Agency for Toxic Substances and Disease Registry
Case Studies in Environmental Medicine
Environmental Triggers of Asthma

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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 people with asthma.
About This and Other Case Studies in Environmental Medicine	This educational case study document is one in a series of self-instructional publications 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: www.atsdr.cdc.gov/csem/. 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.
How to Apply for and Receive Continuing Education Credit	See Internet address www2.cdc.gov/atsdrce/ for more information about continuing medical education credits, continuing nursing education credits, and other continuing education units.

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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 this educational monograph, ATSDR has made diligent effort to ensure the accuracy and currency of the information presented, but makes no claim that the document comprehensively addresses all possible situations related to this substance. This monograph is intended as an educational resource for physicians and other health professionals in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider. The document must be interpreted in light of specific information regarding the patient and in conjunction with other sources of authority.

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Agency for Toxic Substances and Disease Registry
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How to Use This Course

Introduction

The goal of *Case Studies in Environmental Medicine* (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.

Available Versions

Two versions of the Environmental Triggers of Asthma CSEM are available.

- The online version (http://www.atsdr.cdc.gov/csem/asthma/) provides the same 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, we recommend that you

- 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; and
- 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 with which you are already familiar. 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	Enables you to test yourself to determine whether you have mastered the learning objectives.
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:

Topic	Objectives
Overview of Asthma	• Define asthma • Identify environmental factors that trigger asthma
Differential Diagnosis of Asthma	• 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	• Identify five indoor triggers of an acute asthma episode • Identify five outdoor triggers of an acute asthma episode • Describe the effect 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 • Identify the role of allergy testing in managing asthma • Describe questions regarding occupational asthma triggers that should be included in the standard medical history
Treatment, Management, and Prevention	• Describe the general treatment and management goals for patients with asthma • Describe modifying factors that might affect how environmental triggers cause or exacerbate asthma • Identify at least three things patients should do to decrease exposure to allergens and irritants

Initial Check (Case Study)

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 resolve spontaneously. During soccer games, the girl has recurrent episodes of cough and wheezing, which 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 as follows: temperature 98.6ºF (37.0ºC), respiratory rate 17, heart rate 82, blood pressure 118/75 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 asthma, wheezing solely associated with respiratory infections, foreign body aspiration, and wheezing associated with gastroesophageal reflux. Less likely diagnoses include cystic fibrosis, immunodeficiency states, congenital abnormalities, and vocal cord dysfunction.

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

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 section "Differential Diagnosis of Asthma".

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, seasonal variation in symptoms, relationship of symptoms to specific exposures, and exacerbation with exposure to nonspecific triggers such as cigarette or woodstove smoke or household cleaning products. The home environment should be reviewed, with a focus on the patient's environment, particularly within his or her bedroom, the presence of furry pets and carpeting, condition of home heating and cooling system, past water damage or leakage, and smoking within the home. The environment outside the home should also be reviewed, including a potential relationship of symptoms and school and recreational activities.

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

4. If there is any question about the diagnosis, consider referral to a pulmonologist or allergy/asthma specialist. In the pulmonologist's office, the measurement of forced expiratory volume in 1 second (FEV_1) before and after short-acting bronchodilator therapy can be

used 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) Simple peak expiratory flow monitoring in the general practitioner's office can be used, although variability in peak expiratory flow limits its application in screening for asthma. Chest radiographs should be performed for individuals with systemic symptoms such as fever and signs suggestive of a localized lung abnormality. 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 section "Clinical Assessment".

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. Asthma assessment, monitoring, pharmacotherapy, and patient education have been covered at length in many excellent texts and articles (Williams *et al.* 2003).
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 2003).
Incidence and Prevalence	Allergic diseases such as asthma, rhinitis, and eczema are increasing in prevalence. They affect up to 15% of populations in Westernized countries (Robinson *et al.* 2004). The increasing incidence and prevalence of asthma in many parts of the world continues to make it a global health concern (NHLBI 2004). Asthma is one of a few diseases in the United States that are increasing in incidence among children and adults. This is despite scientific advances in improving treatment outcomes and understanding the pathogenesis (IOM 2000). The last several decades have seen a significant rise in the rate of pediatric asthma. From 1980 to 1996, asthma prevalence among children increased by an average of 4.3% per year. As of 2002, 9 million U.S. children under 18 years of age (12%) had at some time in their lives been diagnosed with asthma. Of those, 4.2 million had experienced an asthma attack within the previous year (Dey *et al.* 2004).

