CLINICAL REPORT Guidance for the Clinician in Rendering Pediatric Care

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

Indoor Environmental Control Practices and Asthma Management

Elizabeth C. Matsui, MD, MHS, FAAP, Stuart L. Abramson, MD, PhD, AE-C, FAAP, Megan T. Sandel, MD, MPH, FAAP, SECTION ON ALLERGY AND IMMUNOLOGY, COUNCIL ON ENVIRONMENTAL HEALTH

abstract

Indoor environmental exposures, particularly allergens and pollutants, are major contributors to asthma morbidity in children; environmental control practices aimed at reducing these exposures are an integral component of asthma management. Some individually tailored environmental control practices that have been shown to reduce asthma symptoms and exacerbations are similar in efficacy and cost to controller medications. As a part of developing tailored strategies regarding environmental control measures, an environmental history can be obtained to evaluate the key indoor environmental exposures that are known to trigger asthma symptoms and exacerbations, including both indoor pollutants and allergens. An environmental history includes questions regarding the presence of pets or pests or evidence of pests in the home, as well as knowledge regarding whether the climatic characteristics in the community favor dust mites. In addition, the history focuses on sources of indoor air pollution, including the presence of smokers who live in the home or care for children and the use of gas stoves and appliances in the home. Serum allergen-specific immunoglobulin E antibody tests can be performed or the patient can be referred for allergy skin testing to identify indoor allergens that are most likely to be clinically relevant. Environmental control strategies are tailored to each potentially relevant indoor exposure and are based on knowledge of the sources and underlying characteristics of the exposure. Strategies include source removal, source control, and mitigation strategies, such as high-efficiency particulate air purifiers and allergen-proof mattress and pillow encasements, as well as education, which can be delivered by primary care pediatricians, allergists, pediatric pulmonologists, other health care workers, or community health workers trained in asthma environmental control and asthma education.

FREE

This document is copyrighted and is property of the American Academy of Pediatrics and its Board of Directors. All authors have filed conflict of interest statements with the American Academy of Pediatrics. Any conflicts have been resolved through a process approved by the Board of Directors. The American Academy of Pediatrics has neither solicited nor accepted any commercial involvement in the development of the content of this publication.

Clinical reports from the American Academy of Pediatrics benefit from expertise and resources of liaisons and internal (AAP) and external reviewers. However, clinical reports from the American Academy of Pediatrics may not reflect the views of the liaisons or the organizations or government agencies that they represent.

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

All clinical reports from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

DOI: 10.1542/peds.2016-2589

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

FINANCIAL DISCLOSURE: The authors have indicated they do not have a financial relationship relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

To cite: Matsui EC, Abramson SL, Sandel MT, AAP SECTION ON ALLERGY AND IMMUNOLOGY AAP COUNCIL ON ENVIRONMENTAL HEALTH. Indoor Environmental Control Practices and Asthma Management. *Pediatrics.* 2016;138(5):e20162589

INTRODUCTION

Asthma is one of the most common chronic childhood illnesses, affecting as many as 10% of children across the United States, with prevalence rates as high as 25% in some communities.[1] Self-reported black race, Puerto Rican ethnicity, and poverty are the major risk factors for asthma in US populations.[1] Children with asthma who are sensitized and exposed to indoor allergens, including dust mite,[2] rodent,[3,4] cockroach, and pet allergens,[5,6] have worse asthma control and lung function and greater airway inflammation and morbidity than those who are either not sensitized or not exposed to these allergens. In addition, exposure to common indoor pollutants, including secondhand smoke (SHS)[7] and nitrogen dioxide (NO_2),[8] exacerbates asthma, regardless of the presence of allergic sensitization.

Children may be vulnerable to these environmental exposures for several potential reasons. First, perhaps because of their airway physiology, they may be exposed to larger doses of airborne pollutants.[9–11] Second, it is possible that their exposure to pollutants, chemicals, and/or allergens is greater because of their proximity to the floor, which can be a reservoir for these exposures.[12] It is also noteworthy that most children with asthma who are at least school-aged have evidence of allergic sensitization, so that allergen exposure likely plays a significant role in childhood asthma. However, an estimated 20% of school-aged children with persistent asthma are not atopic,[13–15] and although they are not susceptible to allergen exposure, they are susceptible to pollutants and irritants, as are children with atopic diseases. For all children with asthma, viruses are a major trigger of exacerbations.

The purpose of this report is to raise awareness among pediatricians regarding the need to assess for and implement indoor environmental control practice measures in the management of asthma and, thereof, to provide guidance. The recently published manual *Pediatric Environmental Health*, known as the "Green Book" (third edition, 2012) from the American Academy of Pediatrics (AAP), has 2 chapters devoted to the discussion of topics included in this report: chapter 20, "Air Pollutants, Indoor," and chapter 43, "Asthma."[16] These chapters provide additional reference material that supports a number of findings discussed in this report. There are no contradictions evident between the 2 resources, but the current report includes some updated references and commentary (eg, electronic nicotine delivery systems [e-cigarettes], practice parameters regarding environmental assessment of and exposure reduction to rodent and cockroach allergens, etc).

Environmental control strategies are tailored to each potentially relevant indoor exposure on the basis of knowledge of the patient's allergic sensitivities and relevant indoor exposures. Serum allergen-specific immunoglobulin E (IgE) antibody tests may be performed, or the patient may be referred to a board-certified allergist for allergy skin testing to identify indoor allergens that are most likely to be clinically relevant for a patient who meets National Asthma Education and Prevention Program criteria for persistent asthma, which include patients taking a long-term controller medication as well as patients having symptoms >2 days per week or nocturnal symptoms more than twice per month.[17] Serum IgE testing and allergy skin testing are both appropriate methods of assessing allergic sensitivities; neither is clearly better than the other in identifying clinically important sensitizations,[18] and there is no lower age limit for either of these tests.

