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The Role of Allergen Exposure and Avoidance in Asthma

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Abstract

Allergy testing and avoidance of allergens plays an important role in asthma control. Increased allergen exposure, in genetically susceptible individuals, can lead to allergic sensitization. Continued allergen exposure can increase the risk of asthma and other allergic diseases. In a patient with persistent asthma, identification of indoor and outdoor allergens and subsequent avoidance can improve symptoms. Often times, a patient will have multiple allergies and the avoidance plan should target all positive allergens. Several studies have shown that successful allergen remediation includes a comprehensive approach including education, cleaning, physical barriers and maintaining these practices.

Introduction

Asthma is a common disease that affects 300 million people of all ages world wide.¹ The prevalence among children in the United States has nearly doubled over the last two decades.² Asthma is influenced by multiple factors including the environment, genetics, infections and diet. Allergen sensitization also plays an important role in disease development, severity and treatment. Patients with “allergic” asthma or IgE sensitization to allergens are more likely to develop chronic, persistent asthma. Recent studies have shown that sensitization to certain allergens such as house dust mite, animal dander, cockroach or fungus is a risk factor for asthma. Also, sensitization to pollens has been associated with seasonal asthma. The basis for an allergen’s role in asthma is that increased exposure can lead to sensitization and re-exposure to the allergens in a sensitized individual will increase the risk of asthma development and other allergic diseases. However, allergen avoidance or desensitization will improve symptoms. This review will describe environmental allergens, their role in asthma and strategies for avoidance.

House Dust Mite

House dust mites are arachnids that are microscopic in size (~0.33mm long). They are found in dust and products with woven material or stuffing such as mattresses, pillows, stuffed animals, and bedding. Their life cycle from egg to adult takes 3 to 4 weeks and they live for 6 to 8 weeks. Females produce 40 to 80 eggs during this time.³ Their natural food source is skin scales or fungi. The most common species are *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*. There are six major groups of house dust mite allergens but the most notable are Der p 1 and Der f 1. Fecal particles contain the most allergen and the highest dust mite concentrations are in mattresses. The major factors affecting dust mite growth are warmth and humidity. Mites absorb water in high humidity and get dehydrated when the humidity falls below 50%.

It has been shown that dust mite exposure in early childhood is an important determinant in asthma development. Sporik et al. showed that 16 of 17 children with asthma were sensitized to dust mite.⁴ Further, the higher the level of dust mite exposure at 1 year old, the earlier the first episode of wheezing occurred. The relative risk of asthma was almost 5-times greater in

the subjects who were exposed to high levels of dust mite allergen (>10 µg/g). These findings were confirmed in a recent study by Celedon et al.⁵ They demonstrated that early exposure to house dust mite was associated with an increased risk of asthma and late onset wheezing. They followed a group of 440 children from birth to 7 years and found that children exposed to high levels of dust mite allergen in their bed at 2 to 3 months old had a 3-fold increase in the odds of asthma at age 7 years old compared with those exposed to low level dust mite allergen.

One intervention to reduce dust mite levels is to encase the mattress and pillows with special covers. There are different fabrics such as plastic covers, permeable synthetic fibers, non-woven synthetics and finely woven fabrics. Woven fabrics with a pore size less than 10 microns block dust mite allergens and a pore size of 6 microns also block immature and adult mites and cat allergen.⁶ However, using dust mite encasings as the only intervention is controversial.

Halken et al. found a benefit from using encasings for long term dust mite control in asthmatic children.⁷ This was a prospective trial in which 60 children with dust mite allergy and asthma received pillow and mattress encasings or sham encasings for 1 year. There was a significant reduction in the amount of dust mite allergen in the treatment group and, after 1 year, inhaled steroids use was also reduced. Other studies have shown that dust mite covers alone, without a comprehensive avoidance plan, may not achieve a clinical benefit in asthmatic patients.^{8–12}

Experts agree that the greatest benefit will come from a comprehensive approach to dust mite avoidance and maintaining this practice (Table 1). Such an approach includes education about the allergen, implementing strategies like the encasings, removing dust mite reservoirs like carpets, upholstered furniture, drapes, and stuffed animals, keeping the humidity below 50% and weekly vacuuming with a HEPA filter.

Washing the bedding weekly and using a heated drier will reduce dust mite levels. Hot water (>130° F) killed all mites while cold water reduced mite concentrations by 90 percent.¹³ Also, using a clothes drier on the hot setting for 10 minutes killed all dust mites in a blanket.¹⁴ Chemicals such as benzl benzoate (kills mites) and tannic acid (denatures protein) have modest effects on reducing mite allergen.

Platts-Mills et al. demonstrated that long term dust mite avoidance in sensitized asthmatics resulted in a significant improvement.¹⁵ In this study, 9 patients used a hospital room as their bedroom for 1 year. They could leave the hospital but not visit houses with animals. By the end of the study, 2 patients were off all asthma medication and 5 patients no longer needed inhaled steroids. Symptoms and peak flows improved and 5 patients showed an 8-fold increase in their bronchial provocation tests.

Pets

Cats and dogs are the most popular animals kept as pets. Unfortunately, in atopic individuals, animal allergies are common. The major cat and dog allergens have been well characterized and will be the focus of this review. Other animal allergens have been extensively reviewed elsewhere.^{16, 17} Fel d 1 is the major cat allergen and is primarily found in cat skin and hair follicles and is produced in sebaceous, anal and salivary glands. All breeds of cat produce Fel d 1 and males produce a higher amount. The allergen can take months to degrade. It can be carried on small airborne particles ranging from 1 µm to over 20 µm.¹⁸

Dogs are similar. Their major allergen is Can f 1. It is found in hair, dander and saliva. Airborne levels and particle size distribution is similar to cat allergen. The small particles of cat and dog allergen can scatter easily in the air and adhere to clothing for further dispersal.

