Animal allergens are common causes of both acute and chronic allergic disease. The most important animal allergens are derived from mammals, principally cats, dogs, rats, mice, horses, and cows, which secrete or excrete allergens into the environment. Allergic sensitization may occur at home or in the workplace. Cat and dog allergens commonly cause allergies in the home and affect the general population. Laboratory animal handlers often have allergic reactions to rats and mice. Cow dander allergy is usually caused by occupational exposure and occurs in farmers and farm workers. Horse allergy occurs among people who regularly handle horses, either professionally or for recreational purposes. Over the past 20 years, the major animal allergens have been defined and characterized with regard to their molecular structure, immunogenicity, and environmental distribution. One remarkable finding has been the fact that most of the mammalian allergens that have thus far been cloned belong to a single family of proteins called the lipocalins. In addition to these molecular similarities, it has also been shown that most of the animal allergens are quite similar with regard to their aerodynamic properties. Although much is yet to be learned, progress is being made in our knowledge regarding the steps that may be necessary to control exposure to these allergens through environmental modifications in both homes and occupational settings. These measures include source control, air filtration devices, barrier devices, removal of carpeting and other reservoirs, and, in some cases, washing of the animal. (J Allergy Clin Immunol 2001;107:S414-21.)

**Key words:** Animal allergens, cat allergens, mammalian allergens

Cat allergen has been the most extensively studied animal allergen in terms of its structure, aerodynamic properties, environmental distribution, and the relationship between allergen exposure and the development of allergic disease and asthma. The immune response to cat allergen has been studied in numerous patient populations, as well as in patients undergoing immunotherapy. Recently, the molecular structure and functions of the other mammalian allergens, which belong to the lipocalin family of proteins, have been determined (Table I).

**CAT ALLERGEN**

The major cat allergen, Fel d 1, was first identified as “Cat-1” by Ohman in the 1970s and has proved to be an ideal marker for immunologic, environmental, and clinical studies of cat allergy. Fel d 1 is a 17-kd heterodimer comprising 2 disulfide-linked peptide chains of 70 and 90–92 amino acids (chain 1 and chain 2, respectively). Under native conditions, 2 of these heterodimers associate together to form an approximately 39-kd glycoprotein. Fel d 1 elicits IgE responses in 90% to 95% of patients with cat allergy and accounts for 60% to 90% of the total allergenic activity of cat extracts. The allergen is primarily produced in sebaceous glands and secreted onto the skin and fur. Other sites of allergen production in cats include the sublingual salivary glands and the anal glands. The production of Fel d 1 is thought to be under hormonal control. Castration reduces Fel d 1 production, and injections of testosterone into castrated cats permits Fel d 1 production to recover.

Both the cDNA and genomic sequences of Fel d 1 have been determined. Fel d 1 chain 1 shares approximately 25% homology to rabbit uteroglobin and human Clara cell protein; however, the relevance of this homology is unclear. In spite of the wealth of structural information about Fel d 1, the biologic function of this major allergen remains unknown. The allergen is secreted in copious amounts and accumulates in house dust at levels of up to 3000 µg/g of dust.

Cat albumin elicits IgE responses in about 20% of patients with cat allergy, and a few patients are selectively sensitive to this allergen. Recently, cystatin (cysteine protease inhibitor) has been cloned from a cat skin cDNA library and caused IgE responses in approximately 10% of patients with cat allergy. A comprehensive study of over 500 subjects with known cat allergies has shown that recombinant Fel d 1 and cat albumin could be used to replace natural cat allergen extracts for diagnostic purposes. Recombinant Fel d 1 produced in *Escherichia coli* needs to be refolded in order to bind IgE antibodies. Fully immunoreactive Fel d 1 has been produced by expressing both chains in baculovirus, and the baculovirus-expressed allergen shows excellent reactivity with both IgE and IgG antibodies. Both cat albumin and cystatin have been cloned and expressed in *Pichia pastoris*.

