# Trigger control to enhance asthma management

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**INTRODUCTION** — Asthma is a chronic lung disease characterized by airway obstruction, inflammation, and hyperresponsiveness [1]. Identifying and avoiding asthma "triggers" is essential in preventing asthma flare-ups. Although "triggers" are often thought of as airborne agents that can bring on an attack, there are a variety of exposures that can cause or exacerbate symptoms, including the following categories of stimuli [1]:

- Inhaled allergens
- Respiratory infections
- Inhaled respiratory irritants (including tobacco smoke and cold dry air)
- Hormonal fluctuations
- Medications
- Physical activity
- Emotional state (eg, anxiety, sudden upsets)

It is recommended that clinicians assess patients for a range of factors that can make asthma worse, including inhalant allergens, tobacco smoke, indoor/outdoor pollutants and irritants, workplace exposures, gastroesophageal reflux and other comorbidities, rhinosinusitis, sulfite sensitivity, and medication intolerances [1]. Such assessment should take into account the amount of exposure, the patient's sensitivity to allergens encountered, and the clinical significance of the exposure/sensitivity in the context of the person's medical history [1]. Exposures at home, daycare, school, work, and regular leisure sites should be reviewed. Questions about work environment are particularly important, as industrial or occupational exposures are responsible for up to 15 percent of cases of adult asthma [2,3].

The recommendations in this topic review are consistent with "The National Asthma Education and Prevention Program: Expert Panel Report 3, Guidelines for the Diagnosis and Management of Asthma – Full Report 2007" [1]. The full text is available at the website of the National Heart, Lung, and Blood Institute [1]. This document provides a set of questions that can be used to determine which triggers and environmental factors contribute to a patient's asthma (table 1).

After the patient has identified those elements that trigger his or her breathing problems, a plan can be devised to enable the patient to reduce the exposure.

Patients may have predictable exposures to avoidable triggers, and in these situations, the following may be possible [1]:

- Complete avoidance of the trigger (eg, do not own pets if allergic to them, mop or wet wipe instead of sweeping or dusting).
- Limit exposure to the trigger if it cannot be completely avoided (eg, leave the room if someone starts smoking, move to another seat if someone with strong perfume sits near you, have someone else dust and clean the house if dust mite allergic).

- Take an extra dose of bronchodilator and an antihistamine before trigger exposure. This approach should be implemented only if the first two options are not feasible. Patients should consult with their clinician to be sure before instituting this approach, and they should be careful not to exceed the amount of normally prescribed medication.

More commonly, trigger exposure occurs on a chronic and unpredictable basis. A description of factors that may contribute to asthma severity followed by suggestions for management of these factors will be reviewed here. A general approach to asthma management is provided separately. (See "An overview of asthma management".)

**RESPIRATORY INFECTIONS** — Colds, influenza, respiratory syncytial virus (RSV), bronchitis, ear infections, sinus infections, and pneumonia are very common asthma triggers because they can cause airway inflammation and increased mucus production [1,4]. Asthma attacks that occur with a respiratory infection are frequently more severe than those occurring at other times.

One study found that clusters of asthma hospitalizations in school-aged children in Canada occurred predictably after they returned to school following summer vacation and other breaks [5]. Specifically, there was a "September asthma epidemic" approximately 18 days after Labor Day, with a lesser increase in attacks two days later in preschool children and six days later in adults. Viral infections were the presumed cause.

**Patient recommendations** — Patients should be advised to wash hands frequently, avoid people with infections when possible, get adequate amounts of sleep, and use prescribed treatments for upper respiratory tract symptoms (eg, intranasal glucocorticoids, decongestants) [6].

Vaccination with inactivated influenza virus does not increase the risk of an asthma exacerbation and yearly vaccination is encouraged in order to reduce the risks of complications of influenza infection (ie, pneumonia) [1,7,8]. In addition, there is evidence that pneumococcal vaccination is beneficial for adults with asthma. (See "Approach to immunizations in healthy adults" and "Pneumococcal vaccination in adults".)

