

Chapter 5—Indoor Air Pollutants and Toxic Materials

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Chapter 5: Indoor Air Pollutants and Toxic Materials

“Walking into a modern building can sometimes be compared to placing your head inside a plastic bag that is filled with toxic fumes.”

John Bower
Founder, *Healthy House Institute*

Introduction

We all face a variety of risks to our health as we go about our day-to-day lives. Driving in cars, flying in airplanes, engaging in recreational activities, and being exposed to environmental pollutants all pose varying degrees of risk. Some risks are simply unavoidable. Some we choose to accept because to do otherwise would restrict our ability to lead our lives the way we want. Some are risks we might decide to avoid if we had the opportunity to make informed choices. Indoor air pollution and exposure to hazardous substances in the home are risks we can do something about.

In the last several years, a growing body of scientific evidence has indicated that the air within homes and other buildings can be more seriously polluted than the outdoor air in even the largest and most industrialized cities. Other research indicates that people spend approximately 90% of their time indoors. Thus, for many people, the risks to health from exposure to indoor air pollution may be greater than risks from outdoor pollution.

In addition, people exposed to indoor air pollutants for the longest periods are often those most susceptible to their effects. Such groups include the young, the elderly, and the chronically ill, especially those suffering from respiratory or cardiovascular disease [1].

Indoor Air Pollution

Numerous forms of indoor air pollution are possible in the modern home. Air pollutant levels in the home increase if not enough outdoor air is brought in to dilute emissions from indoor sources and to carry indoor air pollutants out of the home. In addition, high temperature and humidity levels can increase the concentration of some pollutants. Indoor pollutants can be placed into two groups, biologic and chemical.

Biologic Pollutants

Biologic pollutants include bacteria, molds, viruses, animal dander, cat saliva, dust mites, cockroaches, and

pollen. These biologic pollutants can be related to some serious health effects. Some biologic pollutants, such as measles, chickenpox, and influenza are transmitted through the air. However, the first two are now preventable with vaccines. Influenza virus transmission, although vaccines have been developed, still remains of concern in crowded indoor conditions and can be affected by ventilation levels in the home.

Common pollutants, such as pollen, originate from plants and can elicit symptoms such as sneezing, watery eyes, coughing, shortness of breath, dizziness, lethargy, fever, and digestive problems. Allergic reactions are the result of repeated exposure and immunologic sensitization to particular biologic allergens.

Although pollen allergies can be bothersome, asthmatic responses to pollutants can be life threatening. Asthma is a chronic disease of the airways that causes recurrent and distressing episodes of wheezing, breathlessness, chest tightness, and coughing [2]. Asthma can be broken down into two groups based on the causes of an attack: extrinsic (allergic) and intrinsic (nonallergic). Most people with asthma do not fall neatly into either type, but somewhere in between, displaying characteristics of both classifications. Extrinsic asthma has a known cause, such as allergies to dust mites, various pollens, grass or weeds, or pet danders. Individuals with extrinsic asthma produce an excess amount of antibodies when exposed to triggers. Intrinsic asthma has a known cause, but the connection between the cause and the symptoms is not clearly understood. There is no antibody hypersensitivity in intrinsic asthma. Intrinsic asthma usually starts in adulthood without a strong family history of asthma. Some of the known triggers of intrinsic asthma are infections, such as cold and flu viruses, exercise and cold air, industrial and occupational pollutants, food additives and preservatives, drugs such as aspirin, and emotional stress. Asthma is more common in children than in adults, with nearly 1 of every 13 school-age children having asthma [3]. Low-income African-Americans and certain Hispanic populations suffer disproportionately, with urban inner cities having particularly severe problems. The impact on neighborhoods, school systems, and health care facilities from asthma is severe because one-third of all pediatric emergency room visits are due to asthma, and it is the fourth most prominent cause of physician office visits. Additionally, it is the leading cause of school absenteeism—14 million school

days lost each year—from chronic illness [4]. The U.S. population, on the average, spends as much as 90% of its time indoors. Consequently, allergens and irritants from the indoor environment may play a significant role in triggering asthma episodes. A number of indoor environmental asthma triggers are biologic pollutants. These can include rodents (discussed in Chapter 4), cockroaches, mites, and mold.

Cockroaches

The droppings, body parts, and saliva of cockroaches can be asthma triggers. Cockroaches are commonly found in crowded cities and in the southern United States. Allergens contained in the feces and saliva of cockroaches can cause allergic reactions or trigger asthma symptoms. A national study by Crain et al. [5] of 994 inner-city allergic children from seven U.S. cities revealed that cockroaches were reported in 58% of the homes. The Community Environmental Health Resource Center reports that cockroach debris, such as body parts and old shells, trigger asthma attacks in individuals who are sensitized to cockroach allergen [6]. Special attention to cleaning must be a priority after eliminating the presence of cockroaches to get rid of the presence of any allergens left that can be asthma triggers.

House Dust Mites

Another group of arthropods linked to asthma is house dust mites. In 1921, a link was suggested between asthmatic symptoms and house dust, but it was not until 1964 that investigators suggested that a mite could be responsible. Further investigation linked a number of mite species to the allergen response and revealed that humid homes have more mites and, subsequently, more allergens. In addition, researchers established that fecal pellets deposited by the mites accumulated in home fabrics and could become airborne via domestic activities such as vacuuming and dusting, resulting in inhalation by the inhabitants of the home. House dust mites are distributed worldwide, with a minimum of 13 species identified from house dust. The two most common in the United States are the North American house dust mite (*Dermatophagoides farinae*) and the European house dust mite (*D. pteronyssinus*). According to Lyon [7], house dust mites thrive in homes that provide a source of food and shelter and adequate humidity. Mites prefer relative humidity levels of 70% to 80% and temperatures of 75°F to 80°F (24°C to 27°C). Most mites are found in bedrooms in bedding, where they spend up to a third of their lives. A typical used mattress may have from 100,000 to 10 million mites in it. In addition, carpeted floors, especially long, loose pile carpet, provide a

microhabitat for the accumulation of food and moisture for the mite, and also provide protection from removal by vacuuming. The house dust mite's favorite food is human dander (skin flakes), which are shed at a rate of approximately 0.20 ounces per week.

A good microscope and a trained observer are imperative in detecting mites. House dust mites also can be detected using diagnostic tests that measure the presence and infestation level of mites by combining dust samples collected from various places inside the home with indicator reagents [7]. Assuming the presence of mites, the precautions listed below should be taken if people with asthma are present in the home:

- Use synthetic rather than feather and down pillows.
- Use an approved allergen barrier cover to enclose the top and sides of mattresses and pillows and the base of the bed.
- Use a damp cloth to dust the plastic mattress cover daily.
- Change bedding and vacuum the bed base and mattress weekly.
- Use nylon or cotton cellulose blankets rather than wool blankets.
- Use hot (120°F–130°F [49°C–54°C]) water to wash all bedding, as well as room curtains.
- Eliminate or reduce fabric wall hangings, curtains, and drapes.
- Use wood, tile, linoleum, or vinyl floor covering rather than carpet. If carpet is present, vacuum regularly with a high-efficiency particulate air (HEPA) vacuum or a household vacuum with a microfiltration bag.
- Purchase stuffed toys that are machine washable.
- Use fitted sheets to help reduce the accumulation of human skin on the mattress surface.

HEPA vacuums are now widely available and have also been shown to be effective [8]. A conventional vacuum tends to be inefficient as a control measure and results in a significant increase in airborne dust concentrations, but can be used with multilayer microfiltration collection

bags. Another approach to mite control is reducing indoor humidity to below 50% and installing central air conditioning.

Two products are available to treat house dust mites and their allergens. These products contain the active ingredients benzyl benzoate and tannic acid.

Pets

According to the U.S. Environmental Protection Agency (EPA) [9], pets can be significant asthma triggers because of dead skin flakes, urine, feces, saliva, and hair. Proteins in the dander, urine, or saliva of warm-blooded animals can sensitize individuals and lead to allergic reactions or trigger asthmatic episodes. Warm-blooded animals include dogs, cats, birds, and rodents (hamsters, guinea pigs, gerbils, rats, and mice). Numerous strategies, such as the following, can diminish or eliminate animal allergens in the home:

- Eliminate animals from the home.
- Thoroughly clean the home (including floors and walls) after animal removal.
- If pets must remain in the home, reduce pet exposure in sleeping areas. Keep pets away from upholstered furniture, carpeted areas, and stuffed toys, and keep the pets outdoors as much as possible.

However, there is some evidence that pets introduced early into the home may prevent asthma. Several studies have shown that exposure to dogs and cats in the first year of life decreases a child's chances of developing allergies [10] and that exposure to cats significantly decreases sensitivity to cats in adulthood [11]. Many other studies have shown a decrease in allergies and asthma among children who grew up on a farm and were around many animals [12].

Mold

People are routinely exposed to more than 200 species of fungi indoors and outdoors [13]. These include moldlike fungi, as well as other fungi such as yeasts and mushrooms. The terms “mold” and “mildew” are nontechnical names commonly used to refer to any fungus that is growing in the indoor environment. Mold colonies may appear cottony, velvety, granular, or leathery, and may be white, gray, black, brown, yellow, greenish, or other colors. Many reproduce via the production and dispersion of spores. They usually feed on dead organic

matter and, provided with sufficient moisture, can live off of many materials found in homes, such as wood, cellulose in the paper backing on drywall, insulation, wallpaper, glues used to bond carpet to its backing, and everyday dust and dirt.

Certain molds can cause a variety of adverse human health effects, including allergic reactions and immune responses (e.g., asthma), infectious disease (e.g., histoplasmosis), and toxic effects (e.g., aflatoxin-induced liver cancer from exposure to this mold-produced toxin in food) [14]. A recent Institute of Medicine (IOM) review of the scientific literature found sufficient evidence for an association between exposure to mold or other agents in damp indoor environments and the following conditions: upper respiratory tract symptoms, cough, wheeze, hypersensitivity pneumonitis in susceptible persons, and asthma symptoms in sensitized persons [15]. A previous scientific review was more specific in concluding that sufficient evidence exists to support associations between fungal allergen exposure and asthma exacerbation and upper respiratory disease [13]. Finally, mold toxins can cause direct lung damage leading to pulmonary diseases other than asthma [13].

The topic of residential mold has received increasing public and media attention over the past decade. Many news stories have focused on problems associated with “toxic mold” or “black mold,” which is often a reference to the toxin-producing mold, *Stachybotrys chartarum*. This might give the impression that mold problems in homes are more frequent now than in past years; however, no good evidence supports this. Reasons for the increasing attention to this issue include high-visibility lawsuits brought by property owners against builders and developers, scientific controversies regarding the degree to which specific illness outbreaks are mold-induced, and an increase in the cost of homeowner insurance policies due to the increasing number of mold-related claims. Modern construction might be more vulnerable to mold problems because tighter construction makes it more difficult for internally generated water vapor to escape, as well as the widespread use of paper-backed drywall in construction (paper is an excellent medium for mold growth when wet), and the widespread use of carpeting.

