

Human allergy to environmental pet danders: a public health perspective

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Abstract

The prevalence of human allergy to pet danders has increased rapidly over the past six decades, as a consequence of lifestyle changes that have enhanced ambient exposure to pet allergens. This is a problem of global public health importance, as the morbidity associated with allergic diseases disproportionately affects socio-economically disadvantaged populations, particularly children. Although the public often seeks advice from veterinary health-care professionals regarding healthy pet ownership practices, including strategies for reducing residential pet dander exposure, many misconceptions persist in the public domain regarding pet dander allergy, such as the belief that certain dog and cat breeds are ‘hypoallergenic’ due to their hair/coat type. This review considers the epidemiology of human exposure to the major cat and dog dander allergens, Fel d 1 and Can f 1 respectively, and the sensitization mechanisms to them, including the hygiene hypothesis and the putative role of bacterial endotoxin. The literature regarding primary and secondary exposures in different ambient environments is explored including threshold effects that influence allergen sensitization and elicitation of symptoms, and environmental intervention strategies that seek to reduce allergen exposure. Pet-specific factors, including the aetiopathogenesis of the several cat and dog allergens that have been characterized to the molecular level, individual animal characteristics that influence Fel d 1 and Can f 1 shedding, and pet-directed interventions intended to reduce allergen dispersal, are discussed.

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Introduction

Allergy to pet danders is a problem of major public health importance, especially to children. It is also an occupational hazard for some workers, such as animal health-care professionals. Both the incidence of allergic diseases and the prevalence of pet (dog and/or cat) ownership have increased substantially in industrialized nations over recent decades.^{1–3} Concomitant with these changes have been improvements in indoor climate control and increased use of upholstered furnishings and carpets. Efficient indoor climate control reduces air exchange with the outdoor environment (reducing dilution of indoor allergens), whereas upholstery and carpeting trap indoor allergens and complicate cleaning regimens.³ The net effect has been enhanced ambient exposure to pet danders in both the domestic and public environs. This review considers scientific advances over the past 15 years, which have contributed to understanding of the immunologic, epidemiologic and therapeutic aspects of pet dander allergy.

Search strategy

An electronic search was conducted using OVID multi-database (PubMed MEDLINE®, Agricola, CAB abstracts, BIOSIS previews) with the key word strategy: [Fel d 1 OR Can f 1 OR animal dander OR pet allergen(s)] * [asthma OR allergy OR atopic] for English-language articles. After removing duplicates, 1202 articles were posted. Irrelevant articles were excluded by title, and abstracts examined for 487. After analysis of abstracts, 204 articles were saved for reference purposes.

Aetiology of pet dander allergy

The human diseases most commonly associated with sensitization to environmental allergens include allergic asthma, atopic dermatitis, urticaria and allergic rhinitis/conjunctivitis ('hay fever'). Allergens are typically low-molecular-weight proteins or glycoproteins that arise from ubiquitous plant or animal sources, and are designated by a systematic nomenclature maintained by a subcommittee of the World Health Organization/International Union of Immunological Societies. The designation is derived from the first three letters of the source organism's genus name, the first letter of the species name and an Arabic numeral that indicates the chronology of the allergen's discovery.⁴ For example, the first allergen purified from domestic cat (*Felis domesticus*) dander is Fel d 1, whereas the first allergen purified from domestic dog (*Canis familiaris*) dander is Can f 1.

The term 'dander' refers to particles that are sloughed from animal skin. Although composed primarily of skin

flakes, its physical properties cannot be standardized and defined accurately, and hence the term is used in a generic sense. Allergens, such as Fel d 1 and Can f 1, can be carried on a diverse array of particles,⁵ many of which are of an aerodynamic size ($<3 \mu\text{m}$ in diameter) such that attached allergens remain air-borne for long periods after disturbance (i.e. continuously in occupied homes). Therefore, measuring allergen levels in filtered air may provide a better estimate of environmental load than that in house dust.⁶

Most organisms produce more than one type of allergen that can be recognized by an allergic person's immune system and when $>50\%$ of the allergic population is reactive to a particular allergen, it is referred to as a 'major' allergen; 'minor' allergens are recognized by $<50\%$ of the allergic population.⁷ The immunology of allergen recognition and allergic reactions involves a complicated cascade of events that remains incompletely understood, and a detailed discussion is beyond the scope of this review. However, knowledge of some basic concepts of allergic mechanisms is necessary for understanding of pet dander allergies.

