} Der p 1 and Der f 1 are, however, minor allergens for atopic dogs. Several studies have demonstrated that the major allergens in canine AD are the high molecular weight proteins Der f 15 and Der f 18.^{4,41–43} It would be better to quantify these allergens in dogs' environments, but there are as yet no commercial reagents for this purpose.

CONCLUSIONS

These data confirm that, based on detection of group 1 allergens, *D. pteronyssinus* is common in the sampling area and *D. farinae* populations are negligible. Positive reactions to *D. farinae* are probably due to cross-reaction with *D. pteronyssinus*. Allergen levels are similar in homes with atopic and healthy dogs but a combination of allergen avoidance measures may reduce exposure.

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Résumé *D. farinae* est un allergène fréquemment mis en cause dans la dermatite atopique canine, malgré sa relative rareté dans l'environnement au Royaume Uni, et le but de cette étude était de déterminer si les chiens sont seulement exposés à cette espèce. Der f1 et Der p1 ont été collectés et mesurés par ELISA à partir des tapis du salon, des chambres et du lieu de couchage des chiens dans 13 maisons sans chien, 13 maisons avec un chien sain et 16 maisons avec un chien atopique allergique à *Dermatophagoides*. les niveaux de Der p1 ($\mu\text{g/g}$ poussière) étaient significativement plus élevés que les niveaux de Der f1 dans les salons (Der p1 moyenne = 1.9, 95% IC = 2.05–6.32, $n = 42$; Der f1 moyenne = 0.07, 95% IC = 0.01–0.06, $n = 42$), les chambres (Der p1 moyenne = 4.35, SD = 5.52; Der f1 moyenne = 0.01, 95% IC = 0.001–0.1, $n = 42$) et les lieux de couchage (Der p1 moyenne = 1.04, 95% IC = 1.4–8.1, $n = 29$; Der f1 moyenne = 0.008, 95% IC = 0.01–0.04, $n = 29$) ($P < 0.0001$). Les salons dans les maisons sans chien présentaient significativement plus de Der p1 (moyenne = 7.0, 95% IC = 3.53–15.8, $n = 13$) que les maisons avec des chiens sains (moyenne = 1.19, 95% IC = 0.44–3.49, $n = 13$) ou atopiques (moyenne = 0.78, 95% IC = 0.63–2.42, $n = 16$) ($P = 0.0004$). Un traitement de l'environnement contre les puces dans les salons et le lavage des literies des chiens était associé avec une diminution significative des taux de Der p1. Cette étude confirme que *D. pteronyssinus* est fréquent mais que *D. farinae* est rare dans les zones prélevées. La sensibilisation à *D. farinae* est probablement une réaction croisée. L'utilisation de mesures de contrôle dans l'environnement pourrait réduire l'exposition allergénique.

Resumen A pesar de su reportada escasez en el Reino Unido, *D. farinae* es un frecuente alérgeno causante de dermatitis atópica canina, y el propósito de este estudio fue determinar si el perro está sobreexposto de forma inusual a este organismo. Mediante la técnica de ELISA se analizaron y cuantificaron las proteínas Der f1 and Der p1 en el polvo recogido de moquetas en salones de estar, moquetas en habitaciones y de la cama de los perros, en 13 casas sin perros, 13 con perros sanos y 16 casas con perros con atopía producida por *Dermatophagoides*. Los niveles de Der p1 ($\mu\text{g/g}$ en polvo del hogar) fueron significativamente mayores que los niveles de Der f1 en los salones de estar (media para Der p1 = 1.9, 95% CI = 2.05–6.32, $n = 42$; media para Der f1 = 0.07, 95% CI = 0.01–0.06, $n = 42$), habitaciones (media para Der p1 = 4.35, SD = 5.52; media para Der f1 = 0.01, 95% CI = 0.001–0.1, $n = 42$) y camas de los perros (media para Der p1 = 1.04, 95% CI = 1.4–8.1, $n = 29$; media para Der f1 = 0.008, 95% CI = 0.01–0.04, $n = 29$) ($P < 0.0001$). Los salones de estar de las casas sin perros presentaron niveles significativamente mayores de Der p1 (media = 7.0, 95% CI = 3.53–15.8, $n = 13$) que las casas con perros sanos (media = 1.19, 95% CI = 0.44–3.49, $n = 13$) o que las casas con perros con atopía (media = 0.78, 95% CI = 0.63–2.42, $n = 16$) ($P = 0.0004$). El control de pulgas en los salones de estar y el lavado de las camas de los perros se asoció con una reducción significativa de los niveles de Der p1. Estos datos confirman que *D. pteronyssinus* es frecuente pero *D. farinae* es raro en las áreas donde se realizó el muestreo. La aparente sensibilización a *D. farinae* se debe probablemente a una reacción cruzada. Un combinado de medidas medioambientales podría reducir la exposición a alérgenos.

Zusammenfassung *D. farinae* ist ein häufiges Allergen bei caniner atopischer Dermatitis, obwohl seine Seltenheit in Grossbritannien beschrieben wurde; das Ziel dieser Studie war es herauszufinden, ob nur Hunde dieser Spezies ausgesetzt sind. Der f1 und Der f2 in Staub, der aus Wohnzimmer Teppichen, Schlafzimmerschimmel und

Hundebetten von 13 Häusern ohne Hunde, 13 mit gesunden Hunden, und 16 mit *Dermatophagoides* sensitiven atopischen Hunden gesammelt wurde, wurden mittels ELISA quantitativ bestimmt. Der p 1 Konzentrationen ($\mu\text{g/g}$ Hausstaub) waren signifikant höher als Der f 1 in Wohnzimmern (Der p 1 Median = 1.9, 95% Konfidenzintervall = 2.05–6.32, $n = 42$; Der f 1 Median = 0.07, 95% Konfidenzintervall = 0.01–0.06, $n = 42$), Schlafzimmern (Der p 1 Median = 4.35, Standardabweichung = 5.52; Der f 1 Median = 0.01, 95% Konfidenzintervall = 0.001–0.1, $n = 42$) und Hundebetten (Der p 1 Median = 1.04, 95% Konfidenzintervall = 1.4–8.1, $n = 29$; Der f 1 Median = 0.008, 95% Konfidenzintervall = 0.01–0.04, $n = 29$) ($P < 0.0001$). Die Wohnzimmer in Häusern ohne Hunde hatten signifikant höhere Der p 1 Konzentrationen (Median = 7.0, 95% Konfidenzintervall = 3.53–15.8, $n = 13$) als in Häusern mit gesunden (Median = 1.19, 95% Konfidenzintervall = 0.44–3.49, $n = 13$) oder atopischen Hunden (Median = 0.78, 95% Konfidenzintervall = 0.63–2.42, $n = 16$) ($P = 0.0004$). Die Umgebungsbehandlung zur Flohkontrolle in Wohnzimmern und das Waschen von Hundebetten stand im Zusammenhang mit signifikant reduzierten Der p 1 Konzentrationen. Diese Ergebnisse bestätigen, dass *D. pteronyssinus* häufig vorkommt in den Bereichen, wo die Proben entnommen wurden, während *D. farinae* selten ist. Die offensichtliche Sensibilisierung zu *D. farinae* ist vermutlich auf eine Kreuzreaktion zurückzuführen. Durch eine Kombination von Maßnahmen bei der Umgebungsbehandlung könnte eine Exposition zu den Allergenen vermindert werden.