Allergic Health Effects. Many molds produce numerous protein or glycoprotein allergens capable of causing allergic reactions in people. These allergens have been measured in spores as well as in other fungal fragments. An estimated 6%–10% of the general population and 15%–50% of those who are genetically susceptible are

sensitized to mold allergens [13]. Fifty percent of the 937 children tested in a large multicity asthma study sponsored by the National Institutes of Health showed sensitivity to mold, indicating the importance of mold as an asthma trigger among these children [16]. Molds are thought to play a role in asthma in several ways. Molds produce many potentially allergenic compounds, and molds may play a role in asthma via release of irritants that increase potential for sensitization or release of toxins (mycotoxins) that affect immune response [13].

Toxins and Irritants. Many molds also produce mycotoxins that can be a health hazard on ingestion, dermal contact, or inhalation [14]. Although common outdoor molds present in ambient air, such as *Cladosporium cladosporioides* and *Alternaria alternata*, do not usually produce toxins, many other different mold species do [17]. Genera-producing fungi associated with wet buildings, such as *Aspergillus versicolor*, *Fusarium verticillioides*, *Penicillium aiurantiorisen*, and *S. chartarum*, can produce potent toxins [17]. A single mold species may produce several different toxins, and a given mycotoxin may be produced by more than one species of fungi. Furthermore, toxin-producing fungi do not necessarily produce mycotoxins under all growth conditions, with production being dependent on the substrate it is metabolizing, temperature, water content, and humidity [17]. Because species of toxin-producing molds generally have a higher water requirement than do common household molds, they tend to thrive only under conditions of chronic and severe water damage [18]. For example, *Stachybotrys* typically only grows under continuously wet conditions [19]. It has been suggested that very young children may be especially vulnerable to certain mycotoxins [19,20]. For example, associations have been reported for pulmonary hemorrhage (bleeding lung) deaths in infants and the presence of *S. chartarum* [21–24].

Causes of Mold. Mold growth can be caused by any condition resulting in excess moisture. Common moisture sources include rain leaks (e.g., on roofs and wall joints); surface and groundwater leaks (e.g., poorly designed or clogged rain gutters and footing drains, basement leaks); plumbing leaks; and stagnant water in appliances (e.g., dehumidifiers, dishwashers, refrigerator drip pans, and condensing coils and drip pans in HVAC systems). Moisture problems can also be due to water vapor migration and condensation problems, including uneven indoor temperatures, poor air circulation, soil air entry into basements, contact of humid unconditioned air with cooled interior surfaces, and poor insulation on indoor chilled surfaces (e.g., chilled water lines). Problems can

also be caused by the production of excess moisture within homes from humidifiers, unvented clothes dryers, overcrowding, etc. Finished basements are particularly susceptible to mold problems caused by the combination of poorly controlled moisture and mold-supporting materials (e.g., carpet, paper-backed sheetrock) [15]. There is also some evidence that mold spores from damp or wet crawl spaces can be transported through air currents into the upper living quarters. Older, substandard housing low income families can be particularly prone to mold problems because of inadequate maintenance (e.g., inoperable gutters, basement and roof leaks), overcrowding, inadequate insulation, lack of air conditioning, and poor heating. Low interior temperatures (e.g., when one or two rooms are left unheated) result in an increase in the relative humidity, increasing the potential for water to condense on cold surfaces.

Mold Assessment Methods. Mold growth or the potential for mold growth can be detected by visual inspection for active or past microbial growth, detection of musty odors, and inspection for water staining or damage. If it is not possible or practical to inspect a residence, this information can be obtained using occupant questionnaires. Visual observation of mold growth, however, is limited by the fact that fungal elements such as spores are microscopic, and that their presence is often not apparent until growth is extensive and the fact that growth can occur in hidden spaces (e.g., wall cavities, air ducts).

Portable, hand-held moisture meters, for the direct measurement of moisture levels in materials, may also be useful in qualitative home assessments to aid in pinpointing areas of potential biologic growth that may not otherwise be obvious during a visual inspection [14].

For routine assessments in which the goal is to identify possible mold contamination problems before remediation, it is usually unnecessary to collect and analyze air or settled dust samples for mold analysis because decisions about appropriate intervention strategies can typically be made on the basis of a visual inspection [25]. Also, sampling and analysis costs can be relatively high and the interpretation of results is not straightforward. Air and dust monitoring may, however, be necessary in certain situations, including 1) if an individual has been diagnosed with a disease associated with fungal exposure through inhalation, 2) if it is suspected that the ventilation systems are contaminated, or 3) if the presence of mold is suspected but cannot be identified by a visual inspection or bulk sampling [26].

Generally, indoor environments contain large reservoirs of mold spores in settled dust and contaminated building materials, of which only a relatively small amount is airborne at a given time.

Common methods for sampling for mold growth include bulk sampling techniques, air sampling, and collection of settled dust samples. In bulk sampling, portions of materials with visual or suspected mold growth (e.g., sections of wallboard, pieces of duct lining, carpet segments, or return air filters) are collected and directly examined to determine if mold is growing and to identify the mold species or groups that are present. Surface sampling in mold contamination investigations may also be used when a less destructive technique than bulk sampling is desired. For example, nondestructive samples of mold may be collected using a simple swab or adhesive tape [14].

Air can also be sampled for mold using pumps that pull air across a filter medium, which traps airborne mold spores and fragments. It is generally recommended that outdoor air samples are collected concurrent with indoor samples for comparison purposes for measurement of baseline ambient air conditions. Indoor contamination can be indicated by indoor mold distributions (both species and concentrations) that differ significantly from the distributions in outdoor samples [14]. Captured mold spores can be examined under a microscope to identify the mold species/groups and determine concentrations or they can be cultured on growth media and the resulting colonies counted and identified. Both techniques require considerable expertise.

Dust sampling involves the collection of settled dust samples (e.g., floor dust) using a vacuum method in which the dust is collected onto a porous filter medium or into a container. The dust is then processed in the laboratory and the mold identified by culturing viable spores.

Mold Standards. No standard numeric guidelines exist for assessing whether mold contamination exists in an area. In the United States, no EPA regulations or standards exist for airborne mold contaminants [26]. Various governmental and private organizations have, however, proposed guidance on the interpretation of fungal measures of environmental media in indoor environments (quantitative limits for fungal concentrations).

Given evidence that young children may be especially vulnerable to certain mycotoxins [18] and in view of the potential severity of diseases associated with mycotoxin

exposure, some organizations support a precautionary approach to limiting mold exposure [19]. For example, the American Academy of Pediatrics recommends that infants under 1 year of age are not exposed at all to chronically moldy, water-damaged environments [18].

Mold Mitigation. Common intervention methods for addressing mold problems include the following:

- maintaining heating, ventilating, and air conditioning (HVAC) systems;
- changing HVAC filters frequently, as recommended by manufacturer;
- keeping gutters and downspouts in working order and ensuring that they drain water away from the foundation;
- routinely checking, cleaning, and drying drip pans in air conditioners, refrigerators, and dehumidifiers;
- increasing ventilation (e.g., using exhaust fans or open windows to remove humidity when cooking, showering, or using the dishwasher);
- venting clothes dryers to the outside; and
- maintaining an ideal relative humidity level in the home of 40% to 60%.
- locating and removing sources of moisture (controlling dampness and humidity and repairing water leakage problems);
- cleaning or removing mold-contaminated materials;
- removing materials with severe mold growth; and
- using high-efficiency air filters.

Moisture Control. Because one of the most important factors affecting mold growth in homes is moisture level, controlling this factor is crucial in mold abatement strategies. Many simple measures can significantly control moisture, for example maintaining indoor relative humidity at no greater than 40%–60% through the use of dehumidifiers, fixing water leakage problems, increasing ventilation in kitchens and bathrooms by using exhaust fans, venting clothes dryers to the outside,

reducing the number of indoor plants, using air conditioning at times of high outdoor humidity, heating all rooms in the winter and adding heating to outside wall closets, sloping surrounding soil away from building foundations, fixing gutters and downspouts, and using a sump pump in basements prone to flooding [27]. Vapor barriers, sump pumps, and aboveground vents can also be installed in crawlspaces to prevent moisture problems [28].

Removal and Cleaning of Mold-contaminated Materials.

Nonporous (e.g., metals, glass, and hard plastics) and semiporous (e.g., wood and concrete) materials contaminated with mold and that are still structurally sound can often be cleaned with bleach-and-water solutions. However, in some cases, the material may not be easily cleaned or may be so severely contaminated that it may have to be removed. It is recommended that porous materials (e.g., ceiling tiles, wallboards, and fabrics) that cannot be cleaned be removed and discarded [29]. In severe cases, clean-up and repair of mold-contaminated buildings may be conducted using methods similar to those used for abatement of other hazardous substances such as asbestos [30]. For example, in situations of extensive colonization (large surface areas greater than 100 square feet or where the material is severely degraded), extreme precautions may be required, including full containment (complete isolation of work area) with critical barriers (airlock and decontamination room) and negative pressurization, personnel trained to handle hazardous wastes, and the use of full-face respirators with HEPA filters, eye protection, and disposable full-body covering [26].

Worker Protection When Conducting Mold Assessment and Mitigation Projects.

Activities such as cleaning or removal of mold-contaminated materials in homes, as well as investigations of mold contamination extent, have the potential to disturb areas of mold growth and release fungal spores and fragments into the air. Recommended measures to protect workers during mold remediation efforts depend on the severity and nature of the mold contamination being addressed, but include the use of well fitted particulate masks or respirators that retain particles as small as 1 micrometer or less, disposable gloves and coveralls, and protective eyewear [31].

Following are examples of guidance documents for remediation of mold contamination:

- New York City Department of Health and Mental Hygiene. Guidelines on Assessment and Remediation of Fungi in Indoor Environments

(available from URL: <http://www.nyc.gov/html/doh/html/epi/moldrpt1.shtml>).

- American Conference of Governmental Industrial Hygienists (ACGIH) 1999 document, Biosaerosols: Assessment and Control (can be ordered at URL <http://www.acgih.org/home.htm>).
- American Industrial Hygiene Association (AIHA) 2004 document, Assessment, Remediation, and Post-Remediation Verification of Mold in Buildings (can be ordered at URL www.aiha.org)
- Environmental Protection Agency guidance, Mold Remediation in Schools and Commercial Buildings (includes many general principles also applicable to residential mold mitigation efforts; available at URL: http://www.epa.gov/iaq/molds/mold_remediation.html)
- Environmental Protection Agency guidance, A Brief Guide to Mold, Moisture, and Your Home (for homeowners and renters on how to clean up residential mold problems and how to prevent mold growth; available at URL: <http://www.epa.gov/iaq/molds/images/moldguide.pdf>)
- Canada Mortgage and Housing Corporation, Clean-up Procedures for Mold in Houses, (provides qualitative guidance for mold mitigation; can be ordered at URL: <https://www.cmhc-schl.gc.ca/50104/b2c/b2c/init.do?language=en>).

Figure 5.1 shows mold growth in the home.