**OTHER MAMMALIAN ALLERGENS**

With the exception of dog albumin, most of the other important mammalian allergens that have been cloned...
family. Subsequently, several lipocalin allergens were also have IgE responses to dog albumin. Simi-
tarily, of patients with dog allergy. Some 25% of patients cause IgE responses in nearly 75% and 72.5%, respec-

**Table I. Structure and biologic function of selected mammalian allergens**

<table>
<thead>
<tr>
<th>Species</th>
<th>Allergen</th>
<th>Molecular weight (kd)</th>
<th>Biologic function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>Fel d 1</td>
<td>33-39</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td>Albumin</td>
<td>66</td>
<td>Serum protein</td>
</tr>
<tr>
<td></td>
<td>Cystatin</td>
<td>11</td>
<td>Cysteine protease inhibitor</td>
</tr>
<tr>
<td>Dog</td>
<td>Can f 1</td>
<td>16</td>
<td>Lipocalin</td>
</tr>
<tr>
<td></td>
<td>Can f 2</td>
<td>18</td>
<td>Lipocalin</td>
</tr>
<tr>
<td></td>
<td>Albumin</td>
<td>66</td>
<td>Serum protein</td>
</tr>
<tr>
<td>Rat</td>
<td>Rat n 1</td>
<td>15-17</td>
<td>Lipocalin, pheromone-binding protein</td>
</tr>
<tr>
<td>Mouse</td>
<td>Mus m 1</td>
<td>18</td>
<td>Lipocalin, odorant-binding protein</td>
</tr>
<tr>
<td>Horse</td>
<td>Equ c 1</td>
<td>19</td>
<td>Lipocalin</td>
</tr>
<tr>
<td></td>
<td>Equ c 2</td>
<td>18</td>
<td>Lipocalin</td>
</tr>
<tr>
<td>Cow</td>
<td>Bos d 2</td>
<td>19</td>
<td>Lipocalin</td>
</tr>
<tr>
<td></td>
<td>Bos d 5</td>
<td>20</td>
<td>Lipocalin, β-lactoglobulin</td>
</tr>
</tbody>
</table>

* A heterodimer comprised of 2 amino acid chains of 70 and 92 amino acids.  
† Three-dimensional structure was determined by means of molecular modeling.  
X-ray crystal structure is available at high resolution.  

Lipocalins were first identified as allergens after the cloning of the cockroach allergen Bla g 4. Sequence homology searches revealed that Bla g 4, rodent urinary protein allergens (Rat n 1 and Mus m 1), and milk allergen (β-lactoglobulin) were all members of the lipocalin family. Subsequently, several lipocalin allergens were cloned from dog, cow, and horse salivary glands or skin cDNA libraries (Table II). Lipocalin allergens are 16- to 20-kd proteins that show only 20% to 25% amino acid sequence homology but contain 3 structurally conserved regions containing conserved amino acids involved in ligand binding. The allergens have similar 3-dimensional structures comprising 8 antiparallel β sheets, with an α helix located between the last 2 β sheets, and a C-terminal 310 helix (Fig 1). X-ray crystallography studies of the rat and mouse urinary proteins revealed that the crystals contained a pheromone and suggested that those allergens were pheromone-binding proteins. The crystal structures of Bos d 2 and Equ c 1 have recently been determined. The dog allergens, Can f 1 and Can f 2, show a high degree of homology to human von Ebner’s salivary gland proteins, and Can f 1 may also be a cysteine protease inhibitor. Cow’s milk allergen, β-lactoglobulin (Bos d 5), is a retinol- and palmitate-binding protein. The functions of the other lipocalin allergens have yet to be determined.

In terms of allergenic importance, Can f 1 and Can f 2 cause IgE responses in nearly 75% and 72.5%, respectively, of patients with dog allergy. Some 25% of patients also have IgE responses to dog albumin. Similarly, over 80% of patients with rat or mouse allergy have IgE antibodies to Rat n 1 or Mus m 1, and Bos d 2 and Equ c 1 are equally important allergens in patients with known cow or horse allergy. Secretion of lipocalin allergens into the environment by pets and large mammals is an important cause of sensitization for IgE antibody responses and a risk factor for both domestic and occupational asthma.