These allergens are wide spread. They have been found in homes and public places that do not have pets. Bollinger et al. found cat allergen present in 38 of 40 homes that did not have a cat.¹⁹ Almqvist et al. showed that there is significant exposure to cat allergen in schools as allergen is spread through clothing from homes with cats to classrooms.²⁰ Custovic et al. found dog allergen in dust samples from public places including schools, hotels, cinemas, buses and trains.²¹ Multiple studies have shown that pet owner's clothing is an important source of allergen dispersal.^{20, 22}

The role of exposure and sensitization to these allergens is unclear. Some studies show exposure results in increased development of allergy. Ingram et al. demonstrated that cat and dog allergens are associated with asthma in schoolchildren.²³ This study was done in an area of the country with low dust mite allergen, and they found that dog and cat were the major allergens to which asthmatic children were sensitized. Their results showed that the combination of sensitization and increased exposure levels for either dog or cat had a strong correlation with asthma. Though, there is controversy over whether animal exposure can also be beneficial. Recent studies suggest a protective effect where exposure to animals during infancy may reduce the prevalence of allergic sensitization later in childhood.^{24–29} However, there is agreement that in already sensitized individuals, animal allergens are problematic and can cause airway hyper-responsiveness and increase asthma severity.

The best way to decrease animal allergen exposure is to remove the pet from the home (Table 2). Even after removal, Wood et al. found that it took 20 to 24 weeks after cat removal to achieve allergen concentrations similar to controls.³⁰ Aggressive cleaning such as removal of carpets and upholstered furniture resulted in a more rapid decrease in cat allergen. Another reservoir for animal allergens is the mattresses so encasing the bedding is recommended.

Pets are often considered a member of the family and many owners will not remove them animal from the home. In these cases, recommendations include keeping the animal in a single area of the home and out of the bedroom, removing carpets and upholstered furniture, encasing the mattress and pillows, and using an air cleaner in the bedroom and living room.

In one study looking at HEPA air cleaners in the treatment of cat induced asthma and rhinitis, the investigators found that cat exclusion from the bedroom resulted in decreased allergen levels, though there was no effect on disease activity.³¹ However, van der Heide et al. conducted a 3 month intervention with air cleaners in living rooms and bedrooms and found that this helped significantly decrease airway hyperresponsiveness in twenty asthmatic children with animal sensitization with a pet in the home.³² Recently, the use of air filters has been extensively reviewed.³³ The authors concluded that there is sufficient evidence that air filtration reduces indoor levels of airborne allergens.

Cat and dog washing may be helpful but the effects are short lived. Hodson et al. found that dog washing reduced dog allergen but had to be done at least twice a week.³⁴ Cat washing studies show similar results or no benefit.^{35, 36}

Cockroach

The German cockroach (*Blattella germanica*) and the American cockroach (*Periplaneta americana*) are the most common species to cause allergies. The major allergens have been identified as Bla g 1, Bla g 2 and Per a 1. They are found in the saliva, fecal material, secretions, cast skins, and debris.¹⁸ As with dust mite allergen, these become airborne on aerodynamically large particles >10 µm and quickly settle to the ground.

Factors that increase cockroach infestation include low socioeconomic status, building degradation and high population density. Cockroach allergen is a major problem in inner city

homes and a significant cause of asthma morbidity. As with the other allergens, studies have shown that increased cockroach exposure leads to sensitization. Eggleston et al. studied a subset of 500 asthmatic children from the National Cooperative Inner-City Asthma Study (NCICAS). The children had skin testing to indoor allergens and home dust samples were analyzed. The results showed that the bedroom concentration of cockroach allergen was related to cockroach sensitization.³⁷ Recently, Chew et al. found a dose response relationship between inner-city home cockroach exposure and cockroach sensitization in children.³⁸

As part of the NCICAS, Rosenstreich et al. collected dust samples from 476 bedrooms of asthmatic children in 8 inner city areas.³⁹ Cockroach allergen was present in 85% of bedrooms. Allergy testing showed that approximately one third of children were allergic to cockroach. They found that children who were allergic to cockroach and exposed to high levels in their bedroom, had higher rates of hospitalization, medical visits, days of wheezing, missed school and lost sleep versus other indoor allergens. Gruchalla et al. conducted a similar study evaluating allergic sensitization, bedroom allergen exposure and asthma morbidity in 937 children from the Inner-City Asthma Study (ICAS) which included several metropolitan cities from the Northeast, Southwest and Western United States.⁴⁰ Cockroach allergen levels were highest in the high rise apartments and in New York, Chicago, and the Bronx. They also found that children who were both sensitive to cockroach and exposed to significant amounts of cockroach allergen had more asthma symptoms days, missed school and caretaker interrupted sleep.

Both of these studies demonstrated that sensitivity was related to the levels of cockroach allergen in the child's bedroom and children allergic to cockroach and exposed to cockroach allergen had more asthma symptoms.