For allergic children, an environmental history includes questions regarding the presence of furry pets or pests or evidence of pests in the home. Understanding whether the climatic characteristics of a community favor dust mite growth is important for assessing the likelihood that the patient has significant exposure to dust mites. Except for arid climates, exposure to dust mites is an important consideration. For all children, both allergic and nonallergic, the environmental history focuses on nonallergen exposures as well. Allergens only affect a subset of those who are sensitized to the specific allergen(s), whereas irritants affect all children to variable degrees. For pollutants, the history focuses on sources of indoor air pollution, including smokers who live in the home or care for the child and the use of gas stoves and appliances. Household chemicals, such as those found in air fresheners and cleaning agents, also can be respiratory irritants and trigger asthma symptoms.[16] The specific strategies used to target an exposure depend on knowledge about the sources and underlying characteristics of the exposure. Environmental control strategies include source removal (eradication of the allergen source), source control (controlling the population/amount of the allergen source), and mitigation strategies (reducing the amount of allergen in the air, dust, bedding, etc, that is produced by the source), such as using high-efficiency particulate air (HEPA) purifiers and allergen-proof mattress/box spring and pillow encasements. Individually tailored, multifaceted environmental interventions are endorsed by the National Asthma Education and Prevention Program guidelines and may be similar in terms of efficacy to controller medications.[9] Although a multifaceted approach makes intuitive sense and reflects clinical practice, much of the foundational research has focused on single allergens; therefore, each allergen will be discussed individually in this report.

INDOOR ALLERGENS

Dust Mites

The major dust mite allergens are Der f 1 and Der p 1, from the 2 most common house dust mite species, *Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*, respectively. Dust mites, microscopic members of the Arachnid class, are rare in arid environments, because they require moisture to survive; humid environments such as those found in the southeastern United States and along the coasts are most conducive to dust mite growth. In more humid climates, dust mites are found not only in homes but also in public places such as schools.

Dust mite allergen exposure has been repeatedly linked to worse asthma among those who are dust mite sensitized[2]; and many, but not all, studies indicate that effective reduction in dust mite allergen exposure improves asthma in this patient group.[2,19–21] Approximately 30% to 62% of children with persistent asthma are sensitized to dust mite allergens,[2,22,23] and it is this population who are susceptible to the effects of dust mite allergen exposure. Unlike patients who may describe allergic reactions to furry pet exposure, patients who are allergic to dust mites are unable to identify dust mites, which are microscopic, as an allergic trigger. Therefore, the pediatrician can only rely on an understanding of whether the climatic conditions of the community are conducive to dust mite growth. As an alternative, a family can have a home dust sample tested for dust mite content with the use of a commercially available kit, although this test is not currently reimbursed by third-party payers.

Dust mite allergen exposure reduction strategies focus on source removal (ie, killing the dust mites) and/or removal of the allergen. Strategies that have been attempted include measures that target the bed, including frequent washing of all bed linens in hot water and the use of allergen-impermeable mattress and pillow encasements, and measures that target other allergen reservoirs, such as vacuuming, removal of carpet and stuffed toys from the bed, and application of acaricides or allergen-denaturing agents.[24] Applications of acaricides and allergen-denaturing agents are cumbersome and ineffective, sustained reduction in indoor relative humidity is difficult to achieve, and carpet removal is expensive and of unclear benefit.[24] There are also potential risks when applying chemicals to the indoor environment, and although these risks are small when the agents are handled according to instructions, they are an important consideration. More information about the risks of chemical agents, including pesticides, can be found in the AAP publication *Pediatric Environmental Health*.[16] Because the major dust mite allergens are carried on larger particles (>10 μm), they quickly settle to dependent surfaces after disturbance of the reservoir. For this reason, air filtration is not likely to have any meaningful effect on dust mite allergen exposure. Because dust mites feed on shed human skin, which is abundant in the bed, first-line approaches to reduce dust mite allergen exposure include washing of bed linens and the use of allergen-impermeable mattress and pillow encasements, which can be highly effective in reducing dust mite allergen in the bed.

Although the most recent meta-analysis of dust mite interventions concluded that dust mite interventions had no effect on asthma, this meta-analysis included studies whose interventions had little to no effect on dust mite exposure and whose populations may not have been dust mite allergic.[25] In contrast, most of the studies in children who are very likely to have dust mite–driven asthma found that dust mite interventions that resulted in a substantial reduction in dust mite allergen levels had a beneficial clinical effect. For example, of the 15 studies of bedding encasements in dust mite–sensitized children included in the meta-analysis, 14 included assessments of dust mite allergen exposure, and 7 found at least an 80% reduction in dust mite allergen in the active intervention groups. Of these 7 studies, 5 found that the active intervention group had improvements in asthma.[21,24,26–29]

Cat and Dog Allergens

The most common furry pets found in homes are cats and dogs; the major dog allergen is Can f 1 and the major cat allergen is Fel d 1. Exposure to these allergens has been linked to worse asthma among pet-sensitized asthmatics, and approximately 25% to 65% of children with persistent asthma are sensitized to cat or dog allergens.[2,3,22,23] When patients are asked about the presence of a pet in the home (or in other places where the child spends significant time), an affirmative response confirms significant exposure, but the absence of a pet does not ensure that the patient does not have clinically meaningful pet allergen exposure. It is also important to note that a child with clinically relevant pet sensitization may not have acute allergic symptoms with exposure, so that the absence of these symptoms does not rule out a role for the pet in the child's asthma. Although pet allergens are found in higher concentrations in homes with pets, they are ubiquitous and detected in places such as schools, child care centers, and public transportation. Furry animal allergens adhere to clothing, walls, furniture, etc, and, in contrast to dust mite and cockroach allergens, are predominantly carried on smaller particles (<10–20 μm), so they remain airborne for long periods of time. As a result, they are carried on clothing of people who have a cat or dog and are transferred to environments that do not contain a cat or dog. Indirect exposure to pet allergens through this mechanism can also cause asthma symptoms in sensitized children. For example, cat allergen brought into classrooms by students with cats has been linked to worsening of asthma in cat-sensitized classmates with asthma.[5]