**Immune Response**

The immune response to cat allergen (Fel d 1) has been extensively studied. Most patients with cat allergy make IgE, IgG1, and IgG4 antibodies to Fel d 1 after natural exposure to cat allergen, and Fel d 1 levels as low as 1 to 2 µg/g have been associated with cat sensitization. Nonallergic individuals can also make IgG responses to Fel d 1, and this correlates with the level of allergen exposure. After cat allergen immunotherapy, IgG antibody levels may increase from 10- to 50-fold relative to IgE and are associated with symptomatic improvements. Proliferative T-cell responses to Fel d 1 have been demonstrated in the majority of patients who are allergic to cats. In these patients Fel d 1–specific T-cell lines and clones have been isolated from circulating T cells in the peripheral blood. These studies focused on the identification of T-cell epitopes on Fel d 1 as part of a strategy to develop peptide-based immunotherapy for cat allergy. Counsell et al identified 2 large 27-amino acid peptides on Fel d 1 chain 1 that consistently induced T-cell proliferation (Fel-1, residues 7-33, and Fel-2, residues 29-55) with stimulation indices comparable with those of natural Fel d 1. These peptides were reported to elicit T-cell responses in 98% of patients with cat allergy. In an earlier study T-cell epitopes were also demonstrated on chain 2 of Fel d 1, suggesting a polymorphic response. Interestingly, in that study on a limited number of patients (n = 11), none of the T-cell clones obtained showed a reaction with cat albumin. A large group of patients (n = 95) was treated with Fel d 1 peptides as part of a placebo-controlled immunotherapy study, using doses of 7.5, 75, and 750 µg of the two 27-amino acid peptides. Treated patients showed significant improvements in nasal and lung symp-
tom scores after allergen challenge in a room containing cats, with airborne Fel d 1 levels of greater than 500 ng/m2.

There have been few studies of T-cell responses to lipocalin animal allergens. Gurka et al.40 reported isolation of T-cell lines from patients allergic to the rodent urinary proteins Rat n 1 and Mus m 1. More recently, a detailed study compared T-cell responses to bovine lipocalin allergen (Bos d 2) among Finnish farmers and farm workers.41 Although initial peripheral blood T-cell responses to the allergen were comparatively weak (stimulation index, <2), T-cell lines were obtained by restimulation of the lines with allergen or PHA and rIL-2, and these lines had 10– to 100-fold higher stimulation indices. Another feature of this study was that the T-cell epitopes mapped close to the structurally conserved regions of the lipocalin allergen family.

Although the molecular structure of animal allergens is now well defined, our knowledge of the cellular responses to these allergens is limited. There is an urgent need for larger population-based studies of cellular responses to animal allergens in all groups of sensitized patients. In addition, clinical trials of the use of recombinant animal allergens for diagnostic and treatment purposes should be vigorously pursued.

**ENVIRONMENTAL DISTRIBUTION OF ANIMAL ALLERGENS**

A number of studies have investigated the distribution of cat and dog allergens in home environments.42-49 Cat and dog allergen levels can range from less than 1 µg to greater than 3000 µg/g of dust. Using air and settled dust analyses, it has been shown that levels of cat and dog allergen are clearly highest in homes housing these animals. However, it is also clear from a number of studies that the vast majority of homes contain cat and dog allergen, even if a pet has never lived there. Although allergen levels in these homes are typically much lower than those found in homes with pets, they are often high enough to induce sensitization, and it is therefore common to see cat and dog sensitivity even in patients who have never had direct contact with these animals. This widespread distribution of animal allergens had been presumed to occur primarily through passive transfer of allergen from one environment to another, and there are now data from Swedish schools that elegantly demonstrate this process.50 These allergens appear to be very sticky and, unlike dust mite allergens, can be found in high levels on walls and other surfaces within homes.48
The characteristics of airborne cat and dog allergens have also been extensively studied. Cat allergen has been shown to be carried on particles that range from less than 1 µm to greater than 20 µm in mean aerodynamic diameter. Although estimates have varied, studies agree that at least 15% of airborne cat allergen is carried on particles less than 5 µm in size. Airborne levels and particle size distribution for dog allergen appear to be very similar to those for cat allergen, with about 20% of airborne allergen being carried on particles less than 5 µm in diameter. These smaller particles stay airborne for prolonged periods after disturbance, which explains why individuals with cat and dog allergy often experience symptoms on entering environments with these animals.