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 dust mites, cockroaches, animal dander, and molds (Murray *et al.* 2001; Togias 2003; Jaakkola *et al.* 2005),
- irritants, such as environmental tobacco smoke (ETS), and exposures to certain chemical fumes, gases, or vapors (Tager *et al.* 1993; Weitzman *et al.* 1990; Martinez *et al.* 1992; Malo *et al.* 2006), 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, 2003).

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.
- It is 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),
- chronic bronchitis or emphysema,
- congestive heart failure,
- gastroesophageal reflux disease,
- mechanical obstruction of the airways,
- tumor/neoplasm, 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 1997).

Differential Diagnosis in Children

In children, chronic cough is a problem, which needs differentiation asthma, or not asthma? Chronic productive cough with purulent sputum is a reason for concern in children and is not usually a symptom of asthma. The younger the child, the more 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 nonallergic response (Lemanske 2003; Weinberger 2003). Nonallergic 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

- airway obstruction with a foreign body,
- bronchitis,
- pneumonia/bronchiolitis,
- cystic fibrosis,
- bronchopulmonary dysplasia (in premature infants),
- dysmotile cilia syndrome,
- alpha-1-antitrypsin deficiency, and
- immunodeficiencies (NHLBI 1997; NHLBI 2003).

(See **Table 1** below.)

Table 1. Differential Diagnosis Possibilities for Asthma (NHLBI 1997; NHLBI 2003)

Infants and Children	Adults
Upper airway diseases • Allergic rhinitis and sinusitis **Obstructions involving large airways** • Foreign body in trachea or bronchus • Vocal cord dysfunction • Vascular rings or laryngeal webs • Laryngotracheomalacia, tracheal stenosis, or bronchostenosis • Enlarged lymph nodes or tumor **Obstructions involving small airways** • Viral bronchiolitis or obliterative bronchiolitis • Cystic fibrosis • Bronchopulmonary dysplasia • Heart disease **Other causes** • Recurrent cough not due to asthma • Aspiration from swallowing mechanism • Dysfunction or gastroesophageal reflux	• Chronic obstructive pulmonary disease (COPD) • Chronic bronchitis or emphysema • Congestive heart failure • Pulmonary embolism • Laryngeal dysfunction • Mechanical obstruction of the airways (benign and malignant tumors) • Pulmonary infiltration with eosinophilia • Cough secondary to drugs (ACE inhibitors) • Vocal cord dysfunction

Key Points

- Many medical conditions may be confused with asthma at initial diagnosis.

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	Asthma can be triggered and exacerbated by exposure to many environmental factors. The American Academy of Pediatrics has recently 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 fail to 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 (pollen, mold, animal dander, insect parts, and some chemicals) and irritants (smoke, dust, gas or diesel fumes, and chlorine) which can trigger or exacerbate an asthmatic 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 preexisting 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	The importance of allergies and allergens in triggering and exacerbating asthma is supported by several studies. Key findings include the following. • 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 produced improved Pulmonary Function Tests (PFTs) 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 may 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 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 (Etzel 2003). Cockroach droppings may be one of the most under appreciated allergens in the indoor environment. A 36% cockroach sensitization rate has been reported in inner-city asthmatic children. Children with asthma and cockroach allergy who are 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