Chemical Pollutants

Carbon Monoxide

Carbon monoxide (CO) is a significant combustion pollutant in the United States. CO is a leading cause of poisoning deaths [32]. According to the National Fire Protection Association (NFPA), CO-related nonfire deaths are often attributed to heating and cooking equipment. The leading specific types of equipment blamed for CO-related deaths include gas-fueled space heaters, gas-fueled furnaces, charcoal grills, gas-fueled ranges, portable kerosene heaters, and wood stoves.

As with fire deaths, the risk for unintentional CO death is highest for the very young (ages 4 years and younger) and the very old (ages 75 years and older). CO is an odorless, colorless gas that can cause sudden illness and death. It is



Figure 5.1. Mold Growth in the Home

a result of the incomplete combustion of carbon. Headache, dizziness, weakness, nausea, vomiting, chest pain, and confusion are the most frequent symptoms of CO poisoning. According to the American Lung Association (ALA) [33], breathing low levels of CO can cause fatigue and increase chest pain in people with chronic heart disease. Higher levels of CO can cause flulike symptoms in healthy people. In addition, extremely high levels of CO cause loss of consciousness and death. In the home, any fuel-burning appliance that is not adequately vented and maintained can be a potential source of CO. The following steps should be followed to reduce CO (as well as sulfur dioxide and oxides of nitrogen) levels:

- Never use gas-powered equipment, charcoal grills, hibachis, lanterns, or portable camping stoves in enclosed areas or indoors.
- Install a CO monitor (Figure 5.2) in appropriate areas of the home. These monitors are designed to provide a warning before potentially life-threatening levels of CO are reached.
- Choose vented appliances when possible and keep gas appliances properly adjusted to decrease the combustion to CO. (Note: Vented appliances are always preferable for several reasons: oxygen levels, carbon dioxide buildup, and humidity management).
- Only buy certified and tested combustion appliances that meet current safety standards, as certified by Underwriter's Laboratories (UL), American Gas Association (AGA) Laboratories, or equivalent.

- Assure that all gas heaters possess safety devices that shut off an improperly vented gas heater. Heaters made after 1982 use a pilot light safety system known as an oxygen depletion sensor. When inadequate fresh air exists, this system shuts off the heater before large amounts of CO can be produced.



Figure 5.2. Home Carbon Monoxide Monitor
Source: U.S. Navy

- Use appliances that have electronic ignitions instead of pilot lights. These appliances are typically more energy efficient and eliminate the continuous low-level pollutants from pilot lights.
- Use the proper fuel in kerosene appliances.
- Install and use an exhaust fan vented to the outdoors over gas stoves.
- Have a trained professional annually inspect, clean, and tune up central heating systems (furnaces, flues, and chimneys) and repair them as needed.
- Do not idle a car inside a garage.

The U.S. Consumer Product Safety Commission (CPSC) recommends installing at least one CO alarm per household near the sleeping area. For an extra measure of safety, another alarm should be placed near the home's heating source. ALA recommends weighing the benefits of using models powered by electrical outlets versus models powered by batteries that run out of power and need replacing. Battery-powered CO detectors provide continuous protection and do not require recalibration in the event of a power outage. Electric-powered systems do not provide protection during a loss of power and can take up to 2 days to recalibrate. A device that can be easily self-tested and reset to ensure proper functioning should be chosen. The product should meet Underwriters Laboratories Standard UL 2034.

Ozone

Inhaling ozone can damage the lungs. Inhaling small amounts of ozone can result in chest pain, coughing, shortness of breath, and throat irritation. Ozone can also exacerbate

chronic respiratory diseases such as asthma. Susceptibility to the effects of ozone varies from person to person, but even healthy people can experience respiratory difficulties from exposure.

According to the North Carolina Department of Health and Human Services [34], the major source of indoor ozone is outdoor ozone. Indoor levels can vary from 10% of the outdoor air to levels as high as 80% of the outdoor air. The Food and Drug Administration has set a limit of 0.05 ppm of ozone in indoor air. In recent years, there have been numerous advertisements for ion generators that destroy harmful indoor air pollutants. These devices create ozone or elemental oxygen that reacts with pollutants. EPA has reviewed the evidence on ozone generators and states: “available scientific evidence shows that at concentrations that do not exceed public health standards, ozone has little potential to remove indoor air contaminants,” and “there is evidence to show that at concentrations that do not exceed public health standards, ozone is not effective at removing many odor causing chemicals” [35].

Ozone is also created by the exposure of polluted air to sunlight or ultraviolet light emitters. This ozone produced outside of the home can infiltrate the house and react with indoor surfaces, creating additional pollutants.

Environmental Tobacco Smoke or Secondhand Smoke

Like CO, environmental tobacco smoke (ETS; also known as secondhand smoke), is a product of combustion. The National Cancer Institute (NCI) [36], states that ETS is the combination of two forms of smoke from burning tobacco products:

- Sidestream smoke, or smoke that is emitted between the puffs of a burning cigarette, pipe, or cigar; and
- Mainstream smoke, or the smoke that is exhaled by the smoker.

The physiologic effects of ETS are numerous. ETS can trigger asthma; irritate the eyes, nose, and throat; and cause ear infections in children, respiratory illnesses, and lung cancer. ETS is believed to cause asthma by irritating chronically inflamed bronchial passages. According to the EPA [37], ETS is a Group A carcinogen; thus, it is a known cause of cancer in humans. Laboratory analysis has revealed that ETS contains in excess of 4,000 substances, more than 60 of which cause cancer in humans or animals. The EPA also estimates that approximately 3,000 lung cancer deaths occur each year

in nonsmokers due to ETS. Additionally, passive smoking can lead to coughing, excess phlegm, and chest discomfort. NCI also notes that spontaneous abortion (miscarriage), cervical cancer, sudden infant death syndrome, low birth weight, nasal sinus cancer, decreased lung function, exacerbation of cystic fibrosis, and negative cognitive and behavioral effects in children have been linked to ETS [36].

The EPA [37] states that, because of their relative body size and respiratory rates, children are affected by ETS more than adults are. It is estimated that an additional 7,500 to 15,000 hospitalizations resulting from increased respiratory infections occur in children younger than 18 months of age due to ETS exposure. Figure 5.3 shows the ETS exposure levels in homes with children under age 7 years. The following actions are recommended in the home to protect children from ETS:

- if individuals insist on smoking, increase ventilation in the smoking area by opening windows or using exhaust fans; and
- refrain from smoking in the presence of children and do not allow babysitters or others who work in the home to smoke in the home or near children.

Volatile Organic Compounds

In the modern home, many organic chemicals are used as ingredients in household products. Organic chemicals that vaporize and become gases at normal room temperature are collectively known as VOCs.

Examples of common items that can release VOCs include paints, varnishes, and wax, as well as in many cleaning, disinfecting, cosmetic, degreasing, and hobby products. Levels of approximately a dozen common VOCs can be two to five times higher inside the home, as opposed to outside, whether in highly industrialized areas

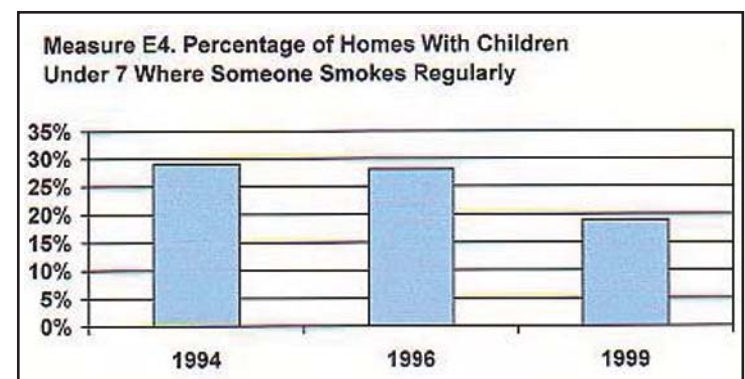


Figure 5.3. Environmental Tobacco Smoke and Children’s Exposure [37]

or rural areas. VOCs that frequently pollute indoor air include toluene, styrene, xylenes, and trichloroethylene. Some of these chemicals may be emitted from aerosol products, dry-cleaned clothing, paints, varnishes, glues, art supplies, cleaners, spot removers, floor waxes, polishes, and air fresheners. The health effects of these chemicals are varied. Trichloroethylene has been linked to childhood leukemia. Exposure to toluene can put pregnant women at risk for having babies with neurologic problems, retarded growth, and developmental problems. Xylenes have been linked to birth defects. Styrene is a suspected endocrine disruptor, a chemical that can block or mimic hormones in humans or animals. EPA data reveal that methylene chloride, a common component of some paint strippers, adhesive removers, and specialized aerosol spray paints, causes cancer in animals [38]. Methylene chloride is also converted to CO in the body and can cause symptoms associated with CO exposure. Benzene, a known human carcinogen, is contained in tobacco smoke, stored fuels, and paint supplies. Perchloroethylene, a product uncommonly found in homes, but common to dry cleaners, can be a pollution source by off-gassing from newly cleaned clothing. Environmental Media Services [39] also notes that xylene, ketones, and aldehydes are used in aerosol products and air fresheners.

To lower levels of VOCs in the home, follow these steps:

- use all household products according to directions;
- provide good ventilation when using these products;
- properly dispose of partially full containers of old or unneeded chemicals;
- purchase limited quantities of products; and
- minimize exposure to emissions from products containing methylene chloride, benzene, and perchlorethylene.

A prominent VOC found in household products and construction products is formaldehyde. According to CPSC [40], these products include the glue or adhesive used in pressed wood products; preservatives in paints, coating, and cosmetics; coatings used for permanent-press quality in fabrics and draperies; and the finish on paper products and certain insulation materials. Formaldehyde is contained in urea-formaldehyde (UF) foam insulation installed in the wall cavities of homes as an energy conservation measure. Levels of formaldehyde increase soon after installation of this product, but these levels

decline with time. In 1982, CPSC voted to ban UF foam insulation. The courts overturned the ban; however, the publicity has decreased the use of this product.

More recently, the most significant source of formaldehyde in homes has been pressed wood products made using adhesives that contain UF resins [41]. The most significant of these is medium-density fiberboard, which contains a higher resin-to-wood ratio than any other UF pressed wood product. This product is generally recognized as being the highest formaldehyde-emitting pressed wood product. Additional pressed wood products are produced using phenol-formaldehyde resin. The latter type of resin generally emits formaldehyde at a considerably slower rate than those containing UF resin. The emission rate for both resins will change over time and will be influenced by high indoor temperatures and humidity. Since 1985, U.S. Department of Housing and Urban Development (HUD) regulations (24 CFR 3280.308, 3280.309, and 3280.406) have permitted only the use of plywood and particleboard that conform to specified formaldehyde emission limits in the construction of prefabricated and manufactured homes [42]. This limit was to ensure that indoor formaldehyde levels are below 0.4 ppm.

