

ATSDR Case Studies in Environmental Triggers of Asthma



**U.S. Department of
Health and Human Services**
Agency for Toxic Substances
and Disease Registry

Agency for Toxic Substances and Disease Registry

Case Studies in Environmental Medicine (CSEM)

Environmental Triggers of Asthma

Course: WB 2490

CE Original Date: November 28, 2014

CE Expiration Date: November 28, 2016

Key Concepts

- Asthma is a chronic inflammatory disease of the airways.
 - Over the past decade, the prevalence of asthma in children and adults has increased in the United States.
 - Environmental exposures to allergens, air pollutants, and environmental tobacco smoke, and workplace exposures can cause and exacerbate asthma.
 - Control of environmental exposures can significantly improve the quality of life of persons with asthma.
-

About This and Other Case Studies in

This educational case study document is one in a series of self-instructional modules designed to increase the primary care provider's knowledge of hazardous substances in the environment and to promote the adoption of medical practices that aid in the evaluation and care of potentially exposed patients. The complete series of Case Studies in Environmental Medicine is located on the ATSDR Web site at URL:

<http://www.atsdr.cdc.gov/csem/csem.html> In addition, the downloadable PDF version of this educational series and other environmental medicine materials provides content in an electronic, printable format, especially for those who may lack adequate Internet service.

Acknowledgments

We gratefully acknowledge the work of the medical writers, editors, and reviewers in producing this educational resource. Contributors to this version of the Case Study in Environmental Medicine are listed below.

Please Note: Each content expert for this case study has indicated that there is no conflict of interest that would bias the case study content.

CDC/ATSDR Author(s): Germania A. Pinheiro, MD, MSc, PhD

CDC/ATSDR Planners: Charlton Coles, Ph.D., Sharon L. Hall, Ph.D.; Delene Roberts, MSMHC; Smith, Julia, MPH, CHES; Germania A. Pinheiro, MD, MSc, PhD.

Peer Reviewers: Dan Middleton, MD; Air Pollution and Respiratory Health Branch/National Center for Environmental Health.

How to Apply for and Receive Continuing Education Credit Including the Assessment and Posttest	Visit http://www.atsdr.cdc.gov/csem/conteduc.html for more information about continuing medical education credits, continuing nursing education credits, and other continuing education units. Access the Assessment and Posttest by selecting http://www2a.cdc.gov/TCEOnline/registration/detailpage.asp?res_id=4730
---	--

Accrediting Organization	Credits Offered
---------------------------------	------------------------

Accreditation Council for Continuing Medical Education (ACCME®)	
--	--

	The Centers for Disease Control and Prevention is accredited by the Accreditation Council for Continuing Medical Education (ACCME®) to provide continuing medical education for physicians.
--	---

	The Centers for Disease Control and Prevention designates this educational activity for a maximum of 1.75 AMA PRA Category 1 Credits™. Physicians should only claim credit commensurate with the extent of their participation in the activity.
--	--

American Nurses Credentialing Center (ANCC), Commission on Accreditation	<p>The Centers for Disease Control and Prevention is accredited as a provider of Continuing Nursing Education by the American Nurses Credentialing Center's Commission on Accreditation.</p> <p>This activity provides 1.75 contact hours.</p>
National Commission for Health Education Credentialing, Inc. (NCHEC)	<p>Sponsored by the Centers for Disease Control and Prevention, a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This program is designed for Certified Health Education Specialists (CHES) and/or Master Certified Health Education Specialists (MCHES) to receive up to 1.5 total Category I continuing education contact hours. Maximum advanced level continuing education contact hours available are 0. CDC provider number GA0082.</p>
International Association for Continuing Education and Training (IACET)	<p>The Centers for Disease Control and Prevention has been approved as an Authorized Provider by the International Association for Continuing Education and Training (IACET), 1760 Old Meadow Road, Suite 500, McLean, VA 22102.</p> <p>The Centers for Disease Control and Prevention is authorized by IACET to offer 0.2 IACET CEU's for this program.</p>

Disclaimer and Disclosure

Disclaimer

The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In developing its educational products, ATSDR has made a diligent effort to ensure the accuracy and the currency of the presented information. ATSDR, however, makes no claim that the environmental medicine and health education resources discussed in these products comprehensively address all possible situations related to various substances. The products are intended for educational use to build the knowledge of physicians and other health professionals in assessing the conditions and managing the treatment of patients potentially exposed to hazardous substances. The products are not a substitute for a health-care

provider's professional judgment. Please interpret the environmental medicine and the health education resources in light of specific information regarding the patient and in conjunction with other medical authorities.

Use of trade names in ATSDR products is for identification purposes only and does not imply endorsement by the Agency for Toxic Substances and Disease Registry or the U.S. Department of Health and Human Services.

Disclosure

In compliance with continuing education requirements, all presenters must disclose any financial or other associations with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters as well as any use of unlabeled product(s) or product(s) under investigational use. CDC, our planners, and the presenters for this seminar do not have financial or other associations with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters. This presentation does not involve the unlabeled use of a product or product under investigational use. There was no commercial support for this activity.



U.S. Department of Health and Human Services

Agency for Toxic Substances and Disease Registry

Division of Toxicology and Human Health Sciences

Environmental Medicine Branch

Table of Contents

Initial Check	8
Overview of Asthma.....	13
Differential Diagnosis of Asthma	16
Environmental Triggers of Asthma	20
Clinical Assessment	34
Case Study (Continued)	42
Treatment, Management, and Prevention	45
Sources of Additional Information.....	59
Literature Cited.....	65
Table of Tables.....	76
Posttest.....	76

How to Use This Course

Introduction	The goal of <i>Case Studies in Environmental Medicine</i> (CSEM) is to increase the primary care provider's knowledge of hazardous substances in the environment and to help in evaluation and treating of potentially exposed patients. This CSEM focuses on environmental triggers of asthma.
Availability	Two versions of the Environmental Triggers of Asthma CSEM are available. <ul data-bbox="526 747 1422 1052" style="list-style-type: none">• The HTML version http://www.atsdr.cdc.gov/csem/csem.asp?csem=32&po=0 provides content through the Internet.• The downloadable PDF version provides content in an electronic, printable format, especially for those who may lack adequate Internet service.• The HTML version offers interactive exercises and prescriptive feedback to the user.
Instructions	To make the most effective use of this course <ul data-bbox="526 1167 1422 1556" style="list-style-type: none">• Take the Initial Check to assess your current knowledge about Environmental Triggers of Asthma.• Read the title, learning objectives, text, and key points in each section.• Complete the progress check exercises at the end of each section and check your answers.• Complete and submit your assessment and posttest response online if you wish to obtain continuing education credit. Continuing education certificates can be printed immediately upon completion.
Instructional Format	This course is designed to help you learn efficiently. Topics are clearly labeled so that you can skip sections or quickly scan sections you are already familiar with. This labeling will also allow you to use this training material as a handy reference. To help you identify and absorb important content quickly, each section is structured as follows

Section Element	Purpose
Title	Serves as a “focus question” that you should be able to answer after completing the section
Learning Objectives	Describes specific content addressed in each section and focuses your attention on important points
Text	Provides the information you need to answer the focus question(s) and achieve the learning objectives
Key Points	Highlights important issues and helps you review
Progress Check exercises	Enables you to test yourself to determine whether you have mastered the learning objectives
Progress Check Answers	Provide feedback to ensure you understand the content and can locate information in the text

Learning Objectives Upon completion of the Environmental Triggers of Asthma CSEM, you will be able to

Content Area	Objectives
Overview of Asthma	<ul style="list-style-type: none"> • Define asthma • Identify environmental factors that trigger asthma
Differential Diagnosis of Asthma	<ul style="list-style-type: none"> • Identify five conditions that may be confused with asthma in children • Identify five conditions that may be confused with asthma in adults
Environmental Triggers of Asthma	<ul style="list-style-type: none"> • Identify five indoor triggers of an acute asthma episode • Identify five outdoor triggers of an acute asthma episode • Describe the impact of occupational exposures on adult asthma prevalence

Clinical
Assessment

- Identify the key signs and symptoms of asthma
- Describe questions regarding environmental asthma triggers that should be included in the standard medical history
- Describe pulmonary function test criteria for diagnosing asthma
- Describe questions regarding occupational exposures that should be included in the standard medical history

Treatment,
Management,
and Prevention

- Describe general management goals for patients with asthma
- Describe modifying factors that might affect how environmental triggers cause/exacerbate asthma
- Identify at least three things you can advise patients to do to decrease exposure to allergens and irritants

Initial Check

Instructions

This Initial Check will help you assess your current knowledge about Environmental Triggers of Asthma. To take the Initial Check, read the case below, and then answer the questions that follow.

Case Study

A 12-year-old girl arrives at your office with her mother for an evaluation of the child's cough. The mother reports that the child has a nocturnal nonproductive cough 2 to 3 times per month for the past 3 months associated with increasing episodes of shortness of breath that usually resolve spontaneously. However, during soccer games, the girl has recurrent episodes of cough and wheezing that are only relieved when she uses a friend's albuterol inhaler.

Past medical history reveals that the patient has had recurrent upper respiratory infections and had bronchitis 2 years ago. The patient has had no hospitalizations or emergency department visits. Current medications include diphenhydramine for her intermittent runny nose and an occasional puff from her friend's albuterol inhaler during soccer games.

Family history reveals that the girl lives with her mother, father, and older sister in a house on the outskirts of the community. The father had a history of seasonal hay fever as a child. Both parents are indoor and outdoor smokers. The mother reports that her husband has had some difficulties with episodic cough and shortness of breath, but has not seen a physician.

A review of systems reveals that the patient has numerous episodes of

- Sneezing,
- Itchy eyes, and
- Clear discharge from the nose.

You ask the mother to leave the examination room. This allows you to ask the patient confidentially if she has been smoking or is around friends who smoke. The patient states that neither she nor any of her friends smoke cigarettes or any other inhaled substances, such as marijuana. In addition, the patient has not reached menarche and she denies sexual activity. The patient has met developmental milestones and followed a 50th-percentile growth curve. She is a 7th grader doing well academically, with no school absences.

Physical examination reveals a young girl, who sits quietly and comfortably, in no apparent distress. Her vital signs are

- Temperature 98.6°F (37.0°C),
- Respiratory rate 17,
- Heart rate 82,
- Blood pressure 118/75 millimeter of mercury (mmHg).

No dyspnea or stridor is evident. Her skin color is normal, without cyanosis. Examination of the nares reveals boggy, red turbinates with moderate congestion, but no sinus tenderness or flaring. The tympanic membranes are mobile and without erythema or air/fluid levels. Inspection of the chest does not show accessory muscle use or intercostal, suprasternal, or supraclavicular retractions. The antero-posterior diameter does not seem to be increased. Pulmonary auscultation reveals inspiratory and expiratory wheezing scattered throughout both lung fields. Her peak expiratory flow rate (PEFR) reading is 285 liters per minute (L/min). You explain to the patient and her mother that her predicted normal should be 360 L/min (give or take 20%), which is the predicted normal PEFR for her age and build. The rest of the physical examination is unremarkable. The fingers are not clubbed, nor are the nail beds cyanotic.

Your primary working diagnosis for this patient is asthma.

Initial Check Questions

1. List the primary and differential diagnosis for wheezing in this patient.
2. What are some risk factors for asthma in this patient and her family?
3. What further questions might you ask about other environmental triggers of asthma in this household?
4. What tests would you order to confirm or rule out your primary diagnosis?

Initial Check Answers

1. The differential diagnosis for wheezing in this patient includes
 - Bronchial asthma (primary diagnosis),
 - Exercise induced bronchospasm,
 - Wheezing solely associated with respiratory
-

-
- infections,
- Foreign body aspiration, and
 - Wheezing associated with gastroesophageal reflux.

Less likely diagnoses include

- Cystic fibrosis,
- Immune deficiency,
- Congenital heart disease or congenital malformation causing narrowing of the intrathoracic airways,
- Vocal cord dysfunction and,
- Chronic rhino-sinusitis.

The information for this answer comes from the "Differential Diagnosis of Asthma" section.

2. This family has a history of atopy. Both parents are smokers. The patient has a history of recurrent upper respiratory infections and bronchitis, as well as a suspicion of allergic rhinitis.

The information for this answer comes from the "Differential Diagnosis of Asthma" section.

3. Ask the parent and patient about possible exposures and events that worsen the wheezing. This information should include

- Exacerbation due to upper respiratory illness,
- Relationship of symptoms to specific exposures,
- Exacerbation with exposure to nonspecific triggers such as cigarette smoke, woodstove smoke or household cleaning products,
- Exacerbation due to use of drugs such as aspirin and certain foods and food additives such as sulfites,
- Emotional stress, and
- Seasonal variation in symptoms.

The home environment should be carefully reviewed, focusing on the possible presence of house-dust

mites, indoor fungi (mold), and smoke:

- The patient's environment, particularly within his or her bedroom due to dust mites,
- The presence of furry pets and carpeting,
- Condition of home heating and cooling system,
- Past water damage or leakage,
- Smoking within the home,
- Wood-burning stoves or fireplaces,
- Other irritants (e.g., perfumes, cleaning agents, sprays), and
- Volatile organic compounds (VOCs) such as new carpeting, particle board, painting.

The environment outside the home should be reviewed, including a potential relationship of symptoms and school and recreational activities.

The information for this answer comes from the "Environmental Triggers of Asthma" section.

