

## ENVIRONMENTAL TRIGGERS OF ASTHMA

### *Environmental Alert*

- Asthma is a chronic inflammatory disease of the airways.
- Over the past decade, the prevalence of asthma in both children and adults has increased in the United States.
- Environmental exposures such as allergens (animal and plant proteins), pollutants released into the environment, and workplace exposures have been linked to exacerbations of asthma.
- Control of the environment can significantly impact the expression and progression of the disease in people with asthma.

#### **Note**

This case study is not intended to teach asthma treatment to primary health care providers, but rather discuss the role environmental factors play in the etiology, triggering, and exacerbation of asthma.

*This monograph 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 aid in the evaluation of potentially exposed patients. This course is also available on the ATSDR Web site, [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/). See page 3 for more information about continuing medical education credits, continuing nursing education units, continuing education units, and continuing health education specialist credits.*



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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 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 exposure. This monograph is intended as an additional 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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**Table of Contents**

Case Study ..... 5

Pretest ..... 5

Definition and Pathogenesis ..... 6

Differential Diagnosis of Asthma ..... 7

Diagnosis and Evaluation ..... 8

Case Study (*Continued*) ..... 11

Follow-Up ..... 12

Environmental Factors, Sources, and Pollutants ..... 13

Treatment, Management, and Prevention ..... 19

References Cited ..... 22

Answers to Pretest and Challenge Questions ..... 23

Evaluation Questionnaire and Posttest, Course Number SS3097 ..... 25

Answer Sheet, Course Number SS3097 ..... 31

**Figure and Tables**

Figure 1. Flow Chart for the Assessment of Asthma by Spirometry in Adults ..... 9

Table 1. Air Quality Index (AQI) and Associated General Health Effects and Cautionary Statements ..... 16

Table 2. Classification of Asthma Disease Severity ..... 20

**Appendix**

Appendix A. National Ambient Air Quality Standards (NAAQS) ..... 33

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**Final responsibility for the contents and views expressed in this monograph resides with ATSDR.**

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# Case Studies in Environmental Medicine (CSEM): Environmental Triggers of Asthma

## Goals and Objectives

The goal of the CSEM is to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients.

After completion of this educational activity, the reader should be able to identify various environmental factors that trigger asthma exacerbations, describe interventions available to mitigate environmental factors in triggering asthma, identify sources of information on the impact of environmental factors on patients with asthma, and identify sources of information on asthma management.

## Accreditation

### Continuing Medical Education (CME)

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 1.5 hours in category 1 credit toward the American Medical Association (AMA) Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

### Continuing Nursing Education (CNE)

This activity for 1.8 contact hours is provided by CDC, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.

### Continuing Education Units (CEU)

CDC has been approved as an Authorized Provider of continuing education and training programs by the International Association for Continuing Education and Training and awards 0.15 continuing education units (CEUs).

### Continuing Health Education Specialist (CHES)

CDC 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 CHES to receive 1.5 category 1 contact hours in health education.

## Instructions

See page 4

The questionnaire and posttest must be completed and returned electronically, by fax, or by mail for eligibility to receive continuing education credit.

## Instructions for Completing CSEM Online

1. Read this CSEM, *Environmental Triggers of Asthma*; all answers are in the text.
2. Link to the MMWR/ATSDR Continuing Education General Information page ([www.cdc.gov/atsdr/index.html](http://www.cdc.gov/atsdr/index.html)).
3. Once you access this page, select the Continuing Education Opportunities link.
4. Once you access the MMWR/ATSDR site online system, select the electronic file and/or register and test for a particular ATSDR course.
  - a. Under the heading “Register and Take Exam,” click on the test type desired.
  - b. If you have registered in this system before, please use the same login and password. This will ensure an accurate transcript.
  - c. If you have not previously registered in this system, please provide the registration information requested. This allows accurate tracking for credit purposes. Please review the CDC Privacy Notice ([www.cdc.gov/privacy.htm](http://www.cdc.gov/privacy.htm)).
  - d. Once you have logged in/registered, select the test and take the posttest.
5. Answer the questions presented. To receive continuing education credit, you must answer all of the questions. Some questions have more than one answer. Questions with more than one answer will instruct you to “indicate all that are true.”
6. Complete the course evaluation and posttest no later than **March 30, 2008**.
7. You will be able to immediately print your continuing education certificate from your personal transcript.

## Instructions for Completing CSEM on Paper

1. Read this CSEM, *Environmental Triggers of Asthma*; all answers are in the text.
2. Complete the evaluation questionnaire and posttest, including your name, mailing address, phone number, and e-mail address, if available.
3. Circle your answers to the questions. To receive your continuing education credit, you must answer all of the questions.
4. Sign and date the posttest.
5. Return the evaluation questionnaire and posttest, no later than **March 1, 2008**, to ATSDR by mail or fax:

**Mail**

Continuing Education Coordinator  
Division of Toxicology and  
Environmental Medicine, ATSDR  
4770 Buford Hwy, NE (Mail Stop F-32)  
Atlanta, GA 30341-3717

**or**

**Fax**

770-488-4178  
ATTN: Continuing Education Coordinator

6. You will receive an award certificate within 90 days of submitting your credit forms. No fees are charged for participating in this continuing education activity.

## 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 two to three 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, 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 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 smokers, and 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, which provides the opportunity for 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 (T) 98.6°F (37.0°C), respiratory rate (RR) 17, heart rate (HR) 82, blood pressure (BP) 118/75 mmHg. No dyspnea or stridor is evident. Her color is normal, without cyanosis. Examination of the nares reveals boggy, red turbinates with moderate congestion without 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 anteroposterior (AP) 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 (Siberry and Iannone 2000). The rest of the

### A 12-year-old girl has a cough

#### Pretest

- What other information should you acquire?
- List the primary and differential diagnosis for wheezing in children and adults.
- What tests would you order to confirm or rule out your primary diagnosis?
- What is your initial management of this patient's symptoms?

physical examination is unremarkable. The fingers are not clubbed, nor are the nail beds cyanotic.

Your primary working diagnosis for this patient is asthma.

## Definition and Pathogenesis

The National Heart, Lung, and Blood Institute (NHLBI) proposed defining asthma as follows:

Asthma is 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 hyperresponsiveness to a variety of stimuli.

The number of people with asthma in the United States is increasing. Asthma is estimated to affect approximately 17.3 million Americans, including 5 million children <18 years of age. Of these 5 million children, 1.3 million are <5 years of age (National Institute of Medicine 2000).

Asthma remains one of a few diseases in the United States of increasing incidence in children and adults despite scientific advances in improving treatment outcomes and understanding the pathogenesis. Numerous factors including allergic, familial, infectious, occupational, socioeconomic, environmental, weather changes, exercise, and psychosocial have been implicated in the induction and exacerbation of asthma; this case study focuses on environmental factors. Agents include pet dander, dust mites, cockroaches, ragweed and other pollens. Occupational exposures can induce asthma in a previously healthy person, whereas nonallergic-type environmental agents such as ozone (O<sub>3</sub>), and sulfur dioxide (SO<sub>2</sub>) can exacerbate preexisting asthma. Environmental tobacco smoke (ETS) can both induce and exacerbate asthma.