Schools are another source of cockroach allergen exposure.^{41–43} Chew et al. found detectable levels of cockroach allergen in 77% of eleven Northeastern schools surveyed.⁴² Amr et al. detected cockroach allergen in 66% of dust samples from eleven downtown Baltimore elementary schools. They also found a positive correlation between the mean levels of cockroach allergen and asthma prevalence rates.⁴¹

Control measures include extermination with insecticides, pesticides or bait traps (Table 3). Food should be stored in sealed containers and clutter removed. Holes or cracks in the home should be sealed. These interventions along with thorough cleaning can significantly reduce allergen levels. Eggleston et al. used a combination of education, extermination, HEPA filters and bed encasings in inner city homes and demonstrated a 51% reduction in cockroach allergen.⁴⁴ Researchers from ICAS demonstrated that a 1 year multifaceted approach aimed at reducing environmental tobacco smoke and multiple indoor allergens could be successful.⁴⁵ The interventions included education, bed encasings, HEPA air filters and vacuum cleaners, and pest control. There was a significant reduction in cockroach and dust mite allergen levels and these significantly correlated with reduced asthma symptoms and morbidity during the intervention year and a year afterward

Mouse

It is clear that mouse allergens are an important cause of asthma morbidity in urban and suburban areas. In the past decade, numerous studies have found that exposure of sensitized children and adults results in an increased risk of asthma and worsened symptoms. Along with cockroach exposure, this has become an important public health problem.

Mouse allergen has been found to be highly prevalent in inner-city and suburban homes.^{46–48} Phipatanakul et al reported that 95% of all homes in a subset of 499 subjects from the

NCICAS had detectable levels of mouse allergen in at least one room and the highest levels were found in the kitchen.⁴⁹

Mus m 1 is the major mouse allergen and is found in mouse urine, hair follicles and dander.⁵⁰ Like other animal allergens, it is carried on particles and can migrate through a facility and can be found in homes without mice infestation⁵¹.

Factors that increase cockroach infestation also increase mouse infestation such as high population density, clutter and integrity of the home. Mice are more likely to be found in high rise apartments or multifamily homes and homes with physical damage.

High levels of mouse allergen can lead to sensitization. Phipatanakul et al. demonstrated that in subjects from the NCICAS, homes with higher mouse allergen concentrations had significantly higher rates of mouse sensitization.⁴⁹

Increased exposure is also a risk factor for asthma morbidity. A study of mouse sensitized children from inner-city Baltimore demonstrated that sensitized children exposed to high levels of home mouse allergen were more likely to have unscheduled physician visits, emergency department visits and hospitalizations.⁵² Household exposure to mice has been shown to be associated with higher rates of asthma in inner cities. A Boston birth cohort study demonstrated that infants whose parents reported exposure to mice in the household had almost twice the odds of developing a wheeze in the first year of life and later in childhood.⁵³ This has also been shown in adults. Women sensitized to mouse allergen were shown to have higher rates of a physician's diagnosis of asthma and asthma morbidity.⁵⁴

Mouse allergen remediation requires multiple interventions and has been termed Integrative Pest Management (IPM). Like dust mite allergen, a comprehensive approach is needed (Table 3). Tactics include education, extermination, cleaning, keeping food and trash covered, air filters and fixing holes and cracks in the home. Phipatanakul et al. used a multifaceted approach to demonstrate that a home environmental intervention can decrease mouse allergen.⁵⁵ Eighteen homes of asthmatic children with mouse sensitization in urban Boston were enrolled in the study. Twelve intervention homes received treatment which included filling holes, vacuuming and cleaning and using low toxicity pesticides and traps. Mouse allergen levels were significantly reduced over a 5 month period.

Pongracic et al. conducted a rodent intervention in a subgroup of children from the ICAS using a similar approach.⁵⁶ They found that mouse allergen reduction was associated with less missed school, sleep disruption and caretaker burden. If maintained, these efforts have been shown to be successful and cost effective.⁵⁷

Schools can also be a source of allergen exposure. Recently, Sheehan et al. found that 89% of dust samples from 4 inner city schools contained detectable levels of mouse allergen.⁵⁸ The school samples had significantly higher levels of mouse allergen when compared to the student's home samples.

Pollen and Mold

Pollen and mold spore exposure can trigger asthma exacerbations or worsen symptoms in sensitized individuals. Pollen's role in nature is to transport the male gamete (i.e. DNA) to the female part of a flower through wind or insect dispersal. In most parts of the United States, trees pollinate in the spring, grass pollinates from late spring to early summer, and weeds pollinate in late summer through the fall. In tropical climates, these seasons can be longer.

Trees are either seed bearing (gymnosperms) or flowering (angiosperms) and most allergenic trees are in the flowering group.⁵⁹ Birch, oak, elm, and maple trees are common in the Northeast and less prevalent in the Southwest.⁶⁰ A single birch tree flower cluster can produce 6 million pollen grains. Tree pollens range in size from 20 to 50 μm .

Ragweed pollen is released in the morning and peaks by noon.⁶¹ A single ragweed plant can expel a million pollen grains in a day. Species like ragweed, lamb's quarter and plantain are found throughout the United States. Weed pollens range in size from 10 to 20 μm .

Grass pollen is a common cause of spring time allergies. It is a larger pollen around 50 μm in size. Northern grasses in the United States include timothy, orchard, meadow, rye, Kentucky, redtop, and vernal, while southern grasses include Bermuda, Bahia and Johnson.

Fungal spores are responsible for both seasonal and perennial allergy symptoms. Outdoor spores peak in the mid-summer and diminish with the first hard frost in regions that experience cold winter seasons. There are dry air spores that peak in the afternoon hours under low humidity which include *Alternaria*, *Cladosporium*, and *Epicoccum*. Wet air spores peak at the pre-dawn hours with high humidity and include ascospores and basidiospores (mushrooms, puffballs). *Alternaria* is the most prevalent mold in dry, warm climates. It is commonly found in soil, seeds, and plants. Several studies have shown associations between *Alternaria* and severe asthma.^{62–65} *Cladosporium* is the most prevalent spore in temperate regions and is the most commonly identified outdoor fungus. It is found on dead plants or vegetable matter. *Aspergillus* is often isolated from house dust. It is also found in compost heaps and dead vegetation. *Penicillium* is found in soil, food and grains, and house dust. It grows in water damaged buildings, wallpaper, and decaying fabrics giving a green "mildew" color. All of these molds induce allergic rhinitis, asthma, and hypersensitivity pneumonitis.

