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SOURCES AND CONCENTRATIONS OF INDOOR AIR POLLUTION

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Indoor air pollution arises from indoor and outdoor sources. This chapter describes indoor sources of air pollution. Information on outdoor sources can be obtained from standard texts such as Lippmann and Schlesinger (1979) and Stern et al. (1984). Indoor pollutants can be categorized by type of source, such as combustion, and by pollutant group, such as volatile organic compounds (VOCs) and fibers. Sources can be characterized by the pollutants emitted, by the source locations, and by the rate and pattern of emissions. In the context of mass-balance models, we consider the roles of source strength, volume of distribution, dilution, and removal in determining indoor concentrations (NRC 1981a). These models do not consider, however, patterns of source use and maintenance, which may be strong determinants of exposure. For some pollutants, the presence of a potential source does not necessarily indicate exposure. For example, the presence of fibrous asbestos-containing material in a building may not result in exposure to asbestos except under circumstances in which the material is disturbed or inadequately maintained.

Pollutant species are typically described under the broad source headings of combustion, evaporation, abrasion, biologic, and radon. Table 2.1 lists the major indoor sources and contaminants. Some sources are potential and contribute to indoor air pollution only when used or operated in an improper or unplanned fashion (e.g., asbestos).

Emission rates for indoor sources are reported in a variety of units. Physical mass-balance emission rates in models typically require units of mass per time, such as micrograms per second or grams per hour. Combustion sources that consume fuel can be characterized by mass released per unit of energy. If the fuel consumption rate and the energy per unit are known, fuel emissions can be reexpressed as mass per time. The emission rates for most conventional combustion

Table 2.1 Sources of Common Indoor Contaminants

Contaminant	Source
Asbestos	
Chrysotile	Some wall and ceiling insulation installed between 1930 and 1950
Crocidolite	Old insulation on heating pipes and equipment
Amosite	Old wood stove door gaskets
Tremolite	Some vinyl floor tiles
	Drywall joint-finishing material and textured paint purchased before 1977
	Cement-asbestos millboard and exterior wall shingles
	Some sprayed and troweled ceiling finishing plaster installed between 1945 and 1973
	Sprayed onto some structural steel beams as fire retardant
Combustion By-products	
Carbon monoxide	Gas range
Nitrogen dioxide	Wood and coal stoves
Sulfur dioxide	Gas and propane engines
Particulate soot	Fireplaces
Nitrogenated compounds	Backdrafting of exhaust flues
	Candles and incense
Tobacco Smoke	
Carbon monoxide	Cigarettes
Nitrogen dioxide	Pipes
Carbon dioxide	Cigars
Hydrogen cyanide	
Nitrosamines	
Aromatic hydrocarbons	
Benzo[a]pyrene	
Particles	
Benzene	
Formaldehyde	
Nicotine	
Formaldehyde	Some particle board, plywood, pressed-board, paneling
	Some carpeting and carpet backing
	Some furniture and dyed materials
	Urea-formaldehyde insulating foam
	Some household cleaners and deodorizers
	Combustion (gas, tobacco, wood)
	Some glues and resins
	Tobacco smoke
	Cosmetics
	Permanent-press textiles
Biologic organisms	
Fungal spores	Mold, mildew, and other fungi
Bacteria	Humidifiers with stagnant water
Viruses	Poorly maintained surfaces and systems
Pollen	Decaying soils and dirt near the air intake system
Protozoa	Drainage pans in refrigerators
	Some thermophilics on dirty heating coils
	Animals
	Rodents
	Insects
	Humans

(*continued*)

Table 2.1 (*Continued*)

Contaminant	Source
Radon	
Radon gas and radon progeny	Radon gas emanating from soil, rocks, and water that diffuses through cracks and holes in the foundation and floor
	Radon in well water
	Radon in natural gas used near the source wells
	Some building materials such as granite
Volatile organic compounds	
Alkanes	Solvents and cleaning compounds
Aromatic hydrocarbons	Paints
Esters	Glues and resins
Alcohols	Spray propellants
Aldehydes	Fabric softeners and deodorizers
Ketones	Combustion
	Dry-cleaning fluids
	Some fabrics and furnishings
	Stored gasoline
	Outgasing from water
	Some building materials
	Waxes and polishing compounds
	Pens and markers
	Binders and plasticizers

sources have been characterized in laboratory settings (Moschandreas 1983). For some sources, *in situ* measurements have been reported, and real-life emissions have been shown to vary considerably from the laboratory conditions.

This chapter does not provide detailed information on emission rates; emission rate data are published in several documents and reports, including the Environmental Protection Agency's *Preliminary Indoor Air Pollution Information Assessment Appendix*, published in June 1987 (EPA/600/8–87/014). This EPA document reports emission rates for combustion appliances and also summarizes emission rates of formaldehyde and other organic vapors from building materials. Emissions from cigarettes have been reported in considerable detail in recent reports (National Research Council [NRC] 1986; U.S. Department of Health and Human Services [U.S. DHHS] 1986).

Describing "steady-state" or even transient emission rates may not provide sufficient information. For the purpose of modeling, sources may be considered as having constant emission rates; but in actual use, emission rates vary. Combustion sources often emit more during start-up than during continued use, and fuel characteristics and fueling rates can cause emissions to vary. Cooking and smoking may take place both periodically and sporadically. Nevertheless, the routine contributions of these sources to indoor pollution can be estimated. For example, the potential for vented appliances to backdraft can be calculated from such factors as chimney height, emission buoyancy, and wind speed over the stack. However, the backdrafting of a water heater and the associated quantity of emissions released into the interior of the building cannot be readily predicted.

Some contaminants are outgased from materials. Although the overall emission rate depends upon the amount of material used, emission rates are usually not constant. For example, the release rate of formaldehyde from urea-formaldehyde foam insulation (UFFI) depends upon partial pressures of the gas, humidity, temperature, and the air exchange rate. Similarly, the leakage of soil gases such as radon into the basement of a home varies not only with the concentration beneath the slab, but with the fluctuation of air pressures in the basement.

Asbestos fibers are released into indoor air episodically and not at a constant rate. Fibers can become airborne when the surface of asbestos-containing materials is mechanically disrupted, but fibers can also be released through other mechanisms. Nicholson et al. (1978) measured an increased asbestos concentration in a school after a heavy rainstorm. The increase was attributed to asbestos washed from asbestos cement walkways and asbestos cement roof panels. Danish investigations have shown that generation of man-made mineral fibers from ceiling tiles depends upon the binders and other factors (Danish Building Research Institute 1987). Apparently, water-based resins absorb moisture and weaken the bond, releasing fibers more readily than epoxy-type binders.

At present, it is nearly impossible to characterize the source rate for biologic materials. Spores can be released periodically or only with an appropriate environmental stimulus (by heat or moisture, for example). Other biologically important material, such as house dust mites or the nonviable, but allergenic, material from fungal organisms, can be present but not airborne. Vacuuming, shaking blankets, or even a child's playing on a carpet can cause these materials to become airborne and result in antigen exposure. Unless samples are taken when the biologic material is actively introduced into the air, antigenic material may not be detected. Such biologic exposures illustrate the potential influence of human activities on source strength; however, it is impossible to predict the source strength in terms of mass of antigenic material released per unit of time for these activities.

CONTAMINANTS BY SOURCE

COMBUSTION

Combustion sources emit inorganic gases (H_2O, NO, NO_2, CO, CO_2) and particles. In addition, depending on fuel type and pyrolysis conditions, combustion sources can also emit hydrocarbon gases, vapors, and organic particles. Most liquid and solid fuels have impurities or additives. As the fuels burn, metals, mercaptans, sulfur oxides, and particles can be emitted. The particles may be partially burned carbon soot or minerals and are readily visible when released from oil-, wood-, or kerosene-fueled appliances. Many of the particles produced are large and settle out, contributing to soiling. However, even gas appliances, if operated under stoichiometric conditions, will emit particles in the submicron size range. Burning tobacco generates particles in the 0.1- to 1-μm range.

Because some combustion sources burn at temperatures high enough (>900°C) to produce atomic nitrogen, these sources can produce nitrogenated species in the effluent. Traynor et al. (1983a, 1983b) have reported formation of nitrated polycyclic aromatic hydrocarbons from both well-tuned and maladjusted kerosene space heaters but at rates 10,000 to 100,000 times lower than the total particulate emission rates.

Gas Combustion Unvented gas combustion is a ubiquitous source of NO_2 and CO in residences. Many studies indicate that gas ranges can increase indoor NO_2 concentrations above ambient levels. The NO_2 levels in homes with gas ranges are nearly always higher than the levels inside homes with electric ranges in the same community (Wade, Cote, and Yocom 1975; Spengler et al. 1979, 1983; Yocom et al. 1982; Moschandreas 1983; Marbury et al. 1988). Figure 2.1 displays week-long NO_2 averaged concentrations collected over a year-long study in Portage, Wisconsin. The ambient levels were between 15 and 20 $\mu g/m^3$. For electric cooking homes, the average indoor concentrations were less than ambient levels; in the gas cooking homes, indoor concentrations always exceeded ambient levels. Kitchen values were twice the bedroom concentrations. A distinct seasonal pattern was present, with higher concentrations in the fall and winter seasons.

In a more recent study, more than five hundred homes in the Los Angeles area were monitored for NO_2 three times during a year (Baker et al. 1987; Colome et al. 1987). In Los Angeles, the ambient levels of NO_2 were more than five times higher than in Portage. Although gas cooking sources were important contributors to indoor NO_2 air pollution, the ambient air had a major influence. A similar large-scale study of five hundred residential units was conducted in Boston (Ryan et al. 1988). Data from this study show the importance of gas pilot lights, which consume about one-third of the gas burned by a cooking stove and contribute about 12 ppb to the indoor NO_2 concentrations.

Most of the studies of indoor NO_2 report the time-averaged concentration over days to weeks. However, under most circumstances, range use usually lasts minutes, and most meals are prepared in less than one hour. Harlos (1988) equipped subjects with personal continuous NO_2 monitors and measured their exposures to NO_2 during the preparation of fifty meals (Figure 2.2). The short-term concentrations of one minute or less were independent of the longer averaging times and reflect the exposures received by a person working near the range or opening the oven door. Personal exposures for averaging times of three minutes and longer correlated with readings of a fixed-location monitor in the kitchen. This correlation indicates that mixing and dilution factors are more important than the individual's behavior in determining concentrations for longer averaging times.

Surveys of indoor NO_2 concentrations document considerable variation among homes, reflecting differences in source use, emission rates, mixing of the air, house volume, and air exchange rates. Most homes, even with unvented combustion sources, do not exceed the national ambient air quality standard for NO_2. Types of sources and home volume have been shown to underlie elevated con-

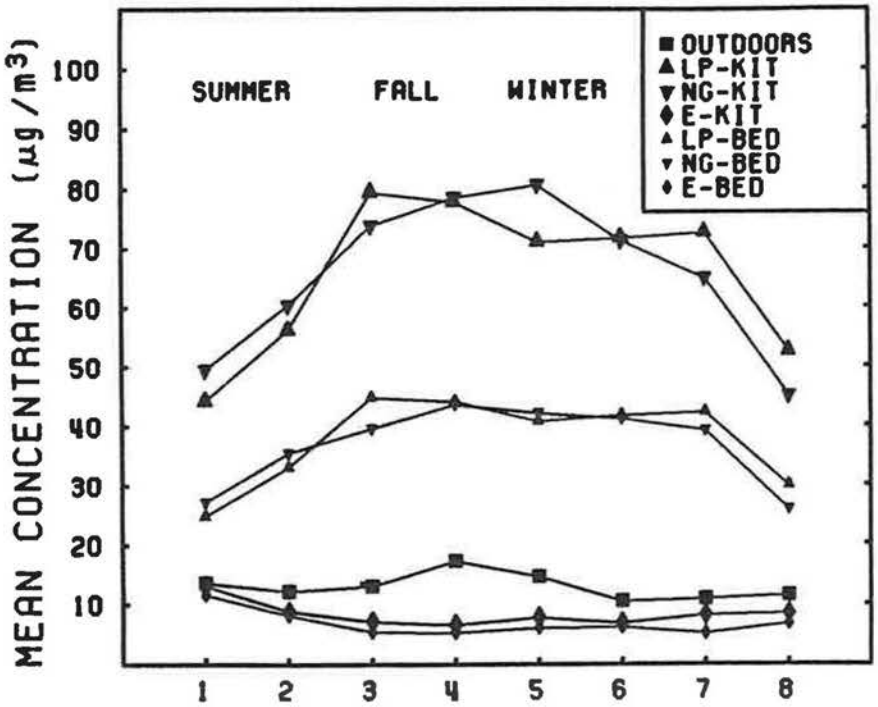

Figure 2.1. Seasonal variation (July 1980–June 1981) NO_2 concentrations (μg/m³) in indoor locations and outdoors in Portage, Wisconsin. Kitchen, bedroom, and outdoors in homes with liquid propane (LP), natural gas (NG), and electricity (E) as cooking fuels. *Source:* Spengler et al. (1983), reprinted with permission.

centrations in some homes. Wall and floor furnaces were associated with increased concentrations in the Los Angeles study. In the Boston study, public housing units tended to have higher concentrations.