# Differential Diagnosis of Asthma

Many cases of recurrent episodes of cough and wheezing in children and adults are due to asthma. Other conditions are often misdiagnosed as asthma.

In children, wheezing can be separated into nonallergic and allergic wheezing. A nonallergic child will often wheeze with viral respiratory infections; this generally will subside with age. Coughing and wheezing in bronchiolitis, a common respiratory virus, is difficult to distinguish from asthma. Bronchiolitis primarily occurs in children under the age of 2 years, with a peak incidence at about 6 months. Asthma is the more likely diagnosis when a patient has a strong family history, has repeated episodes of wheezing, and responds to bronchodilators.

A child with allergic wheezing tends to have more atopic disorders such as atopic dermatitis or allergic rhinitis. In the pediatric population, other causes of wheezing can include nonallergic upper airway disease, diseases associated with nonreversible obstruction of the lower airways, and gastrointestinal disease.

Obstruction of the large airways by a foreign body in the trachea or bronchus should always be considered in children with new-onset wheezing because young children frequently swallow foreign objects or push them into their noses. Less-frequent causes of wheezing include vocal cord dysfunction, vascular rings or laryngeal webs, laryngotracheomalacia, tracheal stenosis, bronchostenosis, and enlarged lymph nodes or tumor.

Wheezing in children occurs during acute infections including viral bronchiolitis. The differential diagnosis of children with frequent lower respiratory infection and wheezing should include cystic fibrosis, bronchopulmonary dysplasia (prevalent in premature infants), dysmotile cilia syndrome, alpha-1-antitrypsin deficiency, and immunodeficiencies. Although it is uncommon, wheezing can occur with pneumonia.

Stimulation of esophageal mucosal receptors by stomach contents resulting in vagally mediated bronchospasm can cause wheezing in children and adults secondary to gastroesophageal reflux. Vomiting followed by cough or a postprandial increase in symptoms suggests a gastrointestinal etiology as the source of asthma. In children, further evaluation by a pediatric gastroenterologist is recommended before the initiation of reflux drug therapy with lifestyle modifications (see Secondary Prevention section).

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, and vocal cord dysfunction. Pulmonary embolism, pulmonary infiltrates with eosinophilia, and medications (e.g., angiotensin-converting enzyme [ACE] inhibitors) are infrequent causes of wheezing. Beta-blockers should be used judiciously because they exacerbate asthma and can be lethal to a patient who has asthma.

## Diagnosis and Evaluation

### Pulmonary Function Testing

The diagnosis of asthma is established by the presence of reversible airway obstruction. This can be demonstrated, especially in adults, through the use of spirometry prebronchodilators and postbronchodilators or, in select cases, a nonspecific bronchoprovocation test (Figure 1). The major indices from spirometry testing are

- forced vital capacity (FVC), which is the total volume of air expired from the lungs during a forced maneuver after a maximum inspiration
- forced expiratory volume in 1 second ( $FEV_1$ )
- $FEV_1/FVC$  (ratio of FVC and  $FEV_1$ ), which is decreased in obstructive lung diseases such as asthma.

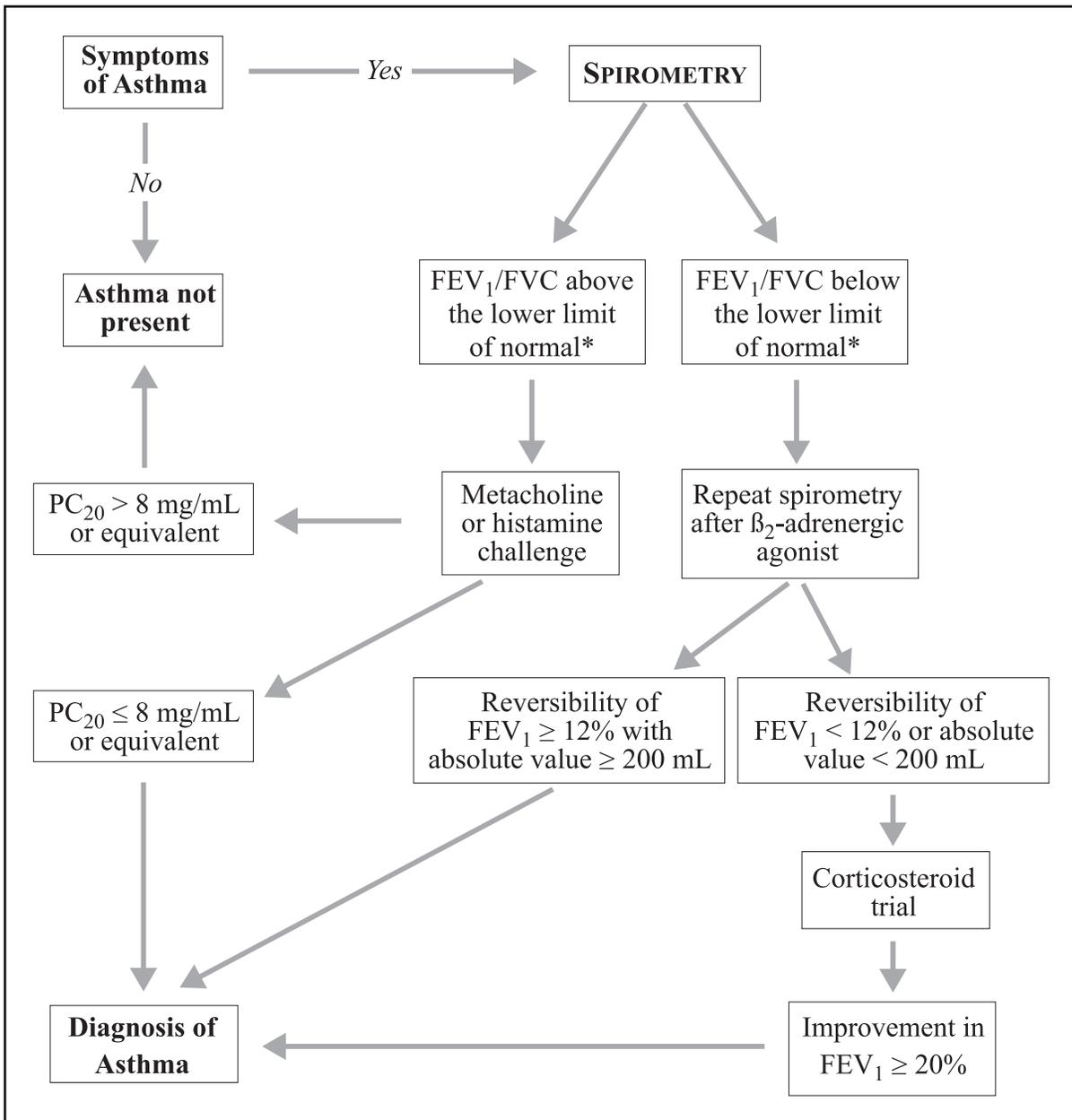
Airway obstruction is generally present when  $FEV_1$  is less than 80% of predicted value and a reduction in the  $FEV_1/FVC$  ratio exists. This threshold should be viewed as a relative value dependent on the patient's clinical picture and should be interpreted in conjunction with an overall pulmonary function assessment.

