
Indoor pollution and its impact on respiratory health

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Learning Objectives: This overview discusses the respiratory complications of indoor air pollution, emphasizing the most common pollutants that individuals are likely to encounter outside the workplace.

Data Sources: Data were obtained from a review of the recent literature.

Study Selection: The expert opinion of the author was used to select and synthesize relevant data on this multifaceted subject.

Results: There have been a number of studies documenting an association between exposure to indoor allergens and development of both sensitization and asthma in children. In addition to classic allergens, chemical indoor air pollution may also exert an adverse effect on both the upper and lower respiratory tract by a variety of nonimmunologic, irritative mechanisms.

Conclusions: Our understanding of the adverse effects of indoor air pollution on health and comfort has broadened in recent years. It has supplied a credible framework for developing and implementing a variety of control strategies.

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INTRODUCTION

Over the last several decades, the prevalence of asthma and allergic disease has risen in many developed countries of the world.^{1–3} This rising prevalence has been associated with remarkable changes in both lifestyles and environmental quality (eg, a shift in the population to urban centers, greater numbers of automobiles on the highways).⁴ There have been a number of studies documenting an association between exposure to indoor allergens and development of both sensitization and asthma in children.⁵ Concern over indoor air has risen to the extent that the Centers for Disease Control and Prevention has identified indoor pollution as a high environmental risk.⁶

Homes have evolved with the purpose of protecting inhabitants from the elements. However, they do not protect dwellers from the effects of pollution. At times, home construction may even facilitate problems with indoor pollution. Modern homes are better insulated, but ventilation rates as low as 0.2 to 0.3 air exchanges/hour are commonplace.⁷ Advances in construction technology have led to a greater dependence on synthetic chemical materials. Pollutants emitted to the indoor air have much less opportunity to become diluted than those emitted outdoors. As a result, individuals encounter a broad range of pollutants as they travel through a succession of microenvironments in the course of their daily activities.^{8,9}

We will discuss the respiratory complications of indoor air pollution, emphasizing the most common pollutants that individuals are likely to encounter outside the workplace.

SOURCES OF INDOOR POLLUTION

The quality of indoor air depends both on the quality of outdoor air and the strength and nature of emissions of

indoor sources. Indoor air quality has been linked to climatic factors. In colder regions, central heating is a necessity, whereas air conditioning predominates in warmer zones. Some areas require both central heating and air conditioning, depending on the season. The proper maintenance and functioning of these mechanical ventilation systems is clearly related to the quality of indoor air. However, they have also led to warmer, more humid homes with decreased amounts of fresh outside air.⁴ A number of investigators have demonstrated that the respiratory health of children may be adversely affected by home dampness and general mold content.^{10,11} The mechanism operative in the latter remains to be elucidated. The sources of indoor pollution can be classified as follows:

Outdoor Air Quality

Buildings are entirely dependent on surrounding air for their source of indoor air. Indoor concentrations of outdoor pollutants such as ozone, carbon monoxide, sulfur dioxide, and respirable particles range between 20 and 80% of outdoor concentrations.¹² It is also possible to carry in other contaminants on clothing, shoes, via pet animals, plants, and Christmas trees (pollen, mold, pesticides, etc).

Biologic Exposures

Biologic contaminants and their by-products are important constituents of indoor air quality. Biologic pollutants include microbial cells such as bacteria and viruses, in addition to fungal spores, protozoans, algae, animal dander and excreta, pollens, and insect fragments and excreta. They rarely exhibit direct toxicity despite their prevalence indoors. More likely, they exert their unwanted effects via immunoglobulin (Ig)E-mediated sensitization or infection.

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Chemical Exposures

Chemical pollutants found in the indoor environment range from simple, ubiquitous agents, such as formaldehyde, carbon monoxide, and nitrogen dioxide, to exotic complex substances, such as remnants of pesticides. The largest component of indoor chemical exposure probably derives from combustion sources. Such sources emit a variety of inorganic gases, hydrocarbon gases, and associated impurities. The second most important group of chemicals related to indoor quality are the volatile organic compounds (VOCs). Among the miscellaneous agents are the pesticides. There are more than 20,000 registered pesticide products in the United States, and their use varies considerably depending upon the geographic location. A large market exists for the use of these chemicals indoors via direct application to structures and/or their contents such as furniture, rugs, and clothing. It has been estimated that nearly 80% of the public will use an insecticide product in their home during any 1 year.¹³

Occupant Activities

The personal habits and activities of building occupants can influence the induction of symptoms. One of the most important sources of indoor pollution is from tobacco smoke. Other contributors to the indoor air quality originate from the use of perfume, cosmetics, room deodorizers, and a variety of products related to hobbies.

