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Indoor Air Pollution: A Public Health Perspective

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Indoor air quality in nonoccupational settings has received relatively little attention from scientists, engineers, regulatory officials, and environmental groups. However, the available information suggests that elevated concentrations of some airborne contaminants are routinely encountered in indoor environments. Time-budget surveys have shown that most of us are at home more than 16 hours per day, with other indoor time divided between the workplace, commercial and public buildings, and transportation microenvironments (for instance, automobiles and subways) (*1, 2*). Even if indoor air pollutant concentrations are low, they may make a substantial contribution to time-weighted exposures. The degree to which indoor air pollution represents a public health hazard has not been established. Nevertheless, concerns about health effects have prompted intervention at local, state, and federal levels of government to limit indoor exposures to sidestream tobacco smoke, asbestos, formaldehyde, and radon. In this article we discuss the scientific basis for recognizing indoor air pollution as a national health concern and the need for a coordinated policy to safeguard indoor air quality.

Summary. Although official efforts to control air pollution have traditionally focused on outdoor air, it is now apparent that elevated contaminant concentrations are common inside some private and public buildings. Concerns about potential public health problems due to indoor air pollution are based on evidence that urban residents typically spend more than 90 percent of their time indoors, concentrations of some contaminants are higher indoors than outdoors, and for some pollutants personal exposures are not characterized adequately by outdoor measurements. Among the more important indoor contaminants associated with health or irritation effects are passive tobacco smoke, radon decay products, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, microorganisms, and aeroallergens. Efforts to assess health risks associated with indoor air pollution are limited by insufficient information about the number of people exposed, the pattern and severity of exposures, and the health consequences of exposures. An overall strategy should be developed to investigate indoor exposures, health effects, control options, and public policy alternatives.

Contaminated indoor air is not new. Soot found on ceilings of prehistoric caves provides evidence of the high levels of pollution associated with inadequate ventilation of open fires. Elevated indoor pollutant concentrations continue to be a fact of life for people who live in impoverished areas and cook over open fires fueled by charcoal, wood, dung, kerosene, or oil. Exposures to gaseous and particulate pollutants for these people greatly exceed measured pollutant concentrations in urban environments (*3*).

In industrialized countries, the more obvious indoor pollution problems have been known for decades. Minimum ventilation requirements, for instance, are a standard component of building codes to control odors and combustion by-products and reduce transmission of respirable diseases. However, the complex nature of indoor nonoccupational environments in technologically advanced nations (including synthetic building materials, energy-efficient buildings, unvented heating and cooking appliances, and cleaning and personal care products) makes possible widespread indoor exposures to a broad spectrum of airborne chemicals. Although many indoor pollutants (such as radon decay products and microorganisms) are below perception thresholds, long-term exposures may cause increased rates of morbidity and mortality.

In the 10 years since Benson *et al.* (*4*) reviewed the subject, a number of reports, symposia, and articles (*5–12*) have been published which contain information on indoor contaminants with potentially negative health effects and on important indoor emission sources (see Table 1). Nevertheless, development of public policy to safeguard indoor air quality is hindered by a paucity of data on distributions of sources, building characteristics, daily activity patterns, indoor concentrations, exposure patterns, and health risks.

Basis for Concern

Increases in energy prices and in costs of new electricity-generating stations have encouraged individuals and institutions to seek alternative fuels and to reduce energy consumption. Because more than one-third of U.S. energy is consumed in buildings (*13*), efforts have been made to reduce energy use in the residential and commercial sectors (*14–16*). Common approaches include: adding insulation, reducing air-exchange rates, and fuel switching.

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Increased sales of wood- and coal-burning stoves and kerosene heaters suggest a national trend away from clean space-heating fuels such as electricity and gas. Sales of wood-burning stoves in the United States increased from fewer than 200,000 in 1972 to approximately 1.5 million in 1981 (*17*). An estimated 3 million kerosene heating units were in use during the 1981–1982 winter, with projections of 8 million to 10 million by 1985 (*18*). Emissions from these heating appliances contain toxic and carcinogenic particles and gases (*19–23*), yet indoor exposures and possible health consequences have not been evaluated adequately.

Because hospitals consume approximately 15 percent of all energy used in the commercial building sector, they have been a target for energy savings (*24, 25*). However, a panel of experts who explored the opportunities and constraints associated with relaxation of Department of Health, Education, and Welfare standards concluded that "the probable limiting constraint on ventilation is control of chemical contaminants. No information exists to adequately characterize the airborne chemical load in the hospital setting at the present time" (*26*).