Exposure to cats is causally related to asthma exacerbations among many children with asthma (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 in the home 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 (Pope *et al.* 1993). 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, sneezing, conjunctivitis, lacrimation, wheezing, chest tightness, and shortness of breath may occur. Among patients studied, children are the most sensitive to mold allergens **(**Etzel 2003).
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 (NO_2), • particulate matter, and • sulfur dioxide (SO_2). 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. Symptoms range from skin eruption to bronchospasm and anaphylaxis. Gloves, balloons, condoms, and various types of sporting equipment may trigger allergic responses around the home (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). Ambient hazardous air pollutants, as well as industrial releases of aldehydes, metals, isocyanates, and others have been shown to cause and trigger asthma (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 dramatic increase in asthma incidence in recent years. (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 (O_3),
- SO_2,
- NO_2,
- CO,
- lead,
- particulate matter <10 microns (PM_{10}) and particulate matter <2.5 microns ($PM_{2.5}$).

The standards are designed to protect the health of all susceptible groups, including asthma. The Air Quality Index (AQI, **Table 2**) provides standardized means of communicating health information associated with daily ambient levels of ground-level O_3, SO_2, NO_2, CO, PM_{10}, 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, the critical pollutant, and the specific groups sensitive to the pollutant (US EPA 1999).

Table 2. Air Quality Index

Air Quality Index (AQI) Values	Levels of Health Concern	Colors
When the AQI is in this range:	*...air quality conditions are:*	*...as symbolized by this color:*
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

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 O_3 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 O_3 has focused on the short-term effects, such as reductions in FEV_1 and forced vital capacity (FVC). Levels of O_3 are usually greatest on hot summer days and tend to peak in the late afternoon (Etzel 2003; Avol *et al.* 1985; Spektor *et al.* 1991).
SO_2	Because of its high solubility, SO_2 mainly irritates the upper airway. The nasal mucosa effectively removes most inspired SO_2 during breathing at rest. Deep penetration to the lung mucosa may occur during moderate exercise. SO_2 has a dose-response association with bronchoconstriction. The amount of SO_2-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 SO_2 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).
NO_2	In contrast to the other pollutants, NO_2 is both an indoor and outdoor air pollutant. Indoor sources of NO_2 include • malfunctioning gas stoves, • furnaces, • fireplaces, and • kerosene space heaters. Most NO_2 health effects are believed to be due to long-term, low-level outdoor exposure. Like the other air pollutants, NO_2 increases bronchial responsiveness during exercise. NO_2 decreases lung function in people with asthma who are exposed to concentrations above 0.3 ppm, although there is not a clear dose-response relationship. Short-term exposure to high concentrations of NO_2 induces terminal bronchiolar changes and diffuse 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; Avol *et al.* 1985; Shima and Adachi 2000).
PM_{10} and $PM_{2.5}$	Particulate matter is a mixture of solid particles and liquid droplets. Particulate matter <10 microns (PM_{10}) is referred to as "course particulate matter" which may result in lower airway exposure (AAPCEH 2003). PM_{10} is the standard measure of particulate air pollution used worldwide. Studies suggest that asthma symptoms can be worsened by increases in the levels of PM_{10}, which is a complex mixture of particle types. PM_{10} has many components and there is no general agreement regarding which component(s) could exacerbate asthma. However, pro-inflammatory effects of transition metals, hydrocarbons, ultrafine particles and endotoxin—all present to varying degrees in PM_{10}—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 (SO_2 and NO_x) and VOCs. $PM_{2.5}$ penetrates deeper into the lung than does PM_{10}, 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 PM_{10} 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).
Work-Related Asthma	The most common occupational respiratory disease in many developed countries is work-related asthma. Approximately 15%–25% of adults with asthma may have work-related asthma (*i.e.*, both occupational asthma that is caused by conditions at work and work-aggravated asthma) (Balmes 2003; Wagner 2006; Henneberger 2007). The two types of occupational asthma are distinguished as shown below. • **Allergy-induced asthma** arises after a latency period that is necessary for acquiring sensitization. • **Irritant-induced asthma** develops because of acute high-level exposure to irritant materials, and may be due to repeated irritant exposures (Malo and Chan-Yeung 2001; Liu 2002; Holla *et al.* 2002; Gautrin 2006).