The immunology of allergy development

The immediate allergic reaction, referred to as type-1 hypersensitivity, likely evolved as a protective mechanism against parasites. However, when directed against otherwise harmless allergens such as pollens and danders, this protective mechanism turns pathogenic. One potential explanation for this redirection of the immune response is referred to as the 'hygiene hypothesis', the basis of which is a skewing of immune function towards allergy-inducing immunoreactivity due to reduced exposure to childhood parasitic, bacterial and viral infections in modern societies. This is characterized immunophenotypically by a shift from the usual dominance of T helper type-1 (T_H1) lymphocytes to dominance by T helper type-2 (T_H2) cells.⁸ In the absence of their usual target (parasites), T_H2 cells are directed against allergens, which are omnipresent in the natural and built environments. The production of T_H2 cytokines leads to priming of the allergic cascade, the phenotypic hallmark of which is production of elevated levels of allergen-specific IgE that can be detected by either serologic or skin-prick testing.⁸

As with any multifaceted biologic system, there is experimental evidence to both support and refute the components of the hygiene hypothesis. Clearly, not all people living in industrialized societies suffer from allergies, despite sharing environments. A genetic predisposition is obvious from familial studies, although the atopic state is a complex trait that is likely influenced by multiple genes.^{9,10} Still, the rapid increase in the incidence of allergic diseases in human populations cannot be explained by genetic models alone; shifts in exposure or immunoreactivity must also be in play.^{9,10} Assuming exposure to be equal, failure to develop clinical allergy must result from either a lack of immune response to allergens or one that induces immunological tolerance.¹¹ As tolerance is the ability of an active immune response to prevent or redirect sensitization to an allergen, and is thought to depend largely on exposure levels,¹¹ the epidemiology of pet

allergen exposure, sensitization and tolerance induction warrants consideration.

Epidemiology of pet dander exposures

The bulk of epidemiologic research on the topic of pet dander allergy has focused on Fel d 1, which is present ubiquitously in the human environment, and because cats are the dominant source of animal-origin allergy of people. Although levels of Fel d 1 are much higher in households with current cat residence,^{12,13} it can be detected in homes without resident cats.^{13–16} In homes where there has never been a resident cat, there is a direct correlation of indoor allergen level with the prevalence of cat ownership in the community at large.¹³ Fel d 1 is also detectible in many public places at levels capable of sensitizing or exacerbating symptoms in susceptible individuals. This is especially relevant and problematic for children in schools and daycare centres,^{17–22} but also occurs in the work place,²³ automobiles,¹⁴ airplanes,²³ cinemas²³ and even human hospitals.²⁴ Secondary exposure (e.g. to a room where a cat has been previously) can be high enough not only to sensitize individuals, but also to provoke acute allergic inflammation.²⁵

The evidence for sensitization and exacerbation of cat dander allergy in school children is compelling. Epidemiologic studies have demonstrated that sensitization of children without regular cat contact may increase in a dose-response fashion according to the degree of contact experienced by their classmates.^{21,22} Elicitation of symptoms is also possible: noncat-owning asthmatic children in high cat-ownership classrooms experienced a doubling of asthmatic symptoms and medication use in the 2 weeks following their return to school from holiday break. Those in low cat-ownership classrooms showed no such increases.²⁶

In schools and daycare centres, dog allergen levels may actually exceed those for cat allergens, and mattresses have been shown to be a source of higher in-school allergen levels than settled dust.^{17,19} Mattresses are a known reservoir for dust mite and pet allergens, and the quantity of Fel d 1 increases with the advancing age of the mattress.²⁷ Even newly purchased mattresses can contain detectible and relevant cat and dog allergen levels, although those wrapped in plastic when purchased are of less concern.^{28,29} A study in Swedish stores that allowed customers to 'try out' floor model mattresses showed highly statistically significant increases in the deposition of dog, cat and horse allergens. For cat and horse allergens, the concentrations detected strongly correlated with the duration of in-store display.²⁹ In non-cat owning homes, a combination of the presence of stuffed animals on the bed, lack of mattress and pillow encasements, and infrequent sheet washing were associated with significantly increased Fel d 1 levels in children's bedrooms.³⁰ The net experience suggests that use of allergen impermeable encasements for mattresses and pillows may be useful components of allergen avoidance strategies, although the efficiency of Fel d 1 exclusion by such materials varies widely³¹ and controlled trials have not been reported. Also of interest is the finding that pillows stuffed with synthetic fibres

trap significantly more pet allergens than feather pillows, an effect likely to be due to the tighter weave used in pillow covers to prevent extrusion of feathers.³²