Reduced air-exchange rates in the presence of emissions from building materials and consumer products may adversely affect human health, welfare, and comfort. Urea formaldehyde foam insulation (UFFI), for example, has been shown to be a significant source of formaldehyde in some instances (*27–29*). Complaints, symptoms, and illnesses have been reported by occupants of buildings with UFFI, and its use has been abandoned in Canada (*27*). The Consumer Products Safety Commission recently proposed a ban on UFFI in the United States (*28*); however, this action is still subject to legislative and judicial review.

Mobile homes and prefabricated housing units are especially prone to indoor air pollution problems. They have lower mean air-exchange rates than conventional homes, are of smaller volume, use proportionally more materials containing volatile organic resins, and are more likely to use propane for cooking fuel. There were 3,722,000 occupied mobile homes in the United States during 1977 (*10*) and about 250,000 new units will be shipped this year, an increase of 15 percent over the past 2 years (*30*). Mobile homes account for about 20 percent of the new housing market, and the number of mobile home occupants is expected to increase as restrictive zoning laws are phased out by many localities and states.

Another reason for concern is the growing number of building-related illnesses that have come to the attention of public health officials. The term "sick building syndrome" refers to health symptoms (for instance, irritation of mucous membranes, headaches, dizziness, nausea, diarrhea, rashes, and abdominal and chest pain) which affect occupants of a building. The Consumer Product Safety Commission has received more than 3000 complaints to date about exposures to contaminant releases from building materials (*29*). The National Institute for Occupational Safety and Health (NIOSH) conducted 115 investigations between mid-1978 and 1980 based on complaints from workers in nonindustrial settings who believed they were victims of building-related pollution (*31*). The New York City Department of Environmental Protection has received many complaints about indoor air pollution, despite the fact there is no established mechanism for receiving and dealing with consumer concerns about indoor air quality (*32*). Systematic investigations of building-associated illness are beginning (*31, 33*), but local, state, and federal agencies often lack explicit authority, funds, and expertise to deal with this issue.

In summary, there is growing evidence that evaluation of indoor as well as outdoor exposures to air pollution is essential for realistic health effects assessment. If indoor exposures are not taken into account in epidemiologic investigations of air pollution, systematic and random biases may give rise to spurious conclusions (*10, 34*). Total personal exposures are often better correlated with indoor than with outdoor concentrations (*35–38*). These findings, along with concerns about reduced air-exchange rates and new indoor pollution sources, challenge the premise that costly controls on sources of ambient pollution are improving public health through reduction of human exposures.

Indoor Pollutants and Sources

Several pollutants from indoor sources affect human health. Sidestream tobacco smoke, radon and radon decay products, asbestos fibers, fiber glass, formaldehyde, combustion by-products (such as polycyclic aromatic hydrocarbons, nitrogen dioxide, carbon monoxide, hydrogen cyanide, and sulfur dioxide), aeropathogens, and allergens are associated with a range of problems from mild irritation of nasal and mucous membranes to irreversible toxic and carcinogenic effects. The available evidence of toxicity, indoor concentrations, and number of people exposed suggests that some indoor air pollutants may constitute significant public health problems. The more important pollutants identified to date are briefly reviewed below.

Combustion by-products. Indoor combustion of fuels can be a source of CO, CO_2, SO_2, formaldehyde, hydrocarbons, nitrogen oxides, and a variety of particles. Numerous studies have reported elevated indoor levels of NO_2, NO, CO, and CO_2 in homes with unvented appliances (*10*). Depending on source use and air-exchange rates, long-term indoor NO_2 averages can exceed the National Ambient Air Quality Standard (NAAQS) of 100 $\mu g/m^3$, even in areas where ambient outdoor values are 15 $\mu g/m^3$ (*34*). Peak hourly NO_2 concentrations between 200 and 700 $\mu g/m^3$ have been measured routinely in kitchens and other rooms of homes during conventional gas cooking. Elevated concentrations of NO_2 or CO have been reported for homes and schools where kerosene heaters and unvented gas heaters are used and in skating arenas with gasoline-powered ice-cleaning equipment (*28, 39*).

Exposures to NO_2 have been associated with toxicological effects including pulmonary edema, bronchoconstriction, and increased infection rates. Some epidemiologic evidence indicates that increased respiratory infection in young children and adult males and lower pulmonary function performance are associated with a history of exposure to gas stove emissions. Other studies failed to show significant effects associated with gas cooking (*40*).