**ALLERGENS**

**Inhalant allergens** — Allergy is a primary cause of asthma symptoms [2]. According to the National Asthma Education and Prevention Program (NAEPP) Guidelines, the "first and most important step in controlling allergen-induced asthma is to reduce exposure to relevant indoor and outdoor allergens" [1]. Thus, the clinician must take a thorough history to determine if the patient has significant allergies. A questionnaire has been published to assist the clinician in assessing the relationship between allergen exposure and the presence of symptoms (table 1) [1].

Allergy testing (ie, skin testing or in vitro immunoglobulin E [IgE] immunoassays) can also be helpful and should be considered in patients with persistent asthma who are exposed to perennial allergens [9]. It is important that only substances to which the patient has been exposed be included in the tests and that the results are interpreted in the context of other diagnostic assessments, including medical history and physical examination, because a positive skin test or in vitro test result only means that the patient is sensitized to that allergen and has the potential to develop symptoms when exposed to that allergen. The diagnosis of allergy requires that the patient also has a history consistent with symptoms following exposure. (See "Overview of skin testing for allergic disease" and "Overview of in vitro allergy tests".)

Common inhalant allergens that can induce asthma exacerbations include [10-15]:

- Animal allergens (both pets and pests: cats, dogs, rodents, birds)
- House-dust mites (present in all but arid or high altitude regions)
- Cockroaches
- Indoor and outdoor fungi (mold, mildew)

- Outdoor plant allergens (tree, grass, ragweed pollen)

Measures for reducing exposure to indoor and outdoor allergens are detailed separately. (See "Allergen avoidance in the treatment of asthma and allergic rhinitis".)

In addition, the NAEPP Guidelines recommend allergen immunotherapy in patients for whom [1]:

- There is clear evidence that exposure to an unavoidable allergen results in symptoms
- Symptoms occur all or most of the year
- There is difficulty in controlling symptoms with pharmacologic management

(See "Subcutaneous immunotherapy for allergic disease: Indications and efficacy".)

**Food allergens** — Food allergens RARELY cause isolated asthma without other symptoms, in contrast to inhalant allergens. However, patients with food allergy may exhibit asthma symptoms as part of food-induced anaphylaxis. Anaphylaxis should be suspected if a patient (especially a child or young adult) develops severe asthma symptoms soon after eating a food allergen, in the absence of other apparent triggers. (See "Clinical manifestations of food allergy: An overview".)

In certain uncommon circumstances, food allergens can be aerosolized in sufficient amounts that highly-sensitive patients with food allergy can develop asthmatic symptoms. Examples include seafood, egg, or cow's milk-allergic patients reacting to steam, vapors, or sprays from the cooking or processing of these foods, or wheat-allergic patients reacting to inhaled flour. The situations in which this has been reported are reviewed separately. (See "Management of food allergy: Avoidance", section on 'Skin contact and inhalation'.)

Sulfite sensitivity, although not an IgE-mediated food allergy, can present with isolated asthma symptoms triggered by the ingestion of foods that are treated with sulfites to prevent spoilage and discoloration. Sulfite sensitivity is rare and largely seen in patients with severe asthma. The foods include wine, vinegar, dried fruits, processed potato products, and others (table 2). However, it is not necessary to advise patients to avoid these foods unless there is clinical suspicion that they have reacted in the past to such foods [16]. (See "Allergic and asthmatic reactions to food additives", section on 'Sulfites and related compounds'.)

**Occupational allergens** — Patients may develop asthma for the first time as a result of exposure to allergens in the workplace. This is referred to as occupational asthma and has been attributed to certain low molecular weight chemicals and high molecular weight organic materials (table 3). Chemical compounds that most commonly aggravate symptoms include toluene diisocyanates, trimellitic anhydrides, enzymes, and wood dusts. Occupational asthma is distinct from "work-aggravated asthma," or preexisting asthma that worsens in the workplace setting. These disorders are reviewed in greater detail separately. (See "Occupational asthma: Definitions, epidemiology, causes, and risk factors".)