CPSC [40] notes that formaldehyde is a colorless, strong-smelling gas. At an air level above 0.1 ppm, it can cause watery eyes; burning sensations in the eyes, nose, and throat; nausea; coughing; chest tightness; wheezing; skin rashes; and allergic reactions. Laboratory animal studies have revealed that formaldehyde can cause cancer in animals and may cause cancer in humans. Formaldehyde is usually present at levels less than 0.03 ppm indoors and outdoors, with rural areas generally experiencing lower concentrations than urban areas. Indoor areas that contain products that release formaldehyde can have levels greater than 0.03 ppm. CPSC also recommends the following actions to avoid high levels of exposure to formaldehyde:

- Purchase pressed wood products that are labeled or stamped to be in conformance with American National Standards Institute criteria ANSI A208.1-1993. Use particleboard flooring marked with ANSI grades PBU, D2, or D3. Medium-density fiberboard should be in conformance with ANSI A208.2-1994 and hardwood plywood with ANSI/HPVA HP-1-1994 (Figure 5.4).
- Purchase furniture or cabinets that contain a high percentage of panel surface and edges that are laminated or coated. Unlaminated or uncoated

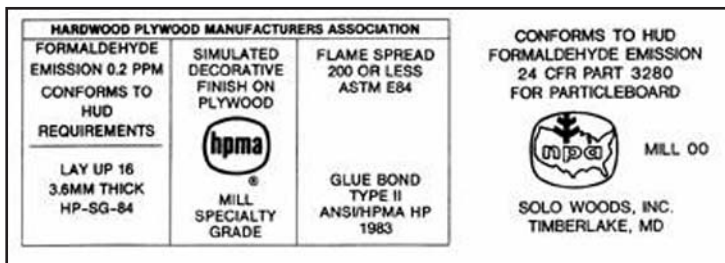


Figure 5.4. Wood Products Label [42]

(raw) panels of pressed wood panel products will generally emit more formaldehyde than those that are laminated or coated.

- Use alternative products, such as wood panel products not made with UF glues, lumber, or metal.
- Avoid the use of foamed-in-place insulation containing formaldehyde, especially UF foam insulation.
- Wash durable-press fabrics before use.

CPSC also recommends the following actions to reduce existing levels of indoor formaldehyde:

- Ventilate the home well by opening doors and windows and installing an exhaust fan(s).
- Seal the surfaces of formaldehyde-containing products that are not laminated or coated with paint, varnish, or a layer of vinyl or polyurethane-like materials.
- Remove products that release formaldehyde in the indoor air from the home.

Radon

According to the EPA [43], radon is a colorless, odorless gas that occurs naturally in soil and rock and is a decay product of uranium. The U.S. Geological Survey (USGS) [44] notes that the typical uranium content of rock and the surrounding soil is between 1 and 3 ppm. Higher levels of uranium are often contained in rock such as light-colored volcanic rock, granite, dark shale, and sedimentary rock containing phosphate. Uranium levels as high as 100 ppm may be present in various areas of the United States because of these rocks. The main source of high-level radon pollution in buildings is surrounding uranium-containing soil. Thus, the greater the level of uranium nearby, the greater the chances are that buildings in the area will have high levels of indoor radon.

Figure 5.5 demonstrates the geographic variation in radon

levels in the United States. Maps of the individual states and areas that have proven high for radon are available at <http://www.epa.gov/iaq/radon/zonemap.html>. A free video is available from the U.S. EPA: call 1-800-438-4318 and ask for EPA 402-V-02-003 (TRT 13.10).

Radon, according to the California Geological Survey [45], is one of the intermediate radioactive elements formed during the radioactive decay of uranium-238, uranium-235, or thorium-232. Radon-222 is the radon isotope of most concern to public health because of its longer half-life (3.8 days). The mobility of radon gas is much greater than are uranium and radium, which are solids at room temperature. Thus, radon can leave rocks and soil, move through fractures and pore spaces, and ultimately enter a building to collect in high concentrations. When in water, radon moves less than 1 inch before it decays, compared to 6 feet or more in dry rocks or soil. USGS [44] notes that radon near the surface of soil typically escapes into the atmosphere. However, where a house is present, soil air often flows toward the house foundation because of

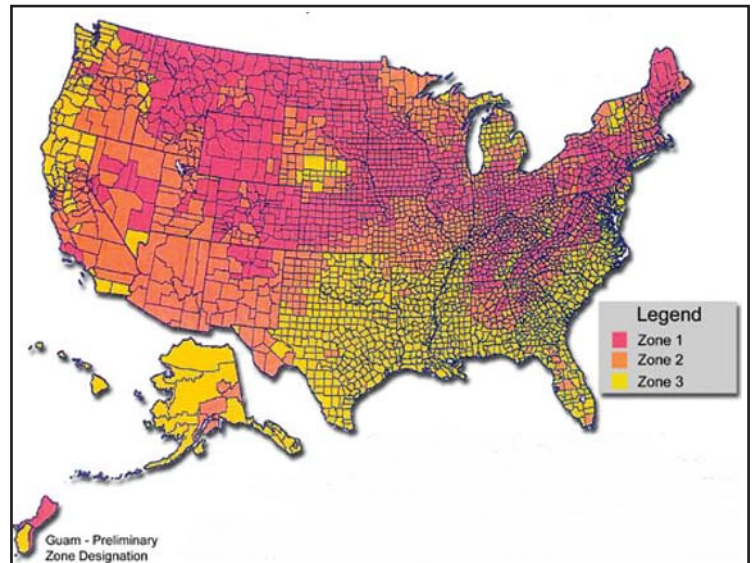


Figure 5.5. EPA Map of Radon Zones [43]

Zone 1: predicted average indoor radon screening level greater than 4 pCi/L [picocuries per liter]

Zone 2: predicted average indoor radon screening level between 2 and 4 pCi/L

Zone 3: predicted average indoor radon screening level less than 2 pCi/L

Important: Consult the EPA Map of Radon Zones document [EPA-402-R-93-071] before using this map. This document contains information on radon potential variations within counties.

EPA also recommends that this map be supplemented with any available local data to further understand and predict the radon potential of a specific area.

- differences in air pressure between the soil and the house, with soil pressure often being higher;
- presence of openings in the house's foundation; and
- increases in permeability around the basement (if present).

Houses are often constructed with loose fill under a basement slab and between the walls and exterior ground. This fill is more permeable than the original ground. Houses typically draw less than 1% of their indoor air from the soil. However, houses with low indoor air pressures, poorly sealed foundations, and several entry points for soil air may draw up to 20% of their indoor air from the soil.

USGS [44] states that radon may also enter the home through the water systems. Surface water sources typically contain little radon because it escapes into the air. In larger cities, radon is released to the air by municipal processing systems that aerate the water. However, in areas where groundwater is the main water supply for communities, small public systems and private wells are typically closed systems that do not allow radon to escape. Radon then enters the indoor air from showers, clothes washing, dishwashing, and other uses of water. Figure 5.6 shows typical entry points of radon.

Health risks of radon stem from its breakdown into “radon daughters,” which emit high-energy alpha particles. These progeny enter the lungs, attach themselves, and may eventually lead to lung cancer. This exposure to radon is believed to contribute to between 15,000 and 21,000 excess lung cancer deaths in the United States each year. The EPA has identified levels greater than 4 picocuries per liter as levels at which remedial action should be taken. Approximately 1 in 15 homes nationwide have radon above this level, according to the U.S. Surgeon General’s recent advisory [46]. Smokers are at significantly higher risk for radon-related lung cancer.

Radon in the home can be measured either by the occupant or by a professional. Because radon has no odor or color, special devices are used to measure its presence. Radon levels vary from day to day and season to season. Short-term tests (2 to 90 days) are best if quick results are needed, but long-term tests (more than 3 months) yield better information on average year-round exposure. Measurement devices are routinely placed in the lowest occupied level of the home. The devices either measure

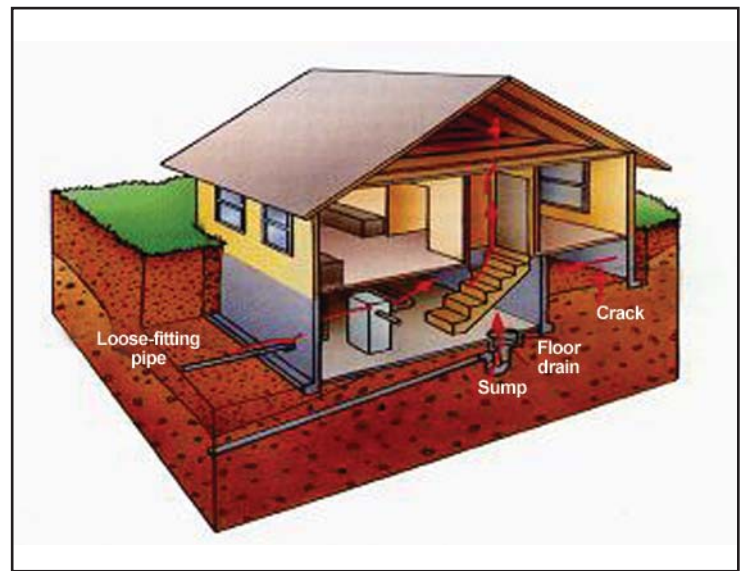


Figure 5.6. Radon Entry [30]

the radon gas directly or the daughter products. The simplest devices are passive, require no electricity, and include a charcoal canister, charcoal liquid scintillation device, alpha track detector, and electret ion detectors [47]. All of these devices, with the exception of the ion detector, can be purchased in hardware stores or by mail. The ion detector generally is only available through laboratories. These devices are inexpensive, primarily used for short-term testing, and require little to no training. Active devices, however, need electrical power and include continuous monitoring devices. They are customarily more expensive and require professionally trained testers for their operation. Figure 5.7 shows examples of the charcoal tester (a; left) and the alpha track detector (b; right).

After testing and evaluation by a professional, it may be necessary to lower the radon levels in the structure. The Pennsylvania Department of Environmental Protection [48] states that in most cases, a system with pipes and a fan is used to reduce radon. This system, known as a subslab depressurization system, requires no major changes to the home. The cost typically ranges from \$500 to \$2,500 and averages approximately \$1,000, varying with geographic region. The typical mitigation system usually has only one pipe penetrating through the basement floor; the pipe also may be installed outside the house. The Connecticut Department of Public Health [49] notes that it is more cost effective to include radon-resistant techniques while constructing a building than to install a reduction system in an existing home. Inclusion of radon-resistant techniques in initial construction costs approximately \$350 to \$500 [50]. Figure 5.8 shows examples of radon-resistant construction techniques.

