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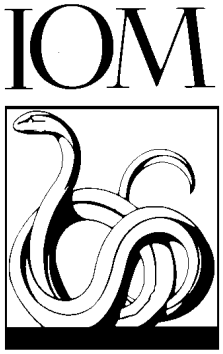
Clearing the Air: Asthma and Indoor Air Exposures

Committee on the Assessment of Asthma and Indoor Air

Division of Health Promotion and
Disease Prevention

INSTITUTE OF MEDICINE



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The serpent has been a symbol of long life, healing, and knowledge among almost all cultures and religions since the beginning of recorded history. The image adopted as a logotype by the Institute of Medicine is based on a relief carving from ancient Greece, now held by the Staatliche Museen in Berlin.

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Executive Summary

The statistics are disturbing.

The Centers for Disease Control and Prevention (CDC) estimates that asthma affected about 17.3 million individuals in the United States in 1998. It is the most common chronic illness among children in the United States and one of the most common chronic illnesses overall in the country. Although by many measures the health of Americans is improving, CDC notes the self-reported prevalence rate for asthma increased 75% from 1980 to 1994. Studies show that asthma mortality is disproportionately high among African Americans and in urban areas that are characterized by high levels of poverty and minority populations. Nor is the phenomenon limited to the United States. The prevalence of asthma in some other parts of the world—including Australia, New Zealand, Ireland, and the United Kingdom—exceeds that of the United States.

Researchers have wondered whether the indoor environment may play a role in the increasing asthma problem. There is ample justification for this speculation. We know, for example, that individuals spend nearly all of their time indoors—most of it in their own homes—and that many of the exposures thought to be associated with asthma occur predominately indoors. If the indoor environment plays a role, then interventions to limit or eliminate exposures there have the potential to help asthmatics and perhaps result in primary prevention of the illness.

Against this backdrop, the U.S. Environmental Protection Agency (EPA) is developing an outreach strategy focused on reducing asthma-related morbidity and mortality potentially associated with exposure to indoor environments. To help ensure that such efforts are based on sound science, EPA requested that the National Academies undertake an assessment of asthma and its relationship to indoor air quality. The EPA charged the committee with two primary objectives:

1. To provide the scientific and technical basis for communications to the public on the health impacts of indoor pollutants related to asthma, and mitigation and prevention strategies to reduce these pollutants.
2. To help determine what research is needed in these areas.

This report presents the results of that assessment.

ORGANIZATION AND FRAMEWORK

The content of this report reflects the committee's goal to speak to a wide-ranging audience of science, health, and engineering professionals; government officials; and interested members of the public. The material presented thus covers a broad range of topics in order to establish a common base of knowledge for the reader. The scope of this material is far too vast for any one book to deal with comprehensively. Other publications, cited throughout the report, go into greater detail on specific issues.

The major topics addressed in the report are the following:

- the definition of asthma and the characteristics of its clinical presentation (Chapter 1);
- methodologic issues in evaluating the evidence regarding indoor air exposures and asthma, including the categorizations used to summarize the evidence and the framework for considering exposure to indoor sources (Chapter 2);
- patterns of asthma morbidity and mortality (Chapter 3);
- the pathophysiology of asthma—that is, the molecular mechanisms that underlie the structural and functional changes in the lungs and airways of asthmatics (Chapter 4);
- the committee's review of the state of the scientific literature regarding indoor air exposures and the exacerbation and development of asthma—Table ES-1 lists the biologic and chemical exposures addressed in this report. (Chapters 5–7);
- the scientific literature on general exposures in indoor environments (Chapters 8–9); and
- how indoor exposures to pollutants associated with the incidence or symptoms of asthma are affected by building ventilation and particle air cleaning (Chapter 10).