The studies by Goldstein et al. (1987) of NO_2 and CO in Harlem, New York, housing showed that excessive stove use and small-volume living units contributed to elevated concentrations. Parkhurst et al. (1988) reported a winter survey of NO_2 in public housing in Chattanooga, Tennessee. In this urban environment, housing [illegible] rate of NO_2. Yet, under typical conditions, concentrations do not exceed 10 ppm

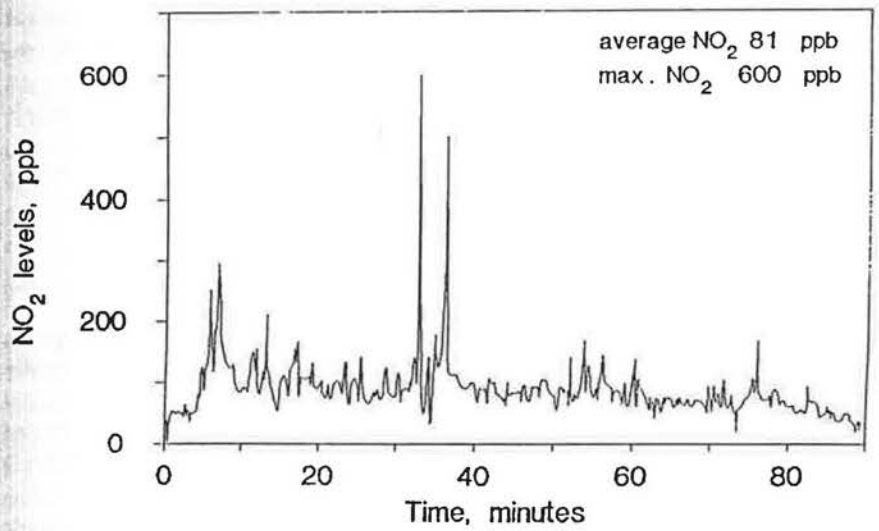

Figure 2.2. Real-time personal exposure to NO_2 concentrations (ppb) during meal preparation using a gas-fueled cooking range. *Source:* Harlos (1988), reprinted with permission.

over the cooking period. Nevertheless, the extreme conditions that result in elevated NO_2 concentrations also cause higher CO levels. Concentrations of 25–50 ppm have been measured in kitchens in New York City (Sterling and Kobayashi 1981). Gas ranges represent a potentially greater source of exposure than unvented space heaters. It is estimated that 45 percent of American homes, about 50 million housing units, use natural gas (U.S. Department of Energy 1987). Most of these units are in urbanized areas where ambient NO_2 and CO levels are elevated by stationary and mobile source emissions.

Space Heaters In contrast, only 11 percent of the U.S. population is potentially exposed to gas or kerosene space heater emissions, which include particles, CO, and NO_2 and SO_2 if sulfur-containing fuel is burned. Leaderer et al. (1984) conducted a survey of indoor air quality in a small number of Connecticut homes. The SO_2 levels were less than 2 μg/m³ inside homes without kerosene space heaters but were 60–150 μg/m³ inside homes using this fuel. Apparently, in Connecticut at the time, commercial kerosene had a measurable amount of sulfur. Kerosene space heaters and gas ranges emit particulate matter (Girman et al. 1985; Traynor et al. 1986, 1987). Studies have shown that kerosene heater [illegible] mutagenic (Sexton et al. 1986).

Wood Burning In some regions of the country, wood stoves are an important source of primary heat in the home; about 6 percent of homes have wood stoves, and 19 percent have fireplaces. Emissions from wood stoves and fireplaces are vented to the outdoors. However, during start-up and stoking, and if the system is not airtight, emissions can contaminate the indoor air. Under such instances, transient particle levels may exceed a few hundred $\mu g/m^3$ (Hawthorne et al. 1988). But, on the average, only a few $\mu g/m^3$ of additional indoor particulate matter can be attributed to the presence of a home wood-burning or coal-burning stove (Sexton, Spengler, and Treitman 1984; Tennessee Valley Authority 1985).

Several investigators have indicated that wood burning produces highly mutagenic emissions. However, indoor air samples in homes with airtight wood stoves showed no mutagenic activity. In contrast, mutagenicity increased significantly with wood burning in open fireplaces and with tobacco smoking (Alfeim and Ramdahl 1984). Reports from the Environmental Protection Agency's integrated air cancer study indicated that the fraction of ambient particulate matter which is wood smoke is mutagenic (Lewtas, Claxton, and Mumford 1987). Wood combustion emissions that leak into the home may be a potential source of mutagenic material.

Other Combustion Sources Significant exposure to combustion products may occur in numerous additional settings. Internal combustion engines running on gasoline or propane are used indoors. Spengler, Stone, and Lilley (1978) showed that CO in ice skating rinks might exceed 50 ppm for several hours due to exhaust emissions from resurfacing machines. Newspapers have published accounts of CO poisoning associated with hockey rinks.

Offices, hotels, hospitals, schools, shopping centers, and department stores often have attached or sublevel garages. Buildings with attached garages and offices connected to warehouses that use gasoline or propane forklifts can have elevated concentrations of combustion-related emissions. Often, sealed high-rise buildings have internal air pressures that are slightly positive on the upper floors but negative on the lower floors with respect to outside atmospheric pressure. Garage or roadside emissions can be entrained into these buildings. Cortese (1976), Ott and Flachsbart (1982), Akland et al. (1985), Flachsbart and Brown (1985a, 1985b), and Flachsbart and Ott (1986) have reported on CO in buildings in Washington, D.C., San Francisco, Honolulu, and Boston. Typically, indoor CO concentrations are lower than roadside ambient concentrations except when automobile emissions are entrained into the building. Under these conditions, the eight-hour (9 ppm) and one-hour (35 ppm) federal outdoor air quality standards can be exceeded indoors.

In more developed countries, nearly everyone spends time in automobiles or buses and is exposed to vehicle exhaust. Emissions can enter the passenger section of the vehicle from holes in the fire wall behind the engine, from faulty exhaust systems beneath the floor, from reentrainment in the negative pressure wake draft behind the car, or through windows and ventilation systems. Levels of CO exceeding 50 ppm may be produced. Cortese and Spengler (1976) estimated that 3–5 percent of all vehicles might produce such conditions. Cracks in the heating manifold have also caused high CO levels in the cockpits of small private airplanes. Even without faulty systems or entrained emission trapped in the wake of a vehicle, higher exposure will be experienced during vehicle transit. Spengler, Billick, and Ryan (1984) summarized exposures to lead particles in vehicles. Several studies have shown levels in vehicles three to ten times higher than concentrations measured at fixed locations in the urban environment.

Harlos (1988) conducted personal NO_2 monitoring in Boston. NO_2 concentrations varied depending on the mode of transportation (walking, bicycle, or automobile), time of day, and route. But in general, in-transit concentrations were 20–100 percent higher than concentrations measured at fixed locations in the city. Similar results were recently reported from a commuter exposure study conducted in Los Angeles (Shikiya et al. 1989). This study showed that other factors, such as time on the freeways and window and ventilation settings, influenced concentrations experienced by the driver.

Several investigators have shown that in-vehicle concentrations of CO are higher than those monitored at fixed urban locations (Cortese and Spengler 1976; Ott and Willits 1981; Ziskind et al. 1981; Akland et al. 1985; Flachsbart and Brown 1985b). Personal driving habits also can influence exposure. At stoplights (or in heavy traffic), CO concentrations can exceed 100 ppm if a driver brings the car close to the vehicle in front. Operating ventilation on *fresh air* intake setting instead of *recirculation* draws exhaust into the car.

Tobacco Combustion The burning of tobacco products is a ubiquitous source of a great number of indoor contaminants. Tobacco burning produces a complex mixture of gases, vapors, and particulate matter. More than 4,500 compounds have been identified from burning tobacco, and 50 of these are known or suspected carcinogens (U.S. DHHS 1986). Tobacco smoke can be categorized as mainstream, sidestream, and environmental tobacco smoke. Mainstream (MS) emissions have been well characterized. These are the gases, vapors, and particles sucked from the "mouth end" of a cigarette. Under MS conditions, the burning cone of the tobacco may reach temperatures of 900°C. At this temperature, N_2 in the air dissociates to atomic nitrogen. In the cooling gas, atomic nitrogen reacts to form NO, NO_2, hydrogen cyanide (HCN), and highly carcinogenic *N*-nitrosamines (Hoffman and Brunneman 1983; U.S. DHHS 1986). Numerous other compounds are also found.

Sidestream smoke (SS) refers to the emissions from the smoldering cigarette; the cone temperature may drop as low as 400°C as the cigarette smolders. In the laboratory, these emissions are captured in close proximity to the cigarette. Approximately half of the tobacco is consumed during smoldering and the remainder during active puffing. Because the pyrolysis conditions are quite different during active puffing and smoldering, the components of MS and SS tobacco smoke differ. Klus and Kuhn (1982) characterized MS and SS smoke; the results are

Table 2.2 Distribution of Constituents in Fresh, Undiluted Mainstream Smoke and Diluted Sidestream Smoke from Nonfilter Cigarettes[a]

Constituent	Amount in Mainstream Smoke	Range in Sidestream/ Mainstream Smoke
Vapor phase[b]		
Carbon monoxide	10–23 mg	2.5–4.7
Carbon dioxide	20–40 mg	8–11
Carbonyl sulfide	18–42 μg	0.03–0.13
Benzene[c]	12–48 μg	5–10
Toluene	100–200 μg	5.6–8.3
Formaldehyde	70–100 μg	0.1–≃50
Acrolein	60–100 μg	8–15
Acetone	100–250 μg	2–5
Pyridine	16–40 μg	6.5–20
3-Methylpyridine	12–36 μg	3–13
3-vinylpyridine	11–30 μg	20–40
Hydrogen cyanide	400–500 μg	0.1–0.25
Hydrazine[d]	32 ng	3
Ammonia	50–130 μg	40–170
Methylamine	11.5–28.7 μg	4.2–6.4
Dimethylamine	7.8–10 μg	3.7–5.1
Nitrogen oxides	100–600 μg	4–10
N-Nitrosodimethylamine[e]	10–40 ng	20–100
N-Nitrosodiethylamine[e]	Nondetectable–25 ng	<40
N-Nitrosopyrrolidine[e]	6–30 ng	6–30
Formic acid	210–490 μg	1.4–1.6
Acetic acid	330–810 μg	1.9–3.6
Methyl chloride	150–600 μg	1.7–3.3
Particulate Phase[b]		
Particulate matter[c]	15–40 mg	1.3–1.9
Nicotine	1–2.5 mg	2.6–3.3
Anatabine	2–20 μg	<0.1–0.5
Phenol	60–140 μg	1.6–3.0
Catechol	100–360 μg	0.6–0.9
Hydroquinone	110–300 μg	0.7–0.9
Aniline	360 ng	30
2-Toluidine	160 ng	19
2-Naphthylamine[c]	1.7 ng	30
4-Aminobiphenyl[c]	4.6 ng	31
Benz[a]anthracene[e]	20–70 ng	2–4
Benzo[a]pyrene[d]	20–40 ng	2.5–3.5
Cholesterol	22 μg	0.9
γ-Butyrolactone[e]	10–22 μg	3.6–5.0
Quinoline	0.5–2 μG	8–11
Harman[f]	1.7–3.1 μg	0.7–1.7
N'-Nitrosonornicotine[e]	200–3,000 ng	0.5–3
NNK[g]	100–1,000 ng	1–4
N-Nitrosodiethanolamine[e]	20–70 ng	1.2
Cadmium	100 ng	7.2
Nickel[d]	20–80 ng	13–30
Zinc	60 ng	6.7
Polonium-210[c]	0.04–0.1 pCi	1.0–4.0
Benzoic acid	14–28 μg	0.67–0.95
Lactic acid	64–174 μg	0.5–0.7
Glycolic acid	37–126 μg	0.6–0.95
Succinic acid	110–140 μg	0.43–0.62

(continued)

Table 2.2 (*Continued*)

[a]Diluted SS is collected with airflow of 25 ml/s, which is passed over the burning cone. Data from Elliot and Rowe (1975); Schmeltz et al. (1979); Hoffmann and Brunnemann (1983), Klus and Kuhn (1982), Sakuma et al. (1983, 1984a, 1984b), Hiller et al. (1982).
[b]Separation into vapor and particulate phases reflects conditions prevailing in mainstream smoke and does not necessarily imply same separation in sidestream smoke.
[c]Human carcinogen (U.S. DHHS 1986).
[d]Suspected human carcinogen (U.S. DHHS 1986).
[e]Animal carcinogen (Vainio, Hemminki, and Wilbourn 1985).
[f]1-Methyl-9H-pyrido(3,4-β)-indole.
[g]NNK = 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone.

summarized as a ratio of SS to MS emissions in Table 2.2. Many of these components have ratios greater than 1, indicating enrichment in the SS phase.