**Patient recommendations** — If the patient suspects an allergic trigger, he/she should initially be instructed to keep a diary (recording food eaten, activity/environment, and peak expiratory flow readings) to see if there is a correlation between symptoms, lung function, and the suspected trigger.

**IRRITANTS (INCLUDING CIGARETTE SMOKE)** — A variety of irritants can induce asthma symptoms, including cigarette smoke, fireplace smoke, ashes, aerosol sprays, perfumes, phthalates [17], cooking odors, musty odors, shower steam, traffic fumes, air pollution, desert dust, and workplace irritants [18-28].

Cigarette smoke is a common airway irritant. Among patients with asthma, smokers have more severe symptoms, increased rates of hospitalization, accelerated decline in lung function, and impaired responses to inhaled and systemic glucocorticoids than nonsmokers [18-20,29-33]. Children with environmental tobacco smoke exposure have a higher risk of developing asthma, more severe asthma symptoms, and more frequent exacerbations [34-36]. Smoking cessation is associated with improved lung function [21]. In addition, exposure to tobacco smoke exacerbates inflammatory airway

responses to allergens [37]. Clinicians should advise patients with asthma not to smoke or be exposed to second-hand smoke [1]. Pediatricians should discuss smoking habits with parents and caregivers of pediatric patients. If a parent is unwilling or unable to quit smoking, discuss not smoking in the same room in which children sleep or play. If feasible, instruct parents and caregivers to restrict their smoking to outside of the home. (See "Overview of smoking cessation management in adults".)

Fumes from unvented gas stoves have been associated with wheezing in children [22]. Changing to a nonpolluting, more effective indoor heat system is associated with an improved sense of well-being and reduced asthma symptoms in children, although not an improvement in lung function [23]. Chlorine-based household cleaners, cleaning fluids, and powders can also trigger asthma. Newly-installed carpeting, paint, and furnishings in homes and offices can release volatile organic compounds and formaldehyde [24,25].

Increases in ambient particulate matter, elemental carbon/soot, nitrogen dioxide, and ozone have been associated with an increase in reports of wheeze, sales of short-acting bronchodilator medication, and hospitalizations for asthma [26,38,39]. Days with high concentrations of desert dust are associated with an increase in asthma hospitalizations [28]. Desert dust contains quartz particles (crystalline silica), which have been associated with respiratory disease in occupationally exposed persons. Dust particles originating from desert dust storms in one location can be transported across the atmosphere to affect wide regions of the globe.

Workplace irritants, such as glacial acetic acid and chlorine, are common among factory workers and professional cleaners. (See "Reactive airways dysfunction syndrome and irritant-induced asthma".)

In addition to these commonly encountered irritants, there may be unusual irritants in certain settings, such as red tide in coastal communities and tourist destinations [40].

**Patient recommendations** — Avoid cigarette smoke by not smoking, not allowing others to smoke inside the home, and avoiding smoke in public buildings. Use unscented and nonaerosol products. Use blankets made of synthetic materials. Do not use wood burning stoves, fireplaces, or unvented stoves or heaters. Work with a clinician on strategies to reduce workplace exposure without threatening loss of employment.

**TEMPERATURE AND WEATHER** — Temperature and weather conditions affect asthma in some patients.

- Temperature and humidity may play a role in exercise-induced asthma [41-43]. While the precise mechanism is not clear, the inhalation of cold and dry air appears to increase bronchoconstriction during or shortly after exercise. One theory suggests that increased breathing during exercise causes water loss in the airways. This water loss leads to an exchange of molecules in the cells lining the airways, causing inflammation that ultimately leads to bronchoconstriction [42].

  A second theory suggests increased breathing during exercise leads to cooling of the airways. This cooling is followed by the rapid flow of blood into airway blood vessels and resultant edema [42,43]. (See "Exercise-induced bronchoconstriction".)