Table ES-1 Indoor Exposures Addressed in This Report

BIOLOGICAL	
Animals	Fungi or molds
Cats	Houseplants
Dogs	Pollen
Rodents	Infectious agents
Cows and horses	Rhinovirus
Domestic birds	Respiratory syncytial virus
Cockroaches	*Chlamydia trachomatis*
House dust mites	*Chlamydia pneumoniae*
Endotoxins	*Mycoplasma pneumoniae*
CHEMICAL	
NO_2, NO_X (nitrogen oxides)	Plasticizers
Pesticides	Volatile organic compounds
Ozone*	Formaldehyde
Particulate matter with sources other than ETS*	Fragrances
SO_2, SO_X (sulfur oxides)*	Environmental Tobacco Smoke (ETS)

*an outdoor air pollutants potentially associated with asthma that can penetrate the indoor environment and that may in some cases have indoor sources. Since the committee's mandate was to address indoor air pollutants, the discussion of this agent is less detailed than others in the report and no conclusions are drawn concerning indoor exposures and asthma outcomes.

The committee faced a significant challenge in conducting its review—research on asthma is burgeoning and significant new papers are constantly being published. Although the committee did its best to paint an accurate picture of the state of the science at the time the report was completed, it is inevitable that research advances will overtake its conclusions.

CONCLUSIONS ABOUT THE RELATIONSHIP BETWEEN INDOOR EXPOSURES AND ASTHMA

The committee used a uniform set of categories to summarize its conclusions regarding the association between exposure to an indoor agent and asthma development and exacerbation, and the effectiveness of exposure mitigation and prevention measures. Box ES-1 lists the definitions of these categories. The distinctions among categories reflect the committee's judgment of the overall strength, quality, and persuasiveness of the scientific literature evaluated. Chapter 2 details the methodologic considerations underlying the categorizations and their definitions.

The sections below are a synopsis of the committee's findings. Chapters 5 through 10 address the reasoning underlying the conclusions and present the findings in greater detail.

Exposure Settings

The indoor exposures considered in this report are highly dependent on the characteristics of the outdoor and indoor environment and its occupants. For example, house dust mites are a very common exposure in temperate and humid regions. They are found primarily within residences, concentrated in the bedroom. Cockroaches, which also thrive in temperate and humid regions, are an important exposure in some urban environments. They are found primarily near food sources. Fungi are ubiquitous and have been the primary source of allergen for several studied populations. Endotoxins may be found in humidifiers and in bacteria from other indoor, as well as outdoor sources. In some environments, exposure to animal allergens; molds; environmental tobacco smoke (ETS); indoor combustion products; and chemicals used in cleaning, building materials, and furnishings may be important. Many of these pollutants are also present in outdoor air, and indoor exposures can result from the infiltration of outdoor air into buildings.

Box ES-1 Categories of Evidence Used in This Report

Sufficient Evidence of a Causal Relationship

Evidence is sufficient to conclude that a causal relationship exists between the action or agent and the outcome. That is, the evidence fulfills the criteria for "Sufficient Evidence of an Association" below and in addition satisfies criteria regarding the strength of association, biologic gradient (dose–response effect), consistency of association, biologic plausibility and coherence, and temporality used to assess causality.

Sufficient Evidence of an Association

Evidence is sufficient to conclude that there is an association. That is, an association between the action or agent and the outcome has been observed in studies in which chance, bias, and confounding can be ruled out with reasonable confidence. For example, if several small studies that are free from bias and confounding show an association that is consistent in magnitude and direction, there may be sufficient evidence of an association.

Limited or Suggestive Evidence of an Association

Evidence is suggestive of an association between the action or agent and the outcome but is limited because chance, bias, and confounding cannot be ruled out with confidence. For example, at least one high-quality study shows a positive association, but the results of other studies are inconsistent.

Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists

The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an association; or no studies exist that examine the relationship. For example, available studies have failed to adequately control for confounding or have inadequate exposure assessment.

Limited or Suggestive Evidence of No Association

Several adequate studies are mutually consistent in not showing an association between the action or agent and the outcome. A conclusion of "no association" is inevitably limited to the conditions, level of exposure, and length of observation covered by the available studies. *In addition, the possibility of a very small elevation in risk at the levels of exposure studied can never be excluded.*

Indoor Air Exposures and Asthma Exacerbation

Studies of asthma can be divided into those dealing with factors leading to the development of asthma and those dealing with factors that exacerbate the illness in known asthmatics. Most of the research on this topic addresses "asthma exacerbation," the onset or worsening of symptoms—some combination of shortness of breath, cough, wheezing, and chest tightness—in someone who already has developed asthma.