IMMUNOLOGICALLY INDUCED RESPIRATORY ILLNESS

In general, indoor air pollution can be divided into agents which can induce respiratory disease immunologically and those which act by a nonimmune, usually irritative, mechanism. Over the last 30 years, major advances have been made in defining allergens in domestic dust which are responsible for IgE-mediated respiratory illness. The major sources of indoor allergens in the United States are house-dust mites, fungi and other microorganisms, do-

mestic pets (cats and dogs), cockroaches, and rodents (Table 1).

House-dust Mite

House-dust mites are small (up to 0.3 μm long), sightless, eight-legged arachnids related to ticks, spiders, and scabies mites. In homes, mites live in dust that accumulates in bedding, carpets, fabrics, and soft furnishings. The dust mite uses skin scales as its major food source. Torey et al¹⁴ were the first to report that aerosolized mite feces were the major source of house-dust allergen. The mattress is the most important site, and infestation is not dependent on the nature of the bedding.¹⁵

The major allergen has been well characterized as antigen Der p 1, 90% of which is associated with the outer membrane of the fecal particles.¹⁶ The major mite allergen is also found in dead mites. The most common species in North America and Europe are *Dermatophagoides farinae* and *D. pteronyssinus*. Their numbers are greatest in coastal areas with high humidity. Mite allergen seems to have seasonal variations tending to increase growth in the fall months concomitant with increases in outdoor humidity. It is known that

80% relative humidity and 25° C temperature are optimal for culturing of the *D. pteronyssinus* dust mite. Mite activity and growth are focused on their requirement to maintain body water. Numbers tend to rise a month after outdoor humidity rises. Both Der p 1 and Der f 1 have been identified as cysteine proteases ranging in size between 10 and 40 μm and do not remain airborne for more than 5 minutes. However, airborne levels in home environments which are highly trafficked can be 1,000 times higher than in undisturbed rooms.¹⁷

The majority of the literature on respiratory disease associated with house-dust mite exposure has focused on bronchial asthma. The presence of sensitization to dust mite is strongly associated with increased airway responsiveness and asthma.^{18,19} Current guidelines indicate that 2 μg of Der p 1 per gram of dust from mattresses and carpets (equivalent to 100 mites/g) should be regarded as a risk factor for sensitization and development of asthma, and that 10 μg of Der p 1 per gram of dust (equivalent to 500 mites/g) should be considered a major risk factor for the development of acute asthma in mite-sensitive individuals.²⁰⁻²²

Hence, there is little doubt that allergic sensitization to house-dust mite is a risk factor for persistent asthma.¹⁸⁻²² In the United States, dwellings with high mite concentrations were found to be associated with sensitivity to mite allergens and an increased prevalence of asthma.²³ In Australian children who had complained of frequent wheezing in the previous year, there was an increased exposure to Der p 1.²⁴ In Sweden, asthma-related symptoms were most prevalent in adults who lived in homes with high mite populations.²⁵ There was also indirect evidence implicating mite allergen exposure as a cause of asthma.²⁶

Despite the evidence that allergic sensitization to mite allergen is a risk factor for persistent asthma, it remains unclear whether the onset of asthma is increased by exposure to mite allergen early in life or prevented by avoidance. Thus far, there have been three longi-