- Hot, humid air can cause bronchoconstriction, as demonstrated in animal studies and a small study of humans [44]. The response to hot, humid air was blunted by pretreatment with inhaled ipratropium, suggesting that the bronchoconstriction is mediated by vagal bronchopulmonary C-fiber sensory nerves.

- Wet conditions and thunderstorms have also been linked to asthma exacerbations. Studies of weather events have found increased levels of respiratory allergens present in the air, particularly pollen. Conditions at the beginning of a thunderstorm can cause pollen grains to rupture, increasing the concentration of pollen debris. This debris can be inhaled into the lower airways, triggering an asthma exacerbation [45,46].

- Conditions related to climate change are predicted to increase exposure to asthma triggers. These may include

more hot sunny days that increase ozone-related asthma symptoms, rises in sea level or altered rainfall that may affect the dampness of indoor environments leading to more dust mite and mold growth, and higher ambient carbon dioxide levels that may increase exposure to allergens by lengthening the pollen season [47].

**Patient recommendations** — Simply being aware of these triggers can help patients prepare for different environments and anticipate problems to gain a better sense of control over their asthma. Masks and scarves that contain devices to trap heat and moisture when a person breathes out and warms inhaled cold air may help persons with asthma who work, exercise, or spend time in cold temperatures [48]. Patients whose asthma is triggered by pollen allergy should be made aware that the conditions at the beginning of a thunderstorm during pollen season can be particularly troublesome. Patients whose asthma is triggered by weather changes can use a rescue medication prior to exercise in cold or dry conditions or at the beginning of storms during pollen seasons.

**PHYSICAL ACTIVITY** — Exercise is a potential asthma trigger that should **not** be avoided. Aerobic exercise strengthens the cardiovascular system and may lessen the sensitivity to asthma triggers [49-51]. However, it is important for persons with asthma who are not in a regular pattern of exercise to build-up their activity level slowly to minimize the risk of inducing asthma. (See "Exercise-induced bronchoconstriction".)

**Patient recommendations** — Take medications on schedule. Warm up gradually before beginning strenuous activity. Consult your clinician about taking medication prior to physical activity. Avoid exercising outdoors in extremely cold weather because cold exposure can trigger asthma. In addition, use of an intermediate-acting inhaled beta-agonist (such as albuterol) 10 minutes before exercise can substantially attenuate exercise-induced symptoms. Other options for the management of exercise-triggered asthma include leukotriene-modifying agents and pretreatment with inhaled cromoglycates (in the United States, only the nebulized form of cromolyn is available). (See "Exercise-induced bronchoconstriction", section on 'Management' and "Beta agonists in asthma: Acute administration and prophylactic use", section on 'Use in exercise-induced asthma' and "Agents affecting the 5-lipoxygenase pathway in the treatment of asthma" and "The use of chromones (cromoglycates) in the treatment of asthma", section on 'Clinical use'.)

**HORMONAL FLUCTUATIONS** — Hormonal fluctuations associated with the menstrual cycle and with pregnancy can affect the frequency and severity of asthma symptoms in some patients.

**Perimenstrual asthma** — Worsening of asthma symptoms prior to or during menstruation, known as perimenstrual asthma, has been reported in 20 to 40 percent of women with asthma [52-55] and in 17 percent of women (ages 12 to 50) with severe asthma [56]. One study found emergency department visits due to asthma occurred slightly more often during the preovulatory phase (days 5 to 11) and the perimenstrual phase of the cycle (day 26 to day 4 of the next cycle) [57]. The pathophysiology responsible for this phenomenon is unclear, although increases in estrogen and progesterone levels have been associated with changes in markers of atopy and asthma, such as fractional exhaled nitric oxide and skin test diameters [58]. Women with hormonally associated asthma tend to have more severe disease than women whose asthma is unaffected by hormone levels, and patients with severe and fatal attacks in association with menstruation have been reported [52,56,59,60].

As women often take nonsteroidal antiinflammatory drugs (NSAIDs) to control premenstrual syndrome and dysmenorrhea, it is important to determine that a perimenstrual flare in asthma symptoms is not caused by ingestion of an NSAID by a patient with aspirin-exacerbated asthma. Aspirin sensitivity may be more prevalent in women with perimenstrual asthma [56]. (See "Aspirin-exacerbated respiratory disease".)