Airway obstruction is considered reversible if an increase of at least 12% in the  $FEV_1$  with a minimal absolute increase of 200 mL occurs after the administration of a short-acting, inhaled  $\beta_2$ -agonist.

In patients with intermittent asthmatic symptoms who have normal or near-normal spirometry results, nonspecific bronchoprovocation testing can help assess the degree of airway hyperreactivity. This test is generally not recommended if the  $FEV_1$  is less than 65% of predicted value. Nonspecific bronchoprovocation testing is done with inhaled methacholine or histamine or with exercise in a specialized laboratory setting. Because of the risk of provoking severe bronchoconstriction during provocation testing, these evaluations should be carried out in the controlled environment of a pulmonologist's office, where equipment and staff are available to treat asthmatic emergencies (Honig and Ingram 2001).

PEFR measurements can also be useful in the diagnosis of patients who have asthma symptoms with normal spirometry. Peak flow meters are commonly used by physicians in office practices to manage patients with asthma. PEFR is the fastest rate that air can move through the airways during a forced expiration, and it can be measured with simple, inexpensive hand-held devices that can be used at home, in a doctor's office, at work,

**Figure 1. Flow Chart for the Assessment of Asthma by Spirometry in Adults**



From the American Lung Association (1993).

FEV<sub>1</sub>: forced expiratory volume in 1 second; FVC: forced vital capacity; PC<sub>20</sub>: provocation concentration to cause a fall in FEV<sub>1</sub> of 20%.

\*Lower limit of normal = below the lowest 5% of the reference population.

or at school. PEFR is generally lowest on first awakening in the morning and highest several hours after awakening (generally between noon and 2:00 PM). NHLBI's guidelines (NHLBI 1997) recommend PEFR monitoring at those two times, including the use of an inhaled short-acting  $\beta_2$ -agonist after the morning measurement and before the afternoon measurement. A 20% difference between the morning and afternoon PEFR measurements suggests asthma, although confirmation by nonspecific bronchoprovocation studies is highly recommended in adults. PEFR monitoring can be used in the long-term, pharmacologic management of asthma as a diagnostic instrument and a means of monitoring control. This technique can also be used to assess occupational asthma (measurements are taken at least four times a day and compared to measurements taken away from work and on weekends).

Allergic asthma occurs in individuals who become sensitized to allergens. There is a latency period from initial exposure to an allergen to actual sensitization. This period can range from months to years. High-molecular-weight (HMW) allergens (>1,000 daltons [d]) such as cat dander, ragweed pollen, dust mite feces, flour or grain dust, and latex typically provoke an IgE-mediated immunologic response in susceptible individuals. 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. 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.

Nonallergic asthma, sometimes referred to as irritant-induced asthma or reactive airways dysfunction syndrome (RADS), is nonspecific airway hyperresponsiveness in individuals without a previous history of asthma but who have had an uncommon, usually accidental, exposure to a high level of a respiratory irritant gas, vapor, fume, aerosol, or dust. Respiratory symptoms consistent with asthma subsequently develop within 24 hours of exposure. The clinical criteria for the diagnosis of RADS are as follows:

- Absence of respiratory complaints before exposure.
- Onset of symptoms following a single, usually accidental, high-level specific exposure.
- Exposure was to a gas, smoke fume, or vapor with irritant qualities and in very high concentrations.
- Onset of symptoms within 24 hours after exposure and persisting for at least 3 months.

- Symptoms simulated asthma, with cough, wheezing, and dyspnea predominating.
- Pulmonary function tests might show airflow obstruction.
- Positive methacholine challenge test (e.g., <8 milligrams per milliliter [mg/mL]).
- Other pulmonary disease has been ruled out.

Atopy, the genetic predisposition to develop IgE antibodies against allergens, is the foremost predisposing factor for the development of asthma. Factors associated with allergies and asthma include allergic rhinitis, nasal polyps, and sinusitis. Asthma and allergic rhinitis are frequently found in the same patient. Studies show that people with asthma are almost three times as likely as people without asthma to have nonseasonal allergies.

## Case Study (*Continued*)

A review of the exposure history for the 12-year-old shows that the family has a long-haired cat that stays in the house and that the patient develops nasal congestion and chest tightness when playing with the pet. Further review reveals that the central heating furnace filters have not been cleaned in the last year and that wall-to-wall carpet is present throughout the house. The home has a wood-burning fireplace, which is occasionally used, and there the shower areas of the bathrooms have some mold. The patient's mother states that she vacuums regularly, she has not seen any insects in the house, and the basement is not damp. Both parents smoke cigarettes indoors, but do not smoke in the children's rooms.

The patient with asthma symptoms underwent peak flow testing in your office. The results demonstrated a 24% increase in PEF after administration of a short-acting  $\beta_2$ -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  $\beta_2$ -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 only the short-acting  $\beta_2$ -agonist is to be used for relief of acute asthma symptoms and that it can also be used before exercise activities. In addition, you prescribe a nonsedating antihistamine to treat the patient's rhinitis, and 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 ( $>130^{\circ}\text{F}$ ;  $55^{\circ}\text{C}$ ) to kill dust mites. Water heaters in homes with young children are frequently set at or below  $120^{\circ}\text{F}$  ( $50^{\circ}\text{C}$ ) 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.

## 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  $\beta_2$ -agonist and corticosteroid inhaler as directed. For the last week, she has not required additional use of the short-acting  $\beta_2$ -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.

### *Challenge*

- 1. Who else in the family is at risk for asthma?*
- 2. The case study suggests several sources of allergens and triggers in the girl's life. What are these sources and how do the pollutants from these sources affect asthma?*
- 3. 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?*
- 4. What are your overall treatment, management, and prevention goals?*

# Environmental Factors, Sources, and Pollutants

Asthma can be exacerbated by exposure to nonspecific inciters. Nonspecific airway hyperresponsiveness is characteristic of allergic and nonallergic asthma and is a reflection of airway inflammation. Nonspecific inciters such as smoke, dust, gas or diesel fumes, or chlorine can trigger an asthmatic attack in individuals with increased airway hyperresponsiveness. Under these circumstances, there is usually no latency from initial exposure to the asthma attack and the patient usually returns to his or her preexisting baseline on removal from exposure.

An environmental pollutant may affect asthma severity in the following ways:

- The pollutant might act as an inciter or trigger, leading to an asthma attack in an individual with hyperresponsive airways.
- The pollutant can exacerbate preexisting airway inflammation, leading to increased airway hyperresponsiveness, which may persist after cessation of exposure.
- The pollutant might augment or modify immune responses to inhaled antigens or intensify the impact of other pollutants in the respiratory tract.

## Indoor Air Pollution

In industrialized countries, adults and children might spend up to 75%–90% of their time indoors. The primary indoor air pollutants associated with asthma exacerbation include the following:

- Biologic allergens, such as those derived from dust mites, cockroaches, and animal dander. The allergen-containing secretions dry on fur, bedding, and clothes and become airborne.
- Environmental tobacco smoke (ETS).
- Heating sources.