Asking an adult asthma patient whether their symptoms improve when away from work and worsen during periods at work is a simple but direct means of detecting potential cases of work-related asthma. Some occupational sensitizers and irritants, such as cleaning agents, epoxy glues, hairdressing products, are also found outside the workplace and may be relevant in non-occupational asthma (Tarlo 2003). However, exposure to these agents in occupational settings will often be more frequent and/or more intense than in non-occupational settings (Friedman-Jimenez *et al.* 2000).

There are well over 300 agents reported to cause occupational asthma (Malo and Chan-Yeung 2006), and an equal or greater number of agents and conditions at work can aggravate existing asthma. Numerous workplace biologic allergens can cause asthma, including natural rubber latex in medical care settings and animal allergens in research laboratories and veterinary offices. A wide range of airborne dusts, gases, fumes, and vapors can cause dose-related symptoms in individuals exposed to them in the workplace. Among the chemicals associated with occupational asthma, the acid anhydrides have been signal agents for study because of their inherent and complex biological activity and the wide range of associated occupational airway disease seen in exposed workers in various occupational settings (Zeiss 2002). In addition to traditional “dirty” workplaces, offices and other non-industrial indoor work environments can pose a risk for asthma. 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 asthmatics (IOM 2004). Since this review was published there has been further work indicating that exposure to damp indoor environments and mold can lead to the development of asthma (Cox-Ganser 2005; Jaakkola 2005).

Based on surveillance for work-related asthma in the United States during 1993-1999, the most common putative agents, in decreasing order, are

- miscellaneous chemicals,
- cleaning materials,
- mineral and inorganic dust,
- indoor air pollutants,
- pyrolysis products,
- isocyanates,
- solvents,
- hydrocarbons,
- polymers, and
- welding exposures (NIOSH 2003).

Diisocyanates are the leading identified cause of occupational asthma worldwide (Johnson *et al.* 2004; Wisnewski 2006).

A number of reports indicate that occupational exposure in health care workers to natural rubber latex can elicit symptoms of

	rhinoconjunctivitis, with or without asthma, in selected individuals who are sensitized (Fish 2002). Further research is needed to determine whether upper airway responses to occupational exposures indicate or predict lower airway responses (Christiani 2006).
Key Points	• A wide range of indoor and outdoor allergens and irritants 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 may be caused by allergens or irritants in the work environment.
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 E. Latex. F. Spider mites. G. Diisocyanates. H. Epoxy. *To review relevant content, see "Work-Related 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,
- identify the role of allergy testing in managing 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 focusing on the upper respiratory tract, chest, and skin, and
- spirometry to demonstrate reversibility.

Additional studies may 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 1997; NHLBI 2003).

Medical History

The focus of the medical history should be on the presence of any of the following (NHLBI 1997; NHLBI 2003):

- 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 1997; NHLBI 2003):

- 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/exphistory).

Physical Exam

The physical examination should focus on the upper respiratory tract, chest, and skin (NHLBI 1997; NHLBI 2003).

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, FEV_1). Airflow obstruction is indicated by reduced FEV_1 and FEV_1/FVC values relative to reference or predicted values.

Spirometry measurements (FEV_1, FVC, and FEV_1/FVC) before and after the patient inhales a short-acting bronchodilator should be taken to help confirm a diagnosis of asthma. This also helps determine whether airflow is obstructed and whether it 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 1997; NHLBI 2003).

Significant reversibility is indicated by an increase of 12% or more and 200 mL or more in FEV_1 after inhaling a short-acting bronchodilator. 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. Abnormalities of lung function are categorized as restrictive and obstructive defects.

- A reduced ratio of FEV_1/FVC (*i.e.*, < 65%*) indicates obstruction to the flow of air from the lungs.
- A reduced FVC with a normal FEV_1/FVC ratio suggests a restrictive pattern.

*<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 1997; NHLBI 2003).

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 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 1997; NHLBI 2003).

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 (NHLBI 1997; NHBLI 2003).