The optimal pharmacologic management of perimenstrual flares of asthma has not been determined [61]:

- In one small trial, hormonal manipulation with oral birth control pills was not helpful [62].
- Several case reports of successful treatment with intramuscular injections of progesterone have also been published [63,64]. Information about treatment with oral progesterone in this setting is lacking and hormonal

interventions should only be considered in consultation with a gynecologist or endocrinologist.

- Leukotriene receptor antagonists appeared protective against perimenstrual worsening of asthma in several small series [65,66].

**Pregnancy** — Pregnancy can aggravate asthma in approximately one-third of women, while one-third experience no change, and one-third experience symptomatic improvement. (See "Physiology and clinical course of asthma in pregnancy".)

**Patient recommendations** — Anticipate possible exacerbations in association with menstruation, increase medication if necessary, and avoid other exacerbating factors. A trial of an antileukotriene agent is suggested. Asthma should be monitored carefully during pregnancy. (See "Management of asthma during pregnancy".)

**MEDICATIONS** — Certain medications can aggravate asthma, including:

- Nonselective beta blockers (see "Treatment of hypertension in asthma and COPD")
- Aspirin and other nonsteroidal antiinflammatory drugs (NSAIDs) (see "Aspirin-exacerbated respiratory disease")

Angiotensin-converting enzyme (ACE) inhibitors can cause cough, which could be mistaken for increased asthma symptoms. Cough related to these agents is reviewed separately. (See "Major side effects of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers", section on 'Cough'.)

For many years, it was believed that excessive drying of bronchial secretions by first generation antihistamines (due to their anticholinergic properties) would adversely affect asthma. However, this effect was never demonstrated to be clinically significant. Newer, nonsedating antihistamines have less of an anticholinergic effect and do not adversely affect asthma [67].

**Patient recommendations** — Check with a clinician before taking any new medications that may contain drugs that aggravate asthma.

**EMOTIONAL FACTORS** — Depression, chronic stress, and stressors, such as exposure to community violence, are associated with greater rates of asthma exacerbations in patients with asthma [68,69]. Parental depression and stress are associated with more severe asthma in children [70].

**Patient recommendations** — Emotional stress is a common trigger for asthma. For some patients, medications to manage depression or anxiety can be helpful. Psychologists and family counselors can assist patients and parents of children with asthma with stress management and provide advice if asthma disrupts a patient's or family's personal life.

**COEXISTENT MEDICAL CONDITIONS** — Clinicians should be vigilant for comorbid conditions in patients with poorly-controlled asthma. These conditions can either contribute to or mimic asthma symptoms.

In children, additional contributing conditions include foreign body aspiration, bronchopulmonary dysplasia, and cystic fibrosis [1]. (See "Approach to wheezing in children".)

In adolescents and adults, these conditions include chronic obstructive pulmonary disease (COPD)/emphysema, allergic bronchopulmonary aspergillosis, gastroesophageal reflux, obesity, obstructive sleep apnea, rhinitis/sinusitis, vocal cord dysfunction, and depression/anxiety. These conditions are reviewed separately. (See "Allergic bronchopulmonary aspergillosis" and "Gastroesophageal reflux and asthma" and "Clinical presentation and diagnosis of obstructive sleep apnea in adults" and "An overview of rhinitis" and "Chronic rhinosinusitis: Clinical manifestations, pathophysiology, and diagnosis".)

**INFORMATION FOR PATIENTS** — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and

they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient information: Avoiding asthma triggers (The Basics)")
- Beyond the Basics topic (see "Patient information: Trigger avoidance in asthma (Beyond the Basics)")

**SUMMARY AND RECOMMENDATIONS** — Identifying and avoiding asthma triggers is essential in preventing and minimizing asthma exacerbations. (See 'Introduction' above.)