Clinically significant reductions in animal allergen levels require source removal, or relocating the pet.[30] Even after removing the pet from the home, it can take several months before significant reductions in allergen levels are observed,[31] so it is important that parents are counseled accordingly. In the only prospective (but nonrandomized) study of pet removal, asthma improved significantly and controller medication needs were reduced substantially in the group who removed the pet but not in the group who kept the pet.[32] Because of the reluctance of patients to give up their pets, there have been several studies examining the efficacy of air filtration in reducing airborne pet allergen levels and improving asthma in sensitized patients.[33–35] Overall, these studies have found that this approach is ineffective at improving asthma outcomes and, at best, only modestly reduces airborne allergen levels. A common patient question is whether certain dog breeds are "hypoallergenic." A recent study found that homes with dogs believed to be hypoallergenic had levels of dog allergen similar to homes with dogs that were not considered to be hypoallergenic.[36] The "Thanksgiving effect" refers to the phenomenon when cat-allergic asthma patients living with, and apparently tolerating, a cat go away to college in the fall and then return home for Thanksgiving and, on reexposure to the cat, develop significant asthma symptoms.[37] Sustained animal allergen exposure as an attempt at inducing tolerance is unlikely to be effective, but pet removal is quite effective.[32]

Rodents

Rodent allergens have been recognized as a cause of occupational allergy and asthma for many decades but have only recently been recognized as an exacerbator of asthma in rodent-sensitized community populations.[38] Mus m 1, the major mouse allergen, is found primarily on small particles, like other furred animal allergens, so it is readily airborne.[39,40] It is found in virtually all inner-city homes, particularly in the northeastern and Midwestern United States.[41–43] Although 75% to 80% of US homes have detectable mouse allergen, concentrations in inner-city homes are as much as 1000-fold higher than those found in suburban homes.[44,45] A report of any evidence of infestation, particularly from a patient living in an urban neighborhood, suggests that there are clinically significant levels of mouse allergen in the home. However, a report of absence of infestation is not a reassurance that there is not clinically significant exposure when the patient resides in an urban neighborhood. Mouse allergen exposure has repeatedly been linked to an increased risk of a range of markers of uncontrolled and more severe asthma among sensitized children.[3,4,42,43]

A substantial reduction in mouse allergen levels can be achieved with a professionally delivered integrated pest-management intervention that includes trap placement, sealing of holes and cracks that can serve as entry points into the home, and application of rodenticide.[46] It is important to weigh the potential benefits of rodenticide against the potential risks, and a discussion of these risks can be found in *Pediatric Environmental Health*.[16] Although a minor mouse infestation can be handled without rodenticide, some homes with serious infestation may require a rodenticide; in this circumstance, bait blocks may be associated with the risk of accidental ingestion by children and pets, and families may have greater benefit from professionally applied rodenticide.

Although there has yet to be a study testing the clinical efficacy of intervention on mouse allergen, mouse-sensitized patients who have any evidence of infestation may benefit from integrated pest management, because reducing mouse allergen would be expected to be clinically efficacious. Integrated pest management targets mice and other pests, such as cockroaches, and includes sealing up entry points for pests and removing sources of food, shelter, and water for pests by storing food in chew-proof containers, cleaning up immediately after eating, fixing leaky faucets and pipes, and taking the trash out on a regular basis. For patients living in rental units, families can work with landlords and/or property managers to address the infestation. Patients with mouse sensitization are very likely to be cat sensitized as well, so the acquisition of a cat may not be a prudent approach to mouse infestation. Rat allergen exposure is less common, because it is detected in approximately one-third of inner-city homes; so, although it is associated with worse asthma, a smaller proportion of children are affected by rat allergen exposure than mouse allergen exposure.

Rodents are also kept as pets, and the more common rodent pets are gerbils, guinea pigs, and hamsters. Rabbits and ferrets, which are not rodents, are also common furry pets. Exposure to these animals can contribute to worse asthma among patients who are sensitized.

Cockroach

The most common cockroaches in US inner cities are the German and American cockroaches; the major German cockroach allergens, Bla g 1 and Bla g 2, are the best studied in terms of health effects. Cockroach allergen exposure among sensitized inner-city children with asthma was first linked to asthma morbidity in the National Cooperative Inner-City Asthma Study report published in 1997.[47] Since then, the link with asthma morbidity has been replicated, and highly effective methods based on integrated pest-management principles to reduce cockroach allergen levels have been identified. Pesticides that come in gel form are preferred to aerosolized pesticides, which likely result in greater pesticide exposure.[48,49] Moreover, in a successful multifaceted environmental intervention in inner-city children with asthma, the degree of reduction in cockroach allergen was correlated with the degree of improvement in asthma symptoms, providing support for cockroach allergen environmental control measures, which include integrated pest management, as an integral part of asthma management in cockroach-sensitized children with asthma.[19,50]

Dampness and Mold

Excessive moisture in homes can contribute to asthma through an increase in mold exposure as well as increased cockroach and dust mite allergen. Excessive moisture may be present because of inadequate ventilation or other building problems or because of a flooding event. Mold exposure occurs mainly as spores become aerosolized, and a substantial number of asthma cases may be attributed to dampness and mold exposure.[51] The prevalence of mold sensitization in children with persistent asthma is approximately 50%, and the most common species to which children are sensitized and exposed are *Alternaria*, *Aspergillus*, *Cladosporium*, and *Penicillium*.[52] Although *Alternaria* and *Aspergillus* are derived from outdoor sources, they are present indoors and may be clinically relevant.[53] The National Survey of Lead and Allergens in Housing found that 56% of homes had levels of some molds above thresholds observed to be associated with asthma symptoms.[54] Children sensitized and exposed to the major indoor molds appear to be at greater risk of asthma exacerbations.[52] Remediation of mold has been shown to reduce symptoms and medication use in several populations, and its effects may not depend on whether the population is sensitized to mold.[55,56]