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

Advantages of Skin Testing	**Advantages of RAST and Other *In Vitro* Test**
• Less expensive than *in vitro* tests • Results are available within 1 hour • More sensitive than *in vitro* tests • Results are visible to the patient (This may encourage compliance with environmental control measures.)	• 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

Occupational asthma is the most prevalent form of work-related lung disease in industrialized nations. (ATS 1995). Increasing numbers of new chemicals are being produced and new manufacturing processes are being introduced. The variety of environments in which individuals may become exposed to respiratory sensitizers and irritants makes diagnosing and treating this illness even more challenging. Clinicians must first document the presence of asthma, and then establish a relationship between asthma and the workplace.

The adult patient's occupational history is the key diagnostic tool. In addition, lung function assessments that include spirometry and bronchial responsiveness are now 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.

Since asthma is an inflammatory disease, a measure of the degree of inflammation would be helpful in quantitating severity and titrating of anti-inflammatory therapy. There is evidence that monitoring eosinophils and neutrophils in induced sputum can help in the management of asthma (Lemiere 2004). A noninvasive method for measuring pulmonary inflammation would be helpful to assist the physician in initial treatment and assist in titration of anti-inflammatory therapy for asthma. Exhaled nitric oxide (NO) assays are being evaluated but are not yet validated for routine use (DeNicola *et al.* 2000; Bates and Silkoff 2003).

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

   I. True.
   J. False.

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

9. The key diagnostic tool in occupational asthma is

   K. Complete blood count.
   L. Chest X-ray.
   M. The occupational exposure history.
   N. Arterial blood gases.

   To review relevant content, see "Diagnosis and Evaluation of Occupational Asthma" in this section.

10. An individual who increases his/her FEV_1 by more that 12% after inhaling a short-acting bronchodilator probably has asthma

    O. True.
    P. 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 PEFR after administration of a short-acting B_2-agonist bronchodilator. At the end of the clinic appointment, the patient is diagnosed with mild persistent asthma. She is given anti-inflammatory therapy consisting of a corticosteroid metered-dose inhaler (MDI) for daily use and a short-acting B_2-agonist MDI for symptomatic relief. The patient 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 to the patient 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 B_2-agonist for rare breakthrough of acute asthma symptoms. She is scheduled for a return visit in 2 to 3 weeks.

You tell the mother that both parents should stop smoking or, at a minimum, they should not smoke in the house or the car. In addition, the furnace filter should be replaced or cleaned on a regular basis. The cat should not be permitted indoors, and wall-to-wall carpeting should be removed from the patient's bedroom (and preferably the whole house if feasible). Mattresses and pillows should be encased in sealed plastic covers and all bedding materials washed 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, so you 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 B_2-agonist and corticosteroid inhaler as directed. For the last week, she has not required additional use of the short-acting B_2-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. At a minimum, they should not smoke in the house or the car.

Progress Check

11. Which is not expected to be an environmental trigger of asthma in this home?

    A. Long-haired cat.
    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 paper 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 Follow-Up" in this section.

Treatment, Management, and Prevention

Learning Objectives

Upon completion of this section, you will be able to

- describe the 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. It 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.

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

- quick-relief medications to treat acute symptoms and exacerbations or
- longer acting medications to achieve control of the asthma and prevent or reduce the frequency of recurrent symptoms.

After confirming the asthma diagnosis and assessing the severity of disease, a stepwise approach is taken for the long-term management of asthma (NHLBI1997; NHLBI 2003; Spahn *et al.* 2002).

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 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 1997; NHLBI 2003; AAP 1999; Lim 2002).

Predisposing Factors

Atopy, the genetic predisposition for the development of an IgE-mediated response to common airborne allergens, is the strongest identifiable predisposing factor for developing asthma (NHLBI 1997; NHLBI 2003; 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). A recent study 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 include the following. ***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 excessive exposure to molds.

Exposure to mold and dampness in homes as much as doubles the risk of asthma development in children (Jaakkola et al. 2005).

Breast-feed infants.

A recent 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.