Cat and dog allergens can also be detected in air samples from all homes with cats and dogs and from many homes that do not house a cat or dog. The levels of airborne Fel d 1 and Can f 1 in homes with these animals typically range from 10 to 200 ng/m³. Bollinger et al detected airborne cat allergen in 10 of 40 air samples from homes without cats, whereas Custovic et al found airborne dog allergen in 11 of 36 homes without dogs. In addition, when a subset of those homes in the Bollinger study were reinvestigated on a weekly basis for 4 weeks, all of them had detectable airborne cat allergen at least once, and when the original 40 air samples were reanalyzed with a more sensitive assay, all 40 homes were found to contain airborne cat allergen.

In an attempt to determine the clinical significance of this unsuspected cat exposure, patients were challenged in an experimental cat exposure facility with varying levels of cat allergen. It was found that allergen levels less than 100 ng/m³ were capable of inducing upper and lower respiratory symptoms, as well as significant pulmonary function changes. These levels are similar to those found in homes with cats, as well as a subset of homes without cats, suggesting that even patients without known cat exposure may be exposed to clinically significant concentrations of airborne cat allergen on a regular basis.

The widespread distribution of cat and dog allergens is further demonstrated by several studies looking at allergen levels in schools and other public buildings. These studies have demonstrated moderately high levels of cat and dog allergen in schools and on the clothing of schoolchildren. A relationship between the number of cat owners in a classroom and the settled dust cat allergen level in that room has also been demonstrated. Most importantly, a strong case was made in a study of Swedish schools by Munir et al that the levels of cat and dog allergens in school classrooms are high enough to induce sensitization and cause perennial symptoms in children with asthma who are sensitized to cats and dogs.

Mouse and rat allergens have been best studied in laboratory settings. Airborne mouse allergen has been shown to reside on particles ranging from 3.3 to 10 µm in one study and from 6 to 18 µm in another study. Ohman et al also found that the particle size distribution was different, ranging from 0.43 to 3.3 µm, in rooms that did not contain mice. Airborne mouse allergen levels in the Ohman study ranged from 16.6 to 563 ng/m³ in rooms with mice and from 1.2 to 2.7 ng/m³ in rooms without mice, with the highest levels being associated with direct mouse contact. In another study levels ranged from 1.8 to 825 ng/m³ and varied with both the number of mice and the degree of work activity in the rooms. A final study demonstrated higher allergen levels in rooms with male mice compared with rooms with female mice (Mus m 1 3050 pg/m³ vs 317 pg/m³).

Airborne rat allergens are carried on particles ranging from less than 1 µm to greater than 20 µm, with the majority of allergens on particles less than 7 µm in diameter. Levels of airborne rat allergen have been studied in a variety of settings, and it is clear that exposure is highly dependent on the type of activity being performed, with cleaning and feeding being associated with the highest levels of exposure.

Studies have also been performed in individuals with rat allergy to determine the levels of exposure that would be expected to induce symptoms. In one study of 12 volunteers with rat allergy, all subjects experienced nasal symptoms, and 5 experienced a decrease in FEV₁ of greater than 10% during a 1-hour exposure, with airborne Rat n 1 levels ranging from less than 1.5 to greater than 310 ng/m³. In a follow-up study, exposures to high allergen levels (cage cleaning, mean Rat n 1 level of 166 ng/m³) were compared with exposures to low allergen levels (quiet sitting in a rat vivarium, mean Rat n 1 level of 9.6 ng/m³) in 17 subjects. Although no firm cutoff point for a safe allergen level could be determined, a clear dose response was demonstrated, with both upper and lower airway responses being highly dependent on airborne allergen levels.