Sources of secondary exposure go beyond just the indoor environment. The clothing of classmates, playmates and co-workers is commonly implicated.^{14,33} In fact, clothing is often the point-source for indoor contamination, as levels of Fel d 1 in the homes of noncat owners have correlated with the level of exposure experienced by their children in school, with the vehicle being clothing.²¹ Even human hair has been shown to serve as a vehicle for Fel d 1 dispersal.^{34,35} In societies where keeping of indoor pets is rare, the prevalence of sensitization to cat allergens is still similar to the rates observed in Western countries, suggesting that feral outdoor cats provide a sufficient degree of dander exposure to induce human allergy.³⁶ On the island of Crista da Cunha, where cats were eliminated in 1974, cat allergen was still present in the environment in 1999, and sensitization in the population continued, perhaps through exposure to visitors' clothing.³⁷

Because of the risk to asthmatic children, reduced-allergen school environments have been studied.^{20,38,39,40} Measures such as banning pet ownership^{20,38} or providing school-issued clothing for students during the school day³⁸ were successful in reducing ambient Fel d 1 concentrations, although the effects on allergic symptoms were not assessed.

Epidemiology of tolerance induction by pet allergen exposure

Immunologic tolerance is the ability of an active immune response to prevent or redirect sensitization to an allergen. This is thought to most commonly occur through natural or induced production of IgG4 blocking antibodies (referred to as a 'modified T_H2 response').¹¹ Induction of blocking antibodies by immunization with allergen vaccines (also known as allergen-specific immunotherapy or 'allergy shots') may be an important medical treatment strategy for individuals with pet dander allergies.³⁹ Although there has been some controversy surrounding the topic of pet allergen tolerance, the bulk of the accrued evidence suggests that high-level exposure to pet allergens has a protective effect against sensitization in children (reviewed by Woodfolk).⁴¹ This is true for both cats^{42–46} and dogs.^{47–49} However, the risk of childhood sensitization and symptoms at or after 3 years of age appears to stratify by maternal history of asthma.^{50,51} Children of asthmatic mothers had increased risk of sensitizing to high-dose Fel d 1 exposure whereas, in children with no maternal history of asthma, high-level exposure appeared to be protective.⁵⁰ Induced childhood tolerance to cat allergen in asthmatics may persist into adolescence and adulthood.⁵²

Mechanisms for tolerance induction: bacterial endotoxin

In accordance with the hygiene hypothesis, epidemiologic reports have suggested that children living in rural and farming environments in Western/industrialized countries show a decreased frequency of hay fever and asthmatic wheeze^{53–58} and allergic conjunctivitis (but not

atopic dermatitis)⁵⁶ than their urban peers. This protective effect against respiratory allergies has correlated with the degree of livestock exposure,⁵³ dog exposure,⁵⁴ amount of time spent on the farm⁵⁷ and sibship,⁵⁶ although the most interesting correlation has been with environmental endotoxin levels.⁵⁸ In farming environments, contact with livestock and unpasteurized milk is known to expose children to high levels of bacterial endotoxin.⁵⁹

Endotoxin is a lipopolysaccharide component of the cell wall of Gram-negative bacteria, which has complex effects on immune regulation. It is found in varying concentrations in both indoor and outdoor environments and can be accurately measured in dust and air.⁶⁰ Several studies have specifically tested for a correlation between endotoxin levels and exposure or sensitization to pet allergens.^{43,47,61–65} The inconsistent results reported between studies likely reflects the fact that the effect of endotoxin on the developing immune response, like most factors involved in immunity, is multifactorial and complicated. It appears to depend upon dose, timing of exposure (age of the host), environmental cofactors and, almost undoubtedly, genetic propensities.⁶⁰

Mechanisms for sensitization and tolerance induction: allergen dose thresholds

The issue of endotoxin exposure and augmentation aside, it is clear that the challenge concentration of the allergen is itself an important factor determining sensitization and symptomatic disease. As explained previously, epidemiologic studies of cat and dog exposure have suggested that low doses provoke sensitization, whereas high doses protect against sensitization. But how much is too much, and how much is just right?