Nonimmunologic occupational asthma can be precipitated by a brief, high-level exposure to a strong irritant. Symptoms occur immediately or within a few hours of the exposure. Multiple lower level exposures to an irritant can also cause asthma. Whether immunologic or nonimmunologic in origin, once the diagnosis of occupational asthma is established, the worker should be removed from further exposure. 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. Jobs that use diisocyanates, enzymes, or latex are especially notorious for producing occupational asthma.

	Prospective surveillance can detect the development of specific IgE antibody before the onset of allergic symptoms. This allows for 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 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 outlines possible preventive measures in Environmental Management of Pediatric Asthma: Guidelines for Health Care Providers. Those environmental intervention guidelines are summarized below (NEETF 2005).
Dust Mites	No matter how clean a home is, dust mites cannot be totally eliminated. However, household interventions can decrease exposure to dust mites and possibly reduce asthma exacerbations (Ehnert *et al.* 1992; Murray and Ferguson 1983). 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). The following steps are recommended 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 covering. • 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 extermination least toxic 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.
- Never attempt to use industrial-strength pesticide sprays that require dilution.

Mold and Mildew

Mold spores are allergens that can be 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 (nonevaporative 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). Simply “smoking outside” is not enough to limit the harm to children from tobacco smoke (CDC 2000). 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.
- 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 being 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 that 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 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

CDC/ATSDR Resources

The following online resources concerning asthma are provided by the Centers for Disease Control and Prevention.

- Agency for Toxic Substances and Disease Registry www.cdc.gov/atsdr
  - For **chemical**, **emergency** situations
    - CDC Emergency Response: 770-488-7100 and request the ATSDR Duty Officer
  - For **chemical**, **non**-emergency situations
    - CDC-INFO (www.bt.cdc.gov/coca/800cdcinfo.asp)
    - 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. Those issues should be addressed 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/

- Centers for Disease Control and Prevention, National Center for Environmental Health, Emergency & Environmental Health Services Branch. Healthy Homes Initiative.

  www.cdc.gov/nche/ehs/Topics/HealthyHomes.htm

  www.healthyhomestraining.org/index.htm

- Environmental Hazards and Health Effects – Asthma

  www.cdc.gov/asthma/default.htm

- National Asthma Control Program

  www.cdc.gov/asthma/nacp.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 - Asthma

www.cdc.gov/HealthyYouth/asthma/

Other Online Resources

The following online resources provide detailed asthma and allergen control and prevention strategies.

- American Academy of Allergy, Asthma and Immunology. Online course on the environmental management of asthma.

  www.aaaai.org/members/cme_ce/environmental_management/notice.asp

- American Academy of Pediatrics

  www.aap.org/healthtopics/asthma.cfm

- National Asthma Education and Prevention Program

  www.nhlbi.nih.gov/health/prof/lung/index.htm#asthma

- The National Environmental Education and Training Foundation, Pediatric Asthma Initiative

  www.neetf.org/Health/asthma.htm

- National Institute of Environmental Health Sciences. Asthma and allergy prevention.

  www.niehs.nih.gov/airborne/prevent/alert.html

- U.S. Environmental Protection Agency. Asthma and indoor environments—health care professionals.

  www.epa.gov/asthma/hcprofessionals.html

- U.S. Environmental Protection Agency. 2004. Asthma home environment checklist. EPA 402-F-03-030.

  permanent.access.gpo.gov/websites/epagov/www.epa.gov/asthma/resources.html

Additional Government Publications

The following government publications on asthma may be helpful:

- National Heart, Lung, and Blood Institute. 1997. Guidelines for the diagnosis and management of asthma. Bethesda (MD): National Institutes of Health. No. 97-4051.

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

- National Heart, Lung, and Blood Institute. 2003. Guidelines for the diagnosis and management of asthma—updates on selected topics

2002. Bethesda (MD): National Institutes of Health. No. 02-5074.

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

- 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.

  www.cdc.gov/mmwr/preview/mmwrhtml/rr5206a1.htm

- National Institute for Occupational Safety and Health (NIOSH). Work-related lung disease surveillance report 2002. DHHS (NIOSH) Publication No. 2003-111, 2003, p191.

  www.cdc.gov/niosh/docs/2003-111/2003-111.html

- National Heart, Lung, and Blood Institute. Global initiative for asthma: global strategy for asthma management and prevention. Bethesda, MD: National Institutes of Health

  www.ginasthma.com.