Environmental tobacco smoke (ETS) is the combination of exhaled MS and SS. The majority of ETS is made up of SS, which undergoes modification with dilution and aging after formation. Components with high vapor pressures may volatilize from particles to vapor phase. For example, nicotine is emitted in the SS particle phase and then evaporates to the vapor phase as it is diluted. The particles may accrete water, form aggregates, and change both in number count and diameter.

ETS cannot be measured directly in its entirety. Concentrations of individual ETS components have been reported: CO, nicotine, nitrogen oxides, aromatic hydrocarbons, acrolein, acetone, benzene, nitrous compounds, benzo[a]pyrene, and respirable suspended particles (RSP). These components have been measured in a variety of locations including homes, offices, restaurants, bars, clubs, arenas, trains, and airplanes, among others (see NRC 1986 and U.S. DHHS 1986 for reviews).

The concentrations of ETS constituents vary among indoor locations and over time. The number of smokers and the pattern of smoking determine the source strength for generation of ETS. An average smoker may smoke two cigarettes per hour over the day. But in some situations, such as in an airplane, smokers tend to light up at predictable times and produce high peak concentrations. In buildings with heating, ventilating, and air conditioning systems, the concentrations of ETS components are influenced by the ventilation rate, mixing, and directional flow of air currents. In homes, the volume of the room and the air exchange rate are determinants of ETS concentration.

Thus, the concentrations of ETS components to which nonsmokers are exposed depends on the degree of dilution of the smoke. As the intact plume of a smoldering cigarette passes by the nose or eyes, acute irritation can be experienced. The concentrations of formaldehyde and acrolein can still be several ppm a few feet from the burning cone of tobacco if the plume has not been mixed by air turbulence (Ayer and Yeager 1982).

More typically, however, ETS exposure occurs after SS and exhaled MS have been diluted several thousand times. Under these conditions, the concentrations experienced depend mostly on ventilation rates, smoking frequency, and room volume. In smoky bars, waiting areas, restaurants, automobiles, airplanes, or even in the home, short-term concentrations of ETS can be quite high. ETS particulate matter can range from 100 to more than 1,000 $\mu g/m^3$. Under these conditions, nicotine in closed environments can range from a few $\mu g/m^3$ to more than 100 $\mu g/m^3$. Nicotine concentrations up to 1,000 $\mu g/m^3$ have been reported (U.S. DHHS 1986, 155).

For some health end points, such as cancer and susceptibility to respiratory infection, a longer averaging time for assessing ETS impact on indoor environments may be most relevant. Comparatively few studies have sampled for ETS over days and months in an attempt to characterize long-term conditions. However, a rather consistent pattern emerges from the limited data available. Respirable particle levels in homes are associated with the number of cigarettes smoked (Spengler et al. 1981). On average, about 1 $\mu g/m^3$ of respirable-size particles is added to the long-term indoor concentrations for each cigarette smoked per day (Dockery and Spengler 1981). Figure 2.3 displays the results of repeated monitoring in 80 homes in six cities. On average, a home with one smoker had about 20 $\mu g/m^3$ greater concentration of RSP than one without smokers. Personal monitoring of nonsmokers confirms the importance of ETS exposure in determining personal exposures to particulates (Figure 2.4) (Spengler et al. 1985).

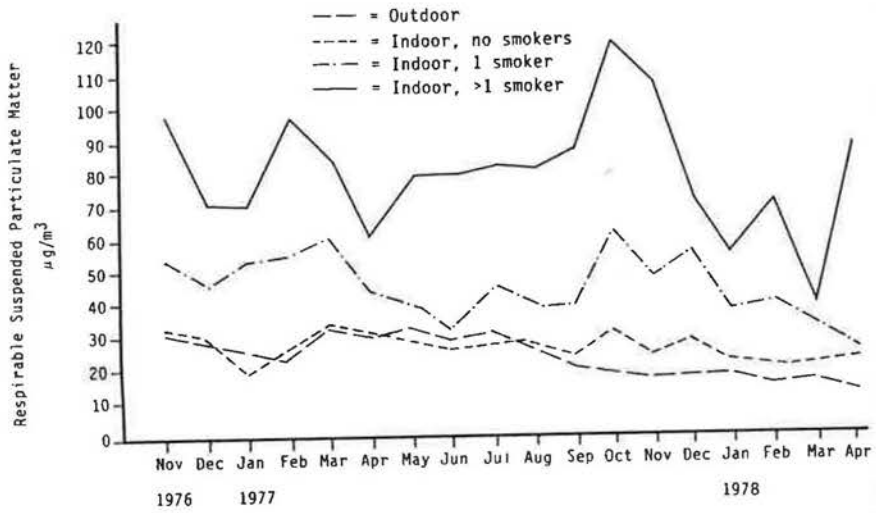

Figure 2.3. Monthly mean mass respirable particulate concentrations ($\mu g/m^3$) in homes with smokers and without smokers and in the outdoors across six cities. *Source:* Reprinted with permission from *Atmospheric Environment* 15, Spengler JD et al., Long-term measurements of respirable sulfates and particles inside and outside homes, Copyright 1981, Pergamon Press PLC.

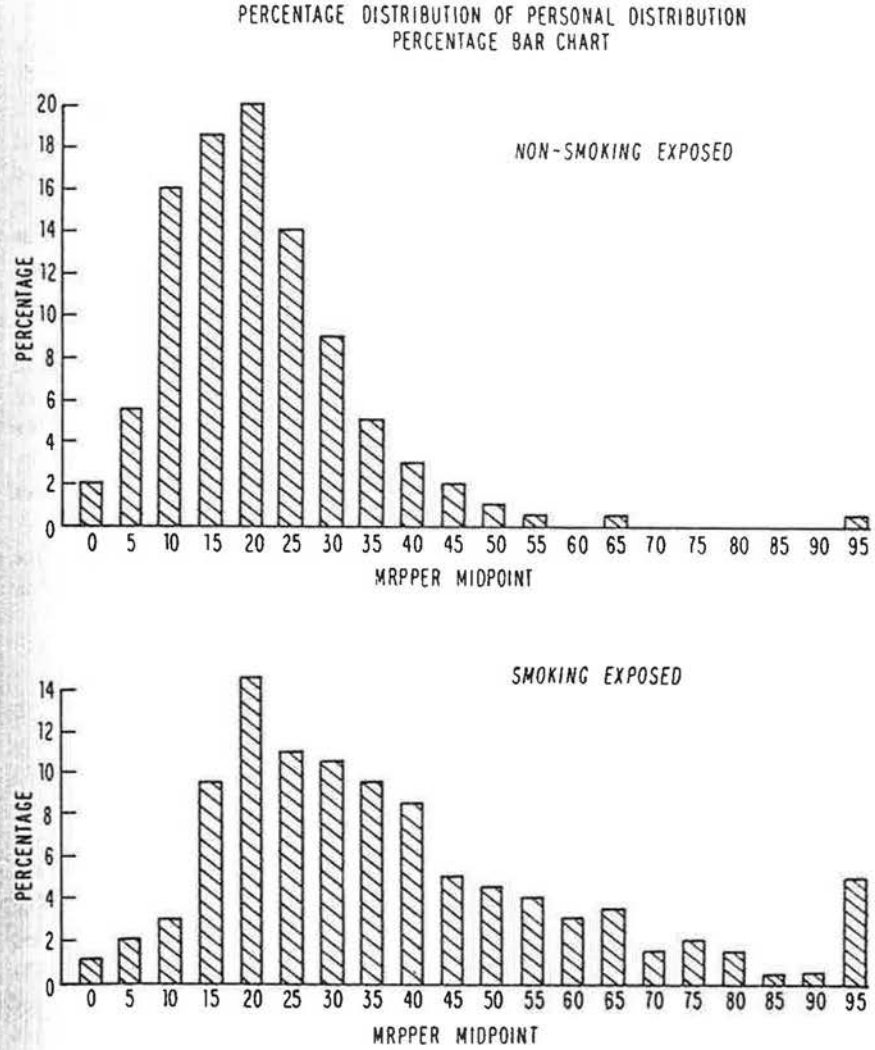

Figure 2.4. Percentage distribution of personal exposures to respirable particles with and without exposures to environmental (passive) tobacco smoke. *Source:* Spengler and Tosteson (1981), reprinted with permission.

Increases in levels of other ETS components have also been documented in association with cigarette smoking. Nitrogen dioxide is increased by a few $\mu g/m^3$ in homes with smokers. Studies on volatile organic vapors conducted in the United States and Germany indicated that benzene is 1–2 ppb higher in homes with smokers. Wallace et al. (1987) reported an average of 4 ppb benzene in homes of nonsmokers and 6 ppb in homes with smokers.

General Sources Modern furnishings, construction materials, and consumer products contaminate indoor air with numerous vapor-phase organic compounds (Table 2.3). In a recent Environmental Protection Agency study of air quality in ten public access buildings more than five hundred VOCs were identified (Sheldon et al. 1988); this study and others (Miksch, Hollowell, and Schmidt 1982; Molhave 1982; Özkaynak 1987; Wallace 1987) have described emission rates of VOCs from hundreds of materials and products, as mass of VOC per mass of material or as emission rate per unit.

Although VOC emission rates from materials and other sources may be used to predict indoor concentration, this approach is limited because emission rates may decrease as materials age. In one study, concentrations of VOCs from construction materials decreased with half-lives of two to twenty weeks after completion of the building (Sheldon et al. 1988). The concentrations of many VOCs decrease after a new building is occupied (Table 2.4), but concentrations of VOCs produced by furnishings, equipment, and occupant use can increase over time. Emission rates may also fluctuate with temperature and humidity (Girman et al. 1987).

Volatile and semivolatile organic compounds overlap in characteristics to some extent. The VOCs are compounds that exist as vapors over the normal range of air temperatures and pressures. Semivolatiles are compounds that exist as liquids or solids but also evaporate. Termiticides such as chlordane and heptachlor are injected into the ground as liquids but are effective against termites because of emanating vapors. Many semivolatile insecticides and pesticides behave in a similar fashion. Some higher molecular weight organic molecules are in the form of particulate matter in air.

Benzo[a]pyrene is one of the most widely known polycyclic aromatic hydrocarbons. These compounds, formed by the combustion of coal, oil, kerosene, gasoline, wood, and tobacco, may condense as particles. Such organic particles and some inorganic particles can absorb gases and vapors and undergo chemical transformation. For example, dioxins may be formed in the cooling combustion gases of municipal waste incinerators.

Because many organic compounds that evaporate at ambient temperatures undergo adsorption and/or chemical reactions, sampling and identification are challenging, and no single sampling method is suitable for all organic compounds (see Chapters 4 and 11).