### Biologic Allergens

Biologic allergens are ubiquitous throughout home, school, and workplace environments, although concentrations of dust mites, cockroaches, and cat dander allergens vary with geographic location. In a study of Baltimore allergy clinic patients, dust mite allergen was found in over 99% of homes occupied by allergic patients, and cat and dog allergens were found in 100% of the homes, including homes where there were no residential furry pets. The dust mite grows optimally at temperatures around 70°F (21.1°C) and with humidity greater than 50% in cloth-covered objects such as soft toys, upholstered furniture, bedding, mattresses, and carpets. In dry

climates, such as Los Alamos, New Mexico, only 30% of children were sensitized to dust mite allergens, but the majority were sensitized to cat dander allergens, which was the most important factor associated with asthma in that population. Food allergies (i.e., to fish, nuts, shellfish, or milk) and latex allergy should be considered and reviewed as part of the exposure assessment.

### **Environmental Tobacco Smoke (ETS)**

The largest impact of exposure to ETS can be seen in children and their respiratory system. The risk for asthma is 2.5 times greater in young children with mothers who smoke more than 10 cigarettes a day indoors as compared to mothers who smoke less or not at all. Children of all ages who live in a home with smokers are 63% more likely to have asthma. Overall, 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. In studies of maternal smoking during pregnancy, as little as 10 cigarettes per day have been associated with an increased risk of the child developing asthma later in life. In addition, the children of teenage mothers have a three-fold to five-fold increased risk of developing childhood asthma.

The Third National Health and Nutrition Examination Survey (CDC 1994), conducted from 1988 to 1994, evaluated children from the age of 2 months to 5 years in the United States. Within this group, approximately 38% of the children surveyed were exposed to ETS in the home, and 23.8% had been exposed to ETS by maternal smoking during pregnancy. Exposure to ETS was associated with three or more episodes of wheezing among children 2 months to 2 years old, and it was associated with asthma among children 2 months to 5 years old. In homes where 20 or more cigarettes were smoked daily, the adjusted odds ratio for asthma was 2.1 for children 2 months to 5 years old.

### **Combustion Devices**

Improperly used or malfunctioning heating devices are a major source of combustion pollutants in the indoor environment. Possible sources of contaminants include

- gas ranges, especially if used for home heating;
- improperly vented fireplaces;
- inefficient or malfunctioning furnaces;
- wood or coal stoves; and
- unvented or improperly vented kerosene or gas space heaters.

The combustion products from these devices include carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>), particulate matter, and SO<sub>2</sub>. In combination, these combustion products, with the exception of CO, will often exacerbate asthma symptoms, but have not been associated with the higher asthma prevalence seen with exposure to ETS.

## 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. The National Ambient Air Quality Standards (NAAQS), required by the Clean Air Act (CAA), are set for six criteria pollutants (O<sub>3</sub>, SO<sub>2</sub>, NO<sub>2</sub>, CO, lead, and PM<sub>10</sub> and PM<sub>2.5</sub> [particulate matter ≤10 or 2.5 micrometers in aerodynamic diameter, respectively]). The standards are designed to protect the health of all susceptible groups (Appendix A). For people with asthma, SO<sub>2</sub>, sulfuric acid aerosols, and NO<sub>2</sub> can exacerbate respiratory symptoms in the short term.

The Air Quality Index (AQI), previously known as the Pollution Standards Index (PSI), provides standardized means of communicating health information associated with daily ambient levels of ground-level O<sub>3</sub>, PM<sub>2.5</sub>, PM<sub>10</sub>, CO, SO<sub>2</sub>, and NO<sub>2</sub> (Table 1). Developed by EPA, AQI is reported in all U.S. metropolitan areas with populations exceeding 350,000. For any reported index value greater than 100, 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. The health professional should also be aware of NAAQS sampling limitations for these air pollutants:

- CO and O<sub>3</sub> are sampled and reported on an hourly basis.
- SO<sub>2</sub> is sampled on a 3-hour schedule.
- NO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub> are sampled on a daily basis.

### Mixed Pollutants

Exposure to motor traffic emissions can have a significant effect on respiratory function in children and adults. One study showed that children living within 100 meters of heavily traveled roadways have significantly higher rates of wheezing and diagnosed asthma. Among adults, a study of street cleaners in Copenhagen who were exposed to traffic-related air pollution found an odds ratio of 2.3 for asthma when the street cleaners were compared to a control group of cemetery workers in the same city. In that study, air pollution levels did not exceed World Health Organization's recommended threshold values. Exposures due to releases from industrial facilities can cause sensitization or exacerbation of asthma.

**Table 1. Air Quality Index (AQI) and Associated General Health Effects and Cautionary Statements**

Index Value	AQI Descriptor	General Health Effects	General Cautionary Statements
0 to 50	Good	None for the general population.	None required.
51 to 100	Moderate	Few or none for the general population. Possibility of aggravation of heart or lung disease among persons with cardiopulmonary disease and the elderly with elevations of PM <sub>2.5</sub> .	Unusually sensitive people should consider limiting prolonged outdoor exertion.
101 to 150	Unhealthy for sensitive groups	Mild aggravation of respiratory symptoms among susceptible people.	Active children and adults and people with respiratory disease (such as asthma) and cardiopulmonary disease should avoid prolonged outdoor exertion.
151 to 200	Unhealthy	Significant aggravation of symptoms and decreased exercise tolerance in persons with heart or lung disease. Possible respiratory effects in the general population.	Active children and adults and people with respiratory and cardiovascular disease should avoid prolonged outdoor exertion. Everyone else, especially children, should limit prolonged outdoor exertion.
201 to 300	Very unhealthy	Increasingly severe symptoms and impaired breathing likely in sensitive groups. Increasing likelihood of respiratory effects in the general population.	Active children and adults and people with respiratory and cardiovascular disease should avoid all outdoor exertion. All others should limit outdoor exertion.
301+	Hazardous	Severe respiratory effects and impaired breathing in sensitive groups, with serious risk of premature mortality in persons with cardiopulmonary disease and the elderly. Increasingly severe respiratory effects in the general population.	Elderly and persons with existing respiratory and cardiovascular diseases should stay indoors with the windows closed. Everyone should avoid outside physical exertion.

Excerpted from the U.S. Environmental Protection Agency (1999).  
 PM<sub>2.5</sub>: particulate matter ≤2.5 micrometers in aerodynamic diameter.

**O<sub>3</sub>**

In the United States, a large fraction of ambient O<sub>3</sub> is the product of photochemical reactions between various nitrogen oxides (NO<sub>x</sub>) and volatile organic chemicals (VOCs) emitted from automobiles. In 1991, 69 million Americans resided in areas where O<sub>3</sub> levels exceeded NAAQS. As an airborne irritant, ozone exerts much of its effect on the trachea and bronchi. Most of the health effects research on O<sub>3</sub> has focused on the short-term effects, such as decrements in FEV<sub>1</sub> and FVC.