The evaluation of a patient with persistent asthma includes an assessment of sensitization to the major indoor molds, which can be accomplished with specific IgE testing performed on a blood sample or through referral to a board-certified allergist for skin testing. Air sampling, thermography, moisture meters, environmental history, and direct observation are all useful techniques to identify moisture problems. Although more sophisticated techniques for assessing home dampness and mold exposure are ideal, parental report of dampness, leaks, or mold is helpful and, in a child with sensitization to the major indoor molds, suggests that the parents be counseled to intervene on the home environment (some simple measures are listed in the Supplemental Appendix). A common tool used to assess home mold exposure is air sampling, but it is important to note that molds are ubiquitous, so reports from air sampling are uninterpretable without concomitant indoor and outdoor sampling. More detailed information about dampness and mold can be found in the Institute of Medicine's report *Damp Indoor Spaces and Health*[57] and the World Health Organization's *Guidelines for Indoor Air Quality: Dampness and Mould*.[58]

INDOOR POLLUTANTS

Particulate Matter and SHS Exposure

Particulate matter (PM) simply means airborne particles, and it is often expressed as either $PM_{2.5}$, which is the portion of PM that is 2.5 μm in diameter or less, or PM_{10}, which is the portion of PM that is 10 μm or less. Both allergic and nonallergic children with asthma are susceptible to the effects of indoor PM and SHS.[59] $PM_{2.5}$, also known as fine PM, penetrates further into the airways than larger-sized particles and is capable of entering the alveoli. Indoor PM is composed partly of outdoor PM but mostly of particles generated indoors by smoking and other activities, such as cooking and sweeping.[60–63] PM exposure is associated with lung inflammation, decreased lung function, and respiratory symptoms in children with asthma, regardless of whether they have allergic sensitization. Other sources of indoor PM include wood-burning stoves, fireplaces, biomass burning, electronic nicotine delivery systems (e-cigarettes),[64] cigar smoke, incense, bus idling outside of school, and other substances that are smoked, such as marijuana.[16]

SHS

Cigarette smoke is a major contributor to indoor PM in US homes, because each half-pack of cigarettes smoked in the home is estimated to contribute 4.0 μg/m^3 of PM,[7,60] and approximately 30% of all US children and 40% to 60% of US children in low-income households are exposed to SHS in their homes.[7,65,66] Smoking cessation by close family members and caregivers is the most effective way to reduce or eliminate tobacco smoke exposure. Although tobacco dependence can be a very severe addiction, tobacco-dependence treatment medications approved by the US Food and Drug Administration are very effective in treating tobacco dependence and allowing the tobacco smoker to stop smoking.[67] State-of-the-art approaches to treating tobacco dependence initiate therapy on the basis of severity of the tobacco dependence and adjust therapy to control nicotine withdrawal symptoms. The AAP Section on Tobacco Control recently published documents that address clinical practice policy as well as public policy to protect children from tobacco, nicotine, and tobacco smoke and provided an accompanying technical report to support evidence-based approaches.[68–70]

If tobacco-dependent family members are not ready to stop smoking or initiate tobacco-dependence treatment, smoke-free home and car policies can reduce but not eliminate a child's tobacco smoke exposure. For families who are not willing to consider smoking cessation, starting tobacco-dependence treatment, or keeping the home smoke free, 2 randomized controlled trials suggest that the use of portable HEPA purifiers in the home may be of some benefit[7,71] but would not be as effective as if occupants of the home stopped smoking or instituted a home smoking ban. HEPA purifiers are also costly, so whether the expenditure is worth the benefit will vary depending on the child's clinical picture, progress in smoking cessation or institution of a home smoking ban, and the financial resources available. It is also important to note that homes are not the only indoor spaces where children are exposed to SHS. SHS exposure in public places has been targeted by legislation that bans smoking in public places, and this legislation has been associated with significant decreases in asthma morbidity in children, including asthma hospitalizations.[72]

Portable HEPA Purifiers

The use of portable HEPA purifiers has been shown to reduce indoor PM concentrations by approximately 25% to 50% and to reduce asthma symptoms and exacerbations.[7,71] Portable HEPA purifiers appear to be effective in reducing PM from tobacco and nontobacco smoke sources, but there is little evidence to support their efficacy in reducing airborne animal allergens or pollen. Although HEPA purifiers are expected to be much less effective than the first-line approach of source removal, if they are suggested, nonionizing HEPA purifiers with clean air delivery rates that are appropriate for the size of the room in which they will be used may be most effective. This information is indicated on the air purifier packaging. It is important to note that cigarette smoke also produces nonparticle, gaseous pollutants, such as nicotine and others, and that HEPA purifiers do not appear to have any effect on air nicotine and possibly other gaseous components of tobacco smoke.[7] As a result, HEPA purifiers will not offer any protection from the adverse health effects of these nonparticle components of SHS.

NO_2

NO_2 is a gas that is a byproduct of combustion, so it is found outdoors as a result of traffic and other combustion activities, and affects both allergic and nonallergic children with asthma.[73] It can be found in concentrations associated with adverse health effects in homes, where the most important source is gas heat and appliances.[8,73] In addition, older wood-burning stoves, unvented space heaters, and other sources of combustion can produce NO_2 and other pollutants. Higher indoor NO_2 concentrations have been linked to worse asthma in children with asthma,[8] although 1 study found that only nonatopic children with asthma were affected.[73] Although data are scant regarding effective interventions for reducing indoor NO_2, ensuring that the stove is properly vented and using the vent while the stove is in use would be expected to reduce indoor NO_2 concentrations. One randomized controlled trial found reductions of 40% to 50% in indoor NO_2 concentrations when a gas stove was replaced with an electric one.[74] Whether this degree of indoor NO_2 reduction results in improvements in asthma remains unclear, however.