Formaldehyde Formaldehyde (HCHO) is one of the most ubiquitous organic vapors indoors (Table 2.5). Long before the EPA's VOC total exposure assessment methodology (TEAM) studies (see Chapter 10) documented the indoor presence of

Table 2.3 Sources of Volatile Organic Compounds in Indoor Air

General Category	Examples	Important Emissions
Construction materials	Foam insulation, carpet glue, paint	Methyl chloroform, formaldehyde, styrene, xylene, tetrachloroethylene, ethylbenzene, benzene, 1,1,1-trichloroethane
Structural components	Particle board, vinyl tile, sheetrock	*n*-Decane, xylene, formaldehyde, acetone, hexanal, benzyl chloride, benzal chloride
Furnishings	Foam, textured carpet, drapery and upholstery fabric	Methyl chloroform, formaldehyde, tetrachloroethylene, *n*-undecane, benzene, 1,1,1-trichloroethane
Cleansers and solvents	Liquid detergent, chlorine bleach, scouring powder, furniture wax	*n*-Dodecane, *n*-undecane, xylene, *n*-decane, chloroform, benzene, 1,1,1-trichloroethane
Personal care products	Eyeliner pencil, deodorant, skin lotion	Methylchloroform, styrene, tetrachloroethylene, trichloroethylene, benzene, lisopene
Insecticides/pesticides	Rodenticide (solid) Insecticide (solid) Insecticide (spray)	*n*-Decane Xylene 1,1,1-Trichloroethane
Electrical equipment	Home computer, cassette tape recorder, VCRs, video cameras, videotapes	Ethyl benzene, chloroform
Combustion	Wood, kerosene	Acrolein, formaldehyde, 1-nitropyrene, 1-nitronaphthalene

Table 2.4 Volatile Organic Compounds in a New Office Building

	Concentration ($\mu g/m^3$)			
	Indoors[a]			Outdoors[b]
Chemical	July	September	December	All Trips
Aliphatics				
Decane	380	38	4	2
Undecane	170	48	13	1
Dodecane	47	19	5	0.2
Aromatics				
m + *p*-Xylene	140	19	9	2
o-Xylene	74	8	4	1
Ethylbenzene	84	6	5	1
Benzene	5	7	7	3
Sytrene	8	7	4	1
Halocarbons				
1,1,1-Trichloroethane	380	100	49	6
Tetrachloroethylene	7	2	3	1
Trichloroethylene	1	38	27	0.3
Carbon tetrachloride	1	1	1	1
Chloroform	1	2	18	6
p-Dichlorobenzene	1	1	1	ND[c]
Total of 14 organics	1,299	296	150	25.5

Source: Adopted from Sheldon et al. (1988).
[a]Mean of six 12-hour averages at five indoor locations.
[b]Mean of eighteen 12-hour averages at one outdoor location.
[c]ND, not determined.

Table 2.5 Examples of Formaldehyde (Aldehyde) Uses and Potential Indoor Sources

Source Categories	Examples
Paper products	Grocery bags, waxed paper, facial tissues, paper towels, disposable sanitary products
Stiffeners, wrinkle resistors, and water repellants	Floor coverings (rugs, linoleum, varnishes, plastics), carpet backings, adhesive binders, fire retardants, permanent-press clothes
Insulation	Urea-formaldehyde foam insulation
Combustion devices	Natural gas, kerosene, tobacco
Pressed-wood products	Plywood, particle board, decorative paneling
Other	Cosmetics, deodorants, shampoos, fabric dyes, inks, disinfectants

Source: Sterling (1985).

organic compounds, HCHO had been recognized as a pungent water soluble gas that causes irritation of the eyes and mucous membranes (NRC 1981b). It is now considered a potential cause of other symptom complaints and some diseases (see Chapter 11). Sources and concentrations of formaldehyde are briefly presented in this chapter.

Formaldehyde is a widely used chemical that can be found in hundreds of products. Formaldehyde is added as a preservative to medicines, cosmetics, toiletries, and some food containers. The largest single use of formaldehyde is in the production of urea- and phenol-formaldehyde resins, which are used to bond laminated wood products and to bind wood chips in particle board. These wood products are used as shelving, counters, bookcases, cabinets, floors, and wall covers common to many homes and offices.

Formaldehyde has been used as a carrier solvent in the dyeing of textiles and paper products and is particularly effective on synthetic fibers. For this reason, most customers visiting fabric or carpeting stores can smell formaldehyde, and contact lens wearers often report acute eye irritation.

Formaldehyde is also released during combustion. Gasoline and, more important, diesel engine emissions, react to form formaldehyde and other aldehydes. Gas stoves and cigarettes are minor indoor sources. Although the HCHO concentrations measured in the intact plume of a cigarette can be as high as 40 ppm, high enough to cause intense eye irritation, dilution of tobacco smoke by room air produces much lower levels. For example, it required intense smoking of seventy cigarettes within thirty minutes to increase the HCHO concentration in an average size room from 0.01 to 0.27 ppm (Gammage and Gupta 1984).

In the mid-1970s, urea-formaldehyde foam insulation became a popular insulation material. To make UFFI, urea-formaldehyde partially polymerized resin is mixed with an acidic hardening agent. The mixed liquid is sprayed into attic and wall cavities by injecting compressed air. Small bubbles form as the resin hardens into a foam. The foam prevents convective air movement within the cavity while the billions of air bubbles form an insulating barrier.

Formaldehyde is emitted from UFFI in an initial burst and then continuously at a lower level (Allan, Dutkiewicz, and Gilmartin 1980). The initial release is unavoidable because of a slight excess of free formaldehyde in the resin. In the past, some installers either used inappropriate formulations of UFFI or installed the UFFI at an inappropriate ambient temperature, thereby increasing the initial release. Allan and colleagues (1980) described a second contribution to the initial fast release of formaldehyde from UFFI. The UFFI reaction forms methylene and diethylene ester bridges; if these links are not formed, a methylol ending group (CH_2OH) is left. These weaker bonds can break and release formaldehyde. In homes with UFFI, the HCHO concentration decays with a half-life of about two years (Dally et al. 1981a). However, because UFFI continues to degrade slowly, HCHO will be released chronically.

In the United States, about 500,000 homes had UFFI installed between 1970 and the early 1980s. In 1982, the Consumer Product Safety Commission (CPSC) banned the sale of UFFI. Even though the U.S. Fifth Circuit Court of Appeals reversed this decision, the controversy over UFFI effectively eliminated its use. In Canada, UFFI was installed in almost 100,000 homes as part of an active program of energy conservation. Extensive formaldehyde monitoring has been conducted in homes insulated with UFFI (Table 2.6). The CPSC reports that the average

Table 2.6 Selected Examples of Observed Formaldehyde Concentrations

Sampling Site	Concentration (ppm) Range	Mean
28 Residences[a]		
UFFI	0.02–0.13	0.07
Control	0.03–0.07	
78 Structures[b]		
Apartments		0.08
UFFI and non-UFFI	0.03–0.20	0.05
Public buildings		0.04
3 Residences[c]		
UFFI	0.11–0.16	
Non-UFFI	0.06–0.08	
Energy-efficient non-UFFI	0.13–0.17	
164 Mobile homes[d]	<0.02–0.78	0.15
65 Mobile homes[e]	<0.01–3.68	0.47[f]
65 Mobile homes[g]	<0.10–0.80	0.16[f]

Source: Sterling (1985).
[a]Godish (1981).
[b]TerKonda and Liaw (1983).
[c]Gammage et al. (1983).
[d]Stock et al. (1984).
[e]Dally et al. (1981a, 1981b).
[f]Median.
[g]Hanrahan et al. (1984).

HCHO concentrations in homes insulated with UFFI were 0.12 ppm in contrast to 0.03 ppm in homes not insulated with UFFI (CPSC 1982; Gupta, Ulsamer, and Preuss, 1982).

Indoor HCHO concentrations do not reach a steady state. Not only can emission rates fluctuate with temperature and humidity, but the absorption rate can also vary. HCHO concentrations in one home in which UFFI was installed three years earlier were tracked over a year. In the winter, the HCHO concentration was about 0.05 ppm; as the outdoor temperature rose to 25°C in the summer, the concentration more than tripled (Schutte et al. 1981). Changes in ventilation (air exchange) and occupant activities also influence concentrations. Figure 2.5 shows diurnal HCHO concentrations in two Oak Ridge, Tennessee, homes (Gammage et al. 1982). Concentrations in the UFFI-containing home fluctuated from 0.1 to nearly 0.18 ppm.

Products formulated with phenol- or urea-formaldehyde resins or treated with formaldehyde to make them crease resistant or to adhere pigments can be found in almost all homes and modern office buildings. Besides the products listed in Table

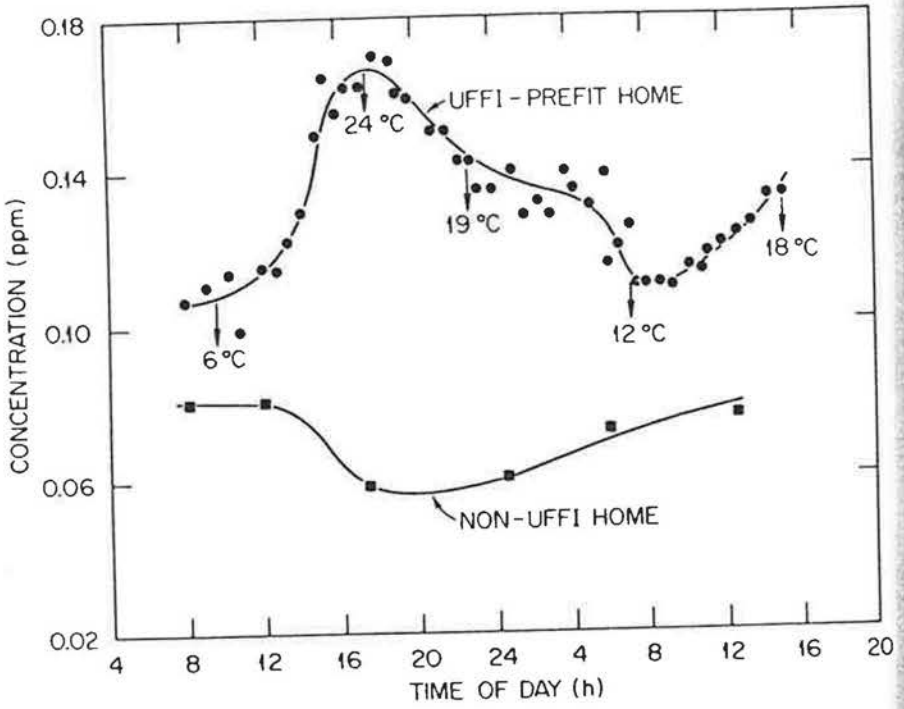

Figure 2.5. Diurnal fluctuations in formaldehyde levels (ppm) inside a three-year-old UFFI-prefit house and a ten-year-old non-UFFI house in Oak Ridge, Tennessee. *Source:* Reprinted with permission from Gammage RB and Gupta KC, Formaldehyde. In *Indoor Air Quality*, ed. Walsh PJ, Dudney CS, Copenhaver ED. Copyright 1984, CRC Press, Inc., Boca Raton, Fla.

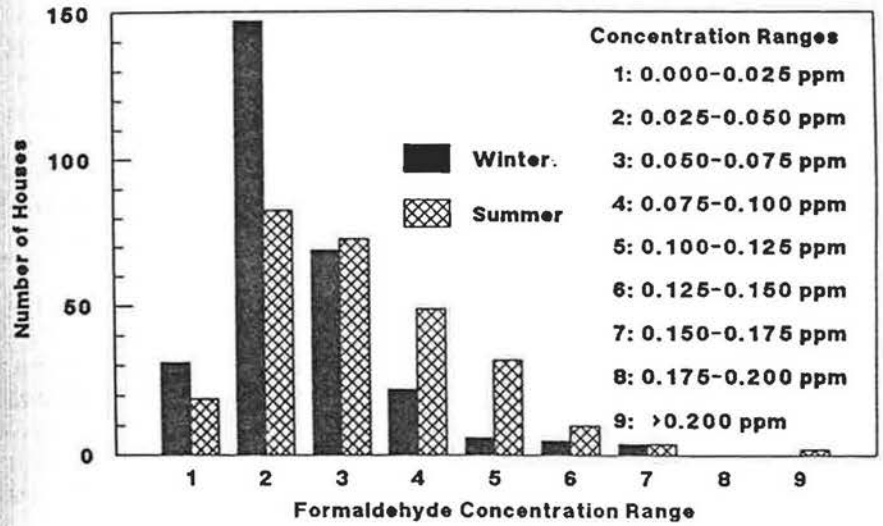

Figure 2.6. Distribution of formaldehyde levels (ppm) during winter and summer in homes in Kingston and Harriman, Tennessee. *Source:* Hawthorne et al. (1988), reprinted with permission.