Epidemiologic investigations, challenge studies, and clinical experience have yielded solid information on the potential for many indoor exposures to exacerbate asthma. The committee found **sufficient evidence to conclude that there is a causal relationship** between

- exposure to the allergens produced by cats, cockroaches, and house dust mites, and exacerbations of asthma in sensitized individuals; and
- ETS exposure and exacerbations of asthma in preschool-aged children.

There is **sufficient evidence of an association** between several exposures and exacerbations of asthma. Dog allergen exposure is associated with exacerbation of asthma in individuals specifically sensitized to these allergens. Fungal exposure is associated with exacerbation in sensitized asthmatics and may be associated with nonspecific chest symptoms. Research indicates that rhinovirus infection is associated with wheezing and exacerbations in

asthmatics. There is also sufficient evidence to conclude that brief high-level[1] exposures to NO_2 and increased airway responses among asthmatic subjects to both nonspecific chemical irritants and inhaled allergens.

Damp conditions are associated with the presence of symptoms considered to reflect asthma; symptom prevalence among asthmatics is also related to dampness indicators. The factors related to dampness that may actually lead to asthma exacerbation are not yet confirmed, but probably relate to dust mite and fungal allergens. There is sufficient evidence that some nonresidential buildings provide exposures that exacerbate asthma. However, the specific agents responsible for such exacerbations are as yet unstudied.

Limited or suggestive evidence was found for an association between exposures to domestic birds and exacerbation of asthma, although it is unclear what portion of this association is attributable to an allergic asthmatic response to the mites harbored by these birds. There is also limited or suggestive evidence of a relationship between

- exposure to the infectious agents respiratory syncytial virus (RSV), *Chlamydia pneumoniae,* and *Mycoplasma pneumoniae,* and exacerbation of asthma;
- chronic ETS exposure and exacerbation of asthma in older children and adults;
- acute ETS exposure and exacerbation of asthma in individuals responsive to this exposure;
- nonacute, nonoccupational formaldehyde exposure and wheezing and other respiratory symptoms; and
- exposure to certain fragrances and the manifestation of respiratory symptoms in asthmatics sensitive to such exposures.

Inadequate or insufficient information was identified to determine whether or not exacerbations of asthma result from nonacute, nonoccupational exposures to cow, horse, and rodent allergens; endotoxins; houseplants[2] or cut flowers; the bacterial agent *Chlamydia trachomatis*; pesticides; plasticizers; and volatile organic compounds (VOCs) other than formaldehyde. Some of these same agents do or may play a role in asthma resulting from exposures in occupational settings, a topic outside the purview of this study.

Although there is sufficient evidence to conclude that pollen exposure is associated with exacerbation of existing asthma in sensitized individuals, and pollen allergens have been documented in both dust and indoor air, there is inadequate or insufficient information to determine whether indoor exposure to pollen is associated with exacerbations of asthma.

These findings are summarized in Table ES-2.

[1] At concentrations that may occur only when gas appliances are used in poorly ventilated kitchens.

[2] Mites and fungi associated with houseplants could be involved in asthma outcomes but no studies document this connection.

Table ES-2 Summary of Findings Regarding the Association between Indoor Biologic and Chemical Exposures and the *Exacerbation* of Asthma in Sensitive Individuals

BIOLOGICAL AGENTS	CHEMICAL AGENTS
Sufficient Evidence of a Causal Relationship	
Cat	ETS (in preschool-aged children)
Cockroach	
House Dust Mite	
Sufficient Evidence of an Association	
Dog	NO_2, NO_X (high-level exposures[a])
Fungi or molds	
Rhinovirus	
Limited or Suggestive Evidence of an Association	
Domestic birds	ETS (in school-aged and older children, and in adults)
Chlamydia pneumoniae	Formaldehyde
Mycoplasma pneumoniae	Fragrances
Respiratory Syncytial Virus (RSV)	
Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists	
Cow and horse	Pesticides
Rodents (as pets or feral animals)	Plasticizers
Chlamydia trachomatis	VOCs
Endotoxins	
Houseplants	
Pollen exposure in indoor environments	
Insects other than cockroaches	
Limited or Suggestive Evidence of No Association	
(no agents met this definition)	

[a] At concentrations that may occur only when gas appliances are used in poorly ventilated kitchens.