## SO<sub>2</sub>

Because of its high solubility, SO<sub>2</sub> irritates primarily the upper airway. The nasal mucosa effectively removes most inspired SO<sub>2</sub> during breathing at rest, but deep penetration to the lung mucosa can occur during moderate exercise. SO<sub>2</sub> is the only regulated outdoor air pollutant that has a dose-response association with bronchoconstriction. The amount of SO<sub>2</sub>-induced bronchoconstriction is dependent on the level of preexisting hyperresponsiveness, so a person without asthma can tolerate a higher concentration of SO<sub>2</sub> before developing symptoms. The bronchoconstrictor response develops within minutes of exposure and resolves within an hour after cessation of exposure. Exposure to SO<sub>2</sub> may present additional problems to people with asthma who exercise. Pretreatment with cromolyn sodium or anticholinergic agents (i.e., ipratropium, a bronchodilator) can partially block SO<sub>2</sub>-induced bronchoconstriction, whereas the bronchoconstriction can be reversed with the administration of inhaled β-adrenergic bronchodilator medications. The SO<sub>2</sub> NAAQS is 0.50 ppm (3-hour average) and 0.14 ppm (24-hour average). In the United States, SO<sub>2</sub> levels have been decreasing over the last 15 years, so it is unlikely that SO<sub>2</sub> alone is responsible for the recent increases in the prevalence of asthma over the last decade.

## NO<sub>2</sub>

In contrast to the other pollutants, NO<sub>2</sub> is both an indoor and outdoor air pollutant. Indoor sources of NO<sub>2</sub> include malfunctioning gas stoves, furnaces, fireplaces, and kerosene space heaters.

Most NO<sub>2</sub> health effects are believed to be due to long-term low level outdoor exposure, in contrast to O<sub>3</sub>, which causes immediate health effects. One study of exposure to NO<sub>2</sub> showed increased bronchial responsiveness if the subjects exercised during the exposure. Other research demonstrated decreased lung function in people with asthma who are exposed to NO<sub>2</sub> concentrations above 0.3 ppm. Although NO<sub>2</sub> is capable of increasing bronchial responsiveness, no consistent evidence exists to suggest that increasing ambient levels contribute to asthma exacerbations.

Short-term exposure to high concentrations of NO<sub>2</sub> induces terminal bronchiolar changes and diffuse alveolar injury. Such high concentrations are generally seen only in accidental exposure, such as within confined spaces in an occupational setting.

## PM<sub>10</sub> and PM<sub>2.5</sub>

Particulate matter is a mixture of solid particles and liquid droplets. PM<sub>10</sub> is the respirable portion of particulate matter that results in lower airway exposure. PM<sub>2.5</sub> is referred to as “fine-particle pollution.” NAAQS includes both PM<sub>10</sub> and PM<sub>2.5</sub>, and the sources and potential health effects are different for each. Sources of PM<sub>10</sub> include dust, street sand, vehicle

exhaust, crushing/grinding operations, wood-burning, and travel on unpaved roads. The major health effects associated with  $PM_{10}$  are exacerbation of existing health problems such as asthma. Sources of  $PM_{2.5}$  include activities such as industrial and residential combustion, vehicle exhaust, and atmospheric reactions between gases (sulfur dioxide and nitrogen oxides) and VOCs.  $PM_{2.5}$  penetrates deeper into the lung than  $PM_{10}$ , resulting in greater health effects. Several recently published community epidemiologic studies show that, when  $PM_{10}$  air concentrations were below NAAQS, but  $PM_{2.5}$  formed a significant portion of the particulate exposure, medication usage, hospital admissions, and number of emergency room visits (seen primarily with elderly patients and individuals with cardiopulmonary disease) increased.

### **Hazardous Air Pollutants**

Exposures due to releases from industrial facilities might cause sensitization or exacerbation of asthma. Although, there is a paucity of research related to hazardous air pollution and asthma in the general environment, some recent investigations suggest a link between such exposures and asthma in the community (White et al. in press). Ambient hazardous air pollutants including aldehydes; metals; isocyanates; and allergens including soy dust, grain dust from harbor ports, and pollen have been shown to cause asthma among occupationally exposed adults (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).

### **Occupational Asthma**

The most common occupational respiratory disease in many developed countries is occupational asthma. Approximately 20% of adults with asthma might have occupational asthma (Milton et al. 1998). The condition is still underdiagnosed and underreported. Obtaining environmental historical information by asking an adult asthma patient whether his or her symptoms improve when away from work and worsen during periods at work is important in detecting potential cases of occupational asthma.

Monitoring serial PEFR monitoring during periods at work and away from work is important in documenting whether asthma is related to work. Challenge testing is used to confirm a specific causative agent in cases when a new agent is suspected, when multiple agents are involved and it is important to establish the exact agent, and when it is not feasible to carry out serial PEFR monitoring. Early diagnosis and removal from further exposure to the causative agent in the workplace will benefit the patient with occupational asthma. Preventive measures are important to protect other workers at risk.

# Treatment, Management, and Prevention

## Treatment

This case study is not intended to teach asthma treatment to primary health care providers, but rather discuss the role environmental factors play in the etiology, triggering, and exacerbation of asthma. For treatment guidelines, please refer to *Harrison's Principles of Internal Medicine*, 14th edition (Fauci et al. 1997), *The Washington Manual of Medical Therapeutics*, 30th edition (Ahya et al. 2001), *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 5th edition (Marx et al. 2001), a pulmonology text, or the NHLBI *National Asthma Education and Prevention Expert Panel Report 2: Guidelines for the Diagnosis and Management of Asthma* (NHLBI 1997).

The treatment of environmental asthma follows the guidelines set forth by NHLBI, with special emphasis on the management of the patient's environment. Pharmaceutical intervention forms the basis of asthma treatment. Asthma medications are generally categorized in two classes: (a) quick-relief medications to treat acute symptoms and exacerbations and (b) longer acting medications to achieve control of the asthma and prevent or reduce the frequency of recurrent symptoms. After assessment of the severity of disease, a step-wise approach is taken for the long-term management of asthma (outlined in NHLBI 1997).

## Management

The goals for the general management of a patient with asthma should include the following (adapted from NHLBI [1997] and American Academy of Pediatrics [1999]):

- confirmation of asthma diagnosis,
- a gauge of severity,
- prevention of chronic asthma symptoms and exacerbations day and night,
- normal activity maintained (including exercise and other physical activities),
- normal or near-normal lung function,
- optimal pharmacotherapy with minimal or no adverse effects,
- careful monitoring, and
- education of the patient and family regarding primary and secondary preventive measures.

The exposure history forms the basis of control for people with asthma to prevent further problems. In children, the home and day care/school environments should be assessed for age of the building; existence of wall-to-wall carpets, drapes, stuffed animals, humidifiers; mold and/or water damage; cockroaches; and/or indoor furry pets; types of hobbies of residents; building location (e.g., urban, rural, or near industrial sites); type of indoor heating sources, including supplemental heat by gas stove; draftiness; and parental and other family members' personal habits (e.g., smoking). Parental occupations should also be assessed with regard to possible home contamination from workplace exposures. In adults and working teenagers with asthma, it is important to collect a history of possible exposures in the occupational as well as in the home environment.

Severity and effect of asthma on the patient's quality of life should be assessed. Severity can be measured by the type and frequency of inhalers used per month; inhaled and oral steroid therapy used in the last year; number of physician visits, emergency room visits, or hospitalizations due to asthma, past admission to an intensive care unit (ICU) and/or endotracheal intubation, whether school/work have been missed, and general overall level of physical activity. Disease severity guidelines set forth by NHLBI are shown in Table 2.