2.5, formaldehyde can be in wall paper, especially the prepasted kind, floor covering, and in the binder used in fiberglass insulation. Sources in offices include partitions, pressed-board desks, wall paneling, and fiberglass-lined ventilation ducts. Even without UFFI, homes and office buildings can have HCHO concentrations of 0.2 ppm or more. Mobile homes, which have small volumes, low air exchange rates, and extensive particle board, may have concentrations of 1 ppm or greater. Hollowell et al. (1980) reported that just adding furniture to an unoccupied house increased HCHO concentrations from 0.07 to 0.19 ppm. Figure 2.6 summarizes week-long winter and summer monitoring in over three hundred homes in eastern Tennessee (Hawthorne et al. 1988), and Table 2.6 summarizes several studies of indoor HCHO concentrations.

ASBESTOS

Asbestos refers to several fibrous inorganic materials characterized by chemical formulation and crystalline structure. Chrysotile asbestos, a serpentine mineral, has been most widely used commercially. The other types of asbestos are amphibole minerals with straight fibers, in contrast to the undulating chrysotile fibers. Of the amphiboles, crocidolite and amosite have been most extensively mined, but most asbestos used in the United States has been chrysotile; both tremolite and cummington-grunerite, also amphiboles, may contaminate other minerals.

The health effects of exposure to asbestos have been amply documented by epidemiologic studies of workers exposed to high concentrations. Asbestos exposure is associated with asbestosis (fibrosis of the lung), pleural effusion and

pleural plaques (fluid in the space around the lung and scarring of the pleura, respectively), mesothelioma (cancer of the pleural sac lining the lung or of the peritoneal sac lining the abdomen), cancer of the lung, cancer of the larynx, and, possibly, cancers of the gastrointestinal tract (CPSC 1983; NRC 1984; Ontario Royal Commission 1984; U.S. EPA 1984; British Health and Safety Commission 1985; Omenn et al. 1986). The epidemiologic studies suggest greater carcinogenicity for the amphiboles, particularly in relation to mesothelioma. The confirmed occupational hazards of asbestos have raised concern that nonoccupational exposure to asbestos may also have adverse consequences.

To date, our understanding of the health risks of indoor asbestos is derived largely from risk assessment, using risk projection models based on occupationally exposed workers. To estimate risks of lung cancer and mesothelioma, these models extrapolate from relatively high workplace exposures to the much lower levels generally encountered indoors. Because the models do not incorporate a threshold exposure that must be exceeded for cancer to develop, some risk is projected for any level of exposure. Diverse models have been developed (recent reviews include Mossman et al. [1990] and Spengler et al. [1989]).

Because of its high tensile strength and thermal properties, asbestos has been used extensively in building materials since the beginning of this century (Table 2.7). The broad use categories are thermal and acoustical insulation, fire protection, and the reinforcement of building products. In addition to its use in acoustical ceiling tiles and vinyl floor tiles, asbestos has been used in paints and wall and ceiling plaster. Until banned in the late 1970s, asbestos materials were used to coat pipes, boilers, and steel structural beams. The use of asbestos in the United States has decreased since 1973, coincident with the banning of certain applications by the EPA (Figure 2.7). However, asbestos-containing materials are still present in many homes, offices, and schools.

Based on surveys, the EPA has estimated that 20 percent of the nation's buildings, about 733,000 not including schools and residential buildings with fewer than ten units, contain some asbestos materials (U.S. EPA 1988a, 1988b). Sixteen percent have thermal insulation containing asbestos. Separate surveys of the city of New York suggest that 67 percent (158,000) of the buildings contain asbestos (Price 1988, U.S. EPA 1988a, 1988b). Concern about the potential health effects of exposure has led to the removal of asbestos-containing materials from private and public buildings. However, asbestos removal is expensive, and the costs are rising. For example, the General Services Administration estimated a cost of over $50 billion to remove asbestos from federal buildings alone (U.S. EPA 1988a). Under Section 6 of the Toxic Substances Control Act, the EPA has banned manufacturing, importing, and processing of most asbestos products. This phased ban will start in 1990 and continue through 1997.

Several issues have arisen regarding the measurement and exposure of asbestos in buildings. A rapidly enlarging data base on airborne asbestos concentrations in buildings demonstrates extremely low average values under normal building use conditions. Early measurements of airborne asbestos levels made by transmission electron microscopy (U.S. EPA 1975, 1980, 1983; Nicholson et al. 1978) were obtained using the indirect filter preparation method. Measurements by indirect transmission electron microscopy are likely to overestimate the number of respir-

Table 2.7 Summary of Asbestos-Containing Building Products

Product	Dates Used	Average Percent Asbestos
Floor tile and sheet	1950–present	20
Asphaltic coatings and sealants	1900–present	10
Cement pipe and sheet	1930–present	30
Roofing felt	1910–present	15
Corrugated paper pipe wrap	1910–present	80
Sprayed insulation	1935–78	50
Troweled insulation	1935–75	70
Preformed pipe wrap	1926–75	50
Insulation board	Unknown	30
Boiler insulation	1930–78	10
Other uses	1900–present	<50

Source: Oppenheim-McMullen, J. and Turner, B. *Progressive Builder* (1986).

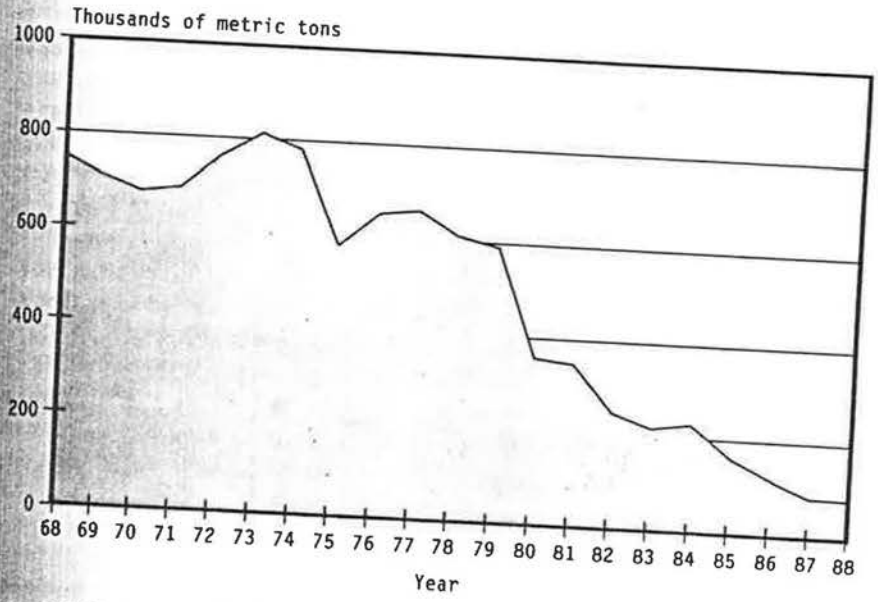

Figure 2.7. U.S. consumption of asbestos in thousands of metric tons per year from 1968 to 1988. *Source:* Bureau of Mines, and Zurer, P. *Chemical & Engineering News*, March 4, 1985, *63*(9), p. 29. Published 1985 by the American Chemical Society.

able asbestos fibers. These measurements were reported in mass units (nanograms per cubic meter of air). A concentration in mass units, however, does not predict potential health risk as directly as concentration measured in fibers per cubic centimeter of air (f/cm^3). More recent data, obtained using transmission electron microscopy and the direct filter preparation method, are reported as f/cm^3 and indicate lower average exposure levels than were implied by the earlier measurements obtained with the indirect method.

The EPA's recent report to Congress concerning asbestos in public and commercial buildings (U.S. EPA 1988a) provides a summary of direct transmission electron microscopy data for forty-one schools and ninety-four buildings other than schools. The average airborne asbestos concentration in nonschool buildings was 0.006 f/cm^3; the average for the school buildings was 0.03 f/cm^3. Since these data were compiled from different air monitoring studies with varying documentation, the averages should be interpreted as representing concentrations for all asbestiform matter visible with transmission electron microscopy (i.e., fibers both longer and shorter than 5 μm, and other asbestoslike materials that may not be considered respirable).

More fully documented data are available for the 43 nonschool buildings with asbestos-containing material studied by the EPA and for seventy-three schools about to undergo asbestos removal studied by asbestos product manufacturers (Price 1988; U.S. EPA 1988b). In the study by the EPA, the average airborne asbestos concentration, counting all fibrous particles, was 0.0007 f/cm^3. Of the 387 samples collected, including forty-eight outdoor samples and samples in six buildings with no friable asbestos-containing material, 83 percent yielded no measurable asbestos fiber counts. Restricting the asbestos count to only those fibers 5 μm in length or longer resulted in an average concentration level approximately eight times lower, 0.00008 f/cm^3. In the seventy-three school buildings, the average airborne concentration, counting all asbestos structures, was 0.01773 f/cm^3. Restricted to fibers 5 μm or longer, the average concentration was 0.00022 f/cm^3. The average of the outdoor measurements in these studies was comparable to the measured indoor level. These indoor concentrations are many orders of magnitude less than were measured in the asbestos-contaminated workplaces that have been associated with disease (e.g., >1 f/cm^3).

Occupant risk is determined by exposures to airborne fibers rather than the presence of asbestos-containing materials in the building. At present, information is lacking on the relationship between the presence of asbestos-containing material and indoor concentrations of airborne asbestos fibers. This relationship cannot be evaluated readily because of the episodic fashion in which much of the asbestos is released into indoor air.

Personal exposures to asbestos are determined by the fiber concentrations in the immediate vicinity of a person. Fixed location monitoring within a structure only provides an estimate of what exposures might be for occupants of that building. For example, the exposure in the breathing zone of a facility maintenance employee during replacement of ceiling panels could be quite different from concentrations monitored at another location. Building occupants have substantially different likelihoods of exposure depending on their activities. The low fiber counts documented in building surveys imply minimal health risks for most persons working in buildings with asbestos-containing materials. However, exposures of persons involved with building maintenance and alteration may be substantially higher than the concentrations measured during surveys.

After asbestos has been removed from a building or an area, monitoring is required before reoccupancy. This EPA requirement states that indoor fiber concentrations cannot exceed outdoor levels. However, settled fibers might be resuspended at a later point. Aggressive sampling in postabatement areas has shown fiber levels increased three- to sixfold (Karaffa et al. 1987). Burdett et al. (1988) found fiber levels increased ten to one hundred times after incomplete dry removal. Further, the comparison with ambient fiber concentrations as the criterion for reoccupancy does not address the change in exposures to occupants before and after removal. It is the net change in estimated lifetime exposures before and after asbestos removal which will determine the efficacy of the mitigation strategy.

RADON

Radon-222, a noble gas, is produced in the decay of naturally occurring uranium–238. It decays with a half-life of 3.8 days into a series of short-lived progeny: polonium–218, lead–214, bismuth–214, and polonium–214, all with half-lives less than thirty minutes (Evans 1969). Polonium–218 and polonium–214 release α-particles during decay; these α-emissions are presumed to produce lung cancer by damaging cells of the tracheobronchial epithelium. The health effects of radon are covered in Chapter 16; Chapter 4 discusses measurement techniques.

The principal source of radon in buildings is naturally occurring gas in the soil. The soil gas penetrates through sump pump wells, drains, cracks, utility access holes, and the foundation into the air in homes. The driving pressure for entry of soil gas comes from the pressure gradient established by a home across the soil; the gradient varies with atmospheric pressure, wind flow over a structure, and/or buoyancy of air within the structure. According to Nero and Nazaroff (1984), soil gas typically contains enough radon so that only 0.1 percent of air infiltrating a home would have to be drawn from the soil to produce potentially significant contamination. Generally, building materials and potable water do not contribute significantly to concentrations of radon indoors. However, potable water drawn from wells in areas in which soils and rocks are enriched in radium may be an important source of radon. Natural gas may also contribute radon if transport and storage times are brief.