### CLINICAL ASPECTS

Sensitivity to cat and dog allergens has been shown to occur in 22% to 67% of asthmatic patients, and in some settings these are clearly the dominant indoor allergens. This fact was best demonstrated in the study by Ingram et al conducted in Los Alamos, New Mexico. In this environment, where cat and dog allergens are common but exposure to dust mite and cockroach allergens is rare, IgE antibody to cat and dog was detected in 62% and 67% of asthmatic children, respectively. The presence of this IgE antibody was highly associated with asthma, whereas sensitivity to mite or cockroach allergen was not associated with asthma.

The effects of early exposure to animal allergens may or may not have a significant effect on eventual sensitization. In one study it was shown that intense exposure to cat allergen early in life leads to an increased risk of cat sensitivity, whereas in another study it was found that the presence of a cat in the home in the first year of life was associated with a reduction in cat allergy in adolescents. On the other hand, it is clear from multiple studies that cat sensitivity is common even in the absence of
obvious exposure. This is most likely related to the widespread distribution of cat and dog allergens, even in homes that do not contain these pets.

It has also been demonstrated that cat sensitivity is associated with asthma in older men and that cat sensitization in this population may predict the development of airway hyperresponsiveness. Litonjua et al\textsuperscript{75} reported that cat sensitivity was much more common in a group of asthmatic men with a mean age of 61 years than in control subjects (23.9\% vs 4.4\%, \(P < .001\)). In addition, they found that the development of new-onset airway hyperresponsiveness to methacholine was more common in subjects with established cat sensitivity than in those without cat sensitivity (18.2\% vs 6.1\%, \(P = .059\)).

The best demonstrations of the relationship of acute asthma to animal allergens come from the previously noted cat\textsuperscript{63} and rat\textsuperscript{69,70} challenge studies. These studies and others\textsuperscript{76-78} clearly demonstrate that asthma symptoms and substantial pulmonary changes are common with acute allergen exposure in sensitized subjects. Airway hyperresponsiveness to methacholine has also been shown to be a strong predictor of an asthmatic response. Although dose responses have been demonstrated in both cat and rat challenges, specific thresholds for airborne allergen levels at which an asthmatic response will either occur or not occur have not been defined.

The specific relationship of cat and dog allergens to chronic asthma has been less well characterized. Clinically, it is clear that many patients with asthma and cat or dog sensitivity have a more severe form of the disease because of continuous ongoing exposure to a family pet. However, it has also become clear from the studies noted above that many patients have significant cat and dog exposure, even though they are not aware of it. It is therefore likely that animal allergens are important causes of chronic airway inflammation, even in patients without known exposure. Further study will be needed to clarify this issue.

It is also clear that individuals who are in regular contact with rats, mice, and other laboratory animals commonly have sensitivity to those animals. As such, laboratory animal allergy represents a major occupational illness to the thousands of technicians, animal caretakers, physicians, and scientists whose work requires such exposure. Allergy to rats and mice is the most common clinical problem, with sensitivity to rats being reported in 12\% to 31\% of laboratory workers\textsuperscript{79-82} and sensitivity to mice occurring in 10\% to 32\% of such workers\textsuperscript{82,83}.

Although allergy to other animals in the workplace is less common than allergy to rats and mice, it is primarily because these other animals are used less often and not because they are necessarily less allergenic. Allergy to guinea pigs, rabbits, hamsters, gerbils, dogs, cats, pigs, cows, horses, sheep, and monkeys will therefore occur in workers exposed to these animals. In a very large epidemiologic study involving over 5000 laboratory animal workers in Japan,\textsuperscript{82} symptoms were reported in 26\% of workers exposed to mice compared with 25\% for rats, 31\% for guinea pigs, 30\% for rabbits, 26\% for hamsters, 25\% for dogs, 30\% for cats, and 24\% for monkeys.