- Common triggers for asthma exacerbations include respiratory tract infections, airborne allergens (for patients with allergic asthma), inhaled irritants (eg, tobacco smoke), extremes of temperature and humidity (eg, very cold, dry air), and physical exercise. (See 'Respiratory infections' above and 'Allergens' above and 'Irritants (including cigarette smoke)' above and 'Temperature and weather' above and 'Physical activity' above.)
- Other triggers that are important to certain subsets of patients include stress and extremes of emotion, hormonal fluctuations, certain weather events (ie, thunderstorms), medications (eg, nonsteroidal antiinflammatory drugs [NSAIDs], nonselective beta blockers), and sulfites added to commercially-prepared foods. (See 'Emotional factors' above and 'Hormonal fluctuations' above and 'Medications' above and "Allergic and asthmatic reactions to food additives".)
- Simply being cognizant of which triggers are important for an individual can help clinicians and patients prepare for different environments and anticipate problems, leading to improved asthma control. In some cases, behavior modification is the primary intervention (ie, avoidance of cigarette smoke). In others, specific medical interventions may be appropriate (eg, allergen immunotherapy for patients with allergic triggers or strict avoidance of NSAIDs in patients sensitive to these drugs).



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## GRAPHICS

### Assessment questions* for environmental and other factors that can make asthma worse

| Inhalant allergens |
|---|
| **Does the patient have symptoms year round? (If yes, ask the following questions. If no, see next set of questions).** |
| Does the patient keep pets indoors? What type? |
| Does the patient have moisture, dampness, or a moldy odor in any room of his or her home (eg, basement)? (Suggests house dust mites, molds). |
| Does the patient have mold visible in any part of his or her home? (Suggests molds). |
| Has the patient seen cockroaches or rodents in his or her home in the past month? (Suggests significant exposure). |
| Assume exposure to house dust mites unless patient lives in a semiarid region. However, if a patient living in a semiarid region uses a swamp cooler, exposure to house dust mites must still be assumed. |
| **Do symptoms get worse at certain times of the year? (If yes, ask when symptoms occur).** |
| Early spring? (Trees). |
| Late spring? (Grasses). |
| Late summer to autumn? (Weeds). |
| Summer and fall? (Alternaria, Cladosporium, mites). |
| Cold months in temperate climates? (Suggests indoor allergens such as animal dander). |
| **Tobacco smoke** |
| Does the patient smoke? |
| Does anyone smoke at home or work? |

| Workplace exposures |
|---|
| Does the patient cough or wheeze during the week, but not on weekends when away from work? |
| Do the patient's eyes and nasal passages get irritated soon after arriving at work? |
| Do coworkers have similar symptoms? |
| What substances are used in the patient's worksite? (Assess for sensitizers). |
| **Rhinitis** |
| Does the patient have constant or seasonal nasal congestion, runny nose, and/or postnasal drip? |
| **Gastroesophageal reflux disease (GERD)** |
| Does the patient have heartburn? |
| Does food sometimes come up into the patient's throat? |
| Has the patient had coughing, wheezing, or shortness of breath at night in the past four weeks? |
| Does the infant vomit, followed by cough, or have wheezy cough at night? Are symptoms worse after feeding? |
| **Sulfite sensitivity ¶** |
| Does the patient have wheezing, coughing, or shortness of breath after eating shrimp, dried fruit, or processed potatoes or after drinking beer or wine? |
| **Medication sensitivities and contraindications** |
| What medications does the patient use now (prescription and nonprescription)? |
| Does the patient use eye drops ¶? What type? |

| | |
|---|---|
| Does anyone smoke at the child's daycare? | Does the patient use any medications that contain beta-blockers or ACE inhibitors¶? |
| **Indoor/outdoor pollutants and irritants** | Does the patient ever take aspirin or other nonsteroidal antiinflammatory drugs? |
| Is a wood-burning stove or fireplace used in the patient's home? | Has the patient ever had symptoms of asthma after starting or taking any of these medications? |
| Are there unvented stoves or heaters in the patient's home? | |
| Does the patient have contact with other smells or fumes from perfumes, cleaning agents, or sprays? | |
| Have there been recent renovations or painting in the home? | |

ACE: angiotensin-converting enzyme.
* These questions are examples and do not represent a standardized assessment or diagnostic instrument. The validity and reliability of these questions have not been assessed.
¶ Rare issue in children.