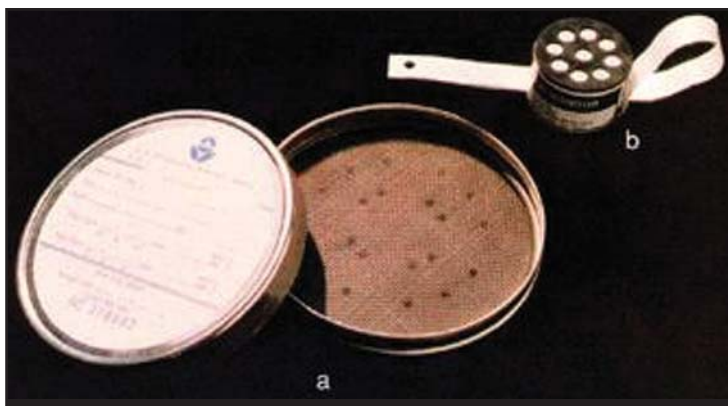


Figure 5.7. Home Radon Detectors [31]

A passive radon-resistant system has five major parts:

1. A layer of gas-permeable material under the foundation.
2. The foundation (usually 4 inches of gravel).
3. Plastic sheeting over the foundation, with all openings in the concrete foundation floor sealed and caulked.
4. A gas-tight, 3- or 4-inch vent pipe running from under the foundation through the house to the roof.
5. A roughed-in electrical junction box for the future installation of a fan, if needed.

These features create a physical barrier to radon entry. The vent pipe redirects the flow of air under the foundation, preventing radon from seeping into the house.

Pesticides

Much pesticide use could be reduced if integrated pest management (IPM) practices were used in the home. IPM is a coordinated approach to managing roaches,

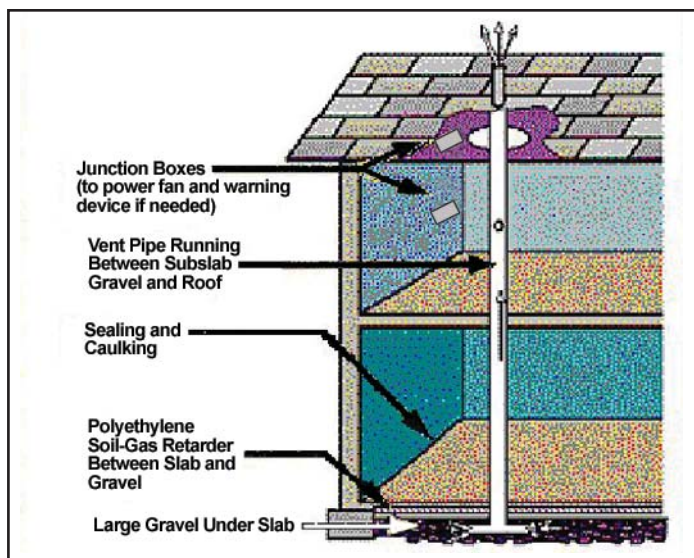


Figure 5.8. Radon-resistant Construction [50]

rodents, mosquitoes, and other pests that integrates inspection, monitoring, treatment, and evaluation, with special emphasis on the decreased use of toxic agents. However, all pest management options, including natural, biologic, cultural, and chemical methods, should be considered. Those that have the least impact on health and the environment should be selected. Most household pests can be controlled by eliminating the habitat for the pest both inside and outside, building or screening them out, eliminating food and harborage areas, and safely using appropriate pesticides if necessary.

EPA [51] states that 75% of U.S. households used at least one pesticide indoors during the past year and that 80% of most people's exposure to pesticides occurs indoors. Measurable levels of up to a dozen pesticides have been found in the air inside homes. Pesticides used in and around the home include products to control insects (insecticides), termites (termiticides), rodents (rodenticides), fungi (fungicides), and microbes (disinfectants). These products are found in sprays, sticks, powders, crystals, balls, and foggers.

Delaplane [52] notes that the ancient Romans killed insect pests by burning sulfur and controlled weeds with salt. In the 1600s, ants were controlled with mixtures of honey and arsenic. U.S. farmers in the late 19th century used copper actoarsenite (Paris green), calcium arsenate, nicotine sulfate, and sulfur to control insect pests in field crops. By World War II and afterward, numerous pesticides had been introduced, including DDT, BHC, aldrin, dieldrin, endrin, and 2,4-D. A significant factor with regard to these pesticides used in and around the home is their impact on children. According to a 2003 EPA survey, 47% of all households with children under the age of 5 years had at least one pesticide stored in an unlocked cabinet less than 4 feet off the ground. This is within easy reach of children. Similarly, 74% of households without children under the age of 5 also stored pesticides in an unlocked cabinet less than 4 feet off the ground. This issue is significant because 13% of all pesticide poisoning incidents occur in homes other than the child's home. The EPA [53] notes a report by the American Association of Poison Control Centers indicating that approximately 79,000 children were involved in common household pesticide poisonings or exposures.

The health effects of pesticides vary with the product. However, local effects from most of the products will be on eyes, noses, and throats; more severe consequences, such as on the central nervous system and kidneys and on

cancer risks, are possible. The active and inert ingredients of pesticides can be organic compounds, which can contribute to the level of organic compounds in indoor air. More significantly, products containing cyclodiene pesticides have been commonly associated with misapplication. Individuals inadvertently exposed during this misapplication had numerous symptoms, including headaches, dizziness, muscle twitching, weakness, tingling sensations, and nausea. In addition, there is concern that these pesticides may cause long-term damage to the liver and the central nervous system, as well as an increased cancer risk. Cyclodiene pesticides were developed for use as insecticides in the 1940s and 1950s. The four main cyclodiene pesticides—aldrin, dieldrin, chlordane, and heptachlor—were used to guard soil and seed against insect infestation and to control insect pests in crops. Outside of agriculture they were used for ant control; farm, industrial, and domestic control of fleas, flies, lice, and mites; locust control; termite control in buildings, fences, and power poles; and pest control in home gardens. No other commercial use is permitted for cyclodiene or related products. The only exception is the use of heptachlor by utility companies to control fire ants in underground cable boxes.

An EPA survey [53] revealed that bathrooms and kitchens are areas in the home most likely to have improperly stored pesticides. In the United States, EPA regulates pesticides under the pesticide law known as the Federal Insecticide, Fungicide, and Rodenticide Act. Since 1981, this law has required most residential-use pesticides to bear a signal word such as “danger” or “warning” and to be contained in child-resistant packaging. This type of packaging is designed to prevent or delay access by most children under the age of 5 years. EPA offers the following recommendations for preventing accidental poisoning:

- store pesticides away from the reach of children in a locked cabinet, garden shed, or similar location;
- read the product label and follow all directions exactly, especially precautions and restrictions;
- remove children, pets, and toys from areas before applying pesticides;
- if interrupted while applying a pesticide, properly close the package and assure that the container is not within reach of children;
- do not transfer pesticides to other containers that children may associate with food or drink;

- do not place rodent or insect baits where small children have access to them;
- use child-resistant packaging properly by closing the container tightly after use;
- assure that other caregivers for children are aware of the potential hazards of pesticides;
- teach children that pesticides are poisons and should not be handled; and
- keep the local Poison Control Center telephone number available.

Toxic Materials

Asbestos

Asbestos, from the Greek word meaning “inextinguishable,” refers to a group of six naturally occurring mineral fibers. Asbestos is a mineral fiber of which there are several types: amosite, crocidolite, tremolite, actinolite, anthrophyllite, and chrysotile. Chrysotile asbestos, also known as white asbestos, is the predominant commercial form of asbestos. Asbestos is strong, flexible, resistant to heat and chemical corrosion, and insulates well. These features led to the use of asbestos in up to 3,000 consumer products before government agencies began to phase it out in the 1970s because of its health hazards. Asbestos has been used in insulation, roofing, siding, vinyl floor tiles, fireproofing materials, texturized paint and soundproofing materials, heating appliances (such as clothes dryers and ovens), fireproof gloves, and ironing boards. Asbestos continues to be used in some products, such as brake pads. Other mineral products, such as talc and vermiculite, can be contaminated with asbestos. The health effects of asbestos exposure are numerous and varied. Industrial studies of workers exposed to asbestos in factories and shipyards have revealed three primary health risk concerns from breathing high levels of asbestos fibers: lung cancer, mesothelioma (a cancer of the lining of the chest and the abdominal cavity), and asbestosis (a condition in which the lungs become scarred with fibrous tissue). The risk for all of these conditions is amplified as the number of fibers inhaled increases. Smoking also enhances the risk for lung cancer from inhaling asbestos fibers by acting synergistically. The incubation period (from time of exposure to appearance of symptoms) of these diseases is usually about 20 to 30 years. Individuals who develop asbestosis have typically been exposed to high levels of asbestos for a long time. Exposure levels to asbestos are measured in fibers per cubic centimeter of air. Most individuals are exposed to small amounts of asbestos

in daily living activities; however, a preponderance of them do not develop health problems. According to the Agency for Toxic Substances and Disease Registry (ATSDR), if an individual is exposed, several factors determine whether the individual will be harmed [54]. These factors include the dose (how much), the duration (how long), and the fiber type (mineral form and distribution). ATSDR also states that children may be more adversely affected than adults [54]. Children breathe differently and have different lung structures than adults; however, it has not been determined whether these differences cause a greater amount of asbestos fibers to stay in the lungs of a child than in the lungs of an adult. In addition, children drink more fluids per kilogram of body weight than do adults and they can be exposed through asbestos-contaminated drinking water. Eating asbestos-contaminated soil and dust is another source of exposure for children. Certain children intentionally eat soil and children's hand-to-mouth activities mean that all young children eat more soil than do adults. Family members also have been exposed to asbestos that was carried home on the clothing of other family members who worked in asbestos mines or mills. Breathing asbestos fibers may result in difficulty in breathing. Diseases usually appear many years after the first exposure to asbestos and are therefore not likely to be seen in children. But people who have been exposed to asbestos at a young age may be more likely to contract diseases than those who are first exposed later in life. In the small number of studies that have specifically looked at asbestos exposure in children, there is no indication that younger people might develop asbestos-related diseases more quickly than older people. Developing fetuses and infants are not likely to be exposed to asbestos through the placenta or breast milk of the mother. Results of animal studies do not indicate that exposure to asbestos is likely to result in birth defects.

A joint document issued by CPSC, EPA, and ALA, notes that most products in today's homes do not contain asbestos. However, asbestos can still be found in products and areas of the home. These products contain asbestos that could be inhaled and are required to be labeled as such. Until the 1970s, many types of building products and insulation materials used in homes routinely contained asbestos. A potential asbestos problem both inside and outside the home is that of vermiculite. According to the USGS [55], vermiculite is a claylike material that expands when heated to form wormlike particles. It is used in concrete aggregate, fertilizer carriers, insulation, potting soil, and soil conditioners. This product ceased being mined in 1992, but old stocks may still be available.

Common products that contained asbestos in the past and conditions that may release fibers include the following:

- Steam pipes, boilers, and furnace ducts insulated with an asbestos blanket or asbestos paper tape. These materials may release asbestos fibers if damaged, repaired, or removed improperly.
- Resilient floor tiles (vinyl asbestos, asphalt, and rubber), the backing on vinyl sheet flooring, and adhesives used for installing floor tile. Sanding tiles can release fibers, as may scraping or sanding the backing of sheet flooring during removal.
- Cement sheet, millboard, and paper used as insulation around furnaces and wood-burning stoves. Repairing or removing appliances may release asbestos fibers, as may cutting, tearing, sanding, drilling, or sawing insulation.
- Door gaskets in furnaces, wood stoves, and coal stoves. Worn seals can release asbestos fibers during use.
- Soundproofing or decorative material sprayed on walls and ceilings. Loose, crumbly, or water-damaged material may release fibers, as will sanding, drilling, or scraping the material.
- Patching and joint compounds for walls, ceilings, and textured paints. Sanding, scraping, or drilling these surfaces may release asbestos.
- Asbestos cement roofing, shingles, and siding. These products are not likely to release asbestos fibers unless sawed, drilled, or cut.
- Artificial ashes and embers sold for use in gas-fired fireplaces in addition to other older household products such as fireproof gloves, stove-top pads, ironing board covers, and certain hair dryers.
- Automobile brake pads and linings, clutch facings, and gaskets.