Table 1. Classification of the Major Indoor Allergens

Acarids
Housedust mite
Storage mite
Domestic and other animals
Cat, dog, ferrets, horses, monkey
Birds
Parrot, parakeet, cockatiel, etc
Insects
Cockroaches
Crickets, flies, moths, etc
Fungi
<i>Penicillium</i> , <i>Cladosporium</i>
<i>Aspergillus</i> , <i>Mucor</i>
Pollens
Transmitted from outside (grass, weed and tree pollen)
Rodents
Mice
Gerbil, guinea pig
Other
Indoor plants
Kapok, latex
Low molecular weight chemicals

tudinal studies addressing this issue. In two studies the children had a family history of atopy. Sporik et al²² were unable to show significant associations between early allergen exposure and active wheezing or airway hyperresponsiveness, thought to be surrogates for asthma. Another study showed no association between early allergen exposure and the development of physician-confirmed asthma.²⁷ A recent longitudinal study was especially notable because 62% of the cohort were randomly selected from a population not at high risk of atopy. Children were enrolled as newborns, were seen frequently during the first 2 years of life, then annually until the age of 7. Carpet dust was collected at 6, 18, and 36 months of age and analyzed for the presence of dust mite and cat allergens.²⁸ Sensitization to specific foods and inhaled allergen was evaluated by RAST, and bronchial hyperresponsiveness was evaluated by histamine challenge. There was a significant association between sensitization to mite and/or cat allergen and wheezing from age 3 onward. At age 7, sensitized individuals showed a greater bronchial response than non-sensitized individuals to histamine. There was a relationship between exposure to indoor allergens and sensitization; however, it was not related to any of the asthma indicators that were addressed or tested.²⁸ Pearce et al²⁹ recently reviewed the studies on this topic and concluded there was no evidence that allergen exposure early in life is a major risk factor for the development of asthma. Platts-Mills et al³⁰ differ with this evaluation and maintain that for dust-mite allergens, there is good evidence for a dose-response link joining exposure and both sensitization and asthma.

Cat and Dog Allergens

The allergenic content of household dust is prominently contaminated by animal allergens. Although only 30 to 40% of homes in the United States have pets, animal dander can be detected in almost all homes.^{19,31} Although a larger number of individuals in the population experience allergic

reactions to various animals, the exact prevalence of this common problem has not yet been determined. There are data suggesting that at least 2% of the general population and up to 50% of asthmatic children are sensitive to cat allergen.^{32,33}

The most important cat allergen is Fel d 1. It is produced by feline sebaceous and salivary glands and can be isolated from saliva, pelt, lacrimal glands, and urine. Fel d 1 is produced by all breeds of cats and is especially concentrated at the root of the hair. Fel d 1 is carried on small particles (<2.5 μm) and, unlike mite allergen, is extremely buoyant. It can be airborne for many hours. Cat sensitivity is an important allergy to detect because it has a predilection to cause more severe ocular and respiratory symptoms than those caused by other animals. Cat-allergic individuals tend to develop symptoms rapidly upon entering a contaminated home, supporting the premise that the allergen remains airborne for protracted periods. Symptoms related to cat allergen exposure frequently involve development of acute asthma which may precipitate visits to emergency rooms.³⁴ Levels of cat allergen as low as 2 μg Fel d 1/gram of dust, commonly observed in houses without cats, may be a risk factor for sensitization to Fel d 1,³⁵ but homes with cat levels of 8 μg Fel d 1/gram of dust are associated with respiratory allergy symptoms.³⁴ A recent population-based, cross-sectional study showed that exposure to cat allergen can produce an IgG and IgG1 antibody response without sensitization or risk of asthma.³⁰ This study showed that high exposure levels seemed to be protective for some children and a risk factor for others. This does not seem to be the case for mite allergen and is felt to be of great importance in understanding the factors that influence the prevalence of allergic disease in developed countries.³⁰

There seem to be more dogs than cats in households, and it clearly is the most popular domesticated pet in the United States.¹⁹ Dog allergen has been detected in up to 60% of homes in the Baltimore area.³⁶ Evidence of sensitiv-

ity to dog allergen has been noted in 40% of asthmatic children.³⁷ Differences in sensitivity between cat and dog may be related to the less well characterized antigen for skin testing and the fact that many dog owners keep their dogs outside the home.³⁸

The major dog allergen has been termed Can f 1. Can f 1 is a relatively stable molecule and may persist in dust for extended periods of time. Many dog owners indicate that they are not allergic to their particular breed. There are data indicating that approximately 15% of dog-sensitive patients demonstrate significant differences among skin test responses to different dog breeds. This suggests the presence of breed-specific allergens, but there is no evidence to suggest breeds of dogs that are nonallergenic.^{38,39} There is also some cross-reactivity between dog and cat allergens.