Suggested Reading

The following publications on asthma may be helpful:

- Bernstein DI, Chan-Yeung M, Malo J-L, Bernstein IL, eds. Asthma in the Workplace and Related Conditions, 3rd Edition. New York: Taylor and Francis, 2006.
- Etzel RA. 2003. How environmental exposures influence the development and exacerbation of asthma. Pediatrics 112(1):233–9.

  http://pediatrics.aappublications.org/cgi/content/full/112/1/S1/233

Other CSEMs

Case Studies in Environmental Medicine: Environmental Triggers of Asthma is one monograph in a series. For other publications in this series, please go to: www.atsdr.cdc.gov/csem/

Posttest

Introduction	ATSDR seeks feedback on this course so we can asses its usefulness and effectiveness. We ask you to complete the assessment questionnaire online for this purpose. In addition, if you complete the assessment and posttest online, you can receive continuing education credits as follows.

Accrediting Organization	Credits Offered
Accreditation Council for Continuing Medical Education (ACCME)	The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of **1.75** *AMA PRA Category 1 Credit(s)*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.
American Nurses Credentialing Center (ANCC), Commission on Accreditation	This activity for **1.75** contact hours is provided by the Centers for Disease Control and Prevention, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.
National Commission for Health Education Credentialing, Inc. (NCHEC)	CDC is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. The Centers for Disease Control and Prevention is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for the Certified Health Education Specialist (CHES) to receive **1.5** Category I contact hours in health education, CDC provider number GA0082.
International Association for Continuing Education and Training (IACET)	The Centers for Disease Control and Prevention (CDC) has been reviewed and approved as an Authorized Provider by the International Association for Continuing Education and Training (IACET), Suite 800, McLean, VA 22102. CDC will award **0.15** of CEU's to participants who successfully complete this program.

Disclaimer	In compliance with continuing education requirements, all presenters must disclose any financial or other relationships 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/ATSDR, our planners, and the presenters for this seminar do not have financial or other relationships 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.

Instructions	To complete the assessment and posttest, go to www2.cdc.gov/atsdrce/ and follow the instructions on that page. You can immediately print your continuing education certificate from your personal transcript online. No fees are charged.

Posttest

Please select the best correct answer.

1. Asthma has been defined as

   A. Reversible airway obstruction.
   B. Chronic airway inflammation.
   C. Nonreversible airway obstruction.
   D. A and B.
   E. B and C.

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. Chest radiographs are generally not helpful.
   D. 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.
   E. 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.
   E. Exposure to increased concentrations of dust mite allergens.

4. For biologic allergens, which of these statements is false?

   A. Biologic allergens are ubiquitous in the environment.
   B. Biologic allergens are increased with the presence of carpets and upholstered furniture.
   C. Biologic allergens are associated with 10% humidity in the case of dust mites.
   D. Biologic allergens are associated with water-damaged areas.
   E. 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. Atopic dermatitis.
   E. 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. Wash bed items in hot water (130°f [55°c]).
   D. Limit food consumption to one area of the house.
   E. 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. Using individual bait stations.
   E. 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.

Relevant Content	To review content relevant to the posttest questions, see:

Question	**Location of Relevant Content – Section/Learning Objective**
1	Overview of Asthma
2	Diagnosis and Evaluation
3	Treatment, Management, and Prevention
4	Environmental Triggers of Asthma
5	Diagnosis and Evaluation
6	Treatment, Management, and Prevention
7	Treatment, Management, and Prevention
8	Differential Diagnosis of Asthma
9	Differential Diagnosis of Asthma
10	Environmental Triggers of Asthma
11	Environmental Triggers of Asthma
12	Diagnosis and Evaluation
13	Diagnosis and Evaluation
14	Treatment, Management, and Prevention
15	Treatment, Management, and Prevention
16	Treatment, Management, and Prevention
17	Treatment, Management, and Prevention

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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? Which ones?____ Yes □ /No □ / Not sure □
Does your child sleep with stuffed animals? Yes □ /No □ / Not sure □
Is there carpet in the room where your child sleeps? Yes □ /No □ / Not sure □

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)?