Homeowners who believe material in their home may be asbestos should not disturb the material. Generally, material in good condition will not release asbestos fibers, and there is little danger unless the fibers are released and inhaled into the lungs. However, if disturbed, asbestos material may release asbestos fibers, which can be inhaled into the lungs. The fibers can remain in the lungs for a

long time, increasing the risk for disease. Suspected asbestos-containing material should be checked regularly for damage from abrasions, tears, or water. If possible, access to the area should be limited. Asbestos-containing products such as asbestos gloves, stove-top pads, and ironing board covers should be discarded if damaged or worn. Permission and proper disposal methods should be obtainable from local health, environmental, or other appropriate officials. If asbestos material is more than slightly damaged, or if planned changes in the home might disturb it, repair or removal by a professional is needed. Before remodeling, determine whether asbestos materials are present.

Only a trained professional can confirm suspected asbestos materials that are part of a home's construction. This individual will take samples for analysis and submit them to an EPA-approved laboratory.

If the asbestos material is in good shape and will not be disturbed, the best approach is to take no action and continue to monitor the material. If the material needs action to address potential exposure problems, there are two approaches to correcting the problem: repair and removal.

Repair involves sealing or covering the asbestos material. Sealing or encapsulation involves treating the material with a sealant that either binds the asbestos fibers together or coats the material so fibers are not released. This is an approach often used for pipe, furnace, and boiler insulation; however, this work should be done only by a professional who is trained to handle asbestos safely. Covering (enclosing) involves placing something over or around the material that contains asbestos to prevent release of fibers. Exposed insulated piping may be covered with a protective wrap or jacket. In the repair process, the approach is for the material to remain in position undisturbed. Repair is a less expensive process than is removal.

With any type of repair, the asbestos remains in place. Repair may make later removal of asbestos, if necessary, more difficult and costly. Repairs can be major or minor. Both major and minor repairs must be done only by a professional trained in methods for safely handling asbestos.

Removal is usually the most expensive and, unless required by state or local regulations, should be the last option considered in most situations. This is because removal poses the greatest risk for fiber release. However, removal may be required when remodeling or making major changes to the home that will disturb asbestos material. In addition, removal may be called for if

asbestos material is damaged extensively and cannot be otherwise repaired. Removal is complex and must be done only by a contractor with special training. Improper removal of asbestos material may create more of a problem than simply leaving it alone.

Lead

Many individuals recognize lead in the form often seen in tire weights and fishing equipment, but few recognize its various forms in and around the home. The Merriam-Webster Dictionary [56] defines lead as "a heavy soft malleable ductile plastic but inelastic bluish white metallic element found mostly in combination and used especially in pipes, cable sheaths, batteries, solder, and shields against radioactivity." Lead is a metal with many uses. It melts easily and quickly. It can be molded or shaped into thin sheets and can be drawn out into wire or threads. Lead also is very resistant to weather conditions. Lead and lead compounds are toxic and can present a severe hazard to those who are overexposed to them. Whether ingested or inhaled, lead is readily absorbed and distributed throughout the body.

Until 1978, lead compounds were an important component of many paints. Lead was added to paint to promote adhesion, corrosion control, drying, and covering. White lead (lead carbonate), linseed oil, and inorganic pigments were the basic components for paint in the 18th and 19th centuries, and continued until the middle of the 20th century. Lead was banned by CPSC in 1978. Lead-based paint was used extensively on exteriors and interior trim-work, window sills, sashes, window frames, baseboards, wainscoting, doors, frames, and high-gloss wall surfaces, such as those found in kitchens and bathrooms. The only way to determine which building components are coated with lead paint is through an inspection for lead-based paint. Almost all painted metals were primed with red lead or painted with lead-based paints. Even milk (casein) and water-based paints (distemper and calcimines) could contain some lead, usually in the form of hiding agents or pigments. Varnishes sometimes contained lead. Lead compounds also were used as driers in paint and window-glazing putty. Lead is widespread in the environment. People absorb lead from a variety of sources every day. Although lead has been used in numerous consumer products, the most important sources of lead exposure to children and others today are the following:

- contaminated house dust that has settled on horizontal surfaces,
- deteriorated lead-based paint,

- contaminated bare soil,
- food (which can be contaminated by lead in the air or in food containers, particularly lead-soldered food containers),
- drinking water (from corrosion of plumbing systems), and
- occupational exposure or hobbies.

Federal controls on lead in gasoline, new paint, food canning, and drinking water, as well as lead from industrial air emissions, have significantly reduced total human exposure to lead. The number of children with blood lead levels above 10 micrograms per deciliter ($\mu\text{g}/\text{dL}$), a level designated as showing no physiologic toxicity, has declined from 1.7 million in the late 1980s to 310,000 in 1999–2002. This demonstrates that the controls have been effective, but that many children are still at risk. CDC data show that deteriorated lead-based paint and the contaminated dust and soil it generates are the most common sources of exposure to children today. HUD data show that the number of houses with lead paint declined from 64 million in 1990 to 38 million in 2000 [57].

Children are more vulnerable to lead poisoning than are adults. Infants can be exposed to lead in the womb if their mothers have lead in their bodies. Infants and children can swallow and breathe lead in dirt, dust, or sand through normal hand-to-mouth contact while they play on the floor or ground. These activities make it easier

for children to be exposed to lead. Other sources of exposure have included imported vinyl miniblinds, crayons, children’s jewelry, and candy. In 2004, increases in lead in water service pipes were observed in Washington, D.C., accompanied by increases in blood lead levels in children under the age of 6 years who were served by the water system [58].

In some cases, children swallow nonfood items such as paint chips. These may contain very large amounts of lead, particularly in and around older houses that were painted with lead-based paint. Many studies have verified the effect of lead exposure on IQ scores in the United States. The effects of lead exposure have been reviewed by the National Academy of Sciences [59].

Generally, the tests for blood lead levels are from drawn blood, not from a finger-stick test, which can be unreliable if performed improperly. Units are measured in micrograms per deciliter and reflect the 1991 guidance from the Centers of Disease Control [60]:

- Children: 10 $\mu\text{g}/\text{dL}$ (level of concern)—find source of lead;
- Children: 15 $\mu\text{g}/\text{dL}$ and above—environmental intervention, counseling, medical monitoring;
- Children: 20 $\mu\text{g}/\text{dL}$ and above—medical treatment;
- Adults: 25 $\mu\text{g}/\text{dL}$ (level of concern)—find source of lead; and

Action Levels for Lead

Lead in paint. Differing methods report results in differing units. Lead is considered a potential hazard if above the following levels, but can be a hazard at lower levels if improperly handled. Below are the current action levels identified by HUD [62] and EPA (40 CFR Part 745):

Lab analysis of samples:

5,000 milligram per kilogram (mg/kg) or 5,000 parts per million (ppm)
0.5% lead by weight.

X-ray fluorescence:

1 milligram per square centimeter (mg/cm^2)

Lead in dust:

Floors, 40 micrograms per square foot ($\mu\text{g}/\text{ft}^2$)
Window sills, 250 $\mu\text{g}/\text{ft}^2$
Window troughs, 400 $\mu\text{g}/\text{ft}^2$ (clearance only)

Lead in soil:

High-contact bare play areas: 400 ppm
Other yard areas: 1,200 ppm

- Adults: 50 µg/dL—Occupational Safety and Health Administration (OSHA) standard for medical removal from the worksite.

Adults are usually exposed to lead from occupational sources (e.g., battery construction, paint removal) or at home (e.g., paint removal, home renovations).

In 1978, CPSC banned the use of lead-based paint in residential housing. Because houses are periodically repainted, the most recent layer of paint will most likely not contain lead, but the older layers underneath probably will. Therefore, the only way to accurately determine the amount of lead present in older paint is to have it analyzed.

It is important that owners of homes built before 1978 be aware that layers of older paint can contain a great deal of lead. Guidelines on identifying and controlling lead-based paint hazards in housing have been published by HUD [61].

Controlling Lead Hazards

The purpose of a home risk assessment is to determine, through testing and evaluation, where hazards from lead warrant remedial action. A certified inspector or risk assessor can test paint, soil, or lead dust either on-site or in a laboratory using methods such as x-ray fluorescence (XRF) analyzers, chemicals, dust wipe tests, and atomic absorption spectroscopy. Lists of service providers are

available by calling 1-800-424-LEAD. Do-it-yourself test kits are commercially available; however, these kits do not tell you how much lead is present, and their reliability at detecting low levels of lead has not been determined. Professional testing for lead in paint is recommended. The recommended sampling method for dust is the surface wet wipe. Dust samples are collected from different surfaces, such as bare floors, window sills, and window wells. Each sample is collected from a measured surface area using a wet wipe, which is sent to a laboratory for testing. Risk assessments can be fairly low-cost investigations of the location, condition, and severity of lead hazards found in house dust, soil, water, and deteriorating paint. Risk assessments also will address other sources of lead from hobbies, crockery, water, and work environments. These services are critical when owners are seeking to implement measures to reduce suspected lead hazards in housing and day-care centers or when extensive rehabilitation is planned.

HUD has published detailed protocols for risk assessments and inspections [61].

It is important from a health standpoint that future tenants, painters, and construction workers know that lead-based paint is present, even under treated surfaces, so they can take precautions when working in areas that will generate lead dust. Whenever mitigation work is completed, it is

Definitions Related to Lead

Deteriorated lead-based paint: Paint known to contain lead above the regulated level that shows signs of peeling, chipping, chalking, blistering, alligating, or otherwise separating from its substrate.

Dust removal: The process of removing dust to avoid creating a greater problem of spreading lead particles; usually through wet or damp collection and use of HEPA vacuums.

Hazard abatement: Long-term measures to remove the hazards of lead-based paint through replacement of building components, enclosure, encapsulation, or paint removal.

Interim control: Short-term methods to remove lead dust, stabilize deteriorating painted surfaces, treat friction and impact surfaces that generate lead dust, and repaint surfaces. Maintenance can ensure that housing remains lead-safe.

Lead-based paint: Any existing paint, varnish, shellac, or other coating that is equal to or greater than 1.0 milligrams per square centimeter (mg/cm²) or greater than 0.5% by weight (5,000 ppm, 5,000 micrograms per gram [µg/g], or 5,000 milligrams per kilogram [mg/kg]). For new paint, CPSC has established 0.06% as the maximum amount of lead allowed in new paint. Lead in paint can be measured by x-ray fluorescence analyzers or laboratory analysis by certified personnel and approved laboratories.