Nero (1985) summarized the results of radon entry studies conducted in five countries. Figure 2.8 shows the distribution of radon entry rates; the median entry rate for U.S. single-family homes is about 0.5 pCi/liter/h (about 15–20 Bq/m^3/h). As Nero indicated, this rate is much higher than the estimated rate of emissions from concrete, only 0.07 pCi/liter/h (Ingersoll 1983).

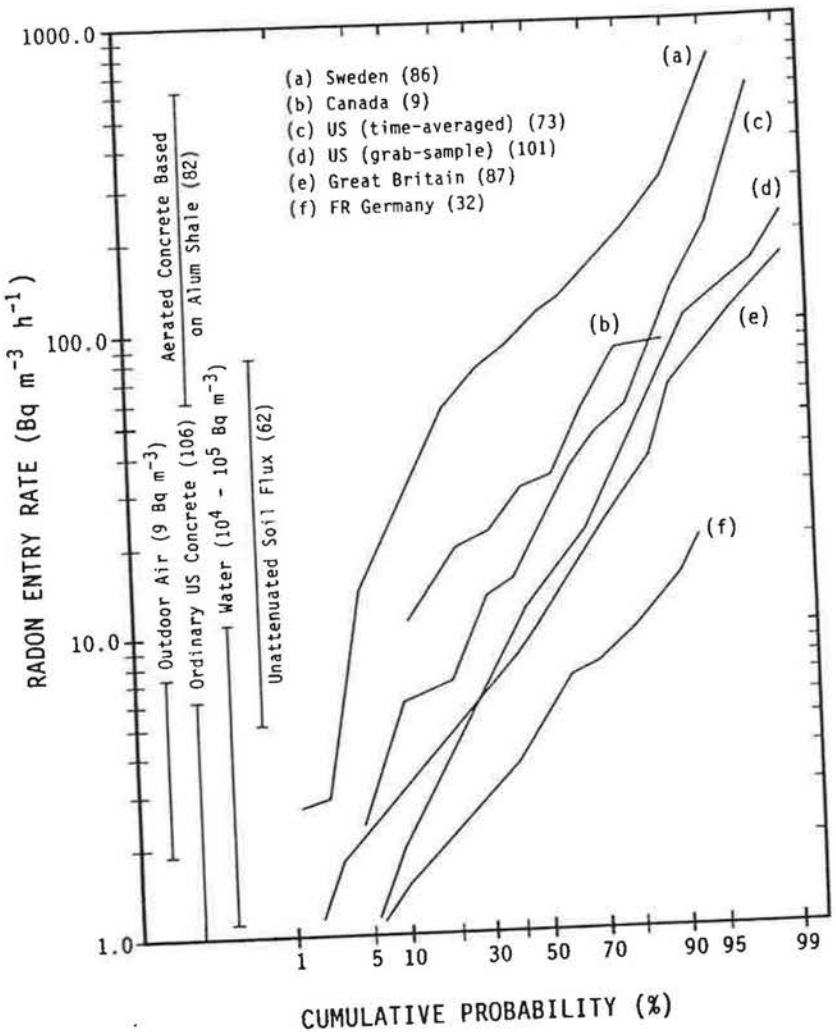

Figure 2.8. Cumulative frequency distribution of radon entry rate determined in dwellings in several countries as the product of simultaneously measured ventilation rate and radon concentration. *Source:* From *Indoor Air and Human Health*, edited by Richard B. Gammage and Stephen V. Kaye. Copyright 1985, Lewis Publishers, Inc., Chelsea, Mich. Used with permission.

On a short-term basis, radon concentrations vary with the ventilation rate of the structure. For example, Figure 2.9 shows radon measurements made in an elementary school over six days. During the week, the perimeter unit ventilators were [illegible] during the day. Radon levels quickly dropped to below 4 pCi/liter with the units operating. When the univents were turned off, levels rose to 20–30 pCi/liter. Over the weekend, without mechanical ventilation, the air exchange rate

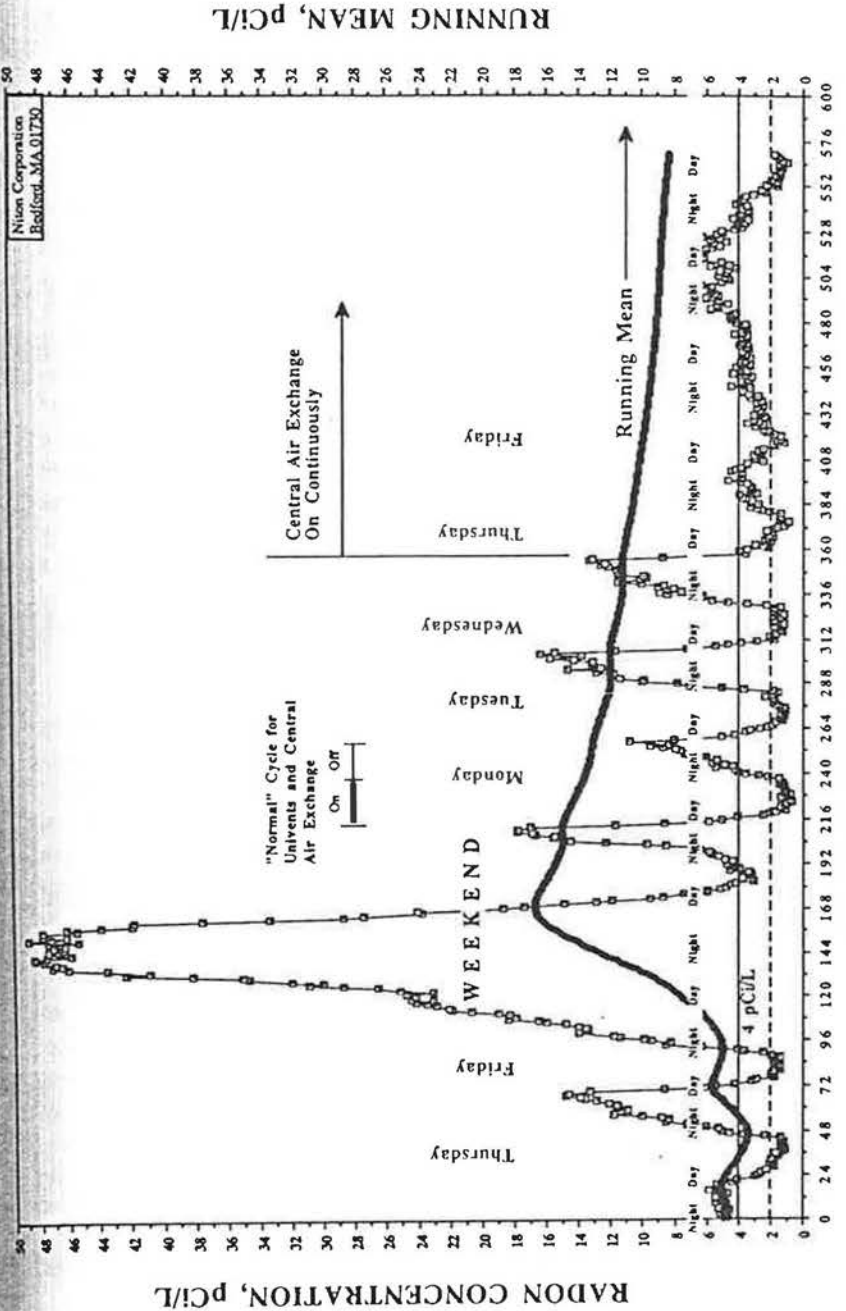

Figure 2.9. Half-hour and running mean indoor radon concentration (pCi/liter) in a school room over twelve days. Variation in concentrations over first week demonstrates the influence of ventilation. *Source:* Grodzins (1990), reprinted with permission.

must be assumed to have dropped sharply. Levels of radon increased to nearly 100 pCi/liter from Friday evening to Sunday morning.

The indoor concentration of radon at any particular time is determined in a complex fashion by the interplay of several dynamic factors including meteorology, soil characteristics, and building use and operations. Therefore, it is not unexpected that radon concentrations vary over several orders of magnitude among homes (0.05 to greater than 100 pCi/liter) and over an order of magnitude for a particular home. Because of variations in entry rates and air exchange rates, homes may have short-term fluctuations in radon concentrations as large as those documented in the public school (Figure 2.9).

Smoothing these diurnal patterns by monitoring radon with integrating samplers reveals temporal variations on the seasonal scale. Researchers at Oak Ridge National Laboratories participated in an indoor air quality study in Kingston and Harriman, Tennessee (Hawthorne et al. 1988). Radon monitoring was conducted in 248 homes over six winter months and in 287 homes during a five-month summer phase. Figure 2.10 presents the distribution of winter and summer concentrations. In this study, the downstairs radon concentrations were higher, by about a factor of two, than the upstairs concentrations. Other studies have shown a similar spatial gradient.

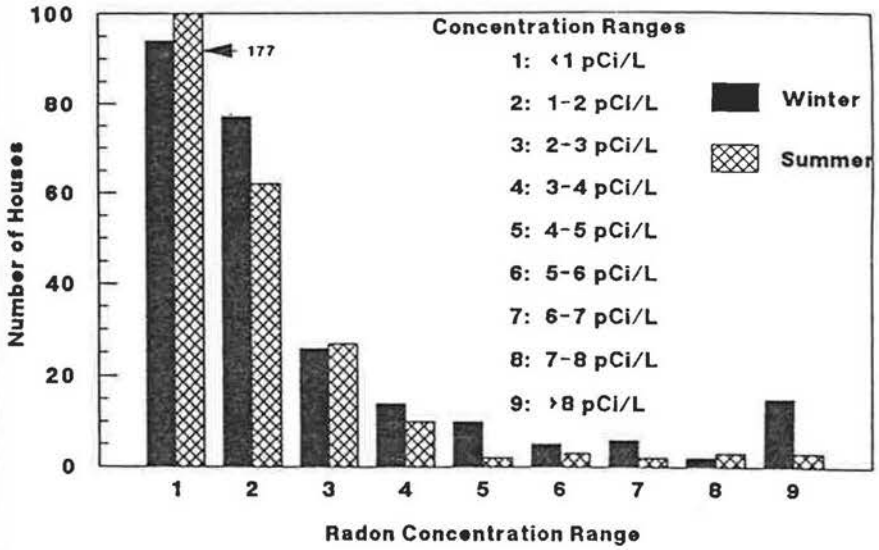

Figure 2.10. Distribution of indoor radon levels (pCi/liter) during winter and summer in homes in Kingston and Harriman, Tennessee. *Source:* Hawthorne et al. (1988), reprinted with permission.

MICROBIOLOGIC CONTAMINANTS

Microbiologic contaminants have a wide variety of forms. Pollens from trees, grasses, and other plants are familiar to most people through their associations with allergens and the clinical syndromes of allergy. Microbiologic contaminants of indoor air also include microbial cells such as viruses and bacteria in addition to fungal spores, protozoans, algae, animal dander and excreta, and insect excreta and fragments. A broad definition of microbiologic contaminants would also include the volatile metabolites of living and decaying organisms. The contaminants may be viable organisms that multiply in an infected host or they may live in dust, soil, water, oil, organic films, food, vegetative debris, or wherever the microclimate provides the correct temperature, humidity, and nutrients for growth. Bathroom walls and window casements as well as damp basements are typical locations in which water condenses. Dust can accumulate on heat exchanger coils of heating and cooling systems and refrigerators as well as in the duct lining of these mechanical devices. During the summer cooling season, moisture condenses on cooling coils and encourages the proliferation of microflora. Similarly, the drainage pans under the condensing coils in building heating, ventilating, and air-conditioning (HVAC) systems often provide favorable conditions for microbial growth. Table 2.8 summarizes the potential occurrence of microbiologic contamination at various locations in HVAC systems. Table 2.9 lists some of the aeroallergens and aeropathogens commonly found indoors.

Microbiologic contamination has also been found in a variety of other locations. For example, the use of cool mist humidifiers in the home can result in microbiologic contamination. Ultrasonic humidifiers can also disseminate bacteria if tap water is used and becomes contaminated. Humidification by steam in a central air handling system usually does not present a problem since biocides are added to most steam supplies. However, the addition of moisture to dust-contaminated ducts can lead to biologic contamination.