Indoor Air Exposures and Asthma Development

The second outcome reviewed by the committee was the development of asthma—the initial onset of the illness. Asthma is defined by the manifestation of a set of symptoms rather than by any one objective test. With asthma symptoms ranging from clearly episodic to nearly continuous, from mild to severe, and from coughing without other respiratory symptoms to a loud wheeze, the initial diagnosis of the illness can be complicated and subject to controversy. It is thus difficult to study the determinants of and influences on asthma development. An additional complication stems from the fact that some of the most provocative evidence regarding development comes from studies of infants. Prior to the age of approximately 3, children may exhibit symptoms that are characteristic of asthma, but they may not exhibit persistent asthmatic symptoms or other related conditions such as bronchial reactivity or allergy later in life. Chapter 2 discusses the definitions of asthma and the characteristics of its clinical presentation.

Saying that a particular agent may be associated with the development of asthma does not mean it is the sole factor determining whether an individual will manifest the illness. Most scientists believe that some individuals have a prior, underlying predisposition that permits the evolution of clinical asthma. The development of this predisposition to asthma is dependent on a complex—and at present poorly understood—combination of factors, which are partially inherited and partially acquired later in life.

After careful consideration of the scientific literature, the committee concluded there is **sufficient evidence of a causal relationship** between exposure to house dust mite allergen and the development of asthma in susceptible children. This conclusion was based on the preponderance of several lines of evidence, including the results of clinical studies and population-based, case-control, and prospective epidemiologic investigations; the consistency of the association in different racial and ethnic groups; and the presence of a dose–response relationship between exposure to dust mite allergen and sensitization. Chapter 5 delineates the reasoning underlying this conclusion in greater detail.

There is **sufficient evidence to conclude that there is an association** between ETS exposure and the development of asthma in younger children. In the limited number of studies that have been able to separate the effects of maternal active smoking during pregnancy from the effects of ETS exposure after birth, evidence suggests that—although both exposures are detrimental—maternal smoking during pregnancy has the stronger adverse effect.

Limited or suggestive evidence exists for associations between

- cockroach allergen exposure and development of asthma in preschool-aged children; and
- infection with RSV and development of asthma in preschool-aged children.

The impact of exposure to these agents has been the subject of great research interest in the past few years, and efforts presently underway may clarify their role in asthma development.

Published case reports, public health surveillance of physician reporting, and cross-sectional studies of building occupants with indoor air quality complaints also provide limited or suggestive evidence of an association between aspects of the nonindustrial indoor environment and the development of asthma, with a building occupancy-related pattern of symptoms and in some instances objective abnormalities. What is lacking for the most part, however, is knowledge of specific etiologic agents in these nonindustrial indoor environments that might be responsible for new work-related asthma cases.

Inadequate or insufficient evidence exists to determine whether or not the other indoor exposures listed in Table ES-1 are associated with the development of asthma. This lack of information points to a gap in present-day knowledge concerning asthma—one that will be challenging to resolve.

There is **limited or suggestive evidence of no association** between infection with rhinovirus—the medical term for the large and ubiquitous group of viruses responsible for a variety of respiratory infections including those referred to as “the common cold”—and asthma development.

Table ES-3 summarizes these findings.