Pollen and fungal spore counts are collected the previous day and then counted thus the reports we see on television or websites are a day late. The National Allergy Bureau (NAB) is part of the American Academy of Allergy, Asthma and Immunology and supplies the public with estimates of pollen and spore counts (www.aaaai.org/nab). The most common type of sampling devices are volumetric samplers that draw pollens and mold spores from the air. Studies have shown that there is variation in pollen counts even between locations in the same city.^{66, 67} Therefore, one pollen count may not represent the entire city, but the information does inform people when their pollen season begins and counts roughly correlate with symptoms.

Intact pollens range from 10 to 100 μm , though most have diameters between 20 and 35 μm while fungal spores range from 2 to 50 μm .⁶⁸ These large particles are removed by the nasal mucosa and upper tracheobronchial passages. However, particles <5 μm generally reach the alveoli of the lungs.⁶⁹ An intact pollen is too large to reach the alveoli but studies have shown that submicronic pollen derived bioaerosols can reach the alveoli. For example, ryegrass pollen ruptures on contact with water and releases microscopic starch particles (0.5–2.5 μm) containing major grass allergen.⁷⁰ Such bioaerosols have been shown to be expelled from pollens and mold spores.^{71–74} While rain can wash pollen grains or spores from the air, the expelled bioaerosols can trigger asthma exacerbations. Bioaerosols are thought to be associated with thunderstorm asthma epidemics.^{69, 71–74} In Melbourne, in 1987, there was a 5-fold increase in asthma ED visits in a 24 hour period after a thunderstorm. In 1989, there was a 10-fold increase with 277 ED visits.^{69, 75} In 1994, in London, 40 patients presented with asthma flares within 24 hours of a storm^{76, 77}.

There are a number of studies describing morbidity secondary to fungal exposure. *Alternaria* sensitivity has been found to be a risk factor for severe asthma attacks and epidemic asthma.^{62, 63} Targonski et al. demonstrated that Chicago asthma deaths were more than 2 times higher on days when there were 1000 spores per cubic meter.⁶⁴ Dales et al. studied the association

between daily emergency department visits for asthma to a children's hospital and concentrations of pollen grains and fungal spores during a 5-year period.⁷⁸ The authors found that the concentration of fungal spores and not grass pollen significantly correlated with ED visits. Sears et al. had similar findings in a group of New Zealand children followed from birth to 13 years of age. The most common allergen sensitization was grass; however, relative risk analysis demonstrated that sensitivity to house dust mite, cat, dog and indoor mold were highly significant risk factors associated with current asthma and grass sensitivity was not.⁶⁵

It is not possible to control the pollen or mold spore level outside the home, but one can control the amount of allergen that gets inside. Two major components of control are keeping windows and doors closed during high counts and bathing to remove allergens in the hair and on the body (Table 4). HEPA filters can reduce pollen in the home³³. Sensitized individuals with allergen-induced asthma can benefit from immunotherapy. A number of trials have shown that immunotherapy is beneficial in treating allergen induced asthma.^{79–84} Abramson et al. conducted a meta-analysis for the Cochrane Database of 20 published prospective, randomized, placebo controlled trials of immunotherapy for asthma between 1960 and 1990 and concluded that immunotherapy reduces asthma symptoms and use of asthma medications and improves bronchial hyper-reactivity.⁸⁰

Conclusion

Allergen exposure plays a significant role in asthma morbidity. We have demonstrated that, in genetically susceptible individuals, increased exposure leads to allergen sensitization and continued exposure is a risk factor for asthma and other allergic diseases. Thus, patients with persistent asthma may benefit from allergy testing to identify indoor and outdoor allergens. Successful allergen remediation involves a comprehensive approach that targets the multiple allergens to which they are exposed. This poses a major problem in inner cities as remediation of mouse and cockroach allergens can be difficult. The role of allergen exposure during infancy and the subsequent development of asthma is currently an active area of research. While this review focused on environmental factors, a recent study also hypothesizes that early life sensitizations, such as to food allergens, may affect functional airway properties.⁸⁵ At this point, more data is needed, but follow up studies may provide insight into asthma prevention.

REFERENCES

1. Masoli M, Fabian D, Holt S, Beasley R. The global burden of asthma: executive summary of the GINA Dissemination Committee report. *Allergy* 2004 May;59(5):469–478. [PubMed: 15080825]
2. Eder W, Ege MJ, von Mutius E. The asthma epidemic. *N Engl J Med* 2006 Nov 23;355(21):2226–2235. [PubMed: 17124020]
3. Denmark, HA.; Cromroy, HL. House Dust Mites, *Dermatophagoides* spp. (Acari: Pyroglyphidae). *Entomology Circular*. 1989 [Accessed December 11, 2009]. <http://www.doacs.state.fl.us/pi/enpp/ento/entcirc/ent314.pdf>
4. Sporik R, Holgate ST, Platts-Mills TA, Cogswell JJ. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study. *N Engl J Med* 1990 Aug 23;323(8):502–507. [PubMed: 2377175]
5. Celedon JC, Milton DK, Ramsey CD, et al. Exposure to dust mite allergen and endotoxin in early life and asthma and atopy in childhood. *J Allergy Clin Immunol* 2007 Jul;120(1):144–149. [PubMed: 17507083]
6. Vaughan JW, McLaughlin TE, Perzanowski MS, Platts-Mills TA. Evaluation of materials used for bedding encasement: effect of pore size in blocking cat and dust mite allergen. *J Allergy Clin Immunol* 1999 Feb;103(2 Pt 1):227–231. [PubMed: 9949312]
7. Halken S, Host A, Niklassen U, et al. Effect of mattress and pillow encasings on children with asthma and house dust mite allergy. *J Allergy Clin Immunol* 2003 Jan;111(1):169–176. [PubMed: 12532114]