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? _____
- 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? Bathroom_____ Bedroom_____ Attic _____ Basement _____Garage _____ Laundry room_____ Other_____
- How large an area is the mold growth? _____
- Do you have problems with moisture or leaks in your home? _____
- Do you frequently have condensation on your windows? _____
- 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 □

Additional Questions:

- Who in the family smokes cigarettes? _____
  - 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? _____
- 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? _____
- Do you ever notice a chemical type smell in your home?_____
- 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? _____
  - an area where trucks or other vehicles idle? _____
  - a major industry with smokestacks? _____
- Is residential or agricultural burning a problem where you live? _____

Appendix 2: Answers to Progress Check Questions

1. The correct answer is B. Asthma causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning.
2. The correct answer is D. These triggers, plus miscellaneous causes such as exercise, food allergies, gastroesophageal reflux, aspirin/NSAID sensitivity, and sulfite sensitivity, may cause or exacerbate asthma.
3. The correct answer is answer C. Hematochezia is not identified as being confused with asthma in children. Foreign body aspiration, enlarged lymph nodes or tumors, and gastroesophageal reflux can be confused with asthma in children.
4. The correct answer is A. Epistaxis is not identified as being confused with asthma in adults. COPD, pulmonary embolism, cough, and wheezing secondary to ACE inhibitors can be confused with asthma in adults.
5. The correct answer is D. Risk of asthma may be increased by living near a heavily traveled roadway, by heavy exercise on a day with an unhealthy for sensitive groups AQI of 130, and by spending more than 1 hour each day riding a diesel-powered bus.
6. The correct answer is C. Diisocyanates are the leading cause of occupational asthma, the most commonly reported lung disease associated with the workplace.
7. The correct answer is D. Medical history questions about environmental asthma triggers should include tobacco smoke, pets, bedding and laundering practices.
8. The correct answer is A. True. Significant reversibility is indicated by an increase of 12% or more and 200 mL or more in FEV_1 after inhaling a short-acting bronchodilator.
9. The correct answer is C. The occupational exposure history is the key diagnostic tool.
10. The correct answer is A. Wheezing is not always a reliable indicator of airflow limitation. In mild intermittent asthma, or between exacerbations, wheezing may be absent.
11. The correct answer is D. Lead paint has not been shown to be an asthma trigger.
12. The correct answer is D. The father should contact his occupational health clinic and/or occupational health care provider per his worksite protocol for clinical evaluation. Workplaces provide material safety data sheets (MSDS) on the materials used at the worksite. Theses are also available online at: http://www.msdssearch.com/msdssearch.htm Potential health hazards and known sensitizers or substances associated with an increased risk of asthma can be discussed with the occupational health care provider. Preventive measures can be discussed as well as clinical evaluation of his health concerns. The key is to stop exposure.
13. The correct answer D. The goals for the general management of a patient with asthma should include all of the answer choices listed in this question plus:
    - normal or near-normal lung function,
    - careful monitoring and adjustment of treatment,
    - prevention of chronic asthma symptoms and exacerbations (day and night), and
    - normal activity capacity (including exercise and other physical activities).
14. The correct answer is B. In general, least toxic methods of roach control should be employed first.

15. The correct answer is D. All of the above. Important moderating factors affecting how environmental exposures may worsen or cause asthma include the age and timing of exposure relative to disease development, the dose and frequency of exposure, genetic predispositions in response, and co-exposures.

16. The correct answer is D. All of the above. Advice you can give to patients to decrease exposure to allergens or irritants in the home include covering mattresses and pillows with zippered plastic cases, avoiding smoking and environmental tobacco smoke and removing wall-to-wall carpets, particularly in bedrooms.