The geometric means of indoor allergen concentrations can differ substantially and seasonally even among similar types of homes.^{13,66} Quantitative measurements are necessary for intervention planning and outcome evaluation; standardization is the key. Allergen levels may be measured in either reservoir house dust or in ambient air, and there are several collection modalities and assay methods commercially available for either purpose, which is problematic as inconsistent quantitative results are attained with them.⁶⁷ However, Liccardi *et al.*³ provide an excellent review of those currently available.

The average level of allergen contamination in reservoir dust of US homes, assessed from a representative sample of the entire US population (comprising 831 homes) was a geometric mean of 4.73 µg/g for Fel d 1 and 4.69 µg/g for Can f 1. When a pet-owning subset of these homes was considered (those with a resident cat or dog within the 6 months prior to sampling), geometric means were 200 and 69 µg/g respectively.¹⁶ In general, dust levels of Fel d 1 reported to be necessary for sensitization range from 4 to 20 µg/g,⁶⁸ although one group of investigators reported risk beginning at much lower levels (<1 µg/g).⁶⁹ For exacerbation of symptomatic airway disease, necessary levels are thought to range from 1 to 8 µg/g (Figure 1). Clearly, levels in the average home are sufficient to both sensitize and elicit symptoms in genetically predisposed individuals. Tolerance induction may occur at >20 µg/g, and is characterized by an IgG4

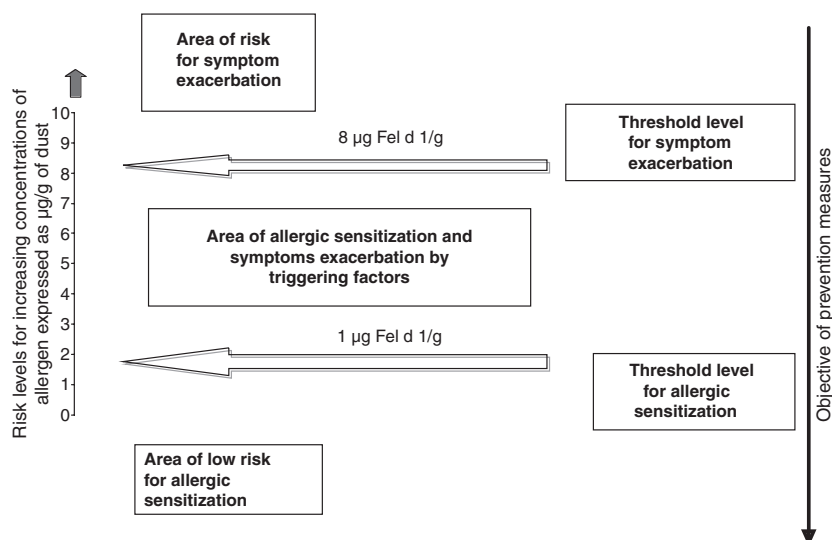


Figure 1. Risk levels for allergic sensitization and/or symptoms, according to the degree of exposure. Reprinted with permission from Ref. 3.

response to Fel d 1 rather than a dominant IgE response.^{8,11} This mechanism may be responsible for both natural tolerance expression and the favourable response that can result from allergen-specific immunotherapy using Fel d 1 vaccines.^{11,39}

Mitigation strategies: avoidance through environmental interventions

Because of the ubiquity of pet allergen dispersal, household and classroom intervention presents a difficult challenge. Several randomized interventional trials have been published and it is clear that environmental reduction of pet dander allergens can be achieved, in some instances with modest clinical benefits.^{72–74} However, the measures necessary – such as extensive home-cleaning regimens, removal of carpeting and upholstered furniture, and encasement of mattresses and pillows with impermeable membranes – can be quite costly and burdensome to sustain. In addition, socio-economic status is highly associated with environmental factors (such as housing characteristics, number of residents, pet ownership and general home hygiene) that strongly influence allergic sensitization,^{70,71,75} and it is apparent that the populations bearing the greatest burden of disease are often those with least access to health care and other resources.