ENVIRONMENTAL CONTROL OF ANIMAL ALLERGENS

At the present time, specific information on the control of animal allergens is still relatively limited, especially when compared with what is known about the control of dust mite allergens. In particular, there are still no convincing studies on the clinical benefits of environmental control measures for animal allergens. Although it is assumed that removing an animal from the home will lead to clinical improvement in patients who have disease related to their pet, even this has not been proven. Even fewer data are available regarding the potential benefits of methods that might be used in lieu of animal removal. Most of the published information about environmental controls relates to cat allergen. Although this information may also be applicable to other allergens, more research will need to be performed before pet removal from homes can be universally recommended.

On the other hand, it should be stated that in any asthmatic patient who is known to be cat sensitive and whose asthma is believed to be related to any significant degree to a pet cat, the most appropriate recommendation is to remove the cat from the home. This is clearly the correct advice from a medical standpoint, and healthcare providers should not shy away from strenuously recommending it.

Once a cat has been removed from the home, it is important to recognize that the clinical benefit may not be seen for a period of at least several months because allergen levels fall quite slowly after cat removal.\textsuperscript{37} In most homes levels in settled dust will have fallen to those seen in homes without cats within 4 to 6 months after cat removal. Levels may fall much more quickly if extensive environmental control measures are undertaken, such as removal of carpets, upholstered furniture, and other reservoirs from the home. Thorough and repeated cleaning will be required once the animal has been removed. Because cat allergen may persist in mattresses for years after a cat has been removed from a home,\textsuperscript{84} the purchase of new bedding or impermeable encasements should also be recommended.

However, a high proportion of patients are either reluctant or completely unwilling to remove a household pet. Therefore these individuals need to consider some alternative measures to control pet allergens in their environment. A number of studies have investigated other measures that might help to reduce cat allergen exposure without removing the animal from the home. De Blay et al\textsuperscript{85} demonstrated significant reductions in airborne Fel d 1 with a combination of air filtration, cat washing, vacuum cleaning, and removal of furnishings, although these results were based on a small sample size and did not include any measure of clinical effect. When cat washing was evaluated separately in that study, dramatic reductions in airborne Fel d 1 levels were seen after cat washes. Subsequent studies, however, have presented conflicting results. Klucka et al\textsuperscript{86} studied both cat washing and Allerpet/\textsuperscript{c} (Allerpet, Inc, New York, NY) and found no
benefit from either treatment. Avner et al\textsuperscript{87} studied 3 different methods of cat washing and found transient reductions in airborne cat allergens after each, but the benefit was not sustained, with levels of allergen returning to baseline within 1 week. A recent study on the effects of washing dogs on dog allergen\textsuperscript{88} produced similar results. Although allergen levels were reduced, the effects were transient, such that washing at least twice a week appeared necessary.

Information is limited as to the clinical benefits of these environmental control measures if one or more cats are allowed to remain in the home. Two recent studies\textsuperscript{89,90} evaluated different combinations of control measures and reached different conclusions. In the first study\textsuperscript{89} adults with asthma or allergic rhinitis and cat sensitivity were treated with a combination of a bedroom air cleaner and covers for mattresses and pillows. A control group was provided with nonactive air cleaners. Cats were restricted from bedrooms in both groups. Although airborne allergen levels were reduced, no significant differences were detected between the groups having active or placebo filters in any clinical parameter, including symptom scores, peak flow rates, medication requirements, pulmonary function studies, or methacholine challenges. In the second study\textsuperscript{90} children with asthma and sensitivity to either cat or dog allergen were treated with either active or placebo air cleaners in both the bedroom and the living room. Airborne allergen levels were not measured. The investigators found that the active treatment group in this study had a significant reduction on airway hyperresponsiveness. Therefore it does appear that, at least in some settings, it may be possible to produce a clinical effect without removing the pet from the home.