Other animals have also been the source of allergens within the home. Asthma triggered by exposure to monkeys has been reported in primate centers and could occur in the home where such animals are kept as pets.⁴⁰ A recent report described horse dander as a "hidden allergen," causing symptoms in children.⁴¹ As with cat allergen, they demonstrated it could be readily transported on clothing. As well, positive skin test reactions to feather extracts, but not fresh feathers, have been reported. This may reflect contamination from dust mites.³⁸ The most common sources of exposure to feathers are pillows, comforters, quilts, down-filled clothing, and feather beds. Pet birds may cause hypersensitivity pneumonitis secondary to sensitivity to bird droppings which dry and are aerosolized in the home.

Rodents

Rodents can be a significant source of allergens in an occupational setting, eg, laboratory workers. A variety of species have also become popular as house pets, eg, hamsters, gerbils, and guinea pigs. Recent studies have found that mouse allergen was strikingly prevalent among inner-city homes, with allergen being detected in >95%

of the homes studied.⁴² In fact, mouse allergen was more prevalent in inner-city homes than dust mite or cat allergens, suggesting that mouse allergen rivals cockroach allergen as a uniquely important allergen in this population. The same investigators reported a prevalence of mouse sensitization of 18%, compared with 37% for cockroach allergen, 35% for dust mite allergen, and 23% for cat allergen in the same study population.⁴³ The degree of exposure and presence of atopy contributed to the development of sensitization, but the relationship of sensitization to mouse allergen and any measures of asthma morbidity was not statistically significant.⁴⁴

Cockroach

Although there are scattered reports of many insect species identified as sources of inhalant allergens (eg, crickets, flies, moths), only the cockroach has been repeatedly recognized as a common source of indoor inhalant allergen.³⁸ Recent studies have shown a significant association between evidence of cockroach infestation on inspection and elevated mouse allergen levels.⁴² Sporik et al⁴⁵ found that the frequency of sensitization to dust mite and cockroach allergen was strongly associated with atopy and increasing domestic concentrations of these allergens, whereas the same relationship was not seen for cat allergen.

Of the seven or eight indoor species, the American cockroach (*Periplaneta americana*) and the German cockroach (*Blattella germanica*) predominate in the United States, whereas the Oriental cockroach (*Blatta orientalis*) is more common in the United Kingdom.¹⁸ Three major allergens have been identified: Bla g 1, Bla g 2, and Per a 1. Sources of cockroach allergens have been identified in body parts as well as fecal extracts. Cockroach sensitivity is the most prevalent allergen in crowded urban dwellings.⁴³ It has a different distribution pattern from cat and dust mite, as it found in kitchen cabinets, kitchen floor dust, bathrooms, and basements. Recently, the National Cooperative Inner-City Asthma Study re-

ported a relationship between cockroach allergen exposure and sensitivity with asthma morbidity in inner-city children with asthma.⁴³

Fungi

Fungi are an ecologically diverse group of organisms, some of which are capable of being infectious agents and most of which are saprophytic. The important indoor fungi are typically saprobes that can use many materials as growth substrates. The environmental requirements of fungi include oxygen, a suitable temperature, and a source of nutrition and moisture.⁴⁵ Preventing access to any one requirement will control fungal growth. Airborne, indoor spores are generally a mixture of spores from both indoor and outdoor sources.⁴⁶ The latter is especially true during the summer and autumn months.

Quantitative sampling for indoor fungi is complex and difficult to execute and is a major impediment to understanding the true health significance of indoor fungi.⁴⁷ Several issues should be addressed whenever sampling is conducted including: 1) whether to study viable versus all particulate fungal components; 2) types of media used; 3) collection of airborne versus settled dust to look for fungal components; and 4) when and where to collect samples. No single fungal sampling strategy is perfect. Several major problems are associated with any major attempt to evaluate the role of fungal exposure in the home with the development and exacerbation of both allergic and nonallergic symptoms. Most previously published studies have only focused on indoor home-based exposures. However, contrary to other indoor allergens discussed previously, it is likely that significant exposure to fungi occurs outdoors, in schools, and other settings.⁴⁸⁻⁵⁰ Ignoring these alternate sources is likely to invalidate any attempt at relating indoor fungal exposure and symptoms or disease. *Penicillium* and *Aspergillus* are the two most important indoor genera. Each of these contains many species whose spores are virtually indistinguishable. *Cladosporium* and *Alternaria* species occur commonly both indoors and outdoors.