Table ES-3 Summary of Findings Regarding the Association Between Indoor Biologic and Chemical Exposures and the *Development* of Asthma

Biologic Agents	Chemical Agents
Sufficient Evidence of a Causal Relationship	
House dust mite	(no agents met this definition)
Sufficient Evidence of an Association	
(no agents met this definition)	ETS (in preschool-aged children)
Limited or Suggestive Evidence of an Association	
Cockroach (in preschool-aged children)	(no agents met this definition)
Respiratory Syncytial Virus (RSV)	
Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists	
Cat	NO_2, NO_X
Cow and horse	Pesticides
Dog	Plasticizers
Domestic birds	VOCs
Rodents	Formaldehyde
Cockroaches (except for preschool-aged children)	Fragrances
Endotoxins	ETS (in school-aged and older children, and in adults)
Fungi or molds	
Chlamydia pneumoniae	
Chlamydia trachomatis	
Mycoplasma pneumoniae	
Houseplants	
Pollen	
Limited or Suggestive Evidence of No Association	
Rhinovirus (adults)	(no agents met this definition)

Effectiveness of Indoor Environmental Interventions in Limiting Exposures and Affecting Asthma Outcomes

Patients with asthma and the parents of children with asthma need reliable information on which measures are likely to be most effective for improving indoor air quality. Specific recommendations are found in each chapter but there are general principles that should be kept in mind. Agents that can exacerbate asthma may generally be thought of in two categories: specific allergens and non-specific respiratory tract irritants. Exposure to non-specific irritants, such as cigarette smoke, may lead to asthma symptoms in any person with asthma; while allergens are only problems for individuals who are allergic to them. For example, if a person with asthma is allergic to cats, exposure to cats may cause wheezing; but if that person is not allergic to cats, exposure to them will not cause any problems. Therefore, reducing indoor airborne exposure to irritants is likely to help all asthmatic individuals to some degree while reductions in allergen exposure would only be expected to help individuals who are allergic to the allergens being reduced.

While the report identifies a number a mitigation strategies that are or may be effective in reducing exposure to potentially problematic agents, the committee found only a small number for which there is presently evidence that proper implementation of the strategy results in an improvement of symptoms or lung function in asthmatics. It is important to remember, though, that the absence of evidence does not mean an absence of effect. The science regarding indoor environmental interventions, exposure limitation, and effects on asthma outcomes is not nearly as well developed as that regarding the health effects of exposures. Exposure assessment[3] is often the weakest link in environmental health studies because it is difficult to do and is given inadequate attention by many researchers.

Nonetheless, the committee was able to identify well-conducted, rigorous studies on which to base conclusions.

Sufficient evidence of an association was found between the use of a combination of physical measures and a reduction in indoor **dust mite** allergen levels in dust samples. As detailed in Chapter 5, strategies for the effective control of mite growth vary by climate. Such measures have been shown to be effective at reducing symptoms in controlled trials and should be part of normal management of asthma in mite-allergic individuals. Several studies now underway are evaluating whether aggressive allergen avoidance regimes have an effect on the subsequent development of asthma. The results of these and other studies will inform the question of whether primary prevention of dust mite-induced asthma is possible. Two related issues that will have to be addressed are (1) the feasibility of implementing such comprehensive interventions and (2) whether these interventions result in lower rates of sensitization to a particular exposure or all exposures.

The committee found limited or suggestive evidence that the combined use of **cockroach** extermination and control of potential reservoirs of allergen in beds, carpets, furnishings, and clothing through cleaning can achieve a short-term decrease in cockroach allergen levels in indoor environments. Extermination alone appears ineffective because significant allergen levels remain in settled dust; cleaning alone in the absence of complete extermination does not eliminate the sources of the allergen. There was inadequate or insufficient evidence to determine whether or not an association exists between any cockroach mitigation or prevention strategy and transient or long-term improvement of symptoms or lung function in cockroach-allergic asthmatics. However, since evidence does suggest that dust mite mitigation strategies result in improvement of symptoms or lung function, mitigation of cockroach exposures would appear to be a sensible course of action in the absence of more definitive information.