One indoor lifestyle change that may have contributed to the rise of human allergic diseases over the past several decades is the increasing prevalence of wall-to-wall fixed carpeting, which cannot be removed for laundering.^{5,68,76} This is exacerbated with the advent of vacuum cleaners of variable efficacy for allergen removal from carpeting; allergen leakage from the machine and the vacuum bag is of major concern, and vacuuming using older models may actually increased air-borne allergen concentrations.⁶⁸ However, in the past decade, improvements such as two- and three-layer microfiltration vacuum bags, and high-efficiency particulate arresting (HEPA) filters in both vacuum machines and room air

cleaners (which claim to capture 99.9% of particles 0.3 µm diameter or larger) have increased allergen uptake and reduced leakage.^{77–79} The use of such units has been correlated with decreased allergen in reservoir dust and improvements in asthmatic patients,⁷⁷ although an evaluation of cat allergen levels after HEPA-filtrated vacuuming showed an actual increase in inhaled Fel d 1 when measured by nasal air sampling.⁷⁸ The authors attribute this to disturbance of allergen adhered to walls and other surfaces by all vacuum models due to beater bar action, the back-and-forth motion of the cleaning head and air exhaust flow from the machine. With regard to HEPA filtered air cleaners, results have also been mixed, with some suggesting excellent utility in reducing air-borne pet allergen levels,^{80–82} but not in reservoir dust,⁸³ and not to a degree where they are likely to be effective alone.^{83,84}

The effect of furniture dusting has also been evaluated in simulation studies, and as might be expected, dry-dusting of wood surfaces releases allergen, whereas the application of a spray polish to the dusting cloth or wood surface significantly reduces aerosolization of Fel d 1.^{85,86}

In addition to house-keeping regimens, laundering of clothing plays an important role in allergen reduction. Liccardi *et al.*⁸⁷ have shown that washing cotton fabrics in water is a simple and highly effective method for Fel d 1 removal. However, dry cleaning of woollens – although significantly reducing Fel d 1 concentrations – does not abolish it entirely, and allergen-free items can actually become contaminated during the dry cleaning process.⁸⁸ In a simulated laundering study using bedding linens, Tovey *et al.*⁸⁹ demonstrated that using detergents (rather than soap or water alone) at 25°C with at least 5 min extraction time is a superior method for removing Fel d 1. Choi *et al.*⁹⁰ demonstrated that the removal of Can f 1 from cotton sheets by mechanical washing machines was highly effective at all temperatures tested, but was significantly better at the highest temperatures (60°C and steam water). Two rinse cycles were the most beneficial; additional rinse cycles provided no additional gain.

Mitigation strategies: pet-directed interventions

It might seem that the most effective pet-directed intervention would be to remove the pet from the household. This very recommendation has been part of several interventional studies, and not surprisingly, compliance has been low. Some physicians and public health professionals will also suggest banishing pets to the outdoors, a recommendation that, especially for cats, is unacceptable to most veterinarians and pet owners. Although cats are often the focus of allergen reduction interventions, it is known that some degree of cross-reactivity occurs between canine and feline dander allergens, and there is a high prevalence of human co-sensitization to dogs and cats.⁹¹ Finally, although the removal of a pet is likely to reduce the overall allergen burden in a home, levels still may not fall below the sensitization or elicitation thresholds (as described above).

These facts considered, studies have been performed to assess the utility of pet bathing in reducing allergen burden. For both dogs and cats, bathing removes significant amounts of allergen from the pet, but must be performed at least twice weekly to maintain a relevant reduction.^{92,93} For dogs, this regimen achieves only a modest reduction of Can f 1 in room air.⁹³ Commercial products Allerpet/C[®] (for cats) and Allerpet/D[®] (for dogs) (Allerpet Inc., New York, NY, USA), available as shampoos (and as moistened wipes for cats), claim to substantially reduce the quantity of pet-related allergens in the home. However, an evaluation study showed that Allerpet/C[®] solution removed significantly less allergen than submersion in water, and no more allergen than application of damp towels.⁹⁴