In general, moisture control of buildings is very important in preventing conditions that lead to fungal or bacterial growth. At relative humidities above 70 percent, fabrics, leather, and wood materials can absorb sufficient water to support fungal growth. Even when air humidities are lower, surfaces may be colder than air temperature or water damaged and hence have higher local relative humidities. Other biologic agents are less dependent on moisture conditions. Animal dander, dust mites, and insect parts can become aerosolized when shaking out bedding or vacuuming rugs. Children playing on carpets or with stuffed animals can be exposed to potentially allergenic antigens. Birds nesting near or in air intake vents can also be a source of exposure.

Chapters 13 and 14 discuss in detail the health consequences of indoor biologic contamination. Many of the airborne allergenic and infectious materials are commonly found in the outdoor and indoor environment. For a building to become contaminated, there needs to be a reservoir in which the organism can colonize and in which there are conditions favorable for its amplification. However, even these are not sufficient if a potential host does not come in close contact with the organism. Contact is facilitated by dissemination of the organism or agents through a ventilation system, air conditioner, or humidifier or by mechanical

Table 2.8 Probabilities of Occurrence of Microbial Contamination at Various Locations in HVAC Systems

HVAC Process or Location	System Time						
	Conventional Heating and Refrigeration	Central Forced Air			Local or Distributed		
		Heat Pump	Evaporative Cooler	Heat Recovery	Window/Wall Air Conditioners	Evaporative Cooler	Heat Recovery
Outdoor air intake	Moderate	Moderate	High	Moderate	Moderate	High	Moderate
Duct lining	Moderate	Moderate	High	Moderate	Moderate	High	Moderate
Air cleaners	Low to moderate	Low to moderate	Low	Low to moderate	Low to moderate	Low	Low to moderate
Humidifier	High	High					
Condensate pans/ drain liner	High	High	Very high	Moderate	High	Very high	Moderate
Return/exhaust air	Moderate to high	Moderate to high		Moderate to high	Low to moderate		Low to moderate

Source: Woods (1989).

Table 2.9 Aeroallergens and Aeropathogens Found Indoors[a]

- Bacteria
  - *Staphylococcus aureus*
  - *Streptococcus faecalis*
  - *Escherichia coli*
  - *Salmonella typhosa*
  - *Salmonella choleraesuis*
  - *Pseudomonas aeruginosa*
  - *Mycobacterium smegmatis*
  - *Mycobacterium tuberculosis*
  - *Streptococcus mutans*
  - *Klebsiella pneumoniae*
  - *Enterobacter agglomerans*
  - *Staphylococcus epidermidis*
  - *Acinetobacter calcoaceticus*
  - *Legionella*
- Yeasts
  - *Saccharomyces cerevisae*
  - *Candida albicans*
- Fungi
  - *Aspergillus niger*
  - *Aspergillus flavus*
  - *Aspergillus terreus*
  - *Chaetomium globosum*
  - *Penicillium funiculosum*
  - *Trichophyton interdigitale*
  - *Aureobasidium pullulans*
- Amoeba
- Arthropods
  - Mites
  - Cockroach
- Dander
  - Cat
  - Dog
  - Hamster

[a]This table represents only a small number of the total microorganisms that may be present in the indoor environments.

disruption. However, it is not limited to these mechanisms alone; the release of spores by some fungal organisms is regulated by biologic mechanisms.

SUMMARY

Concentrations indoors vary not only with the strength of the pollution sources, but with the volume of the polluted space, the rate of air exchange between indoor and outdoor air, and other factors that affect removal. Health risks from indoor pollution depend not only on indoor concentrations, but on patterns of human activity and source use, which, along with indoor concentrations, determine personal exposures (see Chapter 5). For some health effects, short-term patterns of source emissions may be relevant whereas long-term patterns may be more pertinent for

others. Thus, to assess and control the health effects of indoor air pollution, the contributions of indoor sources to personal exposures must be characterized. However, sources of indoor air pollution are myriad and may vary with age, race, and ethnicity of a home's occupants and with climate and geology. For example, inner city residents may have much greater exposure to cockroach antigen than persons residing in the suburbs.

REFERENCES

Akland, G. G., et al. 1985. Measuring human exposure to carbon monoxide in Washington, D.C., and Denver, Colo., during the winter of 1982–1983. *Environ. Sci. Technol.* 19:911–18.

Alfeim, I., and Ramdahl, T. 1984. Contribution of wood combustion to indoor air pollution as measured by mutagenicity in *Salmonella* and polycyclic aromatic hydrocarbon concentration. *Environ. Mutagen* 6:121–30.

Allan, G. G.; Dutkiewicz, J.; and Gilmartin, E. J. 1980. Long-term stability of urea-formaldehyde foam insulation. *Environ. Sci. Technol.* 14:1237.

Ayer, H. E., and Yeager, D. W. 1982. Irritants in cigarette smoke plumes. *Am. J. Public Health* 72:1283–85.

Baker, P. E., et al. 1987. An overview of the residential indoor air quality characterization study of nitrogen dioxide. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate*. Ed. B. Seifert et al., Vol. 1, 395–99. Berlin: Institute for Water, Soil, and Air Hygiene.

British Health and Safety Commission. 1985. Health and Safety Executive, *Asbestos: Effects on health of exposure to asbestos*, Guidance note EH35. London: H. M. Stationery Office.

Burdett, G. J., et al. 1988. Airborne asbestos fiber levels in buildings and their impact on risk management. Paper presented at the symposium on health effects of exposure to asbestos in buildings, 14–16 December, Harvard University.

Colome, S. D., et al. 1987. Analysis of factors associated with indoor residential nitrogen dioxide: Multivariate regression results. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate*. Ed. B. Seifert et al., Vol. 1, 405–7. Berlin: Institute for Water, Soil, and Air Hygiene.

Cortese, A. D. 1976. Ability of fixed monitoring stations to represent personal carbon monoxide exposure. Ph.D. diss., Harvard University School of Public Health, Boston.

Cortese, A. D., and Spengler, J. D. 1976. Ability of fixed monitoring stations to represent personal carbon monoxide exposure. *J. Air. Pollut. Control Assoc.* 26:1144–50.

Dally, K. A., et al. 1981a. A follow-up study of indoor air quality in Wisconsin homes. Paper presented at the international symposium on indoor air pollution, health and energy conservation. 13–16 October, Amherst, Mass.

Dally, K. A., et al. 1981b. Formaldehyde exposure in nonoccupational environments. *Arch. Environ. Health* 36:277–84.

Danish Building Research Institute. 1987. Report on man-made mineral fibers in the indoor air. (In Danish.)

Dockery, D. W., and Spengler, J. D. 1981. Indoor-outdoor relationships of respirable sulfates and particles. *Atmos. Environ.* 15:335–43.

Elliott, L. P., and Rowe, D. R. 1975. Air quality during public gatherings. *J. Air Pollut. Control Assoc.* 25:635–36.

Evans, R. D. 1969. Engineers' guide to the elementary behavior of radon daughters. *Health Phys.* 17:229–52.

Flachsbart, P. G., and Brown, D. E. 1985a. Merchant exposure to CO from motor vehicle exhaust at Honolulu's Ala Moana shopping center. Paper 85-85.3, presented at the seventy-eighth annual meeting of the Air Pollution Control Association, 16–25 June, Pittsburgh, Pa.

Flachsbart, P. G., and Brown, D. E. 1985b. Surveys of personal exposure to vehicle exhaust in Honolulu microenvironments. *Final report under cooperative agreement CR-808541-01-3*. Research Triangle Park, N.C.: U.S. Environmental Protection Agency.

Flachsbart, P. G., and Ott, W. R. 1986. A rapid method for surveying CO concentrations in high-rise buildings. *Environ. Int.* 12:255–64.

Gammage, R. B., and Gupta, K. C. 1984. Formaldehyde. In *Indoor air quality*. Ed. P. J. Walsh, C. S. Dudney, and E. D. Copenhaver, 109–42. Boca Raton, Fla.: CRC Press.

Gammage, R. B., et al. 1982. *Identification and measurement of air pollutants in homes*. U.S. Consumer Product Safety Commission, Oak Ridge National Laboratory, Publication no. CPSC-IAG/81/1403. Monthly Reports for September 1981 and March, May, June, July, August, and October 1982.

Gammage, R. B., et al. 1983. Temporal fluctuations of formaldehyde levels inside residences. *Proceedings of the Air Pollution Control Association specialty conference: Measurement and monitoring of non-criteria (toxic) contaminants in air*, 22–24 March, Chicago.

Girman, J. R., et al. 1982. Pollutant emission rates from indoor appliances and sidestream cigarette smoke. *Environ. Int.* 8:213–21.

Girman, J. R., et al. 1987. Bake-out of an office building. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate*. Ed. B. Seifert et al., Vol. 1, 22–26. Berlin: Institute for Water, Soil, and Air Hygiene.

Godish, T. 1981. Formaldehyde and building related illness. *J. Environ. Health* 44:116–21.

Goldstein, I. F., et al. 1987. Acute exposure to nitrogen dioxide and pulmonary function. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate*. Ed. B. Seifert et al., Vol. 1, 293–97. Berlin: Institute for Water, Soil, and Air Hygiene.

Grodzins, L. 1990. Radon in schools in Massachusetts: A report on testing and mitigation of eighty school systems. *U.S. EPA 1990 international symposium on radon and radon reduction technology*. 19–23 February, Atlanta, Ga.

Gupta, K. C.; Ulsamer, A. G.; and Preuss, P. W. 1982. Formaldehyde in indoor air: Sources and toxicity. *Environ. Int.* 8:349–58.

Hanrahan, L., et al. 1984. Formaldehyde vapor in mobile homes: A cross-sectional survey of concentrations and irritant effects. *Am. J. Public Health* 74:1026–27.

Harlos, D. P. 1988. Acute exposure to nitrogen dioxide during cooking or commuting. Ph.D. diss. Harvard School of Public Health, Boston.

Hawthorne, A. R., et al. 1988. *Indoor air quality in 300 homes in Kingston/Harriman, Tennessee*. Oak Ridge, Tenn.: Oak Ridge National Laboratory, Publication no. ORNL/6401.

Hiller, F. C., et al. 1982. Deposition of sidestream cigarette smoke in the human respiratory tract. *Am. Rev. Respir. Dis.* 125:406–8.

Hoffman, D., and Brunneman, K. D. 1983. Endogenous formation of *N*-nitrosopoline in cigarette smokers. *Cancer Res.* 43:69–83.

Hollowell, C. D., et al. 1980. *Building ventilation and indoor air quality.* LBL Report no. 10391. Berkeley, Calif.: Lawrence Berkeley Laboratory.

Ingersoll, J. G. 1983. A survey of radionuclide contents and radon emanation rates in materials used in the United States. *Health Phys.* 45:363–68.

Karaffa, M. A., et al. 1987. Preliminary evaluation of potential asbestos exposure during carpet vacuuming in buildings containing friable asbestos material. Cincinnati, Ohio: U.S. Environmental Protection Agency, unpublished draft report.

Klus, H., and Kuhn, H. 1982. Distribution of various tobacco smoke constituents in main and sidestream smoke: A review. *Bietr. Tabakforsch. Int.* 15:340–56.

Leaderer, B. P., et al. 1984. Residential exposure to NO_2, SO_2, and HCHO associated with unvented kerosene space heaters, gas appliances, and sidestream tobacco smoke. In B. Berglund, T. Lindvall, and J. Sundell, *Indoor air, Vol. 4: Chemical characterization and personal exposure,* 151–56. Stockholm: Swedish Council for Building Research, Publication no. NTIS/PB85-104214.

Lewtas, J.; Claxton, L. D.; and Mumford, J. L. 1987. Human exposure to mutagens from indoor combustion sources. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate.* Ed. B. Seifert et al., 473–77. Berlin: Institute for Water, Soil, and Air Hygiene.

Lippmann, M., and Schlesinger, R. B. 1979. *Chemical contamination in the human environment.* New York: Oxford University Press.

Marbury, M. C. 1988. Indoor residential NO_2 concentrations in Albuquerque, New Mexico. *J. Air Pollut. Control Assoc.* 38:392–98.