Although the strategy may be unpopular, there is limited or suggestive evidence of an association between removal of a **cat** from the home and improvement of symptoms or lung function in cat-allergic asthmatics. Concomitant removal or isolation of known reservoirs of cat allergen (carpets, upholstery, mattresses, pillows) may be required to diminish allergen levels to those commonly measured in homes without cats. Limited or suggestive evidence indicates that some measures short of removal (e.g.,, washing the animal) may result in transient reduction in allergen levels. However, there is inadequate or insufficient evidence to determine whether or not an association exists between measures short of removal of a cat from the home and improvement in symptoms in cat-allergic asthmatics. Data on the effectiveness of interventions for **other animals** are too sparse to draw informed conclusions.

[3] Classically, "exposure assessment" involves specifying the population that might be exposed to the agent of concern; identifying the routes through which exposure can occur, and estimating the magnitude, duration, and timing of the dose that individuals might receive as a result of their exposure (NAS, 1994).

It is possible to physically remove accessible growing **fungi** from indoor environments. The entry of fungal spores from outdoors can be substantially reduced in mechanically ventilated buildings by pressurizing them and filtering incoming air; closing windows should also reduce indoor concentrations from outdoor sources. Although there is limited or suggestive evidence that such steps may result in a reduction in the levels of fungi in the indoor environment, the health impact of such reduction has not been studied. Fungi are difficult to kill, and dead fungal material probably contains allergens that can become airborne, although this has not been thoroughly tested.

There is relatively little information on the impact of ventilation and air-cleaning measures on indoor **pollen** levels, although it is clear that shutting windows and other measures that generally limit the entry rate of unfiltered outdoor air can be effective.

No general conclusions about means of altering exposure to low levels of **endotoxin** can be made at the present time. However, avoiding the use of cool mist humidifiers would appear to be a simple and effective means of eliminating risk of high-level exposure to endotoxin at home as well as exposure to organisms associated with hypersensitivity pneumonitis.

Source control—that is, stopping smoking—appears to be the only reliably effective means of preventing **environmental tobacco smoke** exposure. There is sufficient evidence to conclude that increased ventilation is *technologically capable* of reducing the indoor concentration of ETS particles and gases, and that particle air-cleaning methods are *technologically capable* of reducing the indoor concentration of ETS particles. However, evidence is lacking on whether interventions designed to encourage the use of the requisite ventilation and air cleaning methods would be associated with a reduction in asthma development or exacerbation.

Control options for **chemical and particulate pollutants** in indoor environments include source modification (removal, substitution, or emission reduction), ventilation (exhaust or dilution), or pollutant removal (filtration). The various forms of pollutant source modification are usually the most effective. For most gaseous pollutants—NO_2 for example—removal via air cleaning is not presently practical.

No intervention studies clearly document that any form of **dampness** control works effectively to reduce symptoms or to reduce the chances of asthma development. However, given its relationship to factors (such as dust mites and fungal growth) associated with asthma, steps to reduce dampness may be appropriate. For homes, these measures include powered mechanical ventilation to remove or dilute occupant-generated moisture, proper installation of vapor barriers, channeling ground water away from foundations, sealing below-ground walls to prevent water intrusion, protecting ground-level concrete slabs from moisture intrusion, and constructing crawl spaces to prevent water intrusion.

There are both theoretical evidence and limited empirical data indicating that feasible modifications in **ventilation** rates can decrease or increase[4] concentrations of some of the indoor pollutants associated with asthma by up to approximately 75%. Limited or suggestive evidence exists to indicate that particle **air cleaning** is associated with a reduction in the exacerbation of asthma symptoms. Theoretical and limited empirical data indicate that particle air cleaners are most likely to be effective in reducing the exacerbation of asthma symptoms associated with particles smaller than approximately 2 µm, such as ETS particles[5] and some airborne cat allergen. There is insufficient evidence to determine whether or not the use of particle air cleaners is associated with decreased asthma development. It should also be noted that microorganisms can grow on some air-cleaning equipment such as filter media; thus, improperly maintained air cleaners are also a potential source of indoor pollutants.

Inadequate or insufficient information was available regarding several other interventions. These are discussed in Chapters 5 through 10.