In families who insist on keeping their pets, the following steps should be recommended, pending more definitive studies. The animals should be restricted to one area of the home and certainly kept out of the patient’s bedroom. High-efficiency particulate air or electrostatic air cleaners should be used, especially in the patient’s bedroom. Carpets and other reservoirs for allergen collection should be removed whenever possible, again focusing on the patient’s bedroom. Finally, mattress and pillow covers should be routinely used. Although tannic acid has been shown to reduce cat allergen levels,\textsuperscript{91,92} the effects are modest and short lived when a cat is present, and therefore this treatment should not be routinely recommended. Similarly, cat and dog washings appear to be of such transient benefit that they are not likely to add significantly to the other avoidance measures.

REFERENCES

59. Dysbenal T, Elays S. Dust from carpeted and smooth floors. V. Cat (Fel d 1) and mite (Der p 1 and Der f 1) allergen levels in school dust. Demonstration of basophil histamine release induced by dust from classrooms. Clin Exp Allergy 1992:22:1100-6.
74. Hesselmar B, Aberg N, Eriksson B, Bjorksten B. Does early exposure to
cat or dog protect against later allergy development. Clin Exp Allergy
75. Litonjua AA, Sparrow D, Weiss ST, O’Connor GT, Long AA, Ohman JL.
Sensitization to cat allergen is associated with asthma in older men and
predicts new-onset airway hyperresponsiveness. Am J Respir Crit Care
76. Sicherer SH, Wood RA, Eggleston PA. Determinants of airway respons-
oses to cat allergen: comparison of environmental challenge to quantitative
nasal and bronchial allergen challenge. J Allergy Clin Immunol
1997;99:798-805.
77. Wood RA, Eggleston PA. Environmental challenges to animal allergens.
In: Spector S, ed. Provocation testing in clinical practice. New York: Mar-
cel Dekker; 1994.
78. Wood RA, Eggleston PA. Effects of intranasal steroids on nasal and pul-
monary responses to cat exposure. Am J Respir Crit Care Med
allergy to laboratory animals: an epidemiologic study. J Occup Med
Taylor AJ. Smoking, atopy, and laboratory animal allergy. Br J Ind Med
toms, sensitization and estimated exposure in workers not previously
82. Aoyama K, Ueda A, Manda F, Matsushita T, Ueda T, Yamauchi C. Aller-
gy to laboratory animals: an epidemiologic study. Br J Ind Med
83. Schumacher MJ, Tait BD, Holmes MC. Allergy to murine antigens in a
84. Van der Brent X, Chaplin D, Hadi E, da Mata P, Vervloet D. Cat removal
85. De Blay F, Chapman MD, Platts-Mills TAE. Airborne cat allergen (Fel d
1): environmental control with the cat in situ. Am Rev Respir Dis
86. Klacka CV, Owney DR, Green J, Zorati E. Cat shedding of Fel d 1 is not
reduced by washings, Allerpet-C spray, or acepromazine. J Allergy Clin
87. Ayner DB, Perzanowski MS, Platts-Mills TAE, Woodfolk JA. Evaluation
of different techniques for washing cats: quantitation of allergen removed
from the cat and effect on airborne Fel d 1. J Allergy Clin Immunol
1997;100:307-12.
88. Hodson T, Custovic A, Simpson A, Chapman MD, Woodcock A, Green
R. Washing the dog reduces allergen levels, but the dog needs to be
89. Wood RA, Flanagan E, Van Natta M, Chen PH, Eggleston PA. A place-
bo-controlled trial of a HEPA air cleaner in the treatment of cat allergy.
90. Van der Heide S, van Aalderen WMC, Kauffman HF, Dubois AEJ, de
Monchy JGR. Clinical effects of air cleaners in homes of children sensi-
91. Woodfolk JA, Hayden ML, Couture N, Platts-Mills TAE. Chemical treat-
Chemical treatment of carpets to reduce allergen: a detailed study of the
effects of tannic acid on indoor allergens. J Allergy Clin Immunol