**Table 2. Classification of Asthma Disease Severity**

	Asthma Severity*			
	Mild Intermittent	Mild Persistent	Moderate Persistent	Severe Persistent
Presence of symptoms	≤2 times per week	>2 per week but <1 per day	Daily	Continuous
Exacerbation	Brief (few hours to few days)	Might affect activity	Affects activity (≥2 times per week)	Frequent (limited physical activity)
Nocturnal asthma symptoms	≤2 times per month	>2 times per month	>1 time per week	Frequent
PEF <sup>†</sup> or FEV <sub>1</sub> PEF variability <sup>‡</sup>	≥80% predicted, variability <20%	≥80% predicted, variability 20%–30%	>60% to <80% predicted, variability >30%	≤60% predicted, variability >30%

Based on the National Heart, Lung, and Blood Institute (1997).

\*Clinical features before treatment. The presence of one of the features of severity is sufficient to place a patient in that category.

<sup>†</sup>PEF: peak expiratory flow; FEV<sub>1</sub>: forced expiratory volume in 1 second.

<sup>‡</sup>Values obtained throughout the day.

## Prevention

### Primary Prevention

Primary prevention of asthma is very important, particularly in children. For children, studies indicate that *in utero* exposure to tobacco smoke products is an important predictor of wheezing within the first year of life. Physicians should strongly discourage smoking during pregnancy, although research has not yet indicated whether decreasing tobacco smoking during pregnancy can reduce the incidence of asthma. 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).

Other studies have indicated that delays in the onset of atopy and asthma in infants of atopic parents may be achieved through intensive allergen source control. In these studies, control measures included prolonged breastfeeding.

Primary prevention of adult-onset asthma relies on smoking cessation and control of workplace exposures. Cigarette smoking enhances the risk of sensitization to environmental and some workplace allergens, and increases the risk of the development of chronic cough, chronic phlegm, persistent wheeze, and breathlessness. It is estimated that 2% to 15% of adult-onset asthma is occupationally related.

### Secondary Prevention

Patients can take a number of steps to reduce or avoid exposure to pollutants, irritants, and allergens that may trigger or exacerbate asthma episodes. During smog alerts and on days with increased amounts of outdoor air pollution or high pollen/mold spore counts, patients with asthma should remain in a controlled environment. To protect children with asthma, adults should try to schedule outdoor activities for times when ozone levels are lowest (typically in the morning). Daily reports of air quality, including air quality index (see Outdoor Air Pollution and Asthma section) and pollen/mold spore counts, are available from county health departments and local news sources.

Seasonal allergies can exacerbate asthma, especially during the spring and fall seasons. Ragweed blooms from mid-August to October in high concentrations throughout the Northeast and Midwest. 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 the late summer months. Sensitive people with asthma might choose to remain indoors between 5:00 AM and 10:00 AM during days with high pollen counts. To help control allergen exposure, these patients should avoid cutting grass and raking leaves.

Modifications to the home environment can significantly reduce the frequency of asthma episodes. Smoking should not be allowed anywhere in the house or cars. Numerous steps can be taken to lower exposure to house dust mites and mite allergens. All sleeping surfaces, such as pillows, mattresses, and box springs should be covered with zippered plastic cases. All bedding (sheets and pillowcases) should be washed weekly in hot water (130°F [55°C]), the most effective method of destroying mite allergen and effectively killing all mites. The fecal pellets of the mite contain the allergic proteins. These proteins can persist in the environment months after the mite is eradicated. Dry cleaning bedding will kill mites and remove dust but will not destroy all allergens. Tumble-drying bedding at temperatures greater than 130°F (55°C) for 10 minutes will effectively kill all mites, but will not destroy or remove dust mite allergens. Freezing items in a home freezer for 24 hours can be used to kill mites on small articles such as stuffed animals, although it does not remove or destroy mite allergens. Management of cockroach allergen should be accomplished first by hygienic measures, such as

- maintaining clean areas and limiting food consumption to only one area, such as the kitchen;
- caulking holes in walls, cupboards, and cabinets;
- storing food in closed containers; and
- placing individual bait stations.

For the rest of the house, humidity levels should be reduced to 30%–50% with a central air conditioner and/or dehumidifier. Avoid room humidifiers. Clean visible mold from walls and ceilings. Vacuum the house on a regular basis with a vacuum cleaner equipped with a microfilter or high-efficiency particulate air (HEPA) vacuum bag. Remove wall-to-wall carpeting where feasible, especially in the bedroom of the person with asthma, and wet-mop the floors once a week.

Pet dander is responsible for a large amount of allergic material within the home. The best solution is to remove pets from the home environment where feasible. If pets remain in the home, they should not be permitted in bedrooms and they should be confined to carpet-free areas of the home. Bathe animals regularly (monthly).

## References Cited

- American Academy of Allergy, Asthma and Immunology. 1999. Pediatric asthma: promoting best practice. Guide for managing asthma in children. Milwaukee (WI): American Academy of Allergy, Asthma and Immunology. Available from URL: [www.aaaai.org/](http://www.aaaai.org/).
- American Academy of Pediatrics. 1999. Clinical practice guidelines of the AAP. A compendium of evidence-based research for pediatric practice. 2nd ed. Elk Grove Village (IL): American Academy of Pediatrics.
- American Lung Association. 1993. Guidelines for the evaluation of impairment/disability in patients with asthma. American Thoracic Society. Medical section of the American Lung Association. *Am Rev Respir Dis* 147:1056–61.
- Ahya SN, Flood K, Paranjothi S, editors. 2001. Washington manual of medical therapeutics. 30th edition. Philadelphia: Lippincott Williams & Wilkins.
- Centers for Disease Control and Prevention. 1994. Third National Health and Nutrition Examination Survey. Hyattsville (MD): National Center for Health Statistics.
- Committee on the Assessment of Asthma and Indoor Air, Division of Health Promotion and Disease Prevention, Institute of Medicine. 2000. Clearing the air: asthma and indoor air exposures. Washington (DC): National Academy Press.

- Dell S, To T. 2001. Breastfeeding and asthma in young children: findings from a population-based study. *Arch Pediatr Adolesc Med* 155(11):1261–5.
- Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, et al., editors. 1997. *Harrison's principles of internal medicine*. 14th edition. New York: McGraw-Hill Professional.
- Honig EG, Ingram RH Jr. 2001. Functional assessment of the lung and diagnostic techniques. In: Dale DC, Federman DD. *Scientific american medicine*. New York: WebMD Corporation.
- Leikauf GD, Kline S, Albert RE, Baxter CS, Bernstein DI, Buncher CR. 1995. Evaluation of a possible association of urban air toxics and asthma. *Environ Health Perspect* 103 (suppl 6):253–71.
- Marx J, Hockberger R, Walls R, Hockberger RS. 2001. *Rosen's emergency medicine: concepts and clinical practice*. 5th edition. St. Louis (MO): Mosby-Year Book.
- Milton DK, Solomon GM, Rosiello RA, Herrick RF. 1998. Risk and incidence of asthma attributable to occupational exposure among HMO members. *Am J Ind Med* 33:1–10.
- National Center for Health Statistics. 1997. *National vital statistics report*. Atlanta: Centers for Disease Control and Prevention. Volume 47, number 4.
- National Heart, Lung, and Blood Institute. 1997. *National Asthma Education and Prevention Expert Panel report 2: guidelines for the diagnosis and management of asthma*. Bethesda (MD): National Institutes of Health. Publ. No. 97-4051. Available from URL: [www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm](http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm).
- Shusterman D. 1992. Critical review: the health significance of environmental odor pollution. *Arch Environ Health* 47:76–87.
- Siberry GK, Iannone R. 2000. *The Harriet Lane handbook. A manual for pediatric house officers*. 15th ed. St. Louis (MO): Mosby.
- US Environmental Protection Agency. 1999. *Guidelines for reporting of daily air quality: air quality index (AQI)*. Washington (DC): US Environmental Protection Agency. EPA-454/R-99-010.
- White MC, Berger-Frank SA, Middleton DC. in press. Addressing community concerns about asthma and air toxics. *Environ Health Perspect*.