4. Consider referral to a pulmonologist or allergy/asthma specialist if there is any question about the diagnosis. In the pulmonologist's office, use the measurement of forced expiratory volume in 1 second (FEV₁) before and after short-acting bronchodilator therapy to demonstrate reversible airway obstruction. This should be done by spirometry (for children who are able to cooperate), preferably using American Thoracic Society guidelines [ATS 1995]. Although variability in peak expiratory flow limits its application in screening for asthma, simple peak expiratory flow monitoring in the general practitioner's office can be used. Perform chest radiographs for individuals with systemic symptoms such as fever and signs suggestive of another lung disease. A total immunoglobulin E (IgE) level, an eosinophil count, and a differential count for eosinophils on nasal or sputum secretions may also provide useful information.

The information for this answer comes from the "Clinical Assessment" section.

Overview of Asthma

Learning Objectives

Upon completion of this section, you will be able to

- Define asthma, and
- Identify environmental factors that trigger asthma.

Purpose of This Case Study

This Case Study in Environmental Medicine focuses specifically on the environmental factors that contribute to asthma expression and severity. The goal is to identify those factors, with the hope of moderating or eliminating exposures or reducing their effect.

This case study is not a comprehensive review of asthma, nor a complete review of asthma management. Many excellent texts and articles have covered this topic. The Global Initiative for Asthma (GINA) updated the [Global Strategy for Asthma Management and Prevention](#) in 2011. This document provides recommendations on

- Asthma assessment,
- Monitoring,
- Pharmacotherapy,
- Asthma education and,
- Implementation of asthma guidelines in health care systems.

The NIH Guidelines for Diagnosis and Management of Asthma provide key recommendations about the disease. This Report was developed by a panel convened by the National Asthma Education and Prevention Program (NAEPP), coordinated by the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health. The third and most recent report, Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma (EPR-3) was released in August 2007. The full report is available online at

www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm

Definition of Asthma

The National Heart, Lung, and Blood Institute defines asthma as "...a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular

- Mast cells,
- Eosinophils,
- T lymphocytes,
- Airway Macrophages,
- Neutrophils, and
- Epithelial cells.

In susceptible individuals, this inflammation causes recurrent episodes of

- Wheezing,
- Breathlessness,
- Chest tightness, and
- Coughing,

particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible, either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyper responsiveness to a variety of stimuli" [NHLBI 2007].

Incidence and Prevalence

Allergic diseases such as asthma, rhinitis, and eczema are increasing in prevalence.

- Asthma is a worldwide problem, with an estimated 300 million affected individuals [GINA 2011].
 - The increasing incidence of asthma in many parts of the world continues to make it a global health concern [NHLBI 2003]. Asthma is one of a few diseases in the United States that is increasing in incidence among children and adults. This is despite scientific advances in improving treatment outcomes and understanding the pathogenesis [IOM 2000].
 - During –2008-2010, an estimated 8.2% of the U.S. population had current asthma.
 - Asthma prevalence was higher among Puerto Rican
-

Hispanics (16.1%) and non-Hispanic blacks (11.2%) than among non-Hispanic whites (7.7%).

- Current asthma prevalence was also higher
 - Among children (9.5%) than among adults (7.7%),
 - Among females (9.2%) than among males (7.0%), and
 - Among persons with family income below 100% of the federal poverty threshold (11.2%) than among persons with family income at or above the federal poverty threshold (8.5% for 100% to less than 250% of the poverty threshold, 7.8% for 250% to less than 450% of the poverty threshold, and 6.7% for at or above 450% of the poverty threshold) for the United States [CDC 2012].

Factors That Contribute to Asthma

Environmental factors that contribute to asthma symptoms and severity include

- Viral infections [Gern 2004; Martinez 2003; Lemanske 2003],
 - Allergens, such as
 - Cockroaches,
 - Dust mites,
 - Pollens,
 - Animal dander, and
 - Molds [Murray et al. 2001; Togias 2003; Jaakkola et al. 2005],
 - Environmental tobacco smoke (ETS) (passive and active smoking),
 - Indoor and outdoor air pollution,
 - Occupational sensitizers: more than 300 substances were identified (including many chemical substances) causing occupational asthma or exacerbating its symptoms such as
 - Isocyanates,
 - Platinum salts and
 - Animal biological products [GINA 2011], and
-

-
- Miscellaneous causes such as
 - Exercise,
 - Food allergies,
 - Gastroesophageal reflux,
 - Aspirin, or
 - Other nonsteroidal anti-inflammatory drug (NSAID) sensitivity, sulfite sensitivity, and others [Weinberger 2003; NHLBI 1997].

This Case Study focuses on preventable environmental asthma triggers and measures that may decrease their effects on patients.

Key Points

- Asthma is a chronic inflammatory disease. It is
 - Increasing in prevalence and
 - Triggered by many various environmental factors.

Progress Check

1. Which of the following is not a symptom of asthma
 - A. Wheezing.
 - B. Seizures.
 - C. Shortness of breath.
 - D. Cough.

To review relevant content, see "Definition of Asthma" in this section.

2. Common environmental triggers of asthma include
 - A. Allergens.
 - B. Irritant chemicals.
 - C. Viral infections.
 - D. All of the above.

To review relevant content, see "Factors Which Contribute to Asthma" in this section.

Differential Diagnosis of Asthma

Learning Objective

Upon completion of this section, you will be able to

- Identify five conditions that may be confused with
-

asthma in children, and

- Identify five conditions that may be confused with asthma in adults.

**Differential
Diagnosis in
Adults**

The first step in dealing with the asthma patient is to make sure it is asthma. Although, many cases of recurrent cough and wheezing in children and adults are due to asthma, other conditions are often misdiagnosed as asthma. In adults, the differential diagnosis of asthma includes

- Chronic obstructive pulmonary disease (COPD),
- Congestive heart failure,
- Gastroesophageal reflux disease,
- Mechanical obstruction of the airways (e.g., tumors, foreign bodies), and
- Vocal cord dysfunction.

Infrequent causes of wheezing include

- Pulmonary embolism,
- Pulmonary infiltrates with eosinophilia, and
- Some medications (e.g., angiotensin-converting enzyme (ACE) inhibitors) [NHLBI 2007].

**Differential
Diagnosis in
Children**

In children, chronic cough is a problem, which needs differentiation between asthma and not asthma. Chronic productive cough with purulent sputum is a reason for concern in children and is not usually a symptom of asthma. Nevertheless, respiratory infection presenting purulent sputum can exacerbate asthma in children previously diagnosed with asthma. The younger the child, the greater the need to exclude underlying disease at an early stage [de Jongste and Shields 2003].

Wheezing in children can be an allergic (i.e., asthma) or non-allergic response [Lemanske 2003; Weinberger 2003]. Non-allergic wheezing in children occurs during acute infections, including viral bronchiolitis. Coughing and wheezing in bronchiolitis is difficult to distinguish from asthma. The differential diagnosis of children with frequent respiratory infection and wheezing should include

- Foreign body aspiration causing airway obstruction,
- Pneumonia/bronchiolitis,
- Cystic fibrosis,
- Bronchopulmonary dysplasia (in premature infants),
- Primary ciliary dyskinesia syndrome, and,
- Immune deficiency [NHLBI 2007; GINA 2011].

Table 1. Differential Diagnosis Possibilities for Asthma [NHLBI, 2007; GINA 2011]

Infants and Children	Adults
<p>Upper airway diseases</p> <ul style="list-style-type: none"> • Allergic rhinitis and sinusitis <p>Obstructions involving large airways</p> <ul style="list-style-type: none"> • Foreign body in trachea or bronchus • Vocal cord dysfunction • Vascular rings or laryngeal webs • Laryngotracheomalacia, tracheal stenosis, or bronchostenosis • Enlarged lymph nodes or tumor <p>Obstructions involving small airways</p> <ul style="list-style-type: none"> • Viral bronchiolitis or obliterative bronchiolitis • Cystic fibrosis • Bronchopulmonary dysplasia <p>Other causes</p> <ul style="list-style-type: none"> • Congenital heart diseases • Recurrent cough not due to asthma • Aspiration from swallowing mechanism 	<ul style="list-style-type: none"> • Chronic obstructive pulmonary disease (COPD) • Hyperventilation syndrome and panic attacks • Congestive heart failure • Pulmonary embolism • Laryngeal dysfunction • Mechanical obstruction of the airways (benign and malignant tumors) • Pulmonary infiltration with eosinophilia • Diffuse parenchymal lung diseases • Cough secondary to drugs (ACE inhibitors) • Vocal cord dysfunction

- | | |
|--|--|
| <ul style="list-style-type: none">• Dysfunction or gastroesophageal reflux | |
|--|--|

Key Points

- Many medical conditions may be confused with asthma at initial diagnosis.
- The most common differential diagnoses of asthma among adults include
 - Chronic obstructive pulmonary disease (COPD),
 - Congestive heart failure,
 - Gastroesophageal reflux disease,
 - Mechanical obstruction of the airways (e.g., tumors, foreign bodies), and
 - Vocal cord dysfunction.
- The most common differential diagnoses of asthma among children are
 - Foreign body aspiration causing airway obstruction,
 - Pneumonia/bronchiolitis,
 - Cystic fibrosis,
 - Bronchopulmonary dysplasia (in premature infants), and
 - Primary ciliary dyskinesia syndrome.

Progress Check

3. Conditions that may be confused with asthma in **CHILDREN** include all of the following **EXCEPT**
- A. Foreign body aspiration.
 - B. Enlarged lymph nodes or tumor.
 - C. Hematochezia.
 - D. Gastroesophageal reflux.

To review relevant content, see "Table 1. Differential Diagnosis Possibilities for Asthma" in this section.

4. Conditions which may be confused with asthma in **ADULTS** include all of the following **EXCEPT**
- A. Epistaxis.
 - B. COPD.
 - C. Pulmonary embolism.
-

D. Cough and wheezing secondary to ACE inhibitors.

To review relevant content, see "Table 1. Differential Diagnosis Possibilities for Asthma" in this section.

Environmental Triggers of Asthma

Learning Objectives

Upon completion of this section, you will be able to

- Identify five indoor triggers of an acute asthma episode,
 - Identify five outdoor triggers of an acute asthma episode, and
 - Describe the impact of occupational exposures on adult asthma prevalence.
-

Introduction

Exposure to many environmental factors can trigger and exacerbate asthma. The American Academy of Pediatrics has published a book about childhood environmental health problems, which states: "Avoiding environmental allergens and irritants is one of the primary goals of good asthma management" [AAPCEH 2003].

Medical and nursing education programs often do not fully incorporate environmental questions and an exposure history into asthma management. A recent study reported that, although over half of practicing pediatricians surveyed had seen a patient with health issues related to environmental exposures, fewer than 1/5th were trained in taking an environmental history [Kilpatrick et al. 2002].

This Case Study focuses on

- Allergens such as
 - Pollen,
 - Mold,
 - Animal dander,
 - Insect parts, and
 - Some chemicals
-

-
- Irritants such as
 - Smoke,
 - Dust,
 - Gas or Diesel fumes, and
 - Chlorine

which can trigger or exacerbate an asthma attack in individuals with increased airway hyper responsiveness.

Models of Effect

How an environmental pollutant may affect asthma severity [IOM 2000].

- The pollutant might act as an inciter or trigger, leading to an asthma attack in an individual with hyper-responsive airways.
- The pollutant can exacerbate pre-existing airway inflammation, leading to increased airway hyper-responsiveness, which may persist after exposure ends.
- The pollutant might augment or modify immune responses to inhaled antigens or intensify the effect of other pollutants in the respiratory tract.

Evidence of Effect

Several studies support the importance of allergies and allergens in triggering and exacerbating asthma. Key findings include

- Sensitization to indoor allergens and the spores of outdoor molds is a risk factor for the development of asthma in children and adults.
- In children and adults, sensitive to indoor allergens, the severity of asthma symptoms may vary with the level of exposure.
- Reduction of exposure to house-dust mites has improved Pulmonary Function Tests (PFTs) results and reduction in airway inflammation and hyper-responsiveness in sensitive children and adults [Nelson 2000; Frew 2003a; Simpson and Custovic 2004].

Taken together, these studies make a strong argument for the importance of allergen and irritant exposure as aggravating factors in asthma in both children and adults. The findings reinforce the importance of the identification

and treatment of these exposures.

**Indoor Air
Pollution**

In industrialized countries, adults and children often spend most of their time indoors [Schwab et al. 1992]. Exposure to indoor air pollutants may have a more important effect on childhood asthma than exposure to outdoor air pollutants [IOM 2000; Etzel 2003]. The primary indoor air pollutants associated with asthma exacerbation include [AAPCEH 2003; Jones 2000]

- Biologic allergens (dust mites, cockroaches, animal dander, mold, etc.),
- Environmental tobacco smoke (ETS),
- Irritant chemicals and fumes, and
- Products from combustion devices.

**Biologic
Allergen
Overview**

Biologic allergens can be found throughout the

- Home,
- School, and
- Work and recreational environments

although concentrations of

- Dust mites,
- Cockroaches, and
- Animal dander allergens (pets, mice, rats) vary with geographic location.

However,

- Dust mite allergen,
- Mold, and
- Cat and dog allergens

can be found in most homes, including homes where there are no pets at present [Togias 2003; Weinberger 2003; Nelson 2000].