Carbon monoxide from faulty furnaces and attached garages is responsible for several fatal accidents each year. Under typical use conditions, emissions from cooking appliances may raise short-term indoor CO concentrations 5 to 10 ppm. When a gas stove is used for heating, a common practice among urban poor in northern climates, concentrations of 25 to 50 ppm have been measured (*41*). Concentrations ranging from 25 to more than 100 ppm have been measured in ice-skating rinks, apartments, and offices with attached or underground garages.

Carbon monoxide forms carboxyhemoglobin (COHb) in the blood and inhibits its oxygen uptake. It is not known at present whether there is a threshold for adverse effects from oxygen deprivation due to COHb. Recent work indicates that exercising adults with angina pectoris are sensitive to COHb concentrations as low as 1 percent (*42*). Community air

pollution and indoor exposures to combustion by-products or sidestream cigarette smoke can raise COHb in nonsmokers to 2 to 3 percent (*43*).

National Ambient Air Quality Standards have been promulgated for CO and NO_2, and emission controls for mobile and stationary sources are required by legislative mandate. A limited number of personal exposure studies showed significant correlations between exposures and indoor concentrations. The evidence suggests that many, if not most, exposures to NO_2 and CO occur indoors and are therefore not represented accurately by outdoor monitors (*10, 34, 44*).

Tobacco smoke. Tobacco smoke is discussed separately for several reasons. First, nearly everyone is exposed at one time or another to tobacco smoke. Although 33 percent of the adult population regularly smoke cigarettes, this figure does not necessarily characterize the population of exposed children. For example, epidemiologic studies in the United States have shown that the percentage of children living in homes with one or more smokers ranged from 54 in Tucson to 76 for a middle-income community in St. Louis (*10*). Second, more than 2000 compounds have been identified in cigarette smoke, many of which are established carcinogens, irritants, and asphyxiants. And third, there is increasing evidence that passive exposures to tobacco smoke may affect respiratory health.

Tobacco combustion indoors contributes to concentrations of respirable particles, nicotine, polycyclic aromatic hydrocarbons, CO, acrolein, NO_2, and many other substances. The resulting concentrations vary widely, depending on the frequency and amount of smoking, air-infiltration rates, air-cleaning devices, and air-distribution systems. Measurements in bars, restaurants, airplanes (smoking section), buses, conference rooms, and offices indicate CO and particulate matter concentrations of 2 to 35 ppm and 10 to 1000 $\mu g/m^3$, respectively (*45*). Analysis of respirable particle samples collected in 80 homes over several years indicates that a "pack-a-day" smoker will contribute approximately 20 $\mu g/m^3$ to 24-hour indoor particle concentrations (*46*). Where two or more heavy smokers resided in a house with low air-exchange rates, the 24-hour NAAQS of 260 $\mu g/m^3$ was also violated.

While the health effects of smoking on smokers have been studied extensively (*47*), the health effects on nonsmokers have received far less emphasis. Many substances in cigarette smoke are irritants, and conjunctival irritation, nasal discomfort, cough, sore throat, and sneezing have been noted in nonsmokers exposed to cigarette smoke (*48*). Measured changes in heart rate, systolic blood pressure, COHb, psychomotor functions, and small airway dysfunction have also been reported in nonsmokers who are exposed to smoke (*43, 49–51*).

Most studies of the effect of parental smoking on respiratory health in children show an association with reported respiratory morbidity in children, especially

Table 1. Summary of indoor pollutants, emission sources, and concentrations [adapted from (*10*)]. Column 3 shows typical ranges of indoor concentrations in the presence of indoor emission sources.