Risk assessment: An on-site investigation to determine the presence and condition of lead-based paint, including limited test samples and an evaluation of the age, condition, housekeeping practices, and uses of a residence.

important to have a clearance test using the dust wipe method to ensure that lead-laden dust generated during the work does not remain at levels above those established by the EPA and HUD. Such testing is required for owners of most housing that is receiving federal financial assistance, such as Section 8 rental housing. A building or housing file should be maintained and updated whenever any additional lead hazard control work is completed. Owners are required by law to disclose information about lead-based paint or lead-based paint hazards to buyers or tenants before completing a sales or lease contract [62].

All hazards should be controlled as identified in a risk assessment.

Whenever extensive amounts of lead must be removed from a property, or when methods of removing toxic substances will affect the environment, it is extremely important that the owner be aware of the issues surrounding worker safety, environmental controls, and proper disposal. Appropriate architectural, engineering, and environmental professionals should be consulted when lead hazard projects are complex.

Following are brief explanations of the two approaches for controlling lead hazard risks. These controls are recommended by HUD in HUD *Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing* [61], and are summarized here to focus on special considerations for historic housing:

Interim Controls. Short-term solutions include thorough dust removal and thorough washdown and cleanup, paint film stabilization and repainting, covering of lead-contaminated soil, and informing tenants about lead hazards. Interim controls require ongoing maintenance and evaluation.

Hazard Abatement. Long-term solutions are defined as having an expected life of 20 years or more and involve permanent removal of hazardous paint through chemicals, heat guns, or controlled sanding or abrasive methods; permanent removal of deteriorated painted features through replacement; removal or permanent covering of contaminated soil; and the use of enclosures (such as drywall) to isolate painted surfaces. The use of specialized encapsulant products can be considered as permanent abatement of lead.

Reducing and controlling lead hazards can be successfully accomplished without destroying the character-defining features and finishes of historic buildings. Federal and

state laws generally support the reasonable control of lead-based paint hazards through a variety of treatments, ranging from modified maintenance to selective substrate removal. The key to protecting children, workers, and the environment is to be informed about the hazards of lead, to control exposure to lead dust and lead in soil and lead paint chips, and to follow existing regulations.

The following summarizes several important regulations that affect lead-hazard reduction projects. Owners should be aware that regulations change, and they have a responsibility to check state and local ordinances as well. Care must be taken to ensure that any procedures used to release lead from the home protect both the residents and workers from lead dust exposure.

Residential Lead-Based Paint Hazard Reduction Act of 1992, Title X [62]. Part of the Housing and Community Development Act of 1992 (Public Law 102-550) [63]. It established that HUD issue *Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing* [61] to outline risk assessments, interim controls, and abatement of lead-based paint hazards in housing. Title X calls for the reduction of lead in federally supported housing. It outlines the federal responsibility toward its own residential units and the need for disclosure of lead in residences, even private residences, before a sale. Title X also required HUD to establish regulations for federally assisted housing (24 CFR Part 35) and EPA to establish standards for lead in paint, dust, and soil, as well as standards for laboratory accreditation (40 CFR Part 745). EPA's residential lead hazard standards are available at <http://www.epa.gov/lead/leadhaz.htm>.

Interim Final Rule on Lead in Construction (29 Code of Federal Regulations [CFR] 1926.62) [64]. Issued by OSHA, these regulations address worker safety, training, and protective measures. The regulations are based in part on personal-air sampling to determine the amount of lead dust exposure to workers.

State Laws. States generally have the authority to regulate the removal and transportation of lead-based paint and the generated waste through the appropriate state environmental and public health agencies. Most requirements are for mitigation in the case of a lead-poisoned child, for protection of children, or for oversight to ensure the safe handling and disposal of lead waste. When undertaking a lead-based paint reduction program, it is important to determine which laws are in place that may affect the project.

Local Ordinances. Check with local health departments, poison control centers, and offices of housing and community development to determine whether any laws require compliance by building owners. Determine whether projects are considered abatements and will require special contractors and permits.

Owner's Responsibility. Owners are ultimately responsible for ensuring that hazardous waste is properly disposed of when it is generated on their own sites. Owners should check with their state government to determine whether an abatement project requires a certified contractor. Owners should establish that the contractor is responsible for the safety of the crew, to ensure that all applicable laws are followed, and that transporters and disposers of hazardous waste have liability insurance as a protection for the owner. The owner should notify the contractor that lead-based paint may be present and that it is the contractor's responsibility to follow appropriate work practices to protect workers and to complete a thorough cleanup to ensure that lead-laden dust is not present after the work is completed. Renovation contractors are required by EPA to distribute an informative educational pamphlet (Protect Your Family from Lead in Your Home) to occupants before starting work that could disturb lead-based paint (<http://www.epa.gov/lead/leadinfo.htm#remodeling>).

Arsenic

Lead arsenate was used legally up to 1988 in most of the orchards in the United States. Often 50 applications or more of this pesticide were applied each year. This toxic heavy metal compound has accumulated in the soil around houses and under the numerous orchards in the country, contaminating both wells and land. These orchards are often turned into subdivisions as cities expand and sprawl occurs. Residues from the pesticide lead arsenate, once used heavily on apple, pear, and other orchards, contaminate an estimated 70,000 to 120,000 acres in the state of Washington alone, some of it in areas where agriculture has been replaced with housing, according to state ecology department officials and others.

Lead arsenate, which was not banned for use on food crops until 1988, nevertheless was mostly replaced by the pesticide dichlorodiphenyltrichloroethane (DDT) and its derivatives in the late 1940s. DDT was banned in the United States in 1972, but is used elsewhere in the world.

For more than 20 years, the wood industry has infused green wood with heavy doses of arsenic to kill bugs and

prevent rot. Numerous studies show that arsenic sticks to children's hands when they play on treated wood, and it is absorbed through the skin and ingested when they put their hands in their mouths. Although most uses of arsenic wood treatments were phased out by 2004, an estimated 90% of existing outdoor structures are made of arsenic-treated wood [65].

In a study conducted by the University of North Carolina Environmental Quality Institute in Asheville, wood samples were analyzed and showed that

- Older decks and play sets (7 to 15 years old) that were preserved with chromated copper arsenic expose people to just as much arsenic on the wood surface as do newer structures (less than 1 year old). The amount of arsenic that testers wiped off a small area of wood about the size of a 4-year-old's handprint typically far exceeds what EPA allows in a glass of water under the Safe Drinking Water Act standard. Figure 5.9 shows a safety warning label placed on wood products.
- Arsenic in the soil from two of every five backyards or parks tested exceeded EPA's Superfund cleanup level of 20 ppm.

Arsenic is not just poisonous in the short term, it causes cancer in the long term. Arsenic is on EPA's short list of chemicals known to cause cancer in humans. According to the National Academy of Sciences, exposure to arsenic causes lung, bladder, and skin cancer in humans, and is suspected as a cause of kidney, prostate, and nasal passage cancer.

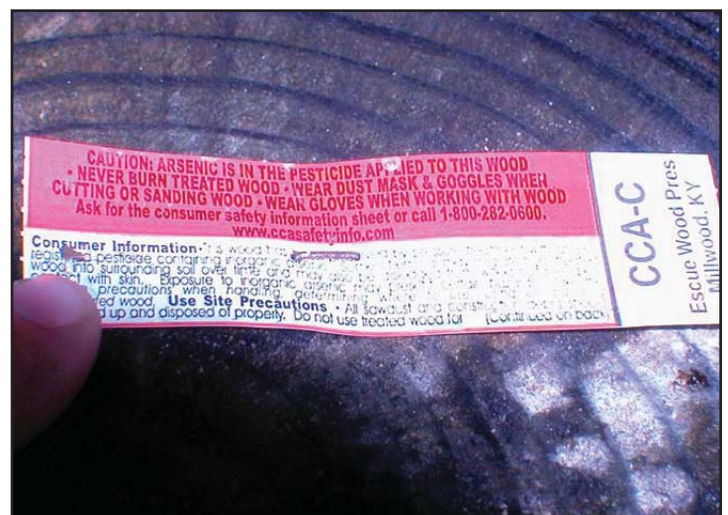


Figure 5.9. Arsenic Label

References

1. US Environmental Protection Agency and the US Consumer Product Safety Commission. The inside story: a guide to indoor air quality. Washington, DC: US Environmental Protection Agency and the US Consumer Product Safety Commission, Office of Radiation and Indoor Air; 1995. Document #402-K-93-007. Available from URL: <http://www.epa.gov/iaq/pubs/insidest.html>.
2. Centers for Disease Control and Prevention. Asthma: speaker's kit for health care professionals; preface. Atlanta: US Department of Health and Human Services; no date. Available from URL: <http://www.cdc.gov/asthma/speakit/intro.htm>.
3. US Environmental Protection Agency. Washington, DC: US Environmental Protection Agency; 2004. Asthma facts. EPA #402-F-04-019. Available from URL: http://www.epa.gov/asthma/pdfs/asthma_fact_sheet_en.pdf.
4. Centers for Disease Control and Prevention. Asthma: speaker's kit for health care professionals; epidemiology. Atlanta: US Department of Health and Human Services; no date. Available from URL: <http://www.cdc.gov/asthma/speakit/epi.htm>.
5. Crain EF, Walter M, O'Connor GT, Mitchell H, Gruchalla RS, Kattan M, et al. Home and allergic characteristics of children with asthma in seven U.S. urban communities and design of an environmental intervention: The Inner-City Asthma Study. *Environ Health Perspect* 2002;119:939–45.
6. Community Environmental Health Resource Center. Cockroaches: tools for detecting hazards. Washington, DC: Community Environmental Health Resource Center; no date. Available from URL: <http://www.cehrc.org/tools/cockroaches/index.cfm>.
7. Lyon WF. Entomology, House Dust Mites HYG 2157-97. Columbus, OH: The Ohio State University Extension; 1997. Available from URL: <http://ohioline.osu.edu/hyg-fact/2000/2157.html>.
8. Morgan WJ, Crain EF, Gruchalla RS, O'Connor GT, Kattan M, et al. Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med* 2004;351:1068–80.
9. US Environmental Protection Agency. Sources of indoor air pollution—biological pollutants. Washington, DC: US Environmental Protection Agency; no date. Available from URL: <http://www.epa.gov/iaq/biologic.html>.
10. Ownby DR. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age. *JAMA* 2002;288(8):963–72.
11. Roost HP. Role of current and childhood exposure to cat and atopic sensitization. *J Allergy Clin Immunol* 1999;104(5):94.
12. Downs SH. Having lived on a farm and protection against allergic diseases in Australia. *Clin Exp Allergy* 2001;31(4):570–5.
13. Institute of Medicine. Clearing the air: asthma and indoor air exposures. Washington, DC: National Academies Press; 2000.
14. American Conference of Governmental and Industrial Hygienists. Macher J, editor. Bioaerosols: assessment and control. Cincinnati, OH: American Conference of Governmental and Industrial Hygienists; 1999.
15. Institute of Medicine. Damp indoor spaces and health. Washington, DC: National Academies Press; 2004.
16. Morgan WJ, Crain EF, Gruchalla RS, O'Connor GT, Kattan M, Evans R 3rd, et al. Results of home-based environmental intervention among urban children with asthma. *N Engl J Med* 2004;351:1068–80.
17. Burge HA, Ammann HA. Fungal toxins and β (1 \rightarrow 3)-D-glucans. In: Macher J, editor. Bioaerosols: assessment and control. Cincinnati, OH: American Conference of Governmental and Industrial Hygienists; 1999.
18. American Academy of Pediatrics, Committee on Environmental Health. Toxic effects of indoor molds. *Pediatrics* 1998;101:712–14.
19. Burge HA, Otten JA. 1999. Fungi. In: Macher J, editor. Bioaerosols: assessment and control. Cincinnati, OH: American Conference of Governmental and Industrial Hygienists; 1999.