Dust Mites

Sensitization to house dust mites is an important risk factor for asthma exacerbations and the development of asthma. The dust mite grows optimally at warm temperatures and with humidity greater than 50% in cloth-covered objects such as

- Soft toys,
- Upholstered furniture,
- Bedding,
- Mattresses, and
- Carpets [Sporik et al. 1990; Platts-Mills et al. 1995; Duffy et al. 1998].

Cockroaches

Cockroach allergens also may increase a child's risk of developing asthma [IOM 2000, Etzel 2003].

- Cockroach droppings may be one of the most under appreciated allergens in the indoor environment.
- There have been reports of 36% cockroach sensitization rates in inner-city asthmatic children.
- Children with asthma and cockroach allergy exposed to cockroach allergens have more
 - Wheezing,
 - Missed school days,
 - Emergency room visits, and
 - Hospitalizations

than nonsensitized or nonexposed children [IOM 2000; Rosenstreich et al. 1997].

Cats

Asthma exacerbation among many children with asthma is causally related to cats [IOM 2000].

- The severity of allergic reactions to cats is greater than reactions to other common domestic pets.
- More than 6 million U.S. residents have allergies to cats, and up to 40% of atopic patients demonstrate skin test sensitivity [Wood and Eggleston 1993].
- However, recent studies have shown that the presence of a cat in the house may decrease the risk of *developing* asthma [Platts-Mills et al. 2001; Nafstad et al. 2001].

Other Animals

Dogs, rodents, birds, and other furry or feathered animals may contribute in varying degrees to the animal allergens within the home.

- Dogs may have breed-specific allergens, and are less uniformly allergenic than cats [Lindren et al. 1988].
 - Rodent allergens can come from pets or pests in the home.
 - Birds and feathers have been suggested as allergenic; however, it may be that the dust mites associated with feathers (including feathers in pillows and clothes) are the culprits [IOM 2000].
-

Molds

Exposure to molds may lead to allergic sensitization and may exacerbate asthma or allergic rhinitis [IOM 2004].

- At least 60 species of molds have spores thought to be allergenic [Burge 1989].
- Species of particular concern are
 - Penicillium,
 - Aspergillus,
 - Cladosporium, and
 - Alternaria.

On exposure to these species:

- Nasal congestion,
- Runny nose,
- Conjunctivitis
- Sneezing,
- Lacrimation,
- Wheezing,
- Chest tightness, and
- Shortness of breath may occur.

Among patients studied, children are the most sensitive to mold allergens [Etzel 2003].

Pollens

Exposure to pollens may cause asthma exacerbations. For pollen to be clinically significant, it must be present in significant numbers and be allergenic. Particles $<7\mu\text{m}$ tend to deposit in the airways [Brooks and Bush 2009]. Ragweed pollen is the most common cause of pollen asthma in the United States. Grass and tree pollens are also a concern in many areas worldwide [Shah and Grammer 2012].

Environmental Tobacco Smoke (ETS)

Exposure to environmental tobacco smoke (ETS) is a risk factor for asthma attacks in children [AAPCEH 1997]. Children with asthma and whose parents smoke have more frequent asthma attacks and more severe symptoms [Weitzman et al. 1990; Martinez et al. 1992; Murray and Morrison 1993]. There is clear evidence of an association between exposure to environmental tobacco smoke and the development and exacerbations of asthma. Exposure to ETS also places children at increased risk for

- Sinusitis,
- Otitis media, and
- Bronchiolitis [IOM 2000; Tager et al. 1993].

Combustion Devices

Improperly used or malfunctioning heating devices are a major source of combustion pollutants indoors. Possible sources of contaminants include

- Gas ranges, especially if used for home heating;
- Improperly vented fireplaces;
- Inefficient or malfunctioning furnaces;
- Stoves burning wood, coal, or other biomass; and
- Unvented or improperly vented kerosene or gas space heaters.

The combustion products from these devices include

- Carbon monoxide (CO),
- Nitrogen dioxide (NO₂),
- Particulate matter, and
- Sulfur dioxide (SO₂).

Although CO is a major health concern, it is not an irritating gas and is not likely by itself to exacerbate asthma. In combination, these combustion products will often exacerbate asthma symptoms [AAPCEH 2003].

Chemical Fumes Some building materials and home furnishings off-gas formaldehyde [US EPA 1994]. Formaldehyde may exacerbate asthma in some infants and children [Krzyzanowski et al. 1990]. At sufficient concentrations in the air, cleaning products such as chlorine and ammonia may also trigger reactions.

Miscellaneous Allergens Latex may cause an allergic response either by direct contact or by inhalation of latex particles [Fish 2002]. Symptoms range from skin eruption to bronchospasm and anaphylaxis. Allergic responses around the home may be triggered by

- Gloves,
- Balloons,
- Condoms, and
- Various types of sporting equipment [Landwehr and Boguniewicz 1996].

Outdoor Air Pollution For the last several decades, high levels of outdoor air pollution have been associated with short-term increases in asthma morbidity and mortality [AAPCEH 1993; Ostro et al. 2001; Tolbert et al. 2000]. Specific exposures to outdoor plant allergens such as organic dusts from

- Castor beans,
- Soybeans, and
- Grains

dramatically illustrate this relationship [Etzel 2003]. Asthma has been shown to be caused and triggered by ambient hazardous air pollutants, as well as industrial releases of

- Aldehydes,
- Metals,
- Uncombusted hydrocarbons,
- Isocyanates, and
- Others [Leikauf et al. 1995].

In some communities, hazardous air pollution is associated with noxious odors, and odors can exacerbate symptoms among some people with asthma [Shusterman

1992].

Air pollution has been implicated as one of the factors responsible for the increase in asthma incidence in most industrialized countries [Salvi 2001]. Clinicians should be aware of the common (criteria) air pollutants that may affect asthmatic patients. The National Ambient Air Quality Standards (NAAQS) are set for six criteria pollutants:

- Ozone (O₃),
- SO₂,
- NO₂,
- CO,
- Particulate matter <10 microns (PM₁₀) and particulate matter <2.5 microns (PM_{2.5}).

The standards are designed to protect the health of all susceptible groups, including persons with asthma. The Air Quality Index (AQI, Table 2) provides standardized means of communicating health information associated with daily ambient levels of ground-level O₃, SO₂, NO₂, CO, PM₁₀, and PM_{2.5} (See Appendix 1). For any reported index value greater than 100, the U.S. Environmental Protection Agency (EPA) determines the index number daily and reports the highest of the:

- Index figures,
- Critical pollutant, and
- Specific groups sensitive to the pollutant [US EPA 1999].

Table 2. Air Quality Index

Air Quality Index (AQI) Values	Levels of Health Concern	Colors
<i>When the AQI is in this range:</i>	<i>...air quality conditions are:</i>	<i>...as symbolized by this color:</i>
0 to 50	Good	Green

51 to 100	Moderate	Yellow
101 to 150	Unhealthy for Sensitive Groups	Orange
151 to 200	Unhealthy	Red
201 to 300	Very Unhealthy	Purple
301 to 500	Hazardous	Maroon

[US EPA 2009]

Ozone

Some children with asthma (and some children without asthma) have decreases in lung function after exposure to ozone. In the United States, a large fraction of ambient O₃ is the product of photochemical reactions between

- Various nitrogen oxides (NO_x),
- Volatile organic chemicals (VOCs), and
- Ultraviolet light.

Most of the health effects research on O₃ has focused on the short-term effects, such as reductions in FEV₁ and forced vital capacity (FVC). Levels of O₃ are usually greatest on hot summer days and tend to peak in the late afternoon [Etzel 2003; Spektor et al. 1991].

SO₂

Because of its high solubility, SO₂ mainly irritates the upper airway. The nasal mucosa effectively removes most inspired SO₂ during breathing at rest. Deep penetration to the lung mucosa may occur during moderate exercise. SO₂ has a dose-response association with bronchoconstriction. The amount of SO₂-induced bronchoconstriction is dependent on the level of pre-existing hyper-responsiveness and exercise of the individual. A person without asthma can tolerate a higher concentration of SO₂ before developing symptoms. The bronchoconstrictor response develops within minutes of exposure and resolves within an hour after exposure ends [Ware et al. 1986; Koenig et al. 1990]. The Donora, PA smog disaster and the Great London Smog, in 1948 and 1952, respectively, are examples of environmental disasters related to air pollution, which claimed many

lives.

NO₂

In contrast to the other pollutants, NO₂ is both an indoor and outdoor air pollutant. Indoor sources of NO₂ include

- Malfunctioning gas stoves,
- Furnaces,
- Fireplaces, and
- Kerosene space heaters.

Most NO₂ health effects are believed to be due to long-term, low-level outdoor exposure. Like the other air pollutants, NO₂ increases bronchial responsiveness during exercise. NO₂ decreases lung function in persons with asthma exposed to concentrations above 0.3 ppm, although there is not a clear dose-response relationship. Short-term exposure to high concentrations of NO₂ induces terminal bronchiolar changes and diffuses alveolar injury. Such high concentrations are generally seen only in accidental exposure, as might occur within confined spaces or in an occupational setting [Etzel 2003; Shima and Adachi 2000].

PM₁₀ and PM_{2.5}

Particulate matter is a mixture of solid particles and liquid droplets. Particulate matter <10 microns (PM₁₀) is referred to as "course particulate matter" which may result in lower airway exposure [AAPCEH 2003]. PM₁₀ is the standard measure of particulate air pollution used worldwide. Studies suggest that asthma symptoms can be worsened by increases in the levels of PM₁₀, which is a complex mixture of particle types. PM₁₀ has many components and there is no general agreement regarding which component(s) could exacerbate asthma. However, the inflammatory effects of

- Transition metals,
- Hydrocarbons,
- Ultrafine particles, and
- Endotoxin

all present to varying degrees in PM₁₀—could be important [Donaldson et al. 2000].

Particulate matter <2.5 microns (PM_{2.5}) is referred to as

“fine-particle matter.” Sources of PM_{2.5} include

- Industrial and residential combustion,
- Vehicle exhaust,
- Forest and vegetation fires, and
- Atmospheric reactions between gases (SO₂ and NO_x) and VOCs.

PM_{2.5} penetrates deeper into the lung than does PM₁₀, potentially causing greater adverse health effects [AAPCEH 2003; Schwartz and Neas 2000]. Several recently published community epidemiologic studies associated adverse effects when PM_{2.5} formed a significant portion of the particulate exposure, even though PM₁₀ air concentrations were below NAAQS. Medication use, hospital admissions, and the number of emergency room visits (seen primarily with elderly patients and individuals with cardiopulmonary disease) increased under those conditions [Ware et al. 1986; Dockery et al. 1989].

**Traffic-Related
Pollutants and
Diesel Exhaust**

Exposure to motor traffic emissions can have a significant effect on respiratory function in children and adults.

- Studies show that children living near heavily traveled roadways have significantly higher rates of wheezing and diagnosed asthma [Ciccone et al. 1998].
- Epidemiologic studies suggest that diesel exhaust may be particularly aggravating to children [Brunekreef et al. 1997].
- A child riding in a school bus may be exposed to as much as 4 times the level of diesel exhaust as one riding in a car [NRDC 2001].

**Occupational
Asthma**

Occupational asthma (OA) is defined as a variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace [Friedman-Jimenez et al. 2000, Mapp et al. 2005].

OA is the most frequently reported respiratory occupational disease in industrialized countries [Bang et

al. 2005; NIOSH 2003].

- The annual incidence of OA ranges from 12 to 170 cases per million workers; the estimated mean is 47 cases per million.
- The prevalence of OA is reported at 5% to 15% across many different industries.

The two main types of Occupational Asthma are

- 1) Immunologic: A latency period that varies from months to years is necessary to acquire immunologically mediated sensitization.
 - IgE mediated: Induced by high molecular weight proteins, and some low molecular weight proteins. Chemicals can form allergens by reacting with cells/proteins and produce IgE. It is associated with other signs and symptoms such as rhinitis and urticaria.
 - Non-IgE: Induced by low molecular weight agents
- 2) Non-immunologic: There is no latency period.
 - The disease occurs after exposure to high concentrations of work place irritant.
 - Known as irritant induced asthma it can occur after single or multiple irritant exposures. There is a clear temporal association between inhalation exposure and the rapid onset of asthmatic symptoms.
 - The diagnosis is never made in individuals with preexisting asthma.
 - The most common form is "reactive airway dysfunction syndrome (RADS)" which occurs after exposure to high levels of an irritating vapor, fume, or smoke.
 - The symptoms arise within 24 hrs after high-level exposure to irritant and accompanied by eye/nasal irritant symptoms and increased airway responsiveness [Mapp et al. 2005].

Work-exacerbated asthma (WEA) is defined as preexisting or concurrent asthma that is exacerbated by

workplace exposure. WEA is common with a median prevalence of 21.5% among adults with asthma [Henneberger et al. 2011].

There are well over 300 agents reported to cause OA [Malo and Chan-Yeung 2006], and an equal or greater number of agents and conditions at work can aggravate existing asthma. Diisocyanates are the leading identified cause of OA worldwide [Johnson et al. 2004; Wisnewski et al. 2006].

Some occupational sensitizers and irritants are

- Aldehydes,
- Animal and vegetable proteins,
- Cleaning agents,
- Detergent enzymes,
- Diisocyanates,
- Epoxy glues,
- Flour,
- Hair dressing products
- Latex,
- Platinum salts, and
- Wood dust.

In 2004, the Institute of Medicine concluded that sufficient evidence exists for associating the presence of mold or other agents in damp buildings to

- Nasal and throat symptoms,
- Cough,
- Wheeze, and
- Asthma symptoms in sensitized people with asthma [IOM 2004].