8. Woodcock A, Forster L, Matthews E, et al. Control of exposure to mite allergen and allergen-impermeable bed covers for adults with asthma. *N Engl J Med* 2003 Jul 17;349(3):225–236. [PubMed: 12867606]
9. Terreehorst I, Hak E, Oosting AJ, et al. Evaluation of impermeable covers for bedding in patients with allergic rhinitis. *N Engl J Med* 2003 Jul 17;349(3):237–246. [PubMed: 12867607]
10. Platts-Mills TA. Allergen avoidance in the treatment of asthma: problems with the meta-analyses. *J Allergy Clin Immunol* 2008 Oct;122(4):694–696. [PubMed: 19014759]
11. Gotzsche PC, Johansen HK. House dust mite control measures for asthma. *Cochrane Database Syst Rev.* 2008;(2) CD001187.
12. Gotzsche PC, Johansen HK. House dust mite control measures for asthma: systematic review. *Allergy* 2008 Jun;63(6):646–659. [PubMed: 18445182]
13. McDonald LG, Tovey E. The role of water temperature and laundry procedures in reducing house dust mite populations and allergen content of bedding. *J Allergy Clin Immunol* 1992 Oct;90(4 Pt 1): 599–608. [PubMed: 1401643]
14. Miller J, Miller A. Ten minutes in a clothes dryer kills all mites in blankets. *J Allergy and Clin Immunol* 1996;97:423.
15. Platts-Mills TA, Tovey ER, Mitchell EB, Moszoro H, Nock P, Wilkins SR. Reduction of bronchial hyperreactivity during prolonged allergen avoidance. *Lancet* 1982 Sep 25;2(8300):675–678. [PubMed: 6126624]
16. Phillips JF, Lockey RF. Exotic pet allergy. *J Allergy Clin Immunol* 2009 Feb;123(2):513–515. [PubMed: 18980777]
17. Chapman MD, Wood RA. The role and remediation of animal allergens in allergic diseases. *J Allergy Clin Immunol* 2001 Mar;107(3 Suppl):S414–S421. [PubMed: 11242602]
18. Phipatanakul W. Environmental factors and childhood asthma. *Pediatr Ann* 2006 Sep;35(9):646–656. [PubMed: 16999298]
19. Bollinger ME, Eggleston PA, Flanagan E, Wood RA. Cat antigen in homes with and without cats may induce allergic symptoms. *J Allergy Clin Immunol* 1996 Apr;97(4):907–914. [PubMed: 8655885]
20. Almqvist C, Larsson PH, Egmar AC, Hedren M, Malmberg P, Wickman M. School as a risk environment for children allergic to cats and a site for transfer of cat allergen to homes. *J Allergy Clin Immunol* 1999 Jun;103(6):1012–1017. [PubMed: 10359879]
21. Custovic A, Green R, Taggart SC, et al. Domestic allergens in public places. II: Dog (Can f1) and cockroach (Bla g 2) allergens in dust and mite, cat, dog and cockroach allergens in the air in public buildings. *Clin Exp Allergy* 1996 Nov;26(11):1246–1252. [PubMed: 8955573]
22. De Lucca SD, O'Meara TJ, Tovey ER. Exposure to mite and cat allergens on a range of clothing items at home and the transfer of cat allergen in the workplace. *J Allergy Clin Immunol* 2000 Nov;106(5): 874–879. [PubMed: 11080709]
23. Ingram JM, Sporik R, Rose G, Honsinger R, Chapman MD, Platts-Mills TA. Quantitative assessment of exposure to dog (Can f 1) and cat (Fel d 1) allergens: relation to sensitization and asthma among children living in Los Alamos, New Mexico. *J Allergy Clin Immunol* 1995 Oct;96(4):449–456. [PubMed: 7560654]
24. Perzanowski MS, Ronmark E, Platts-Mills TA, Lundback B. Effect of cat and dog ownership on sensitization and development of asthma among preteenage children. *Am J Respir Crit Care Med* 2002 Sep 1;166(5):696–702. [PubMed: 12204868]
25. Perzanowski MS, Chew GL, Divjan A, et al. Cat ownership is a risk factor for the development of anti-cat IgE but not current wheeze at age 5 years in an inner-city cohort. *J Allergy Clin Immunol* 2008 Apr;121(4):1047–1052. [PubMed: 18395554]
26. Mandhane PJ, Sears MR, Poulton R, et al. Cats and dogs and the risk of atopy in childhood and adulthood. *J Allergy Clin Immunol* 2009 Oct;124(4):745–750. e744. [PubMed: 19703709]
27. Ownby DR, Johnson CC, Peterson EL. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age. *Jama* 2002 Aug 28;288(8):963–972. [PubMed: 12190366]
28. Ownby DR, Johnson CC. Does exposure to dogs and cats in the first year of life influence the development of allergic sensitization? *Curr Opin Allergy Clin Immunol* 2003 Dec;3(6):517–522. [PubMed: 14612678]