*Reproduced from: National Heart, Blood, and Lung Institute Expert Panel Report 3 (EPR 3): Guidelines for the Diagnosis and Management of Asthma. NIH Publication no. 08-4051, 2007.*

Graphic 80507 Version 9.0

## Sulfite-containing foods

| High content | Low content (<10 ppm)* |
|---|---|
| Dried fruit (excluding dark raisins and prunes) | Corn starch |
| Lemon juice (nonfrozen) | Hominy |
| Lime juice (nonfrozen) | Frozen potatoes |
| Wine | Maple syrup |
| Molasses | Imported jams and jellies |
| Sauerkraut juice | Fresh mushrooms |
| Grape juice (white, white sparkling, pink sparkling, red sparkling) | Malt vinegar |
| **Moderate content** | Dried cod |
| Dried potatoes | Canned potatoes |
| Wine vinegar | Beer |
| Gravies, sauces | Dry soup mix |
| Fruit topping | Soft drinks |
| Maraschino cherries | Instant tea |
| Pectin | Pizza dough (frozen) |
| Shrimp (fresh) | Pie dough |
| Sauerkraut | Sugar (especially beet sugar) |
| Pickled peppers | Gelatin |
| Pickled cocktail onions | Coconut |
| Pickles/relishes | Fresh fruit salad |
| | Domestic jams and jellies |
| | Crackers |
| | Cookies |
| | Grapes |
| | High fructose corn syrup |

* Foods with low sulfite content have not been implicated in inducing reactions in sulfite-sensitive individuals.

*Courtesy of Ronald A Simon, MD.*

Graphic 66943 Version 5.0

## Major causes of occupational asthma

| | Occupation at risk |
|---|---|
| **Low molecular weight chemicals** | |
| Isocyanates (eg, toluene diisocyanate, diphenylmethane diisocyanate, hexamethylene diisocyanate, naphthalene diisocyanate) | Polyurethane workers, roofers, insulators, painters |
| Anhydrides (eg, trimellitic anhydride, phthalic anhydride) | Manufacturers of paint, plastics, epoxy resins |
| Metals (eg, chromic acid, potassium dichromate, nickel sulfate, vanadium, platinum salts) | Platers, welders, metal and chemical workers |
| Drugs (eg, beta lactam agents, opiates, other) | Pharmaceutical workers, farm workers, health professionals |
| Wood dust (eg, Western red cedar, maple, oak, exotic woods) | Carpenters, woodworkers |
| Dyes and bleaches (eg, anthraquinone, carmine, henna extract, persulfate, reactive dyes) | Fabric and fur dyers, hairdressers |
| Amines | Chemists, cleaners, plastic manufacturers |
| Glues and resins (eg, acrylates, epoxy) | Plastic manufacturers |
| Miscellaneous (eg, formaldehyde, glutaraldehyde, ethylene oxide, pyrethrin, polyvinyl chloride vapor) | Laboratory workers, textile workers, paint sprayers, health professionals |
| **High molecular weight organic materials** | |
| Animal proteins (eg, domestic and laboratory animals, fish and seafood) | Farmers, veterinarians, poultry processors, fish and seafood processors |
| Flours and cereals | Bakers, food processors, dock workers |
| Enzymes (eg, pancreatic extracts, papain, trypsin, Bacillus subtilis, bromelain, pectinase, amylase, lipase) | Bakers, food processors, pharmaceutical workers, plastic workers, detergent manufacturers |
| Plant proteins (eg, wheat, grain dust, coffee beans, tobacco dust, cotton, tea, latex, psyllium, various flours) | Bakers, farmers, food and plant processors, health professionals, textile workers |

Graphic 66185 Version 3.0

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