## Answers to Pretest and Challenge Questions

### Pretest

a. Information concerning possible exposures and events that exacerbate the wheezing should be acquired. 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.

b. The differential diagnosis for wheezing in this patient includes bronchial asthma (primary diagnosis), wheezing solely associated with respiratory infections, foreign body aspiration, wheezing associated with gastroesophageal reflux, cystic fibrosis, immunodeficiency states, congenital abnormalities, and vocal cord dysfunction.

c. In the pulmonologist's office, the measurement of FEV<sub>1</sub> before and after short-acting bronchodilator therapy can be used to demonstrate reversible airway obstruction. This should be done by spirometry (for children age 7 or older who are able to cooperate), preferably using American Thoracic Society guidelines. 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 in individuals with systemic symptoms such as fever and signs suggestive of a localized lung abnormality. A total IgE level and eosinophil count should be considered as well as a differential count for eosinophils on nasal and/or sputum secretions.

d. Because the patient's asthma is mildly persistent, treatment with daily inhaled corticosteroids or mast-cell stabilizers (cromolyn or nedocromil) in combination with the use of a short-acting  $\beta_2$ -agonist MDI to provide acute relief is suggested at this time. Spirometry, done in a lung specialist's office with pre- and postbronchodilator studies, should be considered to document the presence of reversible airway obstruction. Serial peak flow measurements can be obtained at home in concert with a diary on symptoms and use of bronchodilators to help to monitor asthma control. Initiation of therapy for allergic rhinitis with a nonsedating antihistamine or inhaled nasal corticosteroids may be initiated on the first visit. The patient should be reevaluated within 2–3 weeks.

### Challenge

1. The father has an atopic background: he had seasonal hay fever as a child, and he smokes. All of these factors, in addition to his exposures to potential asthma-causing agents at work, put him at additional risk for developing asthma.
2. Sources in the home include tobacco smoking by parents, furry pets, a wood-burning stove, an inadequately maintained central heating and cooling system, and wall-to-wall carpeting that serves as a reservoir for dust mites.
3. The father should obtain from his employer the material safety data sheets (MSDS) of the materials with which he works. Any applicable MSDS should be reviewed by his physician with regard to potential health hazards and known sensitizers or associations with an increased risk of asthma. In general, work clothes should be changed at work to prevent home contamination from workplace exposures.
4. The goals for the general management of a patient with asthma should include the following:
  - confirmation of asthma diagnosis,
  - a gauge of severity,
  - prevention of chronic asthma symptoms and exacerbations day and night,
  - normal activity maintained (including exercise and other physical activities),
  - normal or near-normal lung function,
  - optimal pharmacotherapy with minimal or no adverse effects,
  - careful monitoring, and
  - education of the patient and family regarding primary and secondary preventive measures, including smoking cessation.

*Case Studies in Environmental Medicine:*

# Environmental Triggers of Asthma

## Evaluation Questionnaire and Posttest, Course Number SS3097

**Course Goal:** To increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients.

### Objectives

- Identify various environmental factors that trigger asthma exacerbations.
- Describe interventions available to mitigate environmental factors in triggering asthma.
- Identify sources of information on the impact of environmental factors on patients with asthma.
- Identify sources of information on asthma management.

## Tell Us About Yourself

Please carefully read the questions. Provide answers on the answer sheet (page 31). Your credit will be awarded based on the type of credit you select.

**1. What type of continuing education credit do you wish to receive?**

**\*\*Nurses should request CNE, not CEU. See note on page 30.**

- A. CME (for physicians)
- B. CME (for nonattending physicians)
- C. CNE (continuing nursing education)
- D. CEU (continuing education units)
- E. [Not used]
- F. [Not used]
- G. [Not used]
- H. CHES (certified health education specialist)
- I. None of the above

**2. Are you a...**

- A. Physician
- B. Pharmacist
- C. Nurse
- D. Health Educator
- E. None of the above

**3. What is your highest level of education?**

- A. High school or equivalent
- B. Associate (2-year degree)
- C. Bachelor's degree
- D. Master's degree
- E. Doctorate
- F. Other
- G. None of the above

**4. Each year, approximately how many patients with asthma do you see?**

- A. None
- B. 1–5
- C. 6–10
- D. 11–15
- E. More than 15

**5. Which of the following best describes your current occupation?**

- A. Environmental Health Professional
- B. Epidemiologist
- C. Health Educator
- D. Laboratorian
- E. Physician Assistant
- F. Industrial Hygienist
- G. Sanitarian
- H. Toxicologist
- I. Other office of clinic patient care provider
- J. Student
- K. None of the above

**6. Which of the following best describes your current work setting?**

- A. Academic (public and private)
- B. Private health care organization
- C. Public health organization
- D. Environmental health organization
- E. Nonprofit organization
- F. Other work setting

**7. Which of the following best describes the organization in which you work?**

- A. Federal government
- B. State government
- C. County government
- D. Local government
- E. Nongovernmental agency
- F. Other type of organization

## **Tell Us About the Course**

**8. How did you obtain this course?**

- A. Downloaded or printed from Web site
- B. Shared materials with colleague(s)
- C. By mail from ATSDR
- D. Not applicable

- 9. How did you first learn about this course?**
- A. State publication (or other state-sponsored communication)
  - B. *MMWR*
  - C. ATSDR Internet site or homepage
  - D. Public Health Training Network source (PHTN Web site, e-mail announcement)
  - E. Colleague
  - F. Other
- 10. What was the most important factor in your decision to obtain this course?**
- A. Content
  - B. Continuing education credit
  - C. Supervisor recommended
  - D. Previous participation in ATSDR and/or CDC/PHTN training
  - F. Convenience of self-study format
  - G. Other
- 11. How much time did you spend completing the course, evaluation, and posttest?**
- A. 1 to 1.5 hours
  - B. More than 1.5 hours but less than 2 hours
  - C. 2 to 2.5 hours
  - D. More than 2.5 hours but less than 3 hours
  - E. 3 hours or more
- 12. Please rate your level of knowledge before completing this course.**
- A. Great deal of knowledge about the content
  - B. Fair amount of knowledge about the content
  - C. Limited knowledge about the content
  - D. No prior knowledge about the content
  - E. No opinion
- 13. Please estimate your knowledge gain after completing this course.**
- A. Gained a great deal of knowledge about the content
  - B. Gained a fair amount of knowledge about the content
  - C. Gained a limited amount of knowledge about the content
  - D. Did not gain any knowledge about the content
  - E. No opinion

**Please use the scale below to rate your level of agreement with the following statements (questions 14–23) about this course.**

- A. Agree
- B. No opinion
- C. Disagree
- D. Not applicable

- 14. The objectives are relevant to the goal.**
- 15. The tables and figures are an effective learning resource.**
- 16. The content in this course was appropriate for my training needs.**
- 17. Participation in this course enhanced my professional effectiveness.**
- 18. I will recommend this course to my colleagues.**
- 19. Overall, this course enhanced my ability to understand the content.**
- 20. I am confident I can identify various environmental factors that trigger asthma exacerbations.**
- 21. I am confident I can describe interventions available to mitigate environmental factors in triggering asthma.**
- 22. I am confident I can identify sources of information on the impact of environmental factors on patients with asthma.**
- 23. I am confident I can identify sources of information on asthma management.**

## Posttest

If you wish to receive continuing education credit for this program, you must complete this posttest. Each question below contains five suggested answers, of which only one is correct.