It is difficult to draw general conclusions regarding effective indoor environmental interventions. However, the committee is able to offer some observations. For many allergens, effective strategies consist of integrated approaches consistently applied over time. The two primary components of an integrated approach are (1) removal or cleaning of allergen reservoirs and (2) control of new sources of exposure. Source removal—where it is possible—is typically the most effective control measure and may be the only effective measure for some agents.

[4] The indoor concentrations of some pollutants from outdoors—particulate matter and ozone, for example—may increase with the ventilation rate.

[5] Particle air cleaners are <u>not</u> effective in reducing concentrations of the gaseous components of ETS.

Avoidance of exposure through source removal, substitution, or emission reduction is usually the most successful approach for chemical agents.

GENERAL RESEARCH RECOMMENDATIONS AND CONCLUSIONS

Asthma is a complex illness. The many variables that determine its development and severity defy simple summary. Although great strides have been made over the past few years in elucidating mechanisms and understanding the role of environmental and genetic influences, much work remains to be done. Importantly, we still do not know whether or to what extent the reported increases in asthma can be attributed to indoor exposures.

Subsequent chapters of this report contain specific recommendations for further research on the biologic and chemical agents addressed and on the characteristics of indoor environments that may influence asthma outcomes. A digest of these recommendations is contained in Chapter 11. Some general observations are offered below.

The factors that determine the predisposition to sensitivity to certain agents and lead to the development of asthma are still not well understood. There is a great need for studies that rigorously examine the role of prenatal exposure and whether the age of first exposure influences the development of sensitization. The interaction of different environmental exposures with genetic susceptibilities—a topic of great interest but little research progress—also has to be pursued.

A major problem in choosing and implementing an intervention to mitigate an exposure is the generally limited data available. The limitations exist in regard to both the quantity and the quality of research data. Many of the studies reported are not based on rigorous protocols. Definition of clinical outcome (especially in infants), measurement of exposure, rigorous study design, appropriate population selection, and generalizability of the findings are among the issues that are often not adequately addressed. Indoor environments typically include exposures to multiple potentially problematic agents—dust mites and fungi, for example, are ubiquitous. It has proven difficult to assess the individual roles of the factors implicated in existing studies because complete characterization of exposures has not been done. Therefore, it is often not possible to determine with confidence whether any effects noted are indeed the results of specific exposures studied or of confounders.

The poor and inner city residents are vulnerable populations for asthma development, morbidity, and mortality. As such, there is great interest in identifying effective means to address prevalent exposure problems. Although some research on interventions has been directed at these populations, some of the strategies tried may not be practical to implement unless the subjects are part of an organized protocol providing guidance and funds. Further, individuals living in public or rental housing, or in multifamily units, may not have control over parts of their indoor environment that would be desirable to modify, such as carpeting, excessive moisture, and comprehensive pest management. Future research has to address more effectively the feasibility and generalizability of intervention programs on target populations.

Finally, to date there has been little connection between the scientific literature regarding asthma and the scientific literature regarding the characteristics of healthy indoor environments (for example, building design and operation; and sources, transport, control methods, and exposures to indoor pollutants). Relatively little of the existing medical and epidemiologic literature on asthma quantifies indoor environmental conditions such as humidity, ventilation, and pollutant concentrations or exposures in sufficient detail. The effectiveness of exposure limitation strategies in reducing exposures and asthma development or exacerbation has, in general, been inadequately studied. These are areas of research that have the potential to impact public health significantly. The committee believes that better communication between medical, public health, behavioral science, engineering, and building professionals is likely to result in more informed studies on the causes of asthma and the means to limit problematic exposures. The committee encourages efforts to bring these groups together to educate one another on their areas of expertise. Although considerable work has been done and is being done on asthma per se, increased research efforts are needed to address the characteristics of healthy indoor environments. Asthma research clearly needs interdisciplinary involvement—not only of clinicians, immunologists, and researchers in related biologic areas—but also of engineers, architects, materials manufacturers and others who are responsible for the design and function of indoor environments. Collaborations should be fostered, and consideration should be given to formulating model research protocols that include indoor environmental characteristics.

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