There has been further work indicating that exposure to damp indoor environments containing mold can lead to the development of asthma since this review was published [Cox-Ganser et al. 2005; Jaakkola 2005].

Key Points

- A wide range of indoor and outdoor allergens, irritants, as well as cold temperatures, can exacerbate asthma.
- Household exposures to dust mites and cockroach allergens, and the irritant effects of environmental tobacco smoke, contribute significantly to asthma morbidity.
- Occupational asthma is the most common occupational disease in industrialized countries.
- Allergens or irritants in the work environment may cause occupational asthma or exacerbate asthma in those individuals with this preexisting condition.

Progress Check

5. Risk of asthma may be increased by
- A. Living near a heavily traveled roadway.
 - B. Heavy exercise on a day with an AQI of 130.
 - C. Spending more than 1 hour each day riding a diesel-powered bus.
 - D. All of the above.

To review relevant content, see "Traffic-Related Pollutants and Diesel Exhaust" in this section.

6. The leading cause of occupational asthma is exposure to
- A. Latex.
 - B. Spider mites.
 - C. Diisocyanates.
 - D. Epoxy.

To review relevant content, see "Occupational Asthma" in this section.

Clinical Assessment

Learning Objectives

Upon completion of this section, you will be able to

- Identify the key signs and symptoms of asthma,
 - Describe questions regarding environmental asthma triggers that should be included in the standard medical history,
 - Describe pulmonary function test criteria for
-

diagnosing asthma, and

- Describe questions regarding occupational exposures that should be included in the standard medical history.

Key Elements of Diagnosis

To establish a diagnosis of asthma, the clinician should confirm the following key points:

- Episodic symptoms of airflow obstruction are present,
- Airflow obstruction is at least partially reversible, and
- Alternative diagnoses are excluded.

Recommended mechanisms to establish the diagnosis include

- Detailed medical history,
- Physical exam, and
- Measurements of lung function (spirometry or peak expiratory flow) to assess the severity of airflow limitation, its variability, its reversibility and provide confirmation of the diagnosis of asthma.

Additional studies may be needed to diagnosis asthma in children 5 years and younger, in the elderly, and in workers with suspected occupational asthma.

More tests can also be considered to:

- Evaluate alternative diagnoses,
- Identify precipitating factors,
- Assess severity, and
- Investigate potential complications.

Some cases may require referral to a specialist in asthma care for consultation or treatment [NHLBI 2007].

Medical History The focus of the medical history should be on the presence of any of the following [NHLBI 2007]:

- Cough (particularly worse at night),
- Family history of asthma or allergies,
- Recurrent chest tightness,
- Recurrent difficulty in breathing, and
- Recurrent wheeze.

Note whether symptoms occur or worsen in the presence of [NHLBI 2007]:

- Airborne chemicals or dusts,
- Animals with fur or feathers,
- Changes in weather,
- Dust mites (*i.e.*, in mattresses, pillows, upholstered furniture, carpets, bed linens, stuffed animals; note laundering/cleaning practices involving these items),
- Exercise,
- Menses,
- Mold,
- Pollen,
- Smoke (tobacco, wood),
- Strong emotional expression (laughing or crying hard), or
- Viral infection.

A sample environmental trigger exposure history is included in Appendix 1. This tool was developed by The National Environmental Education and Training Foundation for children and adolescents with asthma [NEETF 2005].

A resource for general exposure history taking is "*Case Studies in Environmental Medicine: Taking an Exposure History*"

<http://www.atsdr.cdc.gov/csem/csem.asp?csem=17&po=0>

Physical Exam

The physical examination should focus on the:

- Upper respiratory tract (rhino-sinusitis, nasal polyps),
- Chest, and
- Skin (eczema) [NHLBI 2007].

Physical findings that increase the probability of asthma include

- Appearance of hunched shoulders,
- Atopic dermatitis/eczema or any other manifestation of an allergic skin condition,
- Chest deformity,
- Hyperexpansion of the thorax (especially in children),
- Increased nasal secretion, mucosal swelling, and nasal polyps,
- Prolonged phase of forced exhalation (typical of airflow obstruction),
- Sounds of wheezing during normal breathing, and
- Use of accessory muscles of respiration (neck, back, and chest).

Note—Wheezing during forced exhalation is not always a reliable indicator of airflow limitation. In mild intermittent asthma, or between exacerbations, wheezing may be absent.

**Pulmonary
Function
Testing**

Spirometry typically measures the maximal volume of air forcibly exhaled from the point of maximal inhalation (forced vital capacity, FVC) and the volume of air exhaled during the first second of the FVC (forced expiratory volume in 1 second, FEV₁). Reduced FEV₁ and FEV₁/FVC values relative to reference or predicted values indicate airflow obstruction.

To help confirm a diagnosis of asthma, take spirometry measurements (FEV₁, FVC, and FEV₁/FVC) before and after the patient inhales a short-acting bronchodilator (200–400 µg salbutamol or albuterol). This helps to determine if the airflow obstruction is reversible over the short term. Spirometry is generally valuable in children over 4 years of age; however, some children cannot

conduct the maneuver adequately until after 7 years of age [NHLBI 2007].

An increase of 12% or more and 200 mL or more in FEV₁ after inhaling a short-acting bronchodilator indicates significant reversibility. A 2 to 3-week trial of oral corticosteroid therapy may be required to demonstrate reversibility. The spirometry measures that establish reversibility may not indicate the patient's best lung function. Lung function abnormalities are categorized as restrictive, obstructive, or mixed respiratory impairment.

A reduced ratio of FEV₁/FVC (*i.e.*, < 65%*) indicates obstruction to the flow of air from the lungs.

A reduced FVC with a normal FEV₁/FVC ratio suggests a restrictive pattern.

FVC < 70% is often taken as the normal cut-off. The normal cut-off is age dependent (falling over time).

The severity of abnormal spirometric measurements is evaluated by comparison of the patient's results with reference values that are based on

- Age,
- Height,
- Race, and
- Sex [NHLBI 2007].

Allergy Testing

For patients with persistent asthma who take daily medications, the clinician should identify allergen exposures and consider using skin testing or *in vitro* testing to assess sensitivity to perennial indoor allergens.

Determination of sensitivity to a perennial indoor allergen is often not possible from a patient medical history alone.

- Susceptible individuals tend to be atopic and will demonstrate an immediate wheal-and-flare skin reaction when prick-tested against various common allergens.
 - Skin testing and *in vitro* laboratory results (*e.g.*, radioallergosorbent test (RAST testing), which
-

determine antigen-specific IgE concentration in serum, must be correctly interpreted and correlated with the patient's history and exam (see Table 3).

- The enzyme-linked immunosorbent assay (ELISA) is also used. The demonstration of IgE antibodies to an allergen demonstrates prior exposure, but does not always prove that the patient's allergic symptoms are related to that specific allergen [NHLBI 2007].

The recommendation to do skin or *in vitro* tests for patients with persistent asthma exposed to perennial indoor allergens will result in a limited number of allergy tests for about half of all asthma patients.

- This is based on the prevalence of persistent asthma and the level of exposure to indoor allergens.
- Skin or *in vitro* tests for patients exposed to perennial allergens are essential to justify the expense and effort involved in implementing environmental controls.
- In addition, patients are less likely to maintain environmental controls (*e.g.*, with regard to pets) without proof of their sensitivity to allergens [NHBLI 2007].

Completely negative skin tests to common allergens are rare in childhood asthma but occur in a substantial proportion of patients with adult-onset asthma [Bonner 1984].

Table 3. Comparison of *In Vivo* vs. *In Vitro* Allergy Testing [NHLBI 1997; NHLBI 2003]

Advantages of Skin Testing	Advantages of RAST and Other <i>In Vitro</i> Test
-----------------------------------	--

<ul style="list-style-type: none"> • Less expensive than <i>in vitro</i> tests • Results are available within 1 hour • More sensitive than <i>in vitro</i> tests • Results are visible to the patient (This may encourage compliance with environmental control measures.) 	<ul style="list-style-type: none"> • Does not require knowledge of skin testing technique • Does not require availability of allergen extracts • No risk of systemic reactions • Can be performed on patients who are taking medications that suppress the immediate skin test (antihistamines, antidepressants) • Can be done for patients with extensive eczema
--	--

Diagnosis and Evaluation of Occupational Asthma

The adult patient's occupational history is the key diagnostic tool.

- In addition, lung function assessments that include spirometry and bronchial responsiveness are often coupled with immunological assessment and an evaluation of inflammation in the investigation of occupational asthma.
 - Evaluations may include serial peak expiratory flow rate (PEFR) measurements and nonspecific hypersensitivity challenges with histamine or methacholine.
 - Serial PEFR monitoring while at work and away from work may be important in documenting whether asthma is work-related in selected people, work-environment permitting [Malo and Chan-Yeung 2001].
 - Information about workplace exposures to irritants and sensitizers may be useful.
 - Specific challenge testing at tertiary referral centers providing specialized laboratories can also be helpful [Rabatin and Cowl 2001] but is rarely necessary and may create unnecessary risk.
 - Immunological skin tests may be useful to document immunological sensitization. Induced sputum analysis has been found useful in the assessment of OA [Obata et al. 1999, Lemiere 2004].
-

Key Points

- The medical history should include a set of standard questions addressing factors that worsen the patient's asthma symptoms.
- Asthma is an episodic disease. Physical findings may vary dramatically with time.
- Spirometry measurements before and after a short-acting bronchodilator are extremely helpful in the diagnosis of asthma.
- For those patients with persistent asthma who take medications daily, the clinician should consider using skin testing or *in vitro* testing to assess sensitivity to perennial indoor allergens.
- Occupational asthma is the most prevalent form of work-related lung disease in industrialized nations.

Progress Check

7. Medical history questions about environmental asthma triggers should include

- A. Tobacco smoke.
- B. Pets.
- C. Bedding and laundering practices.
- D. All of the above.

To review relevant content, see "Medical History" in this section.

8. An individual who increases his/her FEV₁ by more than 12% after inhaling a short-acting bronchodilator probably has asthma

- A. True.
- B. False.

To review relevant content, see "Pulmonary Function Testing" in this section.

9. The key diagnostic tool in occupational asthma is

- A. Complete blood count.
- B. Chest X-ray.
- C. The occupational exposure history.
- D. Arterial blood gases.

To review relevant content, see "Diagnosis and

Evaluation of Occupational Asthma” in this section.

10. It is possible to make the diagnosis of asthma without detectable wheezing
- A. True.
 - B. False.

To review relevant content, see “Physical Exam” in this section.

Case Study (Continued)

Case Study (Continued)

A review of the exposure history for the 12-year-old reveals that:

- The family has a long-haired cat that stays in the house,
- The patient develops nasal congestion and chest tightness when playing with the pet,
- The central heating furnace filters have not been cleaned in the last year,
- Wall-to-wall carpet is present throughout the house,
- The home has a wood-burning fireplace, which is occasionally used,
- The shower areas of the bathrooms have some mold, and
- Both parents smoke cigarettes indoors, but do not smoke in the children’s rooms.

In addition, the patient’s mother states that:

- She vacuums regularly,
- She has not seen any insects in the house, and
- The basement is not damp.

The patient with asthma symptoms underwent peak flow testing in your office. The results demonstrated a 24% increase in peak expiratory flow rate (PEFR) after administration of a short-acting B₂-agonist bronchodilator. The patient is diagnosed with mild persistent asthma.

Anti-inflammatory therapy consisting of a corticosteroid metered-dose inhaler (MDI) for daily use and a short-acting B₂-agonist MDI for symptomatic relief is given to the patient; she is instructed on use of the MDIs with the spacer. The patient uses the spacer in front of you to demonstrate that she understands its proper use. You explain that it might take 7 days or more for the corticosteroid inhaler to be effective. You also explain that the goal is to control the asthma with the corticosteroid inhaler and decrease use of the short-acting B₂-agonist for rare breakthrough of acute asthma symptoms. A return visit in 2 to 3 weeks is scheduled.

You tell the mother:

- Both parents should stop smoking or, at a minimum, not smoke in the house or the car.
 - Clean or replace the furnace filter on a regular basis.
 - The cat should not be indoors.
 - Remove the wall-to-wall carpeting from the patient's bedroom (and preferably the whole house if feasible).
 - Encase mattresses and pillows in sealed plastic covers.
 - Wash all bedding materials in hot water (>130°F [>55°C]) to kill dust mites.
 - Water heaters in homes with young children are frequently set at or below 120°F (50°C) to avoid scalding. Suggest that the mother turn the water heater up for short periods to provide the necessary water temperature for washing bedding and area rugs.
-

**Case Study
Follow-Up**

A few weeks later, the father brings his daughter in for her follow-up assessment. The child's cough has subsided and she is able to sleep through the night. The child has been using the short-acting B₂-agonist and corticosteroid inhaler as directed. For the last week, she has not required additional use of the short-acting B₂-agonist. The father relates that his daughter has been more active lately and plays soccer without episodes of shortness of breath. Auscultation of the lungs reveals that both fields are clear without wheezes. You decide to maintain the current medication treatment regimen. The father has an audible wheeze and an intermittent cough. He is wearing his factory work clothes and you smell a strong chemical odor coming from him. You reiterate that both parents should stop smoking.

Progress Check

11. Which is **NOT EXPECTED** to be an environmental trigger of asthma in this home?
- A. Cats.
 - B. Environmental tobacco smoke.
 - C. Mold in the bathroom.
 - D. Lead paint around window sills.