ENVIRONMENTAL EVALUATION AND MANAGEMENT FOR THE PEDIATRIC PATIENT WITH ASTHMA

An assessment of environmental triggers and education regarding evidence-based approaches to exposure reduction are an integral part of asthma management in the pediatric patient.[75] Children are vulnerable to the respiratory effects of indoor environmental exposures because of their respiratory physiology and because any pulmonary effects from these exposures may affect their respiratory health in adulthood. An assessment of allergic sensitization to a panel of indoor allergens is useful for determining whether indoor allergens may be clinically relevant for a patient and, if so, for identifying which allergens may be relevant. Allergen-specific IgE tests can be performed at commercial laboratories, so they can be ordered by a primary care provider. Testing to large panels of allergens is not helpful, because there may be many positive results to allergens that are not relevant to the individual patient's environment and history; instead, testing to selected relevant allergens is preferred. The clinical scenario guides which allergens to include in testing; for pediatric patients with persistent asthma, testing to common indoor allergens, including cat, dog, dust mites (if in a nonarid area of the country), and molds, is appropriate. For children living in a community in which pest infestation is common, testing for mouse and cockroach sensitization would also be appropriate. More information about allergy testing can be found in a recent AAP clinical report on the subject.[18] Alternatively, the patient can be referred to an allergist-immunologist for allergy skin testing, interpretation of results, and education of the family about environmental triggers.

In most cases, a careful exposure history, combined with knowledge about the community and allergy testing, is sufficient to identify the major exposures that may be clinically relevant. The history includes asking parents and patients about exposure to pets, dampness, or mold and whether they have seen evidence of pest infestation. Relevant exposures occur at schools, child care centers, cars/transportation, and relatives' homes, so these other locations of potential exposure are included in the environmental history. Understanding whether the patient lives in an area conducive to dust mite growth or to mouse or cockroach infestation is also an important component of determining the potentially relevant allergens for the patient. For pollutants, which are relevant for both allergic and nonallergic children with asthma, the history includes asking parents and patients first about SHS exposure. An additional history elicits the presence of gas heat and/or appliances, because this finding would suggest that the patient may have clinically relevant NO_2 exposure. Because relevant exposures occur in schools and child care centers, it is important to elicit potential relevant exposures that occur in other settings outside of the home. Often, families can work with schools and child care centers to address relevant exposures, and both the Environmental Protection Agency and the Centers for Disease Control and Prevention have online resources for addressing the school environment (http://www.epa.gov/iaq/schools/managingasthma.html and http://www.cdc.gov/HealthyYouth/asthma/creatingafs/, respectively).

The environmental history and assessment of allergic sensitization will inform a tailored environmental control plan for the patient. It is important to note that environmental interventions that target all relevant exposures are more likely to be successful than those that target only 1 or 2 exposures. For patients sensitized to an allergen, the first-line strategies for targeting indoor exposures discussed previously are likely to result in a reduction in relevant indoor exposures and improvements in asthma. Although the role of allergen-proof mattress and pillow encasements has been debated,[76,77] their efficacy may be greater for children than for adults,[20,21,26–28] and they have been an integral part of successful, individually tailored, multifaceted home environmental interventions.19 For patients with SHS exposure, helping the smoker obtain effective tobacco-dependence treatment so that he or she can stop smoking is the most effective approach, because it eliminates the source of the tobacco smoke. A home smoking ban can reduce, but does not eliminate, the tobacco smoke exposure. In some cases, the parent may not be able to influence the smoking behavior of household members and may not be able to move to another home. In that situation, the use of portable HEPA purifiers may be better than no intervention. Insurance coverage for air purifiers and other goods and services for environmental control is being reevaluated and may be more widespread in the future. For patients with gas heat and appliances, the first-line environmental control strategies would include ensuring that the gas stove is properly vented and the vent is used when the stove is on.

Because each child has his or her own profile of relevant exposures, it is important that the strategies regarding environmental control be tailored to each patient. In addition, each of the child's exposures affects his or her asthma, so targeting all of the exposures, to the extent possible, is important to achieve the maximal benefit. A sample environmental control plan is provided (Supplemental Appendix), which can be used to indicate the child's allergic status, to provide basic background information about indoor environmental exposures, and to list the environmental control practices that the family can implement to reduce the child's exposure to indoor allergens and irritants that are contributing to the child's asthma.

Although public and private insurers may cover environmental assessments and control measures, most do not, despite evidence of their cost-effectiveness. Public and private resources are available, including legal assistance (such as through medical-legal partnerships; www.medical-legalpartnerships.org), to help primary care pediatricians, asthma and allergy specialists, and patients with environmental remediation efforts pertinent to various residential settings. In some states, Medicaid may cover some components of an environmental intervention, such as a home visit for an environmental assessment and education, which can be delivered by health care workers or community health workers trained in asthma environmental control. It is important to note, however, that insurance coverage differs from state to state and among insurers and is expected to change over time, so it is best to seek information from your AAP Chapter, the AAP Department of Practice, or the Asthma and Allergy Foundation to understand what resources, including insurance coverage, are available to support environmental control goods and services.

KEY POINTS

1. Individually tailored environmental control measures have been shown to reduce asthma symptoms and exacerbations, are similar in efficacy to controller medications, and appear to be cost-effective when the aim is to reduce days of symptoms and their associated costs.[75,78] The efficacy of environmental control measures has been sustained for up to 1 year after the intervention.[19]
2. As a part of developing tailored strategies regarding environmental control measures, an environmental history may be obtained to evaluate the key indoor environmental exposures that are known to trigger asthma symptoms and exacerbations, including both indoor pollutants and allergens.
3. The leading indoor environmental contributors to asthma symptoms are indoor allergens (pets, dust mites, mice, rats, cockroaches, molds) and pollutants (airborne PM, SHS, NO_2).
4. An environmental history may include questions regarding the presence of pets or pests, or evidence of pests in the home, as well as knowledge regarding whether the climatic characteristics in the community favor dust mites. In addition, the history may focus on sources of indoor air pollution, including smokers in the home, use of gas stoves and appliances, and presence of mold in the home.
5. Serum allergen-specific IgE antibody tests may be performed or the patient may be referred to a board-certified allergist for evaluation and allergy skin testing to identify indoor allergens that are most likely to be clinically relevant.
6. Environmental control strategies are tailored to each potentially relevant indoor exposure and are based on knowledge of the sources and underlying characteristics of the exposure. Strategies include source removal, source control, and mitigation strategies.