29. Celedon JC, Litonjua AA, Ryan L, Platts-Mills T, Weiss ST, Gold DR. Exposure to cat allergen, maternal history of asthma, and wheezing in first 5 years of life. *Lancet* 2002 Sep 7;360(9335):781–782. [PubMed: 12241839]
30. Wood RA, Chapman MD, Adkinson NF Jr, Eggleston PA. The effect of cat removal on allergen content in household-dust samples. *J Allergy Clin Immunol* 1989 Apr;83(4):730–734. [PubMed: 2708734]
31. Wood RA, Johnson EF, Van Natta ML, Chen PH, Eggleston PA. A placebo-controlled trial of a HEPA air cleaner in the treatment of cat allergy. *Am J Respir Crit Care Med* 1998 Jul;158(1):115–120. [PubMed: 9655716]
32. van der Heide S, van Aalderen WM, Kauffman HF, Dubois AE, de Monchy JG. Clinical effects of air cleaners in homes of asthmatic children sensitized to pet allergens. *J Allergy Clin Immunol* 1999 Aug;104(2 Pt 1):447–451. [PubMed: 10452769]
33. Sublett JL, Seltzer J, Burkhead R, Williams PB, Wedner HJ, Phipatanakul W. Air filters and air cleaners: Rostrum by the American Academy of Allergy, Asthma & Immunology Indoor Allergen Committee. *J Allergy Clin Immunol*. 2009 Nov 10;
34. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. *J Allergy Clin Immunol* 1999 Apr;103(4):581–585. [PubMed: 10200004]
35. Klucka CV, Ownby DR, Green J, Zoratti E. Cat shedding of Fel d 1 is not reduced by washings, Allerpet-C spray, or acepromazine. *J Allergy Clin Immunol* 1995 Jun;95(6):1164–1171. [PubMed: 7797784]
36. Avner DB, Perzanowski MS, Platts-Mills TA, Woodfolk JA. Evaluation of different techniques for washing cats: quantitation of allergen removed from the cat and the effect on airborne Fel d 1. *J Allergy Clin Immunol* 1997 Sep;100(3):307–312. [PubMed: 9314341]
37. Eggleston PA, Rosenstreich D, Lynn H, et al. Relationship of indoor allergen exposure to skin test sensitivity in inner-city children with asthma. *J Allergy Clin Immunol* 1998 Oct;102(4 Pt 1):563–570. [PubMed: 9802363]
38. Chew GL, Perzanowski MS, Canfield SM, et al. Cockroach allergen levels and associations with cockroach-specific IgE. *J Allergy Clin Immunol* 2008 Jan;121(1):240–245. [PubMed: 17936887]
39. Rosenstreich DL, Eggleston P, Kattan M, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 1997 May 8;336(19):1356–1363. [PubMed: 9134876]
40. Gruchalla RS, Pongracic J, Plaut M, et al. Inner City Asthma Study: relationships among sensitivity, allergen exposure, and asthma morbidity. *J Allergy Clin Immunol* 2005 Mar;115(3):478–485. [PubMed: 15753892]
41. Amr S, Bollinger ME, Myers M, et al. Environmental allergens and asthma in urban elementary schools. *Ann Allergy Asthma Immunol* 2003 Jan;90(1):34–40. [PubMed: 12546335]
42. Chew GL, Correa JC, Perzanowski MS. Mouse and cockroach allergens in the dust and air in northeastern United States inner-city public high schools. *Indoor Air* 2005 Aug;15(4):228–234. [PubMed: 15982269]
43. Sarpong SB, Wood RA, Karrison T, Eggleston PA. Cockroach allergen (Bla g 1) in school dust. *J Allergy Clin Immunol* 1997 Apr;99(4):486–492. [PubMed: 9111492]
44. Eggleston PA, Butz A, Rand C, et al. Home environmental intervention in inner-city asthma: a randomized controlled clinical trial. *Ann Allergy Asthma Immunol* 2005 Dec;95(6):518–524. [PubMed: 16400889]
45. Morgan WJ, Crain EF, Gruchalla RS, et al. Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med* 2004 Sep 9;351(11):1068–1080. [PubMed: 15356304]
46. Phipatanakul W, Gold DR, Muilenberg M, Sredl DL, Weiss ST, Celedon JC. Predictors of indoor exposure to mouse allergen in urban and suburban homes in Boston. *Allergy* 2005 May;60(5):697–701. [PubMed: 15813819]
47. Phipatanakul W, Eggleston PA, Wright EC, Wood RA. Mouse allergen. I. The prevalence of mouse allergen in inner-city homes. The National Cooperative Inner-City Asthma Study. *J Allergy Clin Immunol* 2000 Dec;106(6):1070–1074. [PubMed: 11112888]