Miksch, R. R.; Hollowell, C. D.; and Schmidt, H. E. 1982. Trace organic chemical contaminants in office spaces. *Environ. Int.* 8:129–37.

Molhave, L. 1982. Indoor air pollution due to organic gases and vapours of solvents in building materials. *Environ. Int.* 8:117–27.

Moschandreas, D. J. 1983. Emission factors of volatile organic compounds and other air constituents from unvented gas appliances. *Proceedings of the Air Pollution Control Association specialty conference: Measurement and monitoring of noncriteria (toxic) contaminants in air,* 22–24 March, Chicago. Publication no. SP-50.

Mossman, B. T., et al. 1990. Asbestos: Scientific developments and implications for public policy. *Science* 247:294–301.

National Research Council. 1981a. *Indoor pollutants.* Washington, D.C.: National Academy Press.

National Research Council. 1981b. *Formaldehyde and other aldehydes.* Washington, D.C.: National Academy Press.

National Research Council. 1984. *Asbestiform fibers: Nonoccupational health risks.* Washington, D.C.: National Academy Press.

National Research Council. 1986. *Environmental tobacco smoke: Measuring and assessing health effects.* Washington, D.C.: National Academy Press.

Nero, A. V. 1985. Indoor concentrations of radon-222 and its daughters: Sources, range, and environmental influences. In *Indoor air and human health.* Ed. R. B. Gammage and S. V. Kaye. Chelsea, Mich.: Lewis Publishers.

Nero, A. V., and Nazaroff, W. W. 1984. Characterizing the sources of radon indoors. *Radiation Protection Dosimetry* 7:23–39.

Nicholson, W. J., et al. 1978. Control of sprayed asbestos surfaces in school buildings: A feasibility study. *Report to the National Institute of Environmental Health Sciences.*

Omenn, G. S., et al., 1986. Contribution of environmental fibers to respiratory cancer. *Environ. Health Perspect.* 70:51–56.

Ontario Royal Commission. 1984. *Report of the Royal Commission on matters of health and safety arising from the use of asbestos in Ontario,* Vol. 1–3. Toronto, Canada: Ontario Ministry of the Attorney General.

Oppenheim-McMullen, J., and Turner, B. 1986. Facing the asbestos menace. *Progressive Builder,* September:21–22.

Ott, W., and Flachsbart, P. 1982. Measurement of carbon monoxide concentrations in indoor and outdoor locations using personal exposure monitors. *Environ. Int.* 8:295–304.

Ott, W. R., and Willits, N. H. 1981. CO exposures of occupants of motor vehicles: Modeling the dynamic response of the vehicle. *SIMS technical report no. 48.* Stanford, Calif.: Stanford University.

Özkaynak, H., et al. 1987. Sources of emission rates of organic chemical vapors in homes and buildings. In *Indoor air '87: Proceedings of the fourth international conference on indoor air quality and climate.* Ed. B. Seifert et al., Vol. 1, 3–7. Berlin: Institute for Water, Soil, and Air Hygiene.

Parkhurst, W. J., et al. 1988. Influence of indoor combustion sources on indoor air quality. *Environ. Prog.* 7:257–61.

Price, B. 1988. Assessing asbestos exposure potential in buildings. Paper presented at the symposium on health effects of exposure to asbestos in buildings, 14–16 December, Harvard University.

Ryan, P. P., et al. 1988. The Boston residential NO_2 characterization study, I: Preliminary evaluation of the survey methodology. *J. Air Pollut. Control Assoc.* 38:22–27.

Sakuma, H., et al. 1983. The distribution of cigarette smoke components between mainstream and sidestream smoke: 1. Acidic components. *Beiträge Tabakforsch.* 12:63–71.

Sakuma, H., et al. 1984a. The distribution of cigarette smoke components between mainstream and sidestream smoke: 2. Bases. *Beiträge Tabakforsch.* 12:199–209.

Sakuma, H., et al. 1984b. The distribution of cigarette smoke components between mainstream and sidestream smoke: 3. Middle and higher boiling components. *Beiträge Tabakforsch.* 12:251–58.

Schmeltz, I.; Wenger, A.; Hoffman, D.; and Tso, T. C. 1979. On the fate of nicotine during pyrolysis and in a burning cigarette. *Agric. Food Chem.* 27:602–8.

Schutte, W. C., et al. 1981. Problems associated with the use of urea-formaldehyde foam for residential insulation, II. Residential studies in Colorado and Wisconsin. Oak Ridge, Tenn.: Oak Ridge National Laboratory, Publication no. ORNL-SUB/7559/3.

Sexton, K.; Spengler, J. D.; and Treitman, R. D. 1984. Effects of residential wood combustion on indoor air quality: A case study in Waterbury, Vermont. *Atmos. Environ.* 18:1371–83.

Sexton, K., et al. 1986. Characterization of particle composition, organic vapor constituents, and mutagenicity of indoor air pollution emissions. *Environ. Int.* 12:351–62.

Sheldon, L. S., et al. 1988. *Indoor air quality in public buildings, Vol. 1: Project summary.* Washington, D.C.: U.S. Environmental Protection Agency. Publication no. EPA/600/S6-88/009a.

Shikiya, D. C., et al. 1989. *Vehicle air toxins characterization study in the south coast air basin.* El Monte, Calif.: South Coast Air Quality Management District.

Spengler, J. D., and Tosteson, T. D. 1981. Personal exposures to respirable particles.

Presented at Environmetrics '81, April, Society for Industrial and Applied Mathematics, Alexandria, Va.

Spengler, J. D.; Billick, I.; and Ryan, P. B. 1984. Modeling population exposures to airborne lead. In *Indoor Air: Proceedings of the third international conference on indoor air quality and climate*. Ed. B. Berglund, T. Lindvall, and J. Sundell, Vol. 4, 87–94. Stockholm, Sweden: Swedish Council for Building Research. Available from NTIS, Springfield, Va. PB85-104214.

Spengler, J. D.; Stone, K. B.; and Lilley, F. W. 1978. High carbon monoxide levels measured in enclosed skating rinks. *J. Air Pollut. Control Assoc.* 28:776–79.

Spengler, J. D., et al., 1979. Sulfur dioxide levels and nitrogen dioxide levels inside and outside homes and the implications on health effects research. *Environ. Sci. Technol.* 13:1276–80.

Spengler, J. D., et al. 1981. Long-term measurements of respirable sulfates and particles inside and outside homes. *Atmos. Environ.* 15:23–30.

Spengler, J. D., et al., 1983. Nitrogen dioxide inside and outside 137 homes and implications for ambient air quality standards and health effects research. *Environ. Sci. Technol.* 17:164–68.

Spengler, J. D., et al., 1985. Personal exposures to respirable particulates and implications for air pollution epidemiology. *Environ. Sci. Technol.* 19:700–707.

Spengler, J. D., et al., eds. 1989. *Proceedings of the symposium on health aspects of exposure to asbestos in buildings*. Cambridge, Mass.: Harvard University Energy and Environmental Policy Center.

Sterling, D. A. 1985. Volatile organic compounds in indoor air: An overview of sources, concentrations, and health effects. In *Indoor air and human health*. Ed. R. B. Gammage and S. V. Kaye, 387–402. Chelsea, Mich.: Lewis Publishers.

Sterling, T. D., and Kobayashi, D. 1981. Use of the gas ranges for cooking and heating in urban dwellings. *J. Air Pollut. Control Assoc.* 29:238–41.

Stern, A. C., et al. 1984. *Fundamentals of air pollution*. 2d ed. Orlando, Fla.: Academic Press.

Stock, T. H.; Sterling, D. A.; and Monsen, R. M. 1984. A survey of indoor air quality in Texas mobile homes. In *Indoor air: Proceedings of the third international conference on indoor air quality and climate*. Ed. B. Berglund, T. Lindvall, and J. Sundell, Vol. 4, 331–34. Stockholm, Sweden: Swedish Council for Building Research.

Tennessee Valley Authority. 1985. *Indoor air quality study, phase II*. Chattanooga, Tenn.: Tennessee Valley Authority, Division of Conservation and Energy Management.

TerKonda, P., and Liaw, S. 1983. Monitoring of indoor aldehydes. *Proceedings of the Air Pollution Control Association specialty conference: Measurement and monitoring of non-criteria (toxic) contaminants in air*, 22–24 March, Chicago.

Traynor, G. W., et al. 1983a. Pollutant emissions from portable kerosene-fired space heaters. *Environ. Sci. Technol.* 17:369–71.

Traynor, G. W., et al. 1983b. Indoor air pollution from portable kerosene-fired space heaters. Washington, D.C.: U.S. Department of Energy, Office of Energy Research, Office of Health and Environmental Research. Publication no. 20585NTIS DE83-009140.

Traynor, G. W., et al. 1986. Selected organic pollutant emissions from unvented kerosene heaters. Paper 86-52.5, presented at the seventy-ninth annual meeting of the Air Pollution Control Association, 22–27 June, Minneapolis, Minn. Pittsburgh, Pa.: Air Pollution Control Association.

U.S. Consumer Product Safety Commission. 1982. Part IV: Ban of urea-formaldehyde foam insulation, withdrawal of proposed information labeling rule, and denial of petition to issue a standard. *Fed. Reg.* 47:14366.

U.S. Consumer Product Safety Commission. 1983. *Report of the chronic hazard advisory panel on asbestos*. Washington, D.C.: U.S. Consumer Product Safety Commission Directorate for Health Sciences.

U.S. Department of Energy. 1987. *Indoor air quality environmental information handbook: Combustion sources*. Washington, D.C.: U.S. Department of Energy. Publication no. DOE-EV/10450-821.

U.S. Department of Health and Human Services. 1986. *The health consequences of involuntary smoking: A report of the surgeon general*. Washington, D.C.: U.S. Government Printing Office. DHHS Publication no. CDC/87/8398.

U.S. Environmental Protection Agency. 1975. *Asbestos contamination of the air in public buildings*. Research Triangle Park, N.C.: U.S. Environmental Protection Agency. Publication no. EPA/450/3-76/004.

U.S. Environmental Protection Agency. 1980. *Measurement of asbestos air pollution inside buildings sprayed with asbesetos*. Washington, D.C.: U.S. Environmental Protection Agency. Publication no. EPA/560/13-80/026.

U.S. Environmental Protection Agency. 1983. *Airborne asbestos levels in schools*. Washington, D.C.: U.S. Environmental Protection Agency. Publication no. EPA/560/5-81-006.

U.S. Environmental Protection Agency. 1984. *Asbestos in buildings: A national survey of asbestos-containing friable materials*. Washington, D.C.: U.S. Environmental Protection Agency, Office of Toxic Substances. Publication no. EPA/560/5-84/006.

U.S. Environmental Protection Agency. 1987. *Preliminary indoor air pollution information assessment appendix*. Washington, D.C.: U.S. Environmental Protection Agency. Publication no. EPA/600/8-87/014.

U.S. Environmental Protection Agency. 1988a. *Study of asbestos-containing materials in public buildings: A report to Congress*. Washington, D.C.: U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency. 1988b. *Assessing asbestos exposure in public buildings*. Washington, D.C.: U.S. Environmental Protection Agency. Publication no. EPA/560/5-88/002.

Vainio, H.; Hemminki, K.; and Wilbourn, J. 1985. Data on the carcinogenicity of chemicals in the IARC Monographs Programme. *Carcinogenesis* 6:1653–65.

Wade, W. A., III; Cote, W. A.; and Yocum, J. E. 1975. A study of indoor air quality. *J. Air Pollut. Control Assoc.* 25:933–39.

Wallace, L. A., et al. 1987. Emissions of volatile organic compounds from building materials and consumer products. *Atmos. Environ.* 21:385–93.

Woods, J. 1989. *HVAC systems as sources or vectors of microbiological contaminants*. COSC/ALA Workshop on Biological Pollutants in the Home, 10–11 July, Alexandria, Va.

Yocom, J. E. 1982. Indoor-outdoor air quality relationships: A critical review. *J. Air Pollut. Control Assoc.* 32:904–20.

Ziskind, R. A., et al. 1981. Carbon monoxide intrusion into sustained-use vehicles. *Environ. Int.* 5:109–23.

Zurer, P. 1985. Asbestos: The fiber that is panicking America. *Chem. Eng. News* 63:28–41.