To review relevant content, see "Case Study (continued)" in this section.

12. You learn from the girl's father that his place of employment has poor ventilation and no provision for respiratory protection, shower facilities, or changing his work clothes. What advice could you give the girl's father regarding his current work practices?
- A. He should quit his job immediately.
 - B. Don't worry, all facilities regulated by OSHA are safe.
 - C. Wear a disposable dust mask at work and you should have no problems.
 - D. Contact his occupational health clinic or health professional per worksite protocol for evaluation.

To review relevant content, see "Case Study Follow-

Up" in this section.

Treatment, Management, and Prevention

Learning Objectives

Upon completion of this section, you will be able to

- Describe general management goals for patients with asthma,
 - Describe modifying factors that might affect how environmental triggers cause/exacerbate asthma, and
 - Identify at least three things you can advise patients to do to decrease exposure to allergens and irritants.
-

Treatment and Management Overview

This Case Study discusses the role environmental factors play in

- **Causing,**
- **Triggering, and**
- **Exacerbating asthma.**

This Case Study does not comprehensively review asthma treatment and management.

The treatment and management of environmental asthma follow the guidelines set forth by the National Heart, Lung, and Blood Institute, with special emphasis on the management of the patient's environment [NHLBI 2007].

Pharmaceutical intervention forms the basis of asthma treatment. Asthma medications are generally categorized as

- Relievers: medications used on an as-needed basis that act quickly to reverse bronchoconstriction and relieve symptoms or
- Controllers: medications taken daily on a long-term basis to keep asthma under clinical control mainly through their anti-inflammatory effects.

A stepwise approach is taken for the long-term management of asthma after confirming the diagnosis and assessing the severity of disease [NHLBI 2007, GINA

2011].

Goals for the general management of a patient with asthma include

- Preventing chronic asthma symptoms and exacerbations (day and night),
- Maintaining the patient's "normal" activity (including exercise and other physical activities),
- Regaining and maintaining normal or near-normal lung function, and
- Prescribing optimal pharmacotherapy with minimal or no adverse effects.

Management includes careful monitoring of the patient's response to treatment and appropriate adjustments. It also includes educating the patient and family regarding primary and secondary preventive measures [NHLBI 2007; GINA 2011].

Predisposing Factors

Atopy, the genetic predisposition for the development of an immunoglobulin E (IgE)-mediated response to common airborne allergens, is the strongest identifiable predisposing factor for developing asthma [NHLBI 1997; Holla et al. 2002; NHLBI 2007; Busse and Rosenwasser 2003].

Most children with asthma have allergic rhinitis, a major independent risk factor for asthma. Rhinitis and asthma can be viewed as manifestations of one syndrome—the chronic allergic respiratory syndrome—in different parts of the respiratory tract [Togias 2003].

Certain immune system components, such as the T-helper phenotype, are determined in the first year of life by environmental exposure to respiratory infections or environmental allergens in genetically predisposed individuals [Robinson et al. 2004; Luft et al. 2004; Larche et al. 2003; Umetsu et al. 2003].

Exposure to Allergens and Risk of Asthma

Studies of exposure to allergens and risk of asthma have yielded paradoxical results.

- Exposure to some pets appears to increase the risk of asthma and wheezing in older children, yet lower the risk among young children [Apelberg et al. 2001].
 - House dust mite and cockroach allergens appear to have a positive linear relationship, whereas cat allergens appear to act quite differently, with maximum sensitization developing at moderate exposure levels.
 - Very low levels of cat allergen exposure are likely to induce no response; very high levels are likely to develop a form of tolerance [Murray et al. 2001].
 - Decreased exposure to infections and allergens in early childhood has been linked to the increased incidence of asthma in industrialized countries (the "hygiene hypothesis") [Liu and Murphy 2003].
-

Hygiene Hypothesis

The hygiene hypothesis of asthma states that naturally occurring infections and allergen exposures might essentially immunize against the development of asthma and allergic and autoimmune diseases. The modern emphasis on cleanliness or “sanitizing the environment” may have reduced this natural immunotherapy over the past century and might be a factor in the global increase of these conditions [Liu and Murphy 2003]. The differences in health outcomes from exposure are due to important moderating variables, such as

- Age of exposure,
- Timing of exposure relative to disease development,
- Dose and frequency of exposure,
- Co-exposures, and
- Genetic predispositions in response [Song and Liu 2003].

Growing up on a farm may protect against developing asthma and allergic rhinoconjunctivitis [Von Essen 2001]. An important study in 2002 showed that exposure of young children to older children at home or to other children in childcare settings protects against the development of asthma and frequent wheezing later in childhood [Ball et al. 2000].

**Primary
Prevention
Strategies in
Children**

Well-documented primary prevention strategies for asthma.

Avoid smoking and environmental tobacco smoke (ETS).

For children, studies indicate that in utero exposure to tobacco smoke products is an important predictor of wheezing within the first year of life. Exposure to ETS places children at increased risk for the development and exacerbation of asthma as well as

- Sinusitis,
- Otitis media,
- Bronchiolitis, and
- Diminished pulmonary function.

Both in utero and passive (environmental) tobacco smoke exposure adversely affect pulmonary function, and predispose to asthma symptoms and possibly bronchial hyper responsiveness in childhood. Exposure to tobacco smoke products in utero is a risk factor for wheezing in the first year of life [Tager et al. 1993]. Children who have asthma and whose parents smoke have more frequent asthma attacks and more severe symptoms [Weitzman et al. 1990; Martinez et al. 1992].

Avoid exposure to insect allergens.

House dust mite and cockroach allergens have a very close association between exposure and the sensitization of an individual [Murray et al. 2001].

Avoid exposure to molds.

Exposure to mold in homes as much as doubles the risk of asthma development in children [Jaakkola et al. 2005].

Breast-feed infants.

A study demonstrated that exclusively breastfeeding for the first 4 months is associated with a statistically significant decrease in the risk of asthma and wheezing in children until the age of 6 years [Dell and To 2001].

**Primary
Prevention in
Adults**

In adult-onset asthma, primary prevention relies mainly on smoking cessation and control of workplace exposures. Studies of factory workforces in the past decade have provided consistent evidence of exposure-response relationships for both sensitization (IgE production) and asthma [Taylor 2001; Jeebhay et al. 2001].

New-onset occupational asthma may be immunological or nonimmunological in origin. The immunologic variants are usually caused by high molecular-weight allergens such as grain dust and animal or fish protein. Symptoms may take months or years to develop.

A brief, high-level exposure to a strong irritant can precipitate nonimmunologic occupational asthma. Symptoms occur immediately or within a few hours of the exposure. Multiple lower level exposures to an irritant can also cause asthma. The worker should be removed from further exposure once the diagnosis of occupational asthma is established, whether immunologic or nonimmunologic in origin. Continued exposure to sensitizers or irritants following sensitization may cause persistent problems that can lead to permanent impairment. In addition, once sensitized, individuals may have a substantial response to extremely low levels of sensitizers or irritants. If the diagnosis is made in a timely fashion and steps are taken to stop exposure, most workers experience improvement. Prevention is the best therapeutic intervention [Bardana 2003].

Avoidance of exposure to occupational irritants and allergens is the mainstay of primary prevention. Especially notorious for producing occupational asthma are jobs that use

- Isocyanates,
- Enzymes, or
- Latex.

Prospective surveillance can detect the development of specific IgE antibody before the onset of allergic symptoms. This allows continuing interventions to reduce exposures and minimize or eliminate those associated with symptoms. Workers with IgE to specific allergens

can continue to work in the industry symptom-free for their entire careers. This indicates that exposures needed to induce sensitization are different and probably lower than exposures needed to elicit allergic symptoms [Wisnewski et al. 2006; Sarlo and Kirchner 2002].

Secondary Prevention in Children and Adults

Patients can take a number of steps to reduce or avoid exposure to:

- Pollutants,
- Irritants, and
- Allergens

that may trigger or exacerbate asthma episodes [Williams et al. 2003; AAPCEH 2003]. The National Environmental Education and Training Foundation outlined possible preventive measures in Environmental Management of Pediatric Asthma: Guidelines for Health Care Providers. Summarized below are those environmental intervention guidelines [NEETF 2005]. It is important to note that no single intervention will likely achieve sufficient benefits to be cost effective and that a comprehensive environmental intervention may be needed to improve asthma-associated morbidity [GINA 2011].

Dust Mites

No matter how clean the home is, dust mites cannot be eliminated. However, household interventions can decrease exposure to dust mites and possibly reduce asthma exacerbations [Ehnert et al. 1992]. Cleaning with a high-efficiency particulate air (HEPA) filter vacuum is particularly effective in removing allergens and thus decreasing asthma symptoms [McDonald et al. 2002; Platts-Mills et al. 2001].

Listed below are recommended steps to reduce dust mites in the home [NEETF 2005].

- Remove carpet from bedrooms.
 - Use an air conditioner or dehumidifier to reduce household humidity.
 - Remove upholstered furniture.
 - Replace draperies with blinds or other wipeable window coverings.
 - Encase pillow and mattress in allergen impermeable cover.
-

-
- Remove humidifiers.
 - Replace wool or feathered bedding with synthetic materials that will withstand repeated hot water washing.
 - Use a damp mop or rag to remove dust (a dry cloth just stirs up mite allergens).
 - Vacuum regularly using a cleaner with a HEPA filter or a double-layered microfilter bag (try not to vacuum when the asthmatic is in the room).
 - Wash and thoroughly dry stuffed toys weekly in hot water, or freeze them weekly.
 - Wash bedding in hot water (at least 130°F) weekly.

Animal Allergens

Modifications to the home environment can significantly reduce animal allergens and the frequency of asthma episodes [Williams et al. 2003]. The following steps can reduce exposure to animal allergens.

- Find a new home for indoor cats, dogs, and pet rodents that have caused allergy symptoms.
- Keep pets outside.
- Select low-dander pets in place of those with fur or feathers.

If those options are not possible, the following steps **may** help reduce exposure.

- Keep pets out of the bedroom.
- Enclose mattresses and pillows in zippered plastic cases.
- Remove carpets.
- Vacuum regularly using a cleaner with a HEPA filter or a double-layered microfilter bag (try not to vacuum when the asthmatic is in the room).
- Use a portable air cleaner with HEPA filter for the child's bedroom.
- Keep pets off furniture.

Cockroach Allergen

The first step in limiting cockroach allergens is to keep the house clean and in good shape [O'Connor and Gold 1999]. In general, use the least hazardous methods of roach control first.

Food

- Clean up all food items and crumbs.
- Limit spread of food around house, especially bedrooms.
- Restrict food consumption to the kitchen and dining room.
- Store food (including pet food) in closed containers.

Hygiene and maintenance

- Fix water leaks under sinks.
- Mop the kitchen floor and clean countertops at least once a week.
- Check for and plug crevices outside your house that cockroaches may enter.
- Caulk or patch holes in walls, cupboards, and cabinets.

Pest management

- Use the integrated pest management (IPM) approach for least toxic extermination methods first.
 - Use boric acid powder under stoves and other appliances.
 - Use bait stations and gels.
 - Use outdoor treatments as much as possible to prevent insects from entering your house.
 - If those steps are unsuccessful, seek help from a professional, licensed exterminator rather than spraying chemicals yourself.
 - Stay away from the house for several hours after pesticides are applied.
 - Avoid using liquid sprays inside the house, especially near places children crawl, play, or sleep.
-

Mold and Mildew Mold spores are allergens found indoors and outdoors. Outdoor molds are present year-round throughout the West (lower altitudes) and South, and in the North during the fall. Outdoor molds in the North generally peak in late summer. There is no definite seasonal pattern to molds that grow indoors. Moisture control is the key step in limiting indoor mold growth [Krieger and Higgins 2002].

Tips to help keep exposure to mold spores as low as possible.

- Use air-conditioning to cool the house; evaporative coolers are not recommended.
 - When first turning on home or car air-conditioners, leave the room or drive with the windows open for several minutes to allow mold spores to disperse.
 - Use a dehumidifier or air-conditioner (non-evaporative or water-filled type) to maintain relative humidity below 50%.
 - Do not use a humidifier.
 - Check faucets, pipes, and ductwork and repair any that are leaking.
 - Clean mold with chlorine solution diluted 1:10 with water.
 - Do not install carpet and wallpaper in rooms prone to dampness.
 - Leave a light on inside a closet that has mold in it to dry the air.
 - Install and use exhaust fans in the kitchen, bathrooms, and damp areas.
 - Vent bathrooms and clothes dryers to the outside.
 - Remove decaying debris from the yard, roof, and gutters.
 - Avoid raking leaves, mowing lawns, or working with peat, mulch, hay, or dead wood if you are allergic to mold spores.
-

Environmental Tobacco Smoke

Cigarette smoke contains many toxic chemicals and irritants. Approximately 42% of children 2 months to 11 years of age live in a home with at least one smoker [Pirkle et al. 1996]. Children exposed to tobacco smoke have increased asthma exacerbations. Studies suggest that asthma symptoms may be less severe for asthmatic children if parents expose them to less cigarette smoke [Murray and Morrison 1993]. Complete cessation of indoor smoking in the homes of children with asthma may be needed to achieve significant health improvement [Lodrup and Carlsen 2001]. The following are the most important preventive strategies to reduce exposure to environmental tobacco smoke.