Pollutant	Major emission sources	Typical indoor concentrations	Indoor/outdoor concentration ratio
	Origin: predominantly outdoors		
Sulfur oxides (gases, particles)	Fuel combustion, smelters	0–15 $\mu g/m^3$	< 1
Ozone	Photochemical reactions	0–10 ppb	$\ll 1$
Pollens	Trees, grass, weeds, plants	L.V.*	< 1
Lead, manganese	Automobiles	L.V.	< 1
Calcium, chlorine, silicon, cadmium	Suspension of soils, industrial emissions	N.A.†	< 1
Organic substances	Petrochemical solvents, natural sources, vaporization of unburned fuels	N.A.	< 1
	Origin: indoors or outdoors		
Nitric oxide, nitrogen dioxide	Fuel burning	10–120 $\mu g/m^3$‡ 200–700 $\mu g/m^3$§	$\gg 1$
Carbon monoxide	Fuel burning	5–50 ppm	$\gg 1$
Carbon dioxide	Metabolic activity, combustion	2000–3000 ppm	$\gg 1$
Particles	Resuspension, condensation of vapors, combustion products	10–1000 $\mu g/m^3$	1
Water vapor	Biological activity, combustion evaporation	N.A.	> 1
Organic substances	Volatilization, combustion, paint, metabolic action, pesticides	N.A.	$\gg 1$
Spores	Fungi, molds	N.A.	> 1
	Origin: predominantly indoors		
Radon	Building construction materials (concrete, stone), water	0.01–4 pCi/liter	$\gg 1$
Formaldehyde	Particleboard, insulation, furnishings, tobacco smoke	0.01–0.5 ppm	> 1
Asbestos, mineral, and synthetic fibers	Fire retardant materials, insulation	0–1 fiber/ml	1
Organic substances	Adhesives, solvents, cooking, cosmetics	L.V.	> 1
Ammonia	Metabolic activity, cleaning products	N.A.	> 1
Polycyclic hydrocarbons, arsenic, nicotine, acrolein, and so forth	Tobacco smoke	L.V.	$\gg 1$
Mercury	Fungicides, paints, spills in dental-care facilities or labs, thermometer breakage	L.V.	> 1
Aerosols	Consumer products	N.A.	$\gg 1$
Microorganisms	People, animals, plants	L.V.	> 1
Allergens	House dust, animal dander, insect parts	L.V.	$\gg 1$

*L.V., limited and variable (limited measurements, high variation). †N.A., not applicable. ‡Annual average. §One-hour average in homes with gas stoves, during cooking.

during the first 2 years of life (*10, 47, 52*). An association between the number of cigarettes smoked per day by the parents and respiratory symptoms in children has been a consistent finding. However, these relations do not always appear independent of parental symptoms, socioeconomic class, or smoking habits of the children. Some investigators found an association between tobacco smoke exposure of nonsmoking wives whose husbands smoke and increased lung cancer rates (*53–55*).

The National Academy of Sciences stated in its report on indoor pollutants (*10*): "The constituents of tobacco smoke are well-documented as hazardous, the prevalence of population exposure is very high, and there is an increased incidence of respiratory tract symptoms and functional decrements (decreases) in children residing in homes with smokers, compared with those homes without smokers. These considerations and recent evidence of increased lung cancer rates among non-smoking women living with smoking husbands have led us to conclude that indoor exposure to tobacco smoke has adverse effects. Public policy should clearly articulate that involuntary exposure to tobacco smoke ought to be minimized or avoided where possible."

Radon and radon decay products. Radon is a radioactive decay product of radium-226. Radium, a natural trace constituent of rock and soil, is found in building materials made from earth crustal components. Radon-222 is a gas with a half-life of 3.8 days. It decays through polonium-218, lead-214, bismuth-214, and polonium-214 before reaching lead-206, a stable isotope. These decay products are solids and can attach to aerosols, which may become embedded in the lungs and irradiate surrounding tissue. It is usually radon gas which diffuses into indoor air from the ground, building materials, or well water. Higher concentrations are typically measured in basements, crawl spaces, and homes with low air-exchange rates.

Radon and radon decay product concentrations have been measured in conventional and experimental homes in several states (*10, 56*). Typical radon concentrations range from 0.01 to 4 pCi/liter. In "energy-efficient" houses, levels exceeding 20 pCi/liter have been reported. Concentrations ranging from 0.1 to 27 pCi/liter were measured in 52 conventional houses in Maryland (*57*). Integrated samples during the winter in 4000 Swedish homes averaged about three times the concentrations reported for Maine homes and 20 to 30 times the concentrations reported for homes in California and Texas (*58*).

Concerns about adverse health effects of indoor exposure to radon decay products are based on the higher incidence of lung cancer observed in uranium miners. Although indoor home exposures are thought to be considerably lower, risk is proportional to exposure and the number of people exposed is large. Recent reviews of epidemiologic studies of radon and lung cancer in miners estimate that lifetime risks range from 21 to 54 (*59*) to 1000 (*59, 60*) deaths per 10^6 working level months (WLM) (*61*). For the range of in-home radon exposures typically recorded, 0.04 to 0.8 WLM, excess lung cancers in the United States have been estimated to be as high as 10,000 (*56*).