Perhaps the most prevalent, but essentially irrelevant question presented to veterinary health-care professionals in relation to human allergies of pet origin, is which breed of dog or hair coat type will be 'hypoallergenic'. Can f 1 and Can f 2 (a minor allergen) are lipocalin proteins produced by the salivary glands, and neither has been localized to the skin.⁹⁵ Other minor allergens include Can f 3, which is canine serum albumin, and Can f 4, a salivary protein that has not yet been completely defined.⁹⁶ Recently, a new canine allergen – designated Can f 5 – has been characterized from dog urine. A kallikrein protein produced in the prostate, it appears to be recognized by the IgE of more dog-allergic people (70%) than Can f 1 (50%), which may explain the association noted with male sex of dogs in prior studies of dog-allergic people.⁹⁶

The widely held assumption that dogs that shed less (e.g., curly-coated breeds such as poodles, Portuguese water dogs, and Bedlington terriers) produce less allergen was found to be untrue; a comparison of 288 healthy dogs from several breeds (Labrador retriever, poodle, German shepherd, Pyrenean shepherd, Yorkshire terrier, Cocker spaniel, griffon, and spaniel of unspecified breed heritage) revealed that Labrador retrievers, which are notorious for perennial shedding, produced significantly less allergen than other breeds, whereas Yorkshire terriers and poodles produced the most.⁹⁷ Can f 1 did vary significantly with the seborrhic status of each dog, presumably due to increased allergen carrying capacity by a

greater mass of sloughed dander. It was also greater in males, but did not vary according to coat length.

Fel d 1 is a globulin secreted by the feline salivary,^{98,99} sebaceous^{98–100} and perianal¹⁰¹ glands, with the skin and hair serving as reservoirs.^{100,101} Its physiological function remains unknown. It is recognized by specific IgE produced by 85–95% of cat-sensitized people,³ and its production may be influenced by testosterone, as the sebaceous gland output of male cats is reduced after castration.¹⁰² In fact, a study of Fel d 1 levels in reservoir house dust showed reproductive status to be the only cat-dependent variable associated with allergen levels in homes. This was, however, the opposite of what would be expected: the homes of sterilized cats harboured significantly *more* allergen than those housing sexually intact animals (effect size greater for males than females).¹⁰³ Coat length, age, body mass and time spent indoors showed no effect in a multivariable regression model.¹⁰³ Allergen production did, however, vary with anatomical site, with the face producing dramatically higher levels than the chest.¹⁰⁴ This makes sense considering the greater glandular distribution and secretory activity of the facial skin.

Other minor (yet defined) cat allergens include Fel d 2, a serum albumin protein with extensive cross-antigenicity to other mammalian serum albumins, and Fel d 3, a cysteine protease inhibitor produced in the skin (exact structure of origin unknown).¹⁰⁵ These allergens are recognized by specific IgE in about 20% and 10% of cat-sensitized people respectively.³ A lipocalin salivary protein-designated Fel d 4, which was bound by specific IgE in 64% of sera from cat-allergic subjects, was characterized recently.¹⁰⁶ Although the majority of these sera samples harboured relatively low titres of anti-Fel d 4 IgE, 47% of them exceeded the corresponding titres of anti-Fel d 1 IgE. The clinical relevance of Fel d 4 allergen therefore merits additional investigation.

Recently, Allerca[®] Lifestyle Pets (San Diego, CA, USA) has produced and marketed 'hypoallergenic' cats and dogs. These have been developed not through genetic engineering, but rather by natural breeding of individuals deficient in Fel d 1 and Can f 1 production due to putative gene mutations. Although no refereed scientific reports have surfaced to date, the company offers plenty of satisfied client testimonials and claims that blinded clinical trials conducted by a reputable allergist have proven efficacy (<http://www.allerca.com/>, accessed 28 December 2009). Clearly this is not a public health solution (especially at a price ranging from US \$6950 to \$27,000 for a cat, and US \$8950 for a dog), but from a personal health perspective, it could be a welcome solution for individuals who are symptomatic only with direct pet exposure. Yet, as discussed previously, Fel d 1 and Can f 1 are not the only dander allergens recognized by all pet-allergic people, so it is conceivable that these purpose-bred pets will not provide complete benefit for affected people.