Concentrations of airborne fungi outdoors routinely vary over several orders of magnitude, ie, from <10 to >10,000 colony-forming units (CFU)/m³ and from <1,000 to >60,000 spores/m³.⁴⁷ Because indoor fungal levels reflect a combination of what is outside and inside, perturbations in outdoor levels can confound values noted inside. Hourly variations have been noted to be considerable.⁵⁰ In a study of 40 selected Australian houses, both total indoor culturable and total fungal spore levels were observed to be relatively high with 58% of houses with one or more rooms exceeding 1,000 CFU/m³, and 48% exceeding 10,000 CFU/m³. An evaluation of the indoor/outdoor ratios of selected genera indicated that 50% of indoor concentrations could be explained by outdoor levels.⁵¹

No precise threshold values have been established for exposure to indoor fungi. There is some agreement that levels <500 CFU/m³ pose little or no threat to healthy inhabitants. The limited analysis of fungal allergens in indoor environments precludes estimation of a risk level for symptom exacerbation, or even determination of what constitutes a high level, in sensitized patients. Such studies will undoubtedly be facilitated by development of monoclonal antibodies to fungal allergens.^{45,48,49} This issue as well as that of mycotoxins will be discussed in greater detail by Dr. Harriett A. Burge in a subsequent paper in this issue (52-56).

NONIMMUNOLOGICALLY INDUCED RESPIRATORY DISEASE

Aside from the classic allergens, chemical indoor air pollution may exert an adverse effect on both the upper and lower respiratory tract by a variety of nonimmunologic mechanisms. They can exert a direct irritation effect with development of varying degrees of inflammation depending on the nature of the exposure, its concentration, and the duration of the exposure. In addition to a direct irritative effect, chemical air pollutants can result in an increased incidence of respiratory tract infection by adversely affecting specific and nonspecific host defenses of the respi-

ratory tract.⁵² For example, there is indirect evidence that the gas and particulate phase of environmental tobacco smoke (ETS) adversely affects ciliary function in the respiratory tract, as well as humoral and cellular immune defenses.⁵³ This is especially true in children, in whom it has been shown that passive exposure to tobacco smoke will result in more frequent severe exacerbations of asthma, middle ear infections, bronchitis, and pneumonia.⁵⁴ Exposure to chemical air pollutants might also act to increase the severity of respiratory infections mediated by the inflammation of the epithelial surfaces of the tracheobronchial tree caused by direct irritation.

Another mechanism which may be operative with respect to pollution relates to recent observations that diesel particles contribute to the exacerbation of allergic inflammation. Exposure to diesel exhaust particles in the presence of an allergen has led to increased IgE levels specific to that allergen and a shift toward a T helper cell 2-like cytokine pattern.^{55,56}

Combustion Products

There is an increased awareness that pollutants from combustion sources may contaminate indoor environments. Sources of combustion pollutants are numerous in the home environment and include tobacco smoke, gas cooking stoves, pilot lights, unvented kerosene space heaters, wood and coal stoves, and fireplaces. The pollutants that fall into this category include carbon monoxide, nitrogen dioxide, and formaldehyde.

Carbon monoxide (CO). CO is a nonirritating, odorless, colorless, and tasteless gas that is an insidious toxicant. It is not a respiratory irritant, but it does represent a potential threat to health because of its high affinity binding to hemoglobin (200 times greater than oxygen). High indoor-to-outdoor ratios are typically not observed for CO. Indoor concentrations in homes and public buildings for this nonreactive gas are generally similar to ambient levels. CO causes approximately 900 accidental deaths in the United States annually by asphyxiation. Al-

though most of these are related to exposure in homes, at least a portion result from exposure in public access buildings.⁵⁷

Oxidation of methane is the largest natural source of CO in the atmosphere with a significant amount also released from the oceans. Other natural sources include volcanoes, forest and grass fires, marsh gases, and electrical storms. Large amounts of CO are released into the atmosphere secondary to human activities. Combustion of petroleum products accounts for >80% of the emissions.