LEAD AUTHORS

Elizabeth C. Matsui, MD, MHS, FAAP
Stuart L. Abramson, MD, PhD, AE-C, FAAP
Megan T. Sandel, MD, MPH, FAAP

SECTION ON ALLERGY AND IMMUNOLOGY EXECUTIVE COMMITTEE, 2015–2016

Elizabeth C. Matsui, MD, MHS, FAAP, Chair
Stuart L. Abramson, MD, PhD, AE-C, FAAP
Chitra Dinakar, MD, FAAP
Anne-Marie Irani, MD, FAAP
Jennifer S. Kim, MD, FAAP
Todd A. Mahr, MD, FAAP, Immediate Past Chair
Michael Pistiner, MD, FAAP
Julie Wang, MD, FAAP

FORMER EXECUTIVE COMMITTEE MEMBERS

Thomas A. Fleisher, MD, FAAP
Scott H. Sicherer, MD, FAAP
Paul V. Williams, MD, FAAP

STAFF

Debra L. Burrowes, MHA

COUNCIL ON ENVIRONMENTAL HEALTH EXECUTIVE COMMITTEE, 2015–2016

Jennifer A. Lowry, MD, FAAP, Chair
Samantha Ahdoot, MD, FAAP
Carl R. Baum, MD, FAAP
Aaron S. Bernstein, MD, MPH, FAAP
Aparna Bole, MD, FAAP
Heather L. Brumberg, MD, MPH, FAAP
Carla C. Campbell, MD, MS, FAAP
Bruce P. Lanphear, MD, MPH, FAAP
Susan E. Pacheco, MD, FAAP
Adam J. Spanier, MD, PhD, MPH, FAAP
Leonardo Trasande, MD, MPP, FAAP

FORMER EXECUTIVE COMMITTEE MEMBERS

Kevin C. Osterhoudt, MD, MSCE, FAAP
Jerome A. Paulson, MD, FAAP
Megan T. Sandel, MD, MPH, FAAP

LIAISONS

John M. Balbus, MD, MPH – *National Institute of Environmental Health Sciences*
Todd Brubaker, DO – *AAP Section on Medical Students, Residents, and Fellowship Trainees*
Ruth A. Etzel, MD, PhD, FAAP – *US Environmental Protection Agency*
Mary Ellen Mortensen, MD, MS – *Centers for Disease Control and Prevention/National Center for Environmental Health*
Nathaniel G. DeNicola, MD, MSC – *American Congress of Obstetricians and Gynecologists*
Mary H. Ward, PhD – *National Cancer Institute*

STAFF

Paul Spire

ABBREVIATIONS

AAP: American Academy of Pediatrics
HEPA: high-efficiency particulate air
IgE: immunoglobulin E
NO_2: nitrogen dioxide
PM: particulate matter
SHS: secondhand smoke

REFERENCES

1. Keet CA, McCormack MC, Pollack CE, Peng RD, McGowan E, Matsui EC. Neighborhood poverty, urban residence, race/ethnicity, and asthma: rethinking the inner-city asthma epidemic. *J Allergy Clin Immunol*. 2015;135(3):655–662
2. 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;115(3):478–485
3. Ahluwalia SK, Peng RD, Breysse PN, et al Mouse allergen is the major allergen of public health relevance in Baltimore City. *J Allergy Clin Immunol*. 2013;132(4):830–835, e831–e832
4. Torjusen EN, Diette GB, Breysse PN, Curtin-Brosnan J, Aloe C, Matsui EC. Dose-response relationships between mouse allergen exposure and asthma morbidity among urban children and adolescents. *Indoor Air*. 2013;23(4):268–274
5. Almqvist C, Wickman M, Perfetti L, et al. Worsening of asthma in children allergic to cats, after indirect exposure

to cat at school. *Am J Respir Crit Care Med*. 2001;163(3 pt 1):694–698

6. Matsui EC, Sampson HA, Bahnson HT, et al; Inner-city Asthma Consortium. Allergen-specific IgE as a biomarker of exposure plus sensitization in inner-city adolescents with asthma. *Allergy*. 2010;65(11):1414–1422

7. Butz AM, Matsui EC, Breysse P, et al. A randomized trial of air cleaners and a health coach to improve indoor air quality for inner-city children with asthma and secondhand smoke exposure. *Arch Pediatr Adolesc Med*. 2011;165(8):741–748

8. Hansel NN, Breysse PN, McCormack MC, et al. A longitudinal study of indoor nitrogen dioxide levels and respiratory symptoms in inner-city children with asthma. *Environ Health Perspect*. 2008;116(10):1428–1432

9. Bennett WD, Zeman KL. Deposition of fine particles in children spontaneously breathing at rest. *Inhal Toxicol*. 1998;10(9):831–842

10. Bennett WD, Zeman KL, Jarabek AM. Nasal contribution to breathing and fine particle deposition in children versus adults. *J Toxicol Environ Health A*. 2008;71(3):227–237

11. Foos B, Marty M, Schwartz J, et al. Focusing on children's inhalation dosimetry and health effects for risk assessment: an introduction. *J Toxicol Environ Health A*. 2008;71(3):149–165

12. Miller MD, Marty MA, Arcus A, Brown J, Morry D, Sandy M. Differences between children and adults: implications for risk assessment at California EPA. *Int J Toxicol*. 2002;21(5):403–418

13. Childhood Asthma Management Program Research Group. Long-term effects of budesonide or nedocromil in children with asthma. *N Engl J Med*. 2000;343(15):1054–1063

14. Covar RA, Spahn JD, Murphy JR, Szefler SJ; Childhood Asthma Management Program Research Group. Progression of asthma measured by lung function in the childhood asthma management program. *Am J Respir Crit Care Med*. 2004;170(3):234–241

15. 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;102(4 pt 1):563–570

16. Etzel RA, Balk SJ, eds. *Pediatric Environmental Health*. 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012

17. National Asthma Education and Prevention Program. Expert Panel Report 3 (EPR-3): guidelines for the diagnosis and management of asthma—summary report 2007. *J Allergy Clin Immunol*. 2007;120(5 suppl):S94–S138

18. Sicherer SH, Wood RA; American Academy of Pediatrics Section on Allergy and Immunology. Allergy testing in childhood: using allergen-specific IgE tests. *Pediatrics*. 2012;129(1):193–197