The human–animal bond is an undeniable facet of human health, both psychiatric and physical,¹⁰⁷ and it is clear that pets are in our hearts and homes to stay. Despite significant advances in understanding of the epidemiology and immunology of pet dander allergies over the past two decades, little progress has occurred in

mitigating this problem on a population-wide basis. Fortunately, medical therapies for use on an individual basis have improved significantly over this same time frame,¹⁰⁸ although access to health care remains a major road-block in many human populations. Considering the sheer magnitude of morbidity that allergic diseases create – especially paediatric asthma – the primary focus for public health professionals, now and in the foreseeable future, should be the elimination of disparities in healthcare access. As a significant amount of misinformation persists among the general public and human healthcare providers, the primary role of veterinarians will, for the foreseeable future, continue to be education regarding healthy pet ownership practices.

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Résumé La prévalence de l'allergie humaine aux animaux de compagnie s'est accrue rapidement ses six dernières décennies en raison des changements du mode de vie qui augmentent l'exposition ambiante à ces allergènes. Ceci est un problème de santé publique. La morbidité associée aux allergies affecte de façon inversement proportionnelle les populations socio-économiques désavantagées, en particulier les enfants. Alors que le public réclame souvent l'avis des vétérinaires en tant que professionnels de santé, y compris dans les stratégies permettant de diminuer la pression allergénique des animaux, de nombreuses fausses idées persistent dans le domaine public à propos des allergies liées aux animaux comme, par exemple, le fait que certaines races de chiens ou de chats seraient « hypoallergéniques » grâce à leur type de pelage.

Resumen La prevalencia de la alergia a la caspa de los animales domésticos en humanos se ha incrementado rápidamente en las pasadas décadas, como consecuencia de los cambios en la forma de vida que han aumentado la exposición a alérgenos de animales domésticos. Este es un problema de gran importancia

en la salud a nivel global, ya que la morbilidad asociada con la enfermedades alérgicas afecta de forma desproporcionada a poblaciones con desventajas socioeconómicas, especialmente los niños. Mientras que el público a menudo busca consejo de los profesionales veterinarios acerca de las prácticas ideales para mantener los animales domésticos, incluyendo estrategias para reducir la exposición a caspa de animales, existen muchas concepciones erróneas en el dominio público acerca de la alergia a animales domésticos, tal como la creencia de que ciertas razas de perros y gatos son 'hipoalergénicas' debido al tipo de pelo que tienen.

Zusammenfassung Die Prävalenz der Humanallergie auf Tierschuppen hat in den letzten sechzig Jahren als Konsequenz von Veränderungen im Lebensstil, wodurch eine Exponierung zu Haustierallergenen in der Umgebung vermehrt vorkommt, rapide zugenommen. Es handelt sich dabei um ein globales Gesundheitsproblem der Bevölkerung, da die Morbidität, die mit allergischen Erkrankungen im Zusammenhang steht, unverhältnismäßig oft sozialökonomisch minderprivilegierte Bevölkerungsgruppen, vor allem Kinder, betrifft. Während die Bevölkerung oftmals Rat von veterinärmedizinischem Gesundheitspersonal im Bezug auf eine gesunde Tierhaltung einholt, oft auch bezüglich der Strategien zur Vermeidung einer Exposition zu Tierschuppen im Haushalt, bestehen weiterhin in der Öffentlichkeit viele Missverständnisse im Bezug auf Tierschuppenallergie, wie zum Beispiel der Glaube, dass gewisse Hunde- und Katzenrassen aufgrund der Art ihres Haar- bzw. Fellkleides „hypoallergen“ sind.

要約 ペットアレルギーに暴露する機会が増えるというライフスタイルの変化に従い、ペットのフケにアレルギーを示す人は、過去 60 年で急激に増加した。アレルギー疾患の罹患率は社会経済的に恩恵を受けていない地域の子供に偏って発生していることから、世界的規模の公衆衛生上の重要な問題といえる。一般の人々に残存するペットのフケへの暴露を減らす方法を含む健康なペットの飼育法に関するアドバイスを、ヘルスケアの専門家である獣医師に要求する一方、ペットのフケアレルギーに関して特定のイヌやネコの種類がそれらの被毛のタイプによって“低アレルギー”であるという迷信など多くの誤解が社会全体に存続している。