- Keep your home and car smoke-free.
 - If you smoke, do not smoke near children or other nonsmokers.
 - Seek support to quit smoking; consider aids such as nicotine gum, patch, and medication from your doctor to help you in quitting.
 - Change clothes after smoking while you are in the process of cutting down on the number of cigarettes.
 - Choose smoke-free childcare and social settings.
 - Seek smoke-free environments in restaurants, theaters, and hotel rooms.
-

Indoor Air Pollution

For **indoor air pollution**, the two **best approaches to reducing indoor air pollution** are **source control** and **ventilation**. Listed below are specific steps for improving indoor air quality.

- Limit use of products and materials that emit strong odors and irritants, such as
 - Air freshener sprays,
 - Chalk dust,
 - Cleaning products,
 - Hair sprays,
 - Insect sprays.
 - Paint fumes,
 - Sawdust,
 - Smoke,
 - Strong perfumes, and
 - Talcum powder.

 - Moderate indoor humidity and moisture (relative humidity between 35-50%).
 - Use good housekeeping practices to reduce the presence of airborne particles.
 - Install an exhaust fan close to the source of airborne contaminants or odors and vent it to the outside.
 - Properly ventilate the room in which a fuel-burning appliance is used.
 - Ensure that the doors of wood-burning stoves fit tightly.
 - Follow manufacturer's instructions when using an unvented kerosene or gas space heater.
 - Ensure fireplaces are properly vented so smoke escapes through the chimney.
 - Never use a gas-cooking appliance as a heating source.
 - Open windows, especially when pollutant sources are in use (this option must be balanced against the concern of mold allergy or other plant allergens and outdoor air pollution).
-

Outdoor Air Pollution

Outdoor air pollution, especially ozone and particulate matter, can increase asthma symptoms. Ways to limit exposure to outdoor air pollution.

- Monitor air quality and pollen levels and keep children indoors when pollutants are high.
- Avoid sustained contact with vehicle exhaust emissions and diesel fumes (such as student exposure to idling school buses).
- Use High-Efficiency Particulate Air (HEPA) filters in household vents.
- If possible, move to a less polluted location.
- Schedule outdoor activities for times when ozone levels are lowest, typically in the morning

Desensitization

For some cases, consider desensitization—especially if environmental control fails to decrease asthma exacerbations.

- Specific immunotherapy involves the administration of allergen extracts to achieve clinical tolerance of the allergens that cause symptoms in patients with allergic conditions.
- Immunotherapy can be effective in patients with mild forms of allergic disease, and in those who do not respond well to standard drug therapy.
- Effects of specific immunotherapy take longer to manifest, but once established, specific immunotherapy may give long-lasting relief of allergic symptoms, whereas the benefits of drugs only last as long as they are continued [Frew 2003b; Nelson 2003].

Key Points

- Every practitioner who treats asthma patients should have general goals for management.
- Environmental triggers can cause or exacerbate asthma.
- Patients can take a number of steps to reduce or avoid exposure to the pollutants, irritants, and allergens that may trigger or exacerbate asthma episodes.

Progress Check

13. Your overall treatment, management, and prevention

goals might include

- A. Confirmation of asthma diagnosis and gauge of severity.
- B. Optimal pharmacotherapy with minimal or no adverse effects.
- C. Education of the patient and family regarding primary and secondary preventive measures, including smoking cessation.
- D. All of the above.

To review relevant content, see "Treatment and Management Overview" in this section.

14. Cockroaches in the home should always be treated with a pesticide
- A. True.
 - B. False.

To review relevant content, see "Cockroach Allergen" in this section.

15. Important moderating factors affecting how environmental exposures may exacerbate asthma include
- A. Age and timing of exposure relative to disease development.
 - B. Dose and frequency of exposure.
 - C. Genetic predispositions in response and co-exposures.
 - D. All of the above.

To review relevant content, see "Hygiene Hypothesis" in this section.

16. Some advice you can give to patients to decrease exposure to allergens or irritants in the home include:
- A. Cover mattresses and pillows with zippered plastic cases.
 - B. Avoid smoking and environmental tobacco smoke.
-

-
- C. Remove wall-to-wall carpets, particularly in bedrooms.
 - D. All of the above.

To review relevant content, see "Secondary Prevention in Children and Adults" in this section.

Sources of Additional Information

Asthma Specific Information

Please refer to the following Web resources for more information on the adverse effects of asthma, the treatment of asthma-associated diseases, and management of persons with asthma.

- Agency for Toxic Substances and Disease Registry (ATSDR) <http://www.atsdr.cdc.gov>
 - For chemical, emergency situations
 - CDC Emergency Response: 770-488-7100 and request the ATSDR Duty Officer
 - For chemical, non- emergency situations
 - CDC-INFO <http://www.cdc.gov/cdc-info/>
 - 800-CDC-INFO (800-232-4636) TTY 888-232-6348 - 24 Hours/Day
 - Email: cdcinfo@cdc.gov

PLEASE NOTE

ATSDR cannot respond to questions about **individual medical cases**, provide second opinions or make specific recommendations regarding therapy. Address these issues directly with your health care provider.

- Centers for Disease Control and Prevention
 - National Center for Environmental Health
 - Potentially effective interventions for asthma <http://www.cdc.gov/asthma/interventions/>
 - Environmental Hazards and Health Effects – Asthma
-

<http://www.cdc.gov/asthma/default.htm>

- National Asthma Control Program
<http://www.cdc.gov/asthma/nacp.htm>
 - Key Clinical Activities for Quality Asthma Care.
<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5206a1.htm>
 - NIOSH
 - Safety and Health Topics – Asthma and Allergies
<http://www.cdc.gov/niosh/topics/asthma/>
 - National Center for Chronic Disease Prevention and Health Promotion; Healthy Youth Health Topics – Adolescent and School Health
<http://www.cdc.gov/HealthyYouth/asthma/>
 - National Institute of Environmental Health Sciences
 - Asthma and allergy prevention
<http://www.niehs.nih.gov/health/topics/conditions/asthma/>
 - National Institutes of Health – National Heart, Lung and Blood Institute
 - National Asthma Education and Prevention Program
<http://www.nhlbi.nih.gov/about/org/naepp/>
 - Asthma Web Sites and Interactive Web Applications
<http://www.nhlbi.nih.gov/health/prof/lung/index.htm#asthma>
 - Guidelines for the diagnosis and management of asthma
<http://www.nhlbi.nih.gov/files/docs/guidelines/asthgdln.pdf>
 - U. S. Environmental Protection Agency
 - Asthma Home Related Checklist
-

<http://permanent.access.gpo.gov/websites/epago/v/www.epa.gov/asthma/resources.html>

- Asthma-related Publications and Resources
 - Global initiative for asthma: global strategy for asthma management and prevention.
<http://www.ginasthma.com>.
- The National Environmental Education and Training Foundation
 - Pediatric Asthma Initiative
<http://www.neefusa.org/health/asthma/index.htm>
- American Academy of Allergy, Asthma and Immunology
 - Online courses on asthma available at no cost from the Continuing Education Center
<http://education.aaaai.org/courses>
- American Academy of Pediatrics
<http://www.healthychildren.org/English/health-issues/conditions/allergies-asthma/Pages/Asthma.aspx>
- Bernstein DI, Chan-Yeung M, Malo J-L, Bernstein IL, eds. 2006. Asthma in the Workplace and Related Conditions, 3rd Edition. New York: Taylor and Francis
- Etzel RA. 2003. How environmental exposures influence the development and exacerbation of asthma. Pediatrics 112(1):233–9
http://pediatrics.aappublications.org/content/112/Supplement_1/233.full

General Environmental Health Information

Please refer to the following Web resources for general information on environmental health.

- Agency for Toxic Substances and Disease Registry
<http://www.atsdr.cdc.gov>
 - Taking an Exposure History CSEM
-

<http://www.atsdr.cdc.gov/csem/csem.asp?csem=17&po=0>

- Taking a Pediatric Exposure History CSEM
<http://www.atsdr.cdc.gov/csem/csem.asp?csem=26&po=0>
 - To view the complete library of CSEMs
<http://www.atsdr.cdc.gov/csem/csem.html>
 - Exposure History Worksheet
<http://www.atsdr.cdc.gov/csem/csem.asp?csem=17&po=19>
 - ATSDR Division of Regional Operations.
 - Through the working relationships they have established with EPA, other federal and state agencies, individual citizens, and community groups, regional representatives are able to maintain current and historic knowledge of the sites and issues in their regions.
 - ATSDR's Regional Offices, along with the states and territories that they cover as well as contact information, can be found at http://www.atsdr.cdc.gov/DRO/dro_contact.html
 - ATSDR State Cooperative Agreement Program
<http://www.atsdr.cdc.gov/states/index.html>
 - The Cooperative Agreement Program provides essential support in communities nationwide to fulfill the mission of the Agency for Toxic Substances and Disease Registry (ATSDR).
 - The program funds 30 states and one tribal government to develop and strengthen their abilities to evaluate and respond to environmental public health issues.
 - Centers for Disease Control and Prevention (CDC)
<http://www.cdc.gov>
 - CDC works to protect public health and the safety of people, by providing information to enhance health decisions, and promotes health through partnerships with state health departments and other organizations.
-

-
- The CDC focuses national attention on developing and applying disease prevention and control (especially infectious diseases), environmental health, occupational safety and health, health promotion, prevention and education activities designed to improve the health of the people of the United States.
 - National Center for Environmental Health (NCEH) <http://www.cdc.gov/nceh>
 - NCEH works to prevent illness, disability, and death from interactions between people and the environment. It is especially committed to safeguarding the health of populations that are particularly vulnerable to certain environmental hazards - children, the elderly, and people with disabilities.
 - NCEH seeks to achieve its mission through science, service, and leadership.
 - National Institute of Health (NIH) <http://www.nih.gov>
 - A part of the U.S. Department of Health and Human Services, NIH is the primary Federal agency for conducting and supporting medical research.
 - National Institute of Occupational Safety and Health (NIOSH) <http://www.cdc.gov/niosh/>
 - NIOSH is in the U.S. Department of Health and Human Services and is an agency established to help assure safe and healthful working conditions for working men and women by providing research, information, education, and training in the field of occupational safety and health.
 - American College of Occupational and Environmental Medicine (ACOEM) <http://www.acoem.org/>
 - ACOEM is the nation's largest medical society dedicated to promoting the health of workers
-

through preventive medicine, clinical care, research, and education.

- Its members are a dynamic group of physicians encompassing specialists in a variety of medical practices united via the College to develop positions and policies on vital issues relevant to the practice of preventive medicine both within and outside of the workplace.
 - American College of Medical Toxicologists (ACMT) <http://www.acmt.net>
 - ACMT is a professional, nonprofit association of physicians with recognized expertise in medical toxicology.
 - The College is dedicated to advancing the science and practice of medical toxicology through a variety of activities.
 - American College of Preventive Medicine (ACPM) <http://www.acpm.org>
 - ACPM is the national professional society for physicians committed to disease prevention and health promotion.
 - ACPM's 2,000 members are engaged in preventive medicine practice, teaching and research.
 - Association of Occupational and Environmental Clinics (AOEC) <http://aoec.org>
 - AOEC is a network of more than 60 clinics and more than 250 individuals committed to improving the practice of occupational and environmental medicine through information sharing and collaborative research.
 - Pediatric Environmental Health Specialty Units (PEHSUs) <http://www.pehsu.net>
 - The PEHSUs have been developed to provide education and consultation for health professionals, public health professionals and others about the topic of children's environmental
-

health.

- The PEHSU staff is available for consultation about potential pediatric environmental health concerns affecting both the child and the family. Health care professionals may contact their regional PEHSU site for clinical advice.
- Poison Control Center
 - The American Association of Poison Control Centers can be contacted for questions about poisons and poisonings. The web site provides information about poison centers and poison prevention. AAPC does not provide information about treatment or diagnosis of poisoning or research information for student papers.
 - American Association of Poison Control Centers may be contacted at 1-800-222-1222 or <http://www.aapcc.org>

Literature Cited

References

[AAPCEH] American Academy of Pediatrics Committee on Environmental Health. 2003. Etzel RA, editor. Pediatric environmental health. 2nd ed. Elk Grove Village IL: American Academy of Pediatrics.

[AAPCEH] American Academy of Pediatrics Committee on Environmental Health. 1997. Environmental tobacco smoke: a hazard to children. *Pediatrics* 99:639–42.

[AAPCEH] American Academy of Pediatrics, Committee on Environmental Health. 1993. Ambient air pollution: respiratory hazards to children. *Pediatrics* 91:1210–3.

[ATS] American Thoracic Society. 1995. Standardization of spirometry 1994 update. *Am J Respir Crit Care Med* 152:1107–1136.

Apelberg BJ, Aoki Y, Jaakkola JJ. 2001. Systematic review: exposure to pets and risk of asthma and asthma-like symptoms. *J Allergy Clin Immunol*

107(3):455–60.

Ball TM, Castro-Rodriguez JA, Griffith KA, Holberg CJ, Martinez FD, Wright AL. 2000. Siblings, day-care attendance, and the risk of asthma and wheezing during childhood. *N Engl J Med* 343:538–43.

Bang KM, Hnizdo E, Doney B. 2005. Prevalence of asthma by industry in the US population: a study of 2001 NHIS data. *Am J Ind Med*; 47:500–8

Bardana EJ Jr. 2003. Occupational asthma and allergies. *J Allergy Clin Immunol* 111(2 Suppl):S530–9.