19. Morgan WJ, Crain EF, Gruchalla RS, et al; Inner-City Asthma Study Group. Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med*. 2004;351(11):1068–1080

20. Carswell F, Birmingham K, Oliver J, Crewes A, Weeks J. The respiratory effects of reduction of mite allergen in the bedrooms of asthmatic children—a double-blind controlled trial. *Clin Exp Allergy*. 1996;26(4):386–396

21. Halken S, Høst 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;111(1):169–176

22. Weiss ST, Horner A, Shapiro G, Sternberg AL; Childhood Asthma Management Program Research Group. The prevalence of environmental exposure to perceived asthma triggers in children with mild-to-moderate asthma: data from the Childhood Asthma Management Program (CAMP). *J Allergy Clin Immunol*. 2001;107(4):634–640

23. Kattan M, Mitchell H, Eggleston P, et al. Characteristics of inner-city children with asthma: the National Cooperative Inner-City Asthma Study. *Pediatr Pulmonol*. 1997;24(4):253–262

24. Portnoy J, Miller JD, Williams PB, et al; Joint Taskforce on Practice Parameters; Practice Parameter Workgroup. Environmental assessment and exposure control of dust mites: a practice parameter. *Ann Allergy Asthma Immunol*. 2013;111(6):465–507

25. Gøtzsche PC, Johansen HK. House dust mite control measures for asthma. *Cochrane Database Syst Rev*. 2008;2:CD001187

26. Ehnert B, Lau-Schadendorf S, Weber A, Buettner P, Schou C, Wahn U. Reducing domestic exposure to dust mite allergen reduces bronchial hyperreactivity in sensitive children with asthma. *J Allergy Clin Immunol*. 1992;90(1):135–138

27. Rijssenbeek-Nouwens LH, Oosting AJ, de Bruin-Weller MS, Bregman I, de Monchy JG, Postma DS. Clinical evaluation of the effect of anti-allergic mattress covers in patients with moderate to severe asthma and house dust mite allergy: a randomised double blind placebo controlled study. *Thorax*. 2002;57(9):784–790

28. Rijssenbeek-Nouwens LH, Oosting AJ, De Monchy JG, Bregman I, Postma DS, De Bruin-Weller MS. The effect of anti-allergic mattress encasings on house dust mite-induced early- and late-airway reactions in asthmatic patients: a double-blind, placebo-controlled study. *Clin Exp Allergy*. 2002;32(1):117–125

29. Colloff MJ, Ayres J, Carswell F, et al. The control of allergens of dust mites and domestic pets: a position paper. *Clin Exp Allergy*. 1992;22(suppl 2):1–28

30. Portnoy J, Kennedy K, Sublett J, et al. Environmental assessment and exposure control: a practice parameter - furry animals. *Ann Allergy Asthma Immunol*. 2012;108(4):223.e1–223.15

31. 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;83(4):730–734

32. Shirai T, Matsui T, Suzuki K, Chida K. Effect of pet removal on pet allergic asthma. *Chest*. 2005;127(5):1565–1571

33. 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;158(1):115–120

34. 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;104(2 pt 1):447–451
35. Sulser C, Schulz G, Wagner P, et al. Can the use of HEPA cleaners in homes of asthmatic children and adolescents sensitized to cat and dog allergens decrease bronchial hyperresponsiveness and allergen contents in solid dust? *Int Arch Allergy Immunol*. 2009;148(1):23–30
36. Vredegoor DW, Willemse T, Chapman MD, Heederik DJ, Krop EJ. Can f 1 levels in hair and homes of different dog breeds: lack of evidence to describe any dog breed as hypoallergenic. *J Allergy Clin Immunol*. 2012;130(4):904–909, e907
37. Erwin EA, Woodfolk JA, Ronmark E, Perzanowski M, Platts-Mills TAE. The long-term protective effects of domestic animals in the home. *Clin Exp Allergy*. 2011;41(7):920–922
38. Phipatanakul W, Matsui E, Portnoy J, et al; Joint Task Force on Practice Parameters. Environmental assessment and exposure reduction of rodents: a practice parameter. *Ann Allergy Asthma Immunol*. 2012;109(6):375–387
39. 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;94(5):810–817
40. Matsui EC, Simons E, Rand C, et al. Airborne mouse allergen in the homes of inner-city children with asthma. *J Allergy Clin Immunol*. 2005;115(2):358–363
41. 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;106(6):1070–1074
42. Pongracic JA, Visness CM, Gruchalla RS, Evans R III, Mitchell HE. Effect of mouse allergen and rodent environmental intervention on asthma in inner-city children. *Ann Allergy Asthma Immunol*. 2008;101(1):35–41
43. 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;97(4):514–520
44. Matsui EC, Wood RA, Rand C, Kanchanaraksa S, Swartz L, Eggleston PA. Mouse allergen exposure and mouse skin test sensitivity in suburban, middle-class children with asthma. *J Allergy Clin Immunol*. 2004;113(5):910–915
45. Cohn RD, Arbes SJ Jr, Yin M, Jaramillo R, Zeldin DC. National prevalence and exposure risk for mouse allergen in US households. *J Allergy Clin Immunol*. 2004;113(6):1167–1171
46. 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;92(4):420–425
47. 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;336(19):1356–1363
48. Arbes SJ Jr, Sever M, Mehta J, et al. Abatement of cockroach allergens (Bla g 1 and Bla g 2) in low-income, urban housing: month 12 continuation results. *J Allergy Clin Immunol*. 2004;113(1):109–114
49. Wood RA, Eggleston PA, Rand C, Nixon WJ, Kanchanaraksa S. Cockroach allergen abatement with extermination and sodium hypochlorite cleaning in inner-city homes. *Ann Allergy Asthma Immunol*. 2001;87(1):60–64
50. Portnoy J, Chew GL, Phipatanakul W, et al Environmental assessment and exposure reduction of cockroaches: a practice parameter. *J Allergy Clin Immunol*. 2013;132(4):802–808, e801–e825
51. Dales R, Liu L, Wheeler AJ, Gilbert NL. Quality of indoor residential air and health. *CMAJ*. 2008;179(2):147–152
52. Pongracic JA, O'Connor GT, Muilenberg ML, et al. Differential effects of outdoor versus indoor fungal spores on asthma morbidity in inner-city children. *J Allergy Clin Immunol*. 2010;125(3):593–599
53. Salo PM, Arbes SJ Jr, Sever M, et al. Exposure to *Alternaria alternata* in US homes is associated with asthma symptoms. *J Allergy Clin Immunol*. 2006;118(4):892–898
54. Salo PM, Arbes SJ Jr, Crockett PW, Thorne PS, Cohn RD, Zeldin DC. Exposure to multiple indoor allergens in US homes and its relationship to asthma. *J Allergy Clin Immunol*. 2008;121(3):678–684, e672
55. Kercsmar CM, Dearborn DG, Schluchter M, et al. Reduction in asthma morbidity in children as a result of home remediation aimed at moisture sources. *Environ Health Perspect*. 2006;114(10):1574–1580
56. Burr ML, Matthews IP, Arthur RA, et al. Effects on patients with asthma of eradicating visible indoor mould: a randomised controlled trial. *Thorax*. 2007;62(9):767–772
57. Institute of Medicine, Board on Health Promotion and Disease Prevention, Committee on Damp Indoor Spaces and Health. *Damp Indoor Spaces and Health*. Washington, DC: National Academies Press; 2004. Available at: www.iom.edu/Reports/2004/Damp-Indoor-Spaces-and-Health.aspx. Accessed January 25, 2016
58. World Health Organization Regional Office for Europe. Guidelines for Indoor Air Quality: Dampness and Mould. Geneva, Switzerland: World Health Organization; 2009. Available at: www.who.int/indoorair/publications/7989289041683/en/. Accessed January 25, 2016
59. McCormack MC, Breysse PN, Matsui EC, et al; Center for Childhood Asthma in the Urban Environment. Indoor particulate matter increases asthma morbidity in children with non-atopic and atopic asthma. *Ann Allergy Asthma Immunol*. 2011;106(4):308–315
60. McCormack MC, Breysse PN, Hansel NN, et al. Common household activities are associated with elevated particulate matter concentrations in bedrooms of inner-city Baltimore pre-school children. *Environ Res*. 2008;106(2):148–155