20. Etzel RA. The “fatal four” indoor air pollutants. *Pediatr Ann* 2000;29(6):344–50. [IEH/documents/HUD%20Mold%20Detection.pdf](http://www.ehponline.org/view/fullarticle.aspx?url=/documents/HUD%20Mold%20Detection.pdf).
21. Dearborn DG, Smith PG, Dahms BB, Allan TM, Sorenson WG, Montana E, et al. Clinical profile of thirty infants with acute pulmonary hemorrhage in Cleveland. *Pediatrics* 2002;110:627–37.
22. Etzel RA, Montana E, Sorenson WG, Kullman GJ, Allan TM, Dearborn DG. Acute pulmonary hemorrhage in infants associated with exposure to *Stachybotrys atra* and other fungi. *Arch Pediatr Adolesc Med* 1998;152:757–62.
23. Flappan SM, Portnoy J, Jones P, Barnes C. Infant pulmonary hemorrhage in a suburban home with water damage and mold (*Stachybotrys atra*). *Environ Health Perspect* 1999;107:927–30.
24. Vesper S, Dearborn DG, Yike I, Allan T, Sobolewski J, Hinkley SF, et al. Evaluation of *Stachybotrys chartarum* in the house of an infant with pulmonary hemorrhage: quantitative assessment before, during, and after remediation. *J. Urban Health Bull N Y Acad Med* 2000;77(1):68–85.
25. US Environmental Protection Agency. A brief guide to mold, moisture, and your home (EPA 402-K-02-003). Washington, DC: US Environmental Protection Agency; 2002. Available from URL: <http://www.epa.gov/iaq/molds/images/moldguide.pdf>.
26. NYC. 2000. Guidelines on assessment and remediation of fungi in indoor environments. New York City Department of Health, Bureau of Environmental & Occupational Disease Epidemiology. Available from URL: <http://www.ci.nyc.ny.us/html/doh/html/epi/moldrpt1.shtml>.
27. Bush RK, Portnoy JM. The role and abatement of fungal allergens in allergic diseases. *J Allerg Clin Immunol Suppl* 2001;107(3 pt 2):430.
28. Department of Housing and Urban Development, Office of Native American Programs. Mold detection and prevention: a guide for housing authorities in Indian Country. Washington, DC: Department of Housing and Urban Development; 2001. Available from URL: <http://www2.ihs.gov/>
29. US Environmental Protection Agency. Mold remediation in schools and commercial buildings (EPA 402-K-01-001). Washington, DC: US Environmental Protection Agency; 2001.
30. Shaughnessy RJ, Morey PR. Remediation of microbial contamination. In: Macher J, editor. *Bioaerosols: assessment and control*. Cincinnati, OH: American Conference of Governmental and Industrial Hygienists; 1999.
31. National Institute of Environmental Health Sciences. Interim final guidelines for the protection and training of workers engaged in maintenance and remediation work associated with mold. Report of a national technical workshop, National Institute of Environmental Health Sciences, New York City, 2004 Jan 27–28.
32. Mott JA, Wolfe MI, Alverson CJ, Macdonald SC, Bailey CR, Ball LB, et al. National vehicle emissions policies and practices and declining US carbon monoxide mortality. *JAMA* 2002;288:988–95.
33. American Lung Association. Carbon monoxide fact sheet. New York: American Lung Association; 2004. Available from URL: <http://www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=35375>.
34. North Carolina Department of Health and Human Services. Indoor ozone. Raleigh, NC: North Carolina Department of Health and Human Services; 2003. Available from URL: <http://www.epi.state.nc.us/epi/oeel/ozone/indoor.html>.
35. US Environmental Protection Agency. Ozone generators that are sold as air cleaners: an assessment of effectiveness and health consequences. Washington, DC: US Environmental Protection Agency; no date. Available from URL: <http://www.epa.gov/iaq/pubs/ozonegen.html>.
36. National Cancer Institute. Secondhand smoke: questions and answers. Bethesda, MD: National Cancer Institute; no date. Available from URL: <http://www.cancer.gov/cancertopics/factsheet/Tobacco/ETS>.

37. US Environmental Protection Agency. What you can do about secondhand smoke as parents, decision-makers, and building occupants. Washington, DC: US Environmental Protection Agency; 1993. Available from URL: <http://www.epa.gov/smokefree/pubs/etsbro.html>.
38. US Environmental Protection Agency. Methylene chloride (dichloromethane). Washington, DC: US Environmental Protection Agency; 1992. Available from URL: <http://www.epa.gov/ttnatw01/hlthef/methylen.html>.
39. Environmental Media Services. Four to avoid. Washington, DC: Environmental Media Services; 2002.
40. US Consumer Product Safety Commission. An update on formaldehyde: 1997 revision. Washington, DC: Consumer Product Safety Commission; 1997. CPSC Document 725. Available from URL: <http://www.cpsc.gov/cpscpub/pubs/725.html>.
41. US Environmental Protection Agency. Sources of indoor air pollution—formaldehyde. Washington, DC: US Environmental Protection Agency. Available from URL: <http://www.epa.gov/iaq/formalde.html>.
42. US Department of Housing and Urban Development. Formaldehyde emission controls for certain wood products. 24 CFR3280.308. Washington, DC: US Department of Housing and Urban Development; 2001. Available from URL: <http://www.hudclips.org/cgi/index.cgi>.
43. US Environmental Protection Agency. Assessment of risks from radon in homes. Washington, DC: US Environmental Protection Agency; 2003. Available from URL: http://www.epa.gov/radon/risk_assessment.html.
44. US Geological Survey. The geology of radon. Reston, VA: US Geological Survey; 1995. Available from URL: <http://energy.cr.usgs.gov/radon/georadon/3.html>.
45. California Geological Survey. Radon. Sacramento, CA: California Geological Survey; no date. Available from URL: http://www.consrv.ca.gov/cgs/minerals/hazardous_minerals/radon/.
46. US Department of Health and Human Services. Surgeon General releases national health advisory on radon. Washington, DC: US Department of Health and Human Services; 2005. Available from URL: <http://www.hhs.gov/surgeongeneral/pressreleases/sg01132005.html>.
47. Brain M, Freudenrich C. How radon works. Atlanta: How Stuff Works; no date. Available from URL: <http://home.howstuffworks.com/radon.htm>.
48. Pennsylvania Department of Environmental Protection. Mitigating your home or office. Harrisburg, PA: Pennsylvania Department of Environmental Protection; no date. Available from URL: http://www.dep.state.pa.us/dep/deputate/airwaste/rp/radon_division/Mitigation_Info.htm.
49. Connecticut Department of Public Health. Why should you build homes with radon-resistant techniques? Hartford, CT: Connecticut Department of Public Health Radon Program; no date. Available from URL: http://www.dph.state.ct.us/BRS/radon/radon_techniques.htm.
50. US Environmental Protection Agency. Radon-resistant new construction. Washington, DC: US Environmental Protection Agency; no date. Available from URL: <http://www.epa.gov/radon/construc.html>.
51. US Environmental Protection Agency. Pesticides and child safety. Washington, DC: US Environmental Protection Agency; no date. Available from URL: <http://www.epa.gov/pesticides/factsheets/childsaf.htm>.
52. Delaplane KS. Pesticide usage in the United States: history, benefits, risks, and trends. Athens, GA: Cooperative Extension Service, The University of Georgia College of Agriculture and Environmental Sciences; no date. Available from URL: <http://pubs.caes.uga.edu/caespubs/pubcd/B1121.htm>.
53. US Environmental Protection Agency. Sources of indoor air pollution—pesticides. Washington, DC: US Environmental Protection Agency; no date. Available from URL: <http://www.epa.gov/iaq/pesticid.html>.

54. Agency for Toxic Substances and Disease Registry. Public health statement for asbestos. Atlanta: US Department of Health and Human Services; 2001. Available from URL: <http://www.atsdr.cdc.gov/toxprofiles/phs61.html>.
55. US Geological Survey. Some facts about asbestos. USGS fact sheet FS-012-01, online version 1.1. Reston, VA: US Geological Survey; March 2001. Available from URL: <http://pubs.usgs.gov/fs/fs012-01/>.
56. Merriam-Webster, Inc. Merriam-Webster dictionary. Springfield, MA: Merriam-Webster, Inc.; no date. Available from URL: <http://www.m-w.com/home.htm>.
57. Jacobs DE, Clickner RP, Zhou JY, Viet SM, Marker DA, Rogers JW, et al. The prevalence of lead-based paint hazards in U.S. housing. *Environ Health Perspect* 2002;100:A599–606.
58. Centers for Disease Control and Prevention. Blood lead levels in residents of homes with elevated lead in tap water—District of Columbia, 2004. *MMWR* 2004; 53(12):268–70. Available from URL: <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5312a6.htm>.
59. National Research Council. Measuring lead exposure in infants, children and other sensitive populations. Washington, DC: National Academy Press; 1993.
60. Centers for Disease Control. Preventing lead poisoning in young children. Report No. 99-2230. Atlanta: US Department of Health and Human Services; 1991.
61. US Department of Housing and Urban Development. HUD technical guidelines for the evaluation and control of lead-based paint hazards in housing. Washington, DC: US Department of Housing and Urban Development; 1995. Available from URL: <http://www.hud.gov/offices/lead/guidelines/hudguidelines/index.cfm>.
62. US Department of Housing and Urban Development. The lead-based paint disclosure rule (Section 1018 of the Residential Lead-Based Paint Hazard Reduction Act of 1992). Washington, DC: US Department of Housing and Urban Development; no date. Available from URL: <http://www.hud.gov/offices/lead/disclosure/index.cfm>.
63. Residential Lead-Based Paint Hazard Reduction Act of 1992, Title X of the Housing and Community Development Act of 1992, Pub. L. No. 102-550 (Oct 28, 1992).
64. Occupational Safety and Health Administration. Lead exposure in construction: interim final rule. *Fed Reg* 1993;58:26590–649.
65. Environmental Working Group. Nationwide consumer testing of backyard decks and play sets shows high levels of arsenic on old wood. Washington, DC: Environmental Working Group; 2002. Available from URL: <http://www.ewg.org/reports/allhandsondeck/>.