Bernstein DI, Chan-Yeung M, Malo J-L, Bernstein IL, eds. 2006. *Asthma in the Workplace and Related Conditions*, 3rd Edition. New York NY: Taylor and Francis.

Bonner JR. 1984. The epidemiology and natural history of asthma. *Clin Chest Med*; 5:557–65.

Brooks GD and Bush RK 2009. Pathogenic and environmental aspects in allergy and asthma. In *Patterson's Allergic Diseases*, 7th ed. Grammer LC, and Greenberger PA (Eds). Philadelphia, PA: Lippincott, Williams & Wilkins, 73–103.

Brunekreef B, Janssen NA, deHartog J, Harssema H, Knape M, van Vliet P. 1997. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology* 8:298–303.

Burge HA. 1989. Airborne allergenic fungi. Classification, nomenclature, and distribution. *Immunol Allergy Clin North Am* 9:307–19.

Busse WW, Rosenwasser LJ. 2003. Mechanisms of asthma. *J Allergy Clin Immunol* 111(3 Suppl):799–804.

[CDC] Centers for Disease Control and Prevention. 2012. *National Surveillance of Asthma: United States, 2001-2010*. National Center for Health Statistics. *Vital Health Stat* 3(35), 2012.

Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, et al. 1998. Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. *Occup Environ Med* 55(11):771–8.

Cox-Ganser JM, White SK, Jones R, Hilsbos K, Storey E, Enright PL, et al. 2005. Respiratory morbidity in office workers in a water-damaged building. *Environ Health Perspect* 113:485-490.

de Jongste JC, Shields MD. 2003. Cough 2: Chronic cough in children. *Thorax* 58(11):998–1003.

Dell S, To T. 2001. Breastfeeding and asthma in young children: findings from a population-based study. *Arch Pediatr Adolesc Med* 155(11):1261–5.

Dey AN, Schiller JS, Tai DA. 2004. Summary health statistics for US children: National Health Interview Survey. 2002. *Vital Health Stat* 10 (221)1–78.

Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. 1989. Effects of inhalable particles on expiratory health of children. *Am Rev Respir Dis* 139:587–94.

Donaldson K, Gilmour MI, MacNee W. 2000. Asthma and PM₁₀. *Respir Res* 1(1):12–5.

Duffy DL, Mitchell CA, Martin NG. 1998. Genetic and environmental risk factors for asthma: a cotwin-control study. *Am J Respir Crit Care Med* 157(3 Pt 1):840–5.

Ehnert B, Lau-Schadendorf S, Weber A, Buettner P, Schou C, Wahn U. 1992. Reducing domestic exposure to dust mite allergen reduces bronchial hyperreactivity in sensitive children with asthma. *J Allergy Clin Immunol* 90:135–8.

Etzel RA. 2003. How environmental exposures influence the development and exacerbation of asthma. *Pediatrics* 112(1 Pt 2):233–9.

Fish JE. 2002. Occupational asthma and

rhinoconjunctivitis induced by natural rubber latex exposure. *J Allergy Clin Immunol* 110(2 Suppl):S75–81.

Frew AJ. 2003a. Advances in environmental and occupational disorders. *J Allergy Clin Immunol* 111(3 Suppl):824–8.

Frew AJ. 2003b. Immunotherapy of allergic disease. *J Allergy Clin Immunol* 111(2 Suppl):712–9.

Friedman-Jimenez G, Beckett WS, Szeinuk J, Petsonk EL. 2000. Clinical evaluation, management, and prevention of work-related asthma. *Am J Ind Med* 37:121–141.

Gern JE. 2004. Viral respiratory infection and the link to asthma. *Pediatr Infect Dis J* 23(1 Suppl):78–86.

[GINA] Global Strategy for Asthma Management and Prevention, Global Initiative for Asthma (GINA) 2011. Available from: <http://www.ginasthma.org/>. Last accessed 9/30/2012

Henneberger PK, Redlich CA, Callahan DB, Harber P, Lemièrè C, Martin J, Tarlo SM, Vandenplas O, Torén K; ATS Ad Hoc Committee on Work-Exacerbated Asthma. 2011. An official american thoracic society statement: work-exacerbated asthma. *Am J Respir Crit Care Med*; 184:368–78.

Holla AD, Roy SR, Liu AH. 2002. Endotoxin, atopy and asthma. *Curr Opin Allergy Clin Immunol* 2(2):141–5.

[IOM] Institute of Medicine, Committee on the Assessment of Asthma and Indoor Air. 2000. Clearing the air: asthma and indoor air exposures. Washington DC: National Academy Press.

[IOM] Institute of Medicine of the National Academies. 2004. Damp Indoor Spaces and Health. Washington DC: The National Academies Press.

Jaakkola JJ, Hwang BF, Jaakkola N. 2005. Home Dampness and Molds, Parental Atopy, and Asthma in

Childhood: A Six-Year Population-Based Cohort Study. *Environ Health Perspect*. 113 (3):357–361.

Jeebhay MF, Robins TG, Lehrer SB, Lopata AL. 2001. Occupational seafood allergy: a review. *Occup Environ Med* 58(9):553–62.

Johnson VJ, Matheson JM, Luster MI. 2004. Animal models for diisocyanate asthma: answers for lingering questions. *Curr Opin Allergy Clin Immunol* 4(2):105–10.

Jones AP. 2000. Asthma and the home environment. *J Asthma* 37:103–24.

Kilpatrick N, Frumkin H, Trowbridge J, Escoffery C, Geller R, Rubin I, et al. 2002. The environmental history in pediatric practice: a study of pediatricians' attitudes, beliefs, and practices. *Environ Health Perspect* 110:823–7.

Koenig JQ, Covert DS, Hanley QS, van Belle G, Pierson WE. 1990. Prior exposure to ozone potentiates subsequent response to sulfur dioxide in adolescent asthmatic subjects. *Am Rev Respir Dis* 141(2):377–80.

Krieger J, Higgins DL. 2002. Housing and health: time again for public health action. *Am J Public Health* 92(5):758–68.

Krzyzanowski M, Quackenboss JJ, Lebowitz MD. 1990. Chronic respiratory effects of indoor formaldehyde exposure. *Environ Res* 52:117–25.

Landwehr LP, Boguniewicz M. 1996. Current perspectives on latex allergy. *J Pediatr* 128:305–12.

Larche M, Robinson DS, Kay AB. 2003. The role of T lymphocytes in the pathogenesis of asthma. *J Allergy Clin Immunol* 111(3):450–63.

Leikauf GD, Kline S, Albert RE, Baxter CS, Bernstein DI, Buncher CR. 1995. Evaluation of a possible association of urban air toxics and asthma. *Environ Health Perspect*

103(Suppl 6):253–71.

Lemanske RF Jr. 2003. Viruses and asthma: Inception, exacerbation, and possible prevention. *J Pediatr* 142(2 Suppl):3–7.

Lemiere C. 2004. The use of sputum eosinophils in the evaluation of occupational asthma. *Curr Opin Allergy Clin Immunol* 4(2):81–5.

Lindren S, Belin L, Dreborg S, Einarsson R, Pahlman I. 1988. Breed-specific dog-dandruff allergens. *J Allergy Clin Immunol* 82:196–204.

Liu AH, Murphy JR. 2003. Hygiene hypothesis: fact or fiction? *J Allergy Clin Immunol* 111(3):471–8.

Lodrup Carlsen KC, Carlsen KH. 2001. Effects of maternal and early tobacco exposure on the development of asthma and airway hyperreactivity. *Curr Opin Allergy Clin Immunol* 1(2):139–43.

Luft C, Hausding M, Finotto S. 2004. Regulation of T cells in asthma: implications for genetic manipulation. *Curr Opin Allergy Clin Immunol* 4(1):69–74.

Malo JL, Chan-Yeung M. 2006. Appendix: Agents causing occupational asthma with key references. In *Asthma in the Workplace and Related Conditions, 3rd Edition*. Edited by Bernstein DI, Chan-Yeung M, Malo J-L, Bernstein IL. New York NY:Taylor and Francis. p. 825–866.

Malo JL, Chan-Yeung M. 2001. Occupational asthma. *J Allergy Clin Immunol* 108(3):317–28.

Mapp CE, Boschetto P, Maestrelli P, Fabbri LM. 2005. Occupational asthma. *Am J Respir Crit Care Med*. 1;172:280–5.

Martinez FD, Cline M, Burrows B. 1992. Increased incidence of asthma in children of smoking mothers. *Pediatrics* 89:21–6.

Martinez FD. 2003. Respiratory syncytial virus bronchiolitis and the pathogenesis of childhood asthma. *Pediatr Infect Dis J* 22(2 Suppl): 76–82.

McDonald E, Cook D, Newman T, Griffith L, Cox G, Guyatt G. 2002. Effect of air filtration systems on asthma: a systematic review of randomized trials. *Chest* 122(5): 1535–42.

Murray AB, Morrison BJ. 1993. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. *J Allergy Clin Immunol* 91: 102–10.

Murray CS, Woodcock A, Custovic A. 2001. The role of indoor allergen exposure in the development of sensitization and asthma. *Curr Opin Allergy Clin Immunol* 1(5): 407–12.

Nafstad P, Magnus P, Gaarder PI, Jaakkola JJ. 2001. Exposure to pets and atopy-related diseases in the first 4 years of life. *Allergy* 56: 307–12.

[NEETF] The National Environmental Education and Training Foundation. 2005. Environmental management of pediatric asthma guidelines. Washington Available from:
<http://www.neefusa.org/health/asthma/asthmaguidelines.htmh> Last accessed 9/30/2012.

[NHLBI] National Heart, Lung, and Blood Institute. 2007. National Asthma Education and Prevention, Expert Panel Report 3: guidelines for the diagnosis and management of asthma. Bethesda MD: National Institutes of Health. Available from:
<http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf> Last accessed 5/31/2013.

[NHLBI] National Heart, Lung, and Blood Institute. 2003. National Asthma Education and Prevention, Update on Selected Topics 2002 Expert Panel Report: guidelines for the diagnosis and management of asthma. Bethesda MD: National Institutes of Health. No.

02-5074.

[NHLBI] National Heart, Lung, and Blood Institute. 1997. National Asthma Education and Prevention Expert Panel report 2: guidelines for the diagnosis and management of asthma. Bethesda MD: National Institutes of Health. No. 97-4051.

[NIOSH] National Institute for Occupational Safety and Health (NIOSH). Work-related lung disease surveillance report 2002. DHHS (NIOSH) Publication No. 2003-111. Cincinnati OH. Available from: <http://www.cdc.gov/niosh/docs/2003-111/pdfs/2003-111.pdf>. Last accessed 9/30/2012

[NRDC] Natural Resources Defense Council. 2001. No breathing in the aisles: diesel exhaust inside school buses. San Francisco CA: National Resources Defense Council.

Nelson HS. 2003. Advances in upper airway diseases and allergen immunotherapy. *J Allergy Clin Immunol* 111(3 Suppl):793–8..

Nelson HS. 2000. The importance of allergens in the development of asthma and the persistence of symptoms. *J Allergy Clin Immunol* 105(6 Pt 2):628–32.

Obata H, Dittrick M, Chan H, Chan-Yeung M. 1999. Sputum eosinophils and exhaled nitric oxide during late asthmatic reaction in patients with western red cedar asthma. *Eur Respir J*; 13: 489–95.

O'Connor GT, Gold DR. 1999. Cockroach allergy and asthma in a 30-year-old man. *Environ Health Perspect* 107:243–7.

Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology* 12:200–8.

Pirkle JL, Flegal KM, Bernert JT, Brady DJ, Etzel RA, Maurer KR. 1996. Exposure of the US population to

environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988–1991. *JAMA* 275:1233–40.

Platts-Mills TA, Sporik RB, Wheatly LM, Heymann PW. 1995. Is there a dose-response relationship between exposure to indoor allergens and symptoms of asthma? *J Allergy Clin Immunol* 96:435–40.

Platts-Mills TA, Vaughan JW, Carter MC, Woodfolk JA. 2001. The role of intervention in established allergy: avoidance of indoor allergens in the treatment of chronic allergic disease. *J Allergy Clin Immunol* 106:787–804.

Pope AM, Patterson R, Burge H, et al, editors. 1993. Indoor allergens: assessing and controlling adverse health effects. Institute of Medicine, Committee on the Health Effects of Indoor Allergens. Washington DC:National Academy Press.

Rabatin JT, Cowl CT. 2001. A guide to the diagnosis and treatment of occupational asthma. *Mayo Clin Proc* 76(6):633–40.

Robinson DS, Larche M, Durham SR. 2004. Tregs and allergic disease. *J Clin Invest* 114(10):1389–97.

Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, et al. 1997. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 336:1356–63.

Salvi S. Pollution and allergic airways disease. 2001. *Curr Opin Allergy Clin Immunol* 1(1):35–41.

Sarlo K, Kirchner DB. 2002. Occupational asthma and allergy in the detergent industry: new developments. *Curr Opin Allergy Clin Immunol* 2(2):97–101.

Schwab M, McDermott A, Spengler JD. 1992. Using longitudinal data to understand children's activity patterns in an exposure context: data from the

Kanawha County Health Study. *Environ Int* 18:173–89.

Schwartz J, Neas LM. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in school children. *Epidemiology* 11:6–10.

Shah R, Grammer RC. 2012. An overview of allergens. *Allergy Asthma Proc* 33:S2–S5.