61. Koenig JQ, Mar TF, Allen RW, et al. Pulmonary effects of indoor- and outdoor-generated particles in children with asthma. *Environ Health Perspect*. 2005;113(4):499–503

62. Delfino RJ, Quintana PJ, Floro J, et al. Association of FEV1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. *Environ Health Perspect*. 2004;112(8):932–941

63. Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. Short-term effects of PM10 and NO2 on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. *Environ Health Perspect*. 2010;118(4):449–457

64. Schober W, Szendrei K, Matzen W, et al. Use of electronic cigarettes (e-cigarettes) impairs indoor air quality and increases FeNO levels of e-cigarette consumers. *Int J Hyg Environ Health*. 2014;217(6):628–637

65. Morkjaroenpong V, Rand CS, Butz AM, et al. Environmental tobacco smoke exposure and nocturnal symptoms among inner-city children with asthma. *J Allergy Clin Immunol*. 2002;110(1):147–153

66. 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;95(6):518–524

67. 2008 PHS Guideline Update Panel, Liaisons, and Staff. Treating tobacco use and dependence: 2008 update U.S. Public Health Service Clinical Practice Guideline executive summary. *Respir Care*. 2008;53(9):1217–1222

68. Farber HJ, Groner J, Walley S, Nelson K; Section on Tobacco Control. Protecting children from tobacco, nicotine, and tobacco smoke [technical report]. *Pediatrics*. 2015;136(5). Available at: www.pediatrics.org/cgi/content/full/136/5/e1439

69. Farber HJ, Walley SC, Groner JA, Nelson KE; Section on Tobacco Control. Clinical practice policy to protect children from tobacco, nicotine, and tobacco smoke [policy statement]. *Pediatrics*. 2015;136(5):1008–1017

70. Farber HJ, Nelson KE, Groner JA, Walley SC; Section on Tobacco Control. Public policy to protect children from tobacco, nicotine, and tobacco smoke [policy statement]. *Pediatrics*. 2015;136(5):998–1007

71. Lanphear BP, Hornung RW, Khoury J, Yolton K, Lierl M, Kalkbrenner A. Effects of HEPA air cleaners on unscheduled asthma visits and asthma symptoms for children exposed to secondhand tobacco smoke. *Pediatrics*. 2011;127(1):93–101

72. Been JV, Nurmatov U, van Schayck CP, Sheikh A. The impact of smoke-free legislation on fetal, infant and child health: a systematic review and meta-analysis protocol. *BMJ Open*. 2013;3(2):e002261

73. Kattan M, Gergen PJ, Eggleston P, Visness CM, Mitchell HE. Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. *J Allergy Clin Immunol*. 2007;120(3):618–624

74. Paulin LM, Diette GB, Scott M, et al. Home interventions are effective at decreasing indoor nitrogen dioxide concentrations. *Indoor Air*. 2014;24(4):416–424

75. Wu F, Takaro TK. Childhood asthma and environmental interventions. *Environ Health Perspect*. 2007;115(6):971–975

76. Woodcock A, Forster L, Matthews E, et al; Medical Research Council General Practice Research Framework. Control of exposure to mite allergen and allergen-impermeable bed covers for adults with asthma. *N Engl J Med*. 2003;349(3):225–236

77. Platts-Mills TA. Allergen avoidance in the treatment of asthma: problems with the meta-analyses. *J Allergy Clin Immunol*. 2008;122(4):694–696

78. 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;116(5):1058–1063

PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Indoor Environmental Control Practices and Asthma Management

Elizabeth C. Matsui, Stuart L. Abramson, Megan T. Sandel, SECTION ON ALLERGY AND IMMUNOLOGY and COUNCIL ON ENVIRONMENTAL HEALTH

Pediatrics 2016;138;
DOI: 10.1542/peds.2016-2589 originally published online October 31, 2016;