48. Crain EF, Walter M, O'Connor GT, et al. Home and allergic characteristics of children with asthma in seven U.S. urban communities and design of an environmental intervention: the Inner-City Asthma Study. *Environ Health Perspect* 2002 Sep;110(9):939–945. [PubMed: 12204830]
49. Phipatanakul W, Eggleston PA, Wright EC, Wood RA. Mouse allergen. II. The relationship of mouse allergen exposure to mouse sensitization and asthma morbidity in inner-city children with asthma. *J Allergy Clin Immunol* 2000 Dec;106(6):1075–1080. [PubMed: 11112889]
50. Phipatanakul W. Rodent allergens. *Curr Allergy Asthma Rep* 2002 Sep;2(5):412–416. [PubMed: 12165208]
51. Ohman JL Jr, Hagberg K, MacDonald MR, Jones RR Jr, Paigen BJ, Kacergis JB. Distribution of airborne mouse allergen in a major mouse breeding facility. *J Allergy Clin Immunol* 1994 Nov;94(5):810–817. [PubMed: 7963149]
52. Matsui EC, Eggleston PA, Buckley TJ, et al. Household mouse allergen exposure and asthma morbidity in inner-city preschool children. *Ann Allergy Asthma Immunol* 2006 Oct;97(4):514–520. [PubMed: 17069107]
53. Phipatanakul W, Celedon JC, Sredl DL, Weiss ST, Gold DR. Mouse exposure and wheeze in the first year of life. *Ann Allergy Asthma Immunol* 2005 May;94(5):593–599. [PubMed: 15948302]
54. Phipatanakul W, Litonjua AA, Platts-Mills TA, et al. Sensitization to mouse allergen and asthma and asthma morbidity among women in Boston. *J Allergy Clin Immunol* 2007 Oct;120(4):954–956. [PubMed: 17590423]
55. Phipatanakul W, Cronin B, Wood RA, et al. Effect of environmental intervention on mouse allergen levels in homes of inner-city Boston children with asthma. *Ann Allergy Asthma Immunol* 2004 Apr;92(4):420–425. [PubMed: 15104193]
56. Pongracic JA, Visness CM, Gruchalla RS, Evans R 3rd, Mitchell HE. Effect of mouse allergen and rodent environmental intervention on asthma in inner-city children. *Ann Allergy Asthma Immunol* 2008 Jul;101(1):35–41. [PubMed: 18681082]
57. Kattan M, Stearns SC, Crain EF, et al. Cost-effectiveness of a home-based environmental intervention for inner-city children with asthma. *J Allergy Clin Immunol* 2005 Nov;116(5):1058–1063. [PubMed: 16275376]
58. Sheehan WJ, Rangsihienchai PA, Muilenberg ML, et al. Mouse allergens in urban elementary schools and homes of children with asthma. *Ann Allergy Asthma Immunol* 2009 Feb;102(2):125–130. [PubMed: 19230463]
59. Esch, R.; Bush, RK. Aerobiology of Outdoor Allergens. In: Adkinson, NF.; Yunginger, JW.; Busse, WW., editors. *Middleton's Allergy Principles & Practice*. Philadelphia, PA: Mosby; 2003. ed.
60. Kagen S, Lewis W, Levetin E. *Aeroallergen PhotoLibrary of North America*. 2004
61. Barnes C, Pacheco F, Landuyt J, Hu F, Portnoy J. Hourly variation of airborne ragweed pollen in Kansas City. *Ann Allergy Asthma Immunol* 2001 Feb;86(2):166–171. [PubMed: 11258685]
62. Pulimood TB, Corden JM, Bryden C, Sharples L, Nasser SM. Epidemic asthma and the role of the fungal mold *Alternaria alternata*. *J Allergy Clin Immunol* 2007 Sep;120(3):610–617. [PubMed: 17624415]
63. O'Hollaren MT, Yunginger JW, Offord KP, et al. Exposure to an aeroallergen as a possible precipitating factor in respiratory arrest in young patients with asthma. *N Engl J Med* 1991 Feb 7;324(6):359–363. [PubMed: 1987459]
64. Targonski PV, Persky VW, Ramekrishnan V. Effect of environmental molds on risk of death from asthma during the pollen season. *J Allergy Clin Immunol* 1995 May;95(5 Pt 1):955–961. [PubMed: 7751516]
65. Sears MR, Herbison GP, Holdaway MD, Hewitt CJ, Flannery EM, Silva PA. The relative risks of sensitivity to grass pollen, house dust mite and cat dander in the development of childhood asthma. *Clin Exp Allergy* 1989 Jul;19(4):419–424. [PubMed: 2758355]
66. Katelaris CH, Burke TV, Byth K. Spatial variability in the pollen count in Sydney, Australia: can one sampling site accurately reflect the pollen count for a region? *Ann Allergy Asthma Immunol* 2004 Aug;93(2):131–136. [PubMed: 15328671]
67. Frenz DA. Interpreting atmospheric pollen counts for use in clinical allergy: spatial variability. *Ann Allergy Asthma Immunol* 2000 May;84(5):481–489. quiz 489–491. [PubMed: 10831000]