Although tobacco smoking accounts for negligible amounts of CO in the atmosphere, it is a significant source of indoor levels. The CO concentration of cigarette smoke ranges from 10,000 to 40,000 ppm and most smokers have carboxyhemoglobin levels of 3 to 10%. The half-life of CO in the body ranges from 2.5 to 4 hours. Most measurements of CO in buildings have been made to assess the contribution from cigarette smoking and generally have been several parts per million. However, smoking has been associated with levels as high as 10 ppm.⁵⁸

Acute effects of exposure are very nonspecific and may be mistaken for flu-like illness. In order of increasing severity, the most commonly reported manifestations are frontal or temporal headache, roaring in the ears, weakness, dizziness, sleepiness, muscle weakness, incoordination, impaired judgment, collapse with increased pulse and respiratory rate, and eventual loss of consciousness. Pulmonary edema is commonly seen as a sequelae of fatal reactions along with ischemia or frank infarction of the brain and cardiac cells. Acute symptoms from low-level exposure without unconsciousness are usually self-limited and most victims recover completely. Well informed observers and researchers throughout the world have differing opinions as to whether chronic carbon monoxide poisoning exists.⁵⁹

Nitrogen dioxide (NO₂). Nitrogen dioxide is an oxidant gas that is soluble in tissues. Uptake studies show that most inhaled NO₂ is retained in the lungs, primarily in the small airways,

with deposition in the alveoli.⁵⁸ NO₂ may be present in the form of a yellowish brown liquid or reddish brown gas with a pungent, acrid odor. It plays an important role in the formation of photochemical smog. In most urban areas of the United States, ambient NO₂ levels are low and the primary contribution to total personal exposure is received at home from unvented gas-fueled cooking stoves and space heaters.⁶⁰ Over the last several decades many surveys of NO₂ concentrations have been performed in dwellings. These studies have shown that during the winter months with windows and doors closed, the average indoor concentrations are 0.5 to 2 times higher than outdoors in homes with gas-fueled appliances.⁶¹ For example, in almost 300 homes with gas stoves in Boston, MA, indoor levels averaged 26 ppb in the bedroom and 39 ppb in the kitchen when outdoor levels were averaging 22 ppb.⁶²

Health effects of NO₂ have been investigated in the laboratory setting in volunteers and with epidemiologic approaches in a community setting. The focus has been to determine potential health effects in children. Despite a large number of studies and approaches, the evidence remains inconclusive with regard to health outcomes.⁵⁸ The evidence at present indicates that NO₂ exposure typically found in indoor environments has minimal clinical implications for most healthy individuals as well as for most people with asthma.

Formaldehyde (CH₂O). Formaldehyde is a ubiquitous airborne pollutant in our modern environment. It is a pungent, highly reactive chemical that cross-links with many organic molecules. Its wide distribution has caused considerable public health concern and debate in the last several decades. Early observations showing that formaldehyde-altered proteins could react as antigens along with later observations demonstrating it to be a good skin sensitizer has led some to postulate its potential role as a respiratory tract sensitizer.⁶³

There are many potential sources of formaldehyde exposure both in the industrial setting as well as in a variety of public and private dwellings. Most manufactured formaldehyde is used in the production of phenolic, urea, melamine, and acetal resins. In turn, these resins are used extensively in the manufacture of textiles, floor coverings, plywood, and carbonless paper. The Environmental Protection Agency estimates the mean conventional home levels to be 0.03 ppm. This will vary considerably based on ventilation, cigarette smoking, and use of woodburning in the home. Modern building offices and day-care centers have been reported to have mean formaldehyde levels of 0.37 and 0.55 ppm, respectively.⁶⁴

Ambient formaldehyde primarily affects the upper airways and ocular tissues. Formaldehyde is associated with a disagreeable odor that can induce "annoyance" symptoms and at higher concentrations can be a transient, completely reversible irritant to the eyes and mucous membranes of the respiratory tract. It is so soluble and rapidly metabolized that it rarely reaches the lower respiratory tract to inflict damage. The exception is in cigarette smokers who actively inhale. Formaldehyde may on rare occasions induce bronchial asthma at relatively high exposure doses. However, there is no conclusive evidence that proves the development of de novo IgE-mediated respiratory tract symptoms secondary to inhalation of formaldehyde vapors.⁶³