Shima M, Adachi M. 2000. Effect of outdoor and indoor nitrogen dioxide on respiratory symptoms in schoolchildren. *Int J Epidemiol* 29:862–70.

Shusterman D. 1992. Critical review: the health significance of environmental odor pollution. *Arch Environ Health* 47(1):76–87.

Simpson A, Custovic A. 2004. Allergen avoidance in the primary prevention of asthma. *Curr Opin Allergy Clin Immunol* 4(1):45–51.

Song BJ, Liu AH. 2003. Metropolitan endotoxin exposure, allergy and asthma. *Curr Opin Allergy Clin Immunol* 3(5):331–5.

Spektor DM, Thurston GD, Mao J, He D, Hayes C, Lippmann M. 1991. Effects of single- and multiday ozone exposures on respiratory function in active normal children. *Environ Res* 55:107–22.

Sporik R, Holgate ST, Platts-Mills TA, Cogswell JJ. 1990. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study. *N Engl J Med* 323:502–7.

Tager IB, Hanrahan JP, Tosteson TD, Castile RG, Brown RW, Weiss ST, et al. 1993. Lung function, pre- and post-natal smoke exposure, and wheezing in the first year of life. *Am Rev Respir Dis* 147(4):811–7.

Taylor AN. 2001. Role of human leukocyte antigen phenotype and exposure in development of occupational

asthma. *Curr Opin Allergy Clin Immunol* 1(2):157–61.

Togias A. 2003. Rhinitis and asthma: evidence for respiratory system integration. *J Allergy Clin Immunol* 111(6):1171–83.

Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, et al. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *Am J Epidemiol* 151(8):798–810.

Umetsu DT, Akbari O, Dekruyff RH. 2003. Regulatory T cells control the development of allergic disease and asthma. *J Allergy Clin Immunol* 112(3):480–7.

[US EPA] US Environmental Protection Agency. 1999. Guidelines for reporting of daily air quality: air quality index (AQI). Washington DC:US Environmental Protection Agency. EPA-454/R-99-010.

[US EPA] US Environmental Protection Agency. Air Quality Index. A guide to air quality and your health. Washington DC [updated 2009 August; accessed 2012 September 30]. Available from:
http://www.njaqinow.net/App_AQI/AQI.en-US.pdf

[US EPA] US Environmental Protection Agency. 1994. Indoor air pollution: An introduction for health professionals. Washington DC [updated 2012 July 3; accessed 2012 September 30]. Available from:
<http://www.epa.gov/iaq/pubs/hpguide.html>.

Von Essen S. 2001. The role of farm exposures in occupational asthma and allergy. *Curr Opin Allergy Clin Immunol* 1(2):151–6.

Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE. 1986. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. *Am Rev Respir Dis* 133:834–42.

Weinberger M. 2003. Clinical patterns and natural history of asthma. *J Pediatr* 142(2 Suppl):15–9.

Weitzman M, Gortmaker S, Walker DK, Sobol A. 1990. Maternal smoking and childhood asthma. *Pediatrics* 85:505–11.

Williams SG, Schmidt DK, Redd SC, Storms W. 2003. Key clinical activities for quality asthma care. Recommendations of the National Asthma Education and Prevention Program. *MMWR* 52(RR-6):1–8.

Wisnewski AV, Redlich CA, Mapp CE, Bernstein DI. 2006. Polyisocyanates and their prepolymers. In *Asthma in the Workplace and Related Conditions*, 3rd Edition. Edited by Bernstein DI, Chan-Yeung M, Malo J-L, Bernstein IL. New York NY: Taylor and Francis. p 481-504.

Wood RA, Eggleston PA. 1993. Management of allergy to animal danders. *Pediatric Asthma, Allergy & Immunology* 7:11,13–22.

Table of Tables

Table Number	Title
1	Differential Diagnosis Possibilities for Asthma
2	Air Quality Index
3	Comparison of <i>In Vivo</i> vs <i>In Vitro</i> Allergy Testing

Posttest

- Posttest**
1. Asthma has been defined as
 - A. Reversible airway obstruction.
 - B. Chronic airway inflammation.
 - C. Nonreversible airway obstruction.
 - D. A and B.
 2. In the diagnosis of asthma in adults, all the following are true **EXCEPT**
 - A. Reversibility of airway obstruction on spirometry testing after bronchodilators, as demonstrated by
-

-
- an increase of 12% in the FEV1 with an absolute minimum improvement of at least 200 mL.
- B. The use of peak flow measurements alone is usually sufficient to diagnose asthma.
 - C. In patients with mild asthma with normal spirometry results, nonspecific provocation testing (e.g., methacholine challenge testing) can be used to demonstrate the presence of hyperresponsive airways.
 - D. Airway obstruction is generally considered present when the FEV1/FVC ratio is $< 65\%$ and the FVC as a percent predicted is normal.
3. Risk factors for the development of asthma include all of the following **EXCEPT**
- A. Personal or family history of atopy.
 - B. Prenatal smoking by the mother.
 - C. Personal or family history of hypertension.
 - D. Chronic allergic rhinitis.
4. For biologic allergens, which of these statements is **FALSE**?
- A. Biologic allergens are ubiquitous in the environment.
 - B. Biologic allergens are associated with 10% humidity in the case of dust mites.
 - C. Biologic allergens are associated with water-damaged areas.
 - D. Biologic allergens are associated with residential furry or feathered pets.
5. Physical examination of a patient with asthma would be **LEAST LIKELY** to reveal
- A. Allergic conjunctivitis and rhinitis.
 - B. Focal persistent wheezing involving the base of one lung.
 - C. Normal findings on chest auscultation.
 - D. Prolonged expiratory phase and diffuse wheezing on chest auscultation.
6. The treatment for dust mite and cockroach allergens
-

includes all of the following **EXCEPT**

- A. Cover mattresses and pillows with allergen impermeable cover.
 - B. Use a professional exterminator as initial step.
 - C. Limit food consumption to one area of the house.
 - D. Remove wall-to-wall carpets, particularly in bedrooms.
7. Management of cockroach allergen should be accomplished first by hygienic measures, such as
- A. Maintaining clean areas and limiting food consumption to only one area, such as the kitchen.
 - B. Caulking holes in walls, cupboards, and cabinets.
 - C. Storing food in closed containers.
 - D. All of the above.
8. Conditions which may be confused with asthma in children include all of the following **EXCEPT**
- A. Foreign body aspiration.
 - B. Enlarged lymph nodes or tumor.
 - C. Hematochezia.
 - D. Gastroesophageal reflux.
9. Conditions which may be confused with asthma in adults include all of the following **EXCEPT**
- A. Epistaxis.
 - B. COPD.
 - C. Pulmonary embolism.
 - D. Cough and wheezing secondary to ACE inhibitors.
10. Risk of asthma may be increased by
- A. Living near a heavily traveled roadway.
 - B. Heavy exercise on a day with an AQI of 130.
 - C. Spending over 1 hour each day riding a diesel-powered bus.
 - D. All of the above.

11. The leading cause of occupational asthma is

exposure to

- A. Latex.
- B. Spider mites.
- C. Diisocyanates.
- D. Epoxy.

12. Medical history questions about environmental asthma triggers should include

- A. Tobacco smoke.
- B. Pets.
- C. Bedding and laundering practices.
- D. All of the above.

13. It is possible to make the diagnosis of asthma without detectable wheezing

- A. True.
- B. False.

14. Your overall treatment, management, and prevention goals might include

- A. Confirmation of asthma diagnosis and gauge of severity.
- B. Optimal pharmacotherapy with minimal or no adverse effects.
- C. Education of the patient and family regarding primary and secondary preventive measures, including smoking cessation.
- D. All of the above.

15. Goals for the general management of a patient with asthma should include

- A. Normal or near-normal lung function.
- B. Careful monitoring prevention of chronic asthma symptoms and exacerbations day and night.
- C. Normal activity maintained (including exercise and other physical activities).
- D. All of the above.

16. Important moderating variables affecting how

environmental exposures may exacerbate or cause asthma include

- A. Age and timing of exposure relative to disease development.
 - B. Dose and frequency of exposure.
 - C. Genetic predispositions in response and co exposures.
 - D. All of the above.
17. The hygiene hypothesis of asthma states that naturally occurring infections and allergen exposures might essentially protect against the development of asthma and allergic and autoimmune diseases
- A. True.
 - B. False.

Appendix 1: Asthma Triggers Exposure History

Adapted from The National Environmental Education and Training Foundation. Environmental Management Of Pediatric Asthma Guidelines. <http://www.neefusa.org/health/asthma/asthmaguidelines.htm>, 2005 Aug.

It is very important to ask about all environments in which a child with asthma may be spending significant amounts of time, including all residences where the child sleeps or spends time, such as the home of a relative, schools, daycare, camp, and college dorms (for 17 – 18 year olds). Ask the questions in the box first. Ask additional questions if indicated.

Dust Mites

Have you noticed whether dust exposure makes your child's asthma worse?

Yes No Not sure

Have you used any means for dust mite control?

Yes No Not sure

Which ones? _____

Additional Questions:

- Do you know that dust exposure can trigger asthma symptoms? _____
- Do you live in a house or an apartment? _____
- If you live in a house, how old is it? _____
- What type of floor coverings are in your house?

- Is there carpet in your child's bedroom? _____
- Do you have a HEPA vacuum cleaner? _____
- Have you tried anything to decrease dust mite exposure? _____
- Have you ever heard of putting special coverings on a pillow or mattress to decrease dust mite exposure? _____
- Are you currently using a mattress or pillow covering on your child's bed? _____
- How often do you wash your child's bed linens? _____
- Do you wash them in hot, warm, or cold water? _____
- Are there stuffed animals in your child's room/bed? _____
- Do you use other ways to decrease dust mite exposure? _____

Animal Allergens

Do you have any furry pets?

Yes No Not sure

Have you seen rats or mice in the home?

Yes No Not sure

Additional Questions:

- What type of furry pet(s) do you have? (and how many of each)

- Is it a
 - strictly indoor pet? _____
 - outdoor? _____
 - indoor/outdoor? _____
- How often do you wash your pet? _____
- How long have you had your pet (s)? _____

- Has your child's asthma become worse since having the pet? _____
- Has your child's asthma become better since moving the pet outside?

- Have you noticed any rodents indoors or outside your home (rats, mice)?

Yes No Not sure

Cockroach Allergen

Have you seen cockroaches in your home on a regular basis?
(i.e., weekly or daily)

Yes No Not sure

Additional Questions:

- Approximately how many cockroaches do you see in your home per day? _____
- Do you see evidence of cockroach droppings?

Yes No Not sure

- How do you get rid of the cockroaches? _____

Mold/Mildew

Do you see or smell mold/mildew in your home?

Yes No Not sure

Is there evidence of water damage in your home?

Yes No Not sure

Do you use a humidifier or swamp cooler?

Yes No Not sure

- Where do you see mold growth in your home?

Attic	_____	Garage	_____
Basement	_____	Laundry room	_____
Bathroom	_____	Other	_____
Bedroom	_____		_____

- How large an area is the mold growth? _____
- Do you have problems with moisture or leaks in your home?
 - Yes No Not sure
- Do you frequently have condensation on your windows?
 - Yes No Not sure
- Do you have either of the following in your home:
 - Humidifier? _____
 - Evaporative-type air conditioner ("swamp cooler")? _____
- How often is it cleaned? _____
- Have you tried using something to decrease the humidity in your home? _____

Environmental Tobacco Smoke

Do any family members smoke?
 Yes No Not sure

Does this person(s) have an interest or desire to quit?
 Yes No Not sure

Does your child/teenager smoke?
 Yes No Not sure

- How many cigarettes per day? _____
- Does he/she (they) smoke in the house? _____

Outside? _____ Both inside and outside? _____ In the car?

- Do you have a smoking ban in the household? _____
- Does anyone smoke in daycare or other childcare setting where the child stays? _____
- Does anyone who spends time at your house smoke? (friends, neighbors, relatives?) _____
- Describe the circumstances when your child may be exposed to smoke? _____

Air Pollution

Have you had new carpets, paint, or other changes made to your house in the past year?

Yes No Not sure

Does your child or another family member have a hobby that uses toxic materials?

Yes No Not sure

Has outdoor air pollution ever worsened your child's asthma?

Yes No Not sure

Does your child play outdoors when an Air Quality Alert (*i.e.*, ozone, particulate) is issued?

Yes No Not sure

Do you use a wood burning fireplace or stove?

Yes No Not sure

Do you use unvented appliances such as a gas stove for heating your home?

Yes No Not sure

Additional Questions:

Indoor Air Pollution Questions

- Does anyone in your house use strong-smelling perfumes, scented candles, hairsprays, or other aerosol substances? _____
- Do you live in a home that was built in the past 1–2 years?

Yes No Not sure

- If you recently made changes to your house—installed new carpets, painted, or other changes—how long ago was that?

- Was there a change in your child's asthma symptoms after moving to a new house or having the work mentioned above done in your home?

Yes No Not sure

- Do you ever notice a chemical type smell in your home?

Yes No Not sure

If you have a wood burning fireplace or stove, how many times per month in the winter do you use it? _____

- Do you use an unvented appliance such as a gas stove for heating your home?

Outdoor Air Pollution Questions

- Do you live within a ½ mile of a major roadway or highway?

Yes No Not sure

- An area where trucks or other vehicles idle?

Yes No Not sure

- A major industry with smokestacks?

Yes No Not sure

- Is residential or agricultural burning a problem where you live?

Yes No Not sure