68. Portnoy J, Barnes C. Clinical relevance of spore and pollen counts. *Immunol Allergy Clin North Am* 2003 Aug;23(3):389–410. vi. [PubMed: 14524382]
69. Suphioglu C. Thunderstorm asthma due to grass pollen. *Int Arch Allergy Immunol* 1998 Aug;116(4):253–260. [PubMed: 9693274]
70. Suphioglu C. Thunderstorm Asthma Due to Grass Pollen. *International Archives of Allergy and Immunology* 1998;116:253–260. [PubMed: 9693274]
71. Taylor PE, Flagan RC, Miguel AG, Valenta R, Glovsky MM. Birch pollen rupture and the release of aerosols of respirable allergens. *Clin Exp Allergy* 2004 Oct;34(10):1591–1596. [PubMed: 15479275]
72. Taylor PE, Flagan RC, Valenta R, Glovsky MM. Release of allergens as respirable aerosols: A link between grass pollen and asthma. *J Allergy Clin Immunol* 2002 Jan;109(1):51–56. [PubMed: 11799365]
73. Taylor PE, Jonsson H. Thunderstorm asthma. *Curr Allergy Asthma Rep* 2004 Sep;4(5):409–413. [PubMed: 15283882]
74. Grote M, Vrtala S, Niederberger V, Valenta R, Reichelt R. Expulsion of allergen-containing materials from hydrated rye grass (*Lolium perenne*) pollen revealed by using immunogold field emission scanning and transmission electron microscopy. *J Allergy Clin Immunol* 2000 Jun;105(6 Pt 1):1140–1145. [PubMed: 10856148]
75. Bellomo R, Gigliotti P, Treloar A, et al. Two consecutive thunderstorm associated epidemics of asthma in the city of Melbourne. The possible role of rye grass pollen. *Med J Aust* 1992 Jun 15;156(12):834–837. [PubMed: 1603007]
76. Davidson AC, Emberlin J, Cook AD, Venables KM. A major outbreak of asthma associated with a thunderstorm: experience of accident and emergency departments and patients' characteristics. Thames Regions Accident and Emergency Trainees Association. *Bmj* 1996 Mar 9;312(7031):601–604. [PubMed: 8595332]
77. Celenza A, Fothergill J, Kupek E, Shaw RJ. Thunderstorm associated asthma: a detailed analysis of environmental factors. *Bmj* 1996 Mar 9;312(7031):604–607. [PubMed: 8595333]
78. Dales RE, Cakmak S, Burnett RT, Judek S, Coates F, Brook JR. Influence of ambient fungal spores on emergency visits for asthma to a regional children's hospital. *Am J Respir Crit Care Med* 2000 Dec;162(6):2087–2090. [PubMed: 11112119]
79. Allergen immunotherapy: a practice parameter second update. *J Allergy Clin Immunol* 2007 Sep;120(3 Suppl):S25–S85. [PubMed: 17765078]
80. Abramson MJ, Puy RM, Weiner JM. Allergen immunotherapy for asthma. *Cochrane Database Syst Rev.* 2003 Apr; CD001186.
81. Durham SR, Walker SM, Varga EM, et al. Long-term clinical efficacy of grass-pollen immunotherapy. *N Engl J Med* 1999 Aug 12;341(7):468–475. [PubMed: 10441602]
82. Moller C, Dreborg S, Ferdousi HA, et al. Pollen immunotherapy reduces the development of asthma in children with seasonal rhinoconjunctivitis (the PAT-study). *J Allergy Clin Immunol* 2002 Feb;109(2):251–256. [PubMed: 11842293]
83. Walker SM, Varney VA, Gaga M, Jacobson MR, Durham SR. Grass pollen immunotherapy: efficacy and safety during a 4-year follow-up study. *Allergy* 1995 May;50(5):405–413. [PubMed: 7573829]
84. Walker SM, Pajno GB, Lima MT, Wilson DR, Durham SR. Grass pollen immunotherapy for seasonal rhinitis and asthma: a randomized, controlled trial. *J Allergy Clin Immunol* 2001 Jan;107(1):87–93. [PubMed: 11149996]
85. Tepper RS, Llapur CJ, Jones MH, et al. Expired nitric oxide and airway reactivity in infants at risk for asthma. *J Allergy Clin Immunol* 2008 Oct;122(4):760–765. [PubMed: 18760452]

Table 1**Dust Mite Allergen Education and Avoidance Measures**

Education

- Dust mites are microscopic in size
- Source of food is skin scales or fungi
- Dust mite particles are heavy and fall to the ground
- Found in woven materials such as mattresses, pillows, stuffed animals, bedding, upholstered furniture and draperies
- The highest levels of allergen are typically found in the mattress
- Increased humidity above 50% increases growth

Avoidance**First Level**

- Encase the pillow and mattress with a pore diameter no larger than 10 microns
- Wash bedding weekly in hot water and dry in a heated drier
- Remove dust mite reservoirs such as toys and stuffed animals from the bed
- Keep humidity less than 50%
- Vacuum with a HEPA filter bag
- Chemicals to kill mites or denature proteins have a modest effect

Second Level

- Remove upholstered furniture, drapery, carpeting or rugs

Third Level

- Move from a damp location such as a basement to a higher floor
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Table 2

Pet Allergen Education and Avoidance Measures

Education

- Cat allergen is found on the skin, hair and saliva
- Males produce a higher amount of allergen
- Dog allergen is found in skin, hair, and saliva
- The allergens can scatter easily in the air and sticks to furniture and clothing
- Cat allergen may take 4 months to denature

AvoidanceControl allergen with pet removed from the home

- Thoroughly clean upholstered furniture, walls and carpet
- Removal of furniture and carpet is ideal

Control allergen with pet in the home

- Remove pet from the bedroom and other rooms where your child spends a lot of time
 - Aggressive cleaning of upholstered furniture, walls and carpet
 - Removal of upholstered furniture and carpet is ideal
 - Encase the mattress and pillows with a pore diameter no larger than 6 microns
 - Keep the pet clean. Washing dogs twice a week may be helpful.
 - Use a HEPA air filter
-

Table 3

Rodent (Cockroach and Mouse) Education and Avoidance Measures

Education

- Cockroach allergen is found in the saliva, fecal material, secretions, cast skins and debris
- These particles are large and settle quickly to the ground
- Mouse allergen is found in mouse urine, hair follicles and dander
- Like other animals, mouse allergen is carried on particles and can migrate through a home or building
- These allergens are commonly found in areas of increased population density
- The highest levels of allergen are typically found in the kitchen

Avoidance

- Inspect to detect hiding places and identify food sources (grease, kitchen debris)
 - Store food in sealed containers. Avoid exposed pet food
 - Remove clutter
 - Exterminate with pesticides or bait traps. Professional extermination is preferred
 - Seal holes or cracks in the home to prevent re-infestation
 - Wash bedding, curtains and clothing
-

Table 4

Pollen and Mold Education and Avoidance Measures

Education

- Trees typically pollinate from March through June
- Grasses typically pollinate from May through early June
- Weeds typically pollinate from August through the first frost and peaks around noon time
- Outdoor mold spores typically peak in June and decrease after the first frost
- Mold spores are found in soil, seeds, and vegetable matter such as grass or leaves
- Indoor molds are prominent in humid environments

Pollen Avoidance

- Keep windows closed
- Bath to remove allergens from hair and body
- HEPA air filtration
- Consider same principles for outdoor mold avoidance

Mold Avoidance

- Locate mold contaminated items and remove them.
 - Clean moist areas as these are prone to mold growth
 - Repair leaks
 - Reduce humidity to less than 50%
-