**24. 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.

**25. 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  $FEV_1$  with an absolute minimum improvement of at least 200 mL.
- B. The use of peak flow measurements alone are almost always 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 present when the  $FEV_1$  is less than 80% of predicted value, the  $FEV_1/FVC$  ratio is reduced, and the FVC as a percent predicted is normal.

**26. 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. Being the youngest sibling in a family.
- D. Chronic allergic rhinitis.
- E. Exposure to increased concentrations of known allergens.

**27. For biologic allergens, all of these statements are true except the following**

- 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 30% humidity in the case of dust mites.
- D. Biologic allergens associated with water-damaged areas.
- E. Biologic allergens are associated with residential furry pets.

**28. Outdoor air pollution**

- A. has not been associated with short-term increases in asthma mortality and morbidity.
- B. as reflected by ozone is not associated with enhanced airway inflammatory response.
- C. standards, such as NAAQS, have not changed over the last 20 years.
- D. as reflected by AQI is reported only in metropolitan areas with populations greater than 350,000.
- E. has been identified as the primary cause for the increased incidence in asthma.

**29. Physical examination of a patient with asthma would be least likely to reveal the following:**

- 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.

**30. The treatment for dust mite and cockroach allergens includes all of the following except**

- A. cover mattresses and pillows with zippered plastic cases.
- 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.

**31. Primary prevention of adult-onset asthma includes**

- A. smoking cessation
- B. not controlling workplace exposures
- C. modifications to the home environment
- D. removing wall-to-wall carpets, particularly in bedrooms
- E. none of the above.

**32. 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. placing individual bait stations
- E. all of the above.

## Note to Nurses

CDC is accredited by the American Nurses Credentialing Center's (ANCC) Commission on Accreditation. ANCC credit is accepted by most State Boards of Nursing.

California nurses should write in "ANCC - Self-Study" for this course when applying for relicensure. A provider number is **not** needed.

Iowa nurses must be granted special approval from the Iowa Board of Nursing. Call 515-281-4823 or e-mail [marmago@bon.state.ia.us](mailto:marmago@bon.state.ia.us) to obtain the necessary application.

*Case Studies in Environmental Medicine:*

# Environmental Triggers of Asthma

## Answer Sheet, Course Number SS3097

**Instructions for submitting hard-copy answer sheet:** Circle your answers. To receive your certificate, you must answer **all** questions. Mail or fax your completed answer sheet to

**Fax:** 770-488-4178, ATTN: Continuing Education Coordinator

**Mail:** Continuing Education Coordinator  
Agency for Toxic Substances and Disease Registry  
Division of Toxicology and Environmental Medicine  
4770 Buford Hwy, NE (Mail Stop F-32)  
Atlanta, GA 30341-3717

**Be sure to fill in your name and address on the back of this form.**

**Remember, you can access the case studies online at [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/) and complete the evaluation questionnaire and posttest online at [www2.cdc.gov/atsdrce/](http://www2.cdc.gov/atsdrce/).**

**Online access allows you to receive your certificate as soon as you complete the posttest.**

1. A B C D E F G H I

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Name: \_\_\_\_\_ E-mail (not required): \_\_\_\_\_

Address: \_\_\_\_\_

Zip code: \_\_\_\_\_

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**Agency for Toxic Substances and Disease Registry**  
**Division of Toxicology and Environmental Medicine**  
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**Access the case studies online at [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/) and complete the evaluation questionnaire and posttest online at [www2.cdc.gov/atsdrce/](http://www2.cdc.gov/atsdrce/).**

**Online access allows you to receive your certificate as soon as you complete the posttest.**

# Appendix A: National Ambient Air Quality Standards (NAAQS)

EPA is required by the Clean Air Act (CAA) to set NAAQS for pollutants considered harmful to public health and the environment. Two types of national air quality standards are established.

- **Primary standards** set limits to protect public health, including the health of sensitive populations such as children, the elderly, and people with asthma.
- **Secondary standards** set limits to protect public welfare, including protection against decreased visibility and damage to animals, crops, vegetation, and buildings.

The EPA Office of Air Quality Planning and Standards has set NAAQS for six principal pollutants, which are called criteria pollutants. The six criteria pollutants are carbon monoxide, nitrogen oxide, ozone, particulate matter, sulfur dioxide, and lead.

## National Ambient Air Quality Standards

Pollutant	Standard Value*	Standard Type
Carbon monoxide (CO)		
8-hour average	9 ppm (10 mg/m <sup>3</sup> )	Primary
1-hour average	35 ppm (40 mg/m <sup>3</sup> )	Primary
Nitrogen dioxide (NO <sub>2</sub> )		
Annual arithmetic mean	0.053 ppm (100 µg/m <sup>3</sup> )	Primary and secondary
Ozone (O <sub>3</sub> )		
1-hour average <sup>†</sup>	0.12 ppm (235 µg/m <sup>3</sup> )	Primary and secondary
8-hour average	0.08 ppm (157 µg/m <sup>3</sup> )	Primary and secondary
Lead (Pb)		
Quarterly average	1.5 µg/m <sup>3</sup>	Primary and secondary
Particulate matter ≤10 micrometers (PM <sub>10</sub> )		
Annual arithmetic mean	50 µg/m <sup>3</sup>	Primary and secondary
24-hour average	150 µg/m <sup>3</sup>	Primary and secondary
Particulate matter ≤2.5 micrometers (PM <sub>2.5</sub> )		
Annual arithmetic mean	15 µg/m <sup>3</sup>	Primary and secondary
24-hour average	65 µg/m <sup>3</sup>	Primary and secondary
Sulfur dioxide (SO <sub>2</sub> )		
Annual arithmetic mean	0.03 ppm (80 µg/m <sup>3</sup> )	Primary
24-hour average	0.14 ppm (365 µg/m <sup>3</sup> )	Primary
3-hour average	0.50 ppm (1,300 µg/m <sup>3</sup> )	Secondary

Last NAAQS revision 1997.

\*Parenthetical value is an approximately equivalent concentration. Units of measure for the standards are parts per million (ppm), milligrams per cubic meter of air (mg/m<sup>3</sup>), and micrograms per cubic meter of air (µg/m<sup>3</sup>).

<sup>†</sup>The ozone 1-hour standard applies only to areas that were designated nonattainment when the ozone 8-hour standard was adopted in July 1997.





**DEPARTMENT OF HEALTH  
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