ETS

One of the most common indoor pollutants worldwide is tobacco smoke. Tobacco smoke is comprised of numerous pollutants which overlap greatly with the VOCs found indoors (Table 2). In 1991, 46.3 million American adults (25.7%) were current cigarette smokers.⁶⁵ Surveys have indicated that 53 to 76% of children's homes had at least one smoker in the home.⁶⁶ Children and other vulnerable segments of the population (the chronically ill and the elderly) spend a great deal of time in the home with the potential for significant exposures to ETS.

Table 2. Selected Constituents in Mainstream Tobacco Smoke

Vapor Phase
Acetic acid
Acetone
Acrolein
Ammonia
Benzene
Carbon dioxide
Carbon monoxide
Dimethylamine
Formaldehyde
Hydrogen cyanide
Hydrogen sulfide
Methyl chloride
Methyl ethyl ketone
Nitrogen oxide
Toluene
Xylene
Particulate Phase
Aniline
Benzoic acid
Benzopyrene
Cadmium
Lactic acid
Nicotine
Particulates
Phenol

Tobacco smoke is composed of more than 3,800 different chemicals, most of which are potent respiratory irritants and/or carcinogens. Because of the complex nature of ETS, measurements of respirable particles are used as markers of indoor concentrations. ETS particles range in size between 0.1 and 1.5 μm . Spengler et al⁶⁷ studied 80 homes to determine the impact on the indoor air quality. They found that a smoker of one pack a day contributed approximately 20 $\mu\text{g}/\text{m}^3$ to the 24-hour indoor concentrations. Because cigarettes are not smoked uniformly throughout the day, the authors concluded that short-term particulate concentrations of 500 to 1,000 $\mu\text{g}/\text{m}^3$ were likely when cigarettes were ignited.⁶⁷

Chest irritation is one of the most common health effects associated with exposure to ETS.⁴ There is considerable evidence that links higher risk of acute childhood lower respiratory tract illnesses and exposure to passive smoking at home.⁵⁴ There is also evidence of a significant increase in the prevalence of persistent otitis media

with childhood exposure to passive smoking. Passive smoke exposure may also predispose to or aggravate childhood asthma.⁶⁸ Passive smoking also increases the incidence of infection and colonization with bacteria.⁶⁹

VOCs

VOCs are organic compounds that contain at least one carbon and one hydrogen atom in their molecular structure and which have a low boiling point. They exist in various structural forms, including straight or branched chain (aliphatic), ring (aromatic), substituted (halogenated), or oxygenated (alcohols, ketones, ethers, aldehydes, esters) compounds (Table 3). Formaldehyde is one of the most widespread of the VOCs. Of a total of more than 900 chemical and biologic substances identified in indoor air, more than 350 VOCs have been detected at concentrations that exceed 1 ppb.⁷⁰ VOCs are typically present in indoor air at concentrations considerably higher than outdoor concentrations, because there are many more potential sources of indoor contamination. The VOCs have numerous residential and commercial applications including building materials, adhesives, cleaning fluids, etc.

The majority of reported building investigations involve very low concentrations of VOCs. However, even at low levels, many VOCs have perceptible, at times unpleasant, odors that precipitate feelings of great concern

Table 3. Selected Indoor VOCs

Aldehydes
Acetaldehyde
Acrolein
Formaldehyde
Propanolol
Aliphatic halogenated hydrocarbons
Carbon tetrachloride
Chloroform
1,1,1-trichloroethane
Trichloroethylene
Aromatic hydrocarbons
Benzene
Toluene
Xylene
Terpenes
Limonene
Pinene

among building occupants. In many cases the presence of odor occurs at extremely low concentrations that are unassociated with adverse health effects.⁷¹ At sufficiently high concentrations, symptoms of transient sensory irritation of the eyes, nose, and throat may be evident. Of importance is the fact that total VOC concentration in most indoor air environments rarely exceeds 1.0 mg/m³, except during or immediately after construction. There is no evidence of either aggravation or de novo induction of asthma secondary to VOC concentrations at or below 15 mg/m³.

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