Microorganisms and allergens. A large variety of biological material is present in indoor environments. Inhalation of biological aerosols discharged by people and animals is a primary mechanism of contagion for most acute respiratory infections (*62*). Tuberculosis, measles, smallpox, and staphylococci are known to be transmitted by air ventilation systems in schools and hospitals. Air-cooling equipment, cool-mist vaporizers, humidifiers, nebulizers, flush toilets, ice machines, and carpeting can incubate and distribute bacterial aerosols indoors. Legionnaire's disease (*Legionella pneumophila*) and humidifier fever are well-known examples of air-conditioning–related bacteria (*63, 64*).

According to the National Health Survey (*65*), respiratory ailments (predominantly upper respiratory disease and influenza) account for more than half of all acute conditions, including illnesses and injuries. The incidence of respiratory conditions is just under one per person per year, and they typically restrict activity for 4.5 days. Higher incidences are observed among younger children and the elderly. Considering the loss of time from work and school, as well as medical costs, the impact of indoor contagion is probably enormous (*66*). There is a lack of data with which to evaluate the relation between infection and ventilation rates. If the primary mechanism is by contact rather than inhalation, the prevalence of respiratory infection may be unaffected by changes in ventilation.

Pollen, molds, mites, chemical additives, animal dander, fungi, algae, and insect parts are known indoor allergens. Sources of indoor allergenic materials include pets, detergents, humidifier and air-cooling fluids, growth of molds and fungi on surfaces, and insects that live in dust and vents. Temperature and humidity conditions are important for many indoor aeroallergens. For example, house mites flourish at temperatures around 25°C and relative humidities above 45 percent. Because high humidity favors the growth of molds and fungi, tightly sealed buildings in humid climates are more prone to allergenic problems.

Reduced ventilation and increased use of untreated recirculated air may increase concentrations of microorganisms. Prolonged exposure to some chemicals and antigens can cause sensitization. Therefore, reduced fresh air in buildings might lead to increased rates of infection and allergy. However, little is known about sources, concentrations, and survival rates of many aeropathogens indoors.

Formaldehyde and other organic compounds. Building materials (plywood, particleboard, and so on), furnishings (carpets, draperies), and some types of foam insulation contain formaldehyde resins, the most common of which is urea formaldehyde. Excess formaldehyde in these products can be released over a considerable period. Outgassing rates are higher for new materials and are directly influenced by humidity and temperature. Although few longitudinal studies have been done, the half-life for formaldehyde emissions is approximately 4.4 years. Unvented gas combustion and tobacco smoking are other sources of indoor formaldehyde.

Indoor formaldehyde sampling has been conducted principally in locations where higher concentrations were suspected. Measurements in Denmark, the Netherlands, the Federal Republic of Germany, Sweden, and the United States have shown that formaldehyde concentrations often exceed 0.1 ppm. In 23 Danish homes the average formaldehyde concentration was 0.5 ppm and the range was 0.07 to 1.9 ppm (*67*). In response to occupant complaints, formaldehyde concentrations in more than 200 mobile homes in Washington State were measured and were found to range from 0.03 to 2.4 ppm (*68*). Similar findings were reported for mobile homes in Minnesota and Wisconsin (*69*). Formaldehyde concentrations of 0.1 to 0.5 ppm have been measured in conventional homes and schools without obvious sources. Concentrations in excess of 0.1 ppm appear to be common in homes insulated with UFFI.

Adverse effects from formaldehyde may result from inhalation, ingestion, or contact. The compound showed mutagenic activity in a variety of microorganisms and produced nasopharyngeal carcinoma in laboratory rats and mice (*68*). Chamber studies with humans showed

Table 2. Control measures for indoor air pollutants.

Control measure description	Pollutant	Example
Ventilation: Dilution of indoor air with fresh outdoor air or recirculated filtered air, using mechanical or natural methods to promote localized, zonal, or general ventilation	Radon and radon progeny; combustion by-products; tobacco smoke; biological agents (particles)	Local exhaust of gas stove emissions; air-to-air heat exchangers; building ventilation codes
Source removal or substitution: Removal of indoor emission sources or substitution of less hazardous materials or products	Organic substances; asbestiform minerals; tobacco smoke	Restrictions on smoking in public places; removal of asbestos
Some modification: Reduction of emission rates through changes in design or processes; containment of emissions by barriers or sealants	Radon and radon progeny; organic substances; asbestiform minerals; combustion by-products	Plastic barriers to reduce radon levels; containment of asbestos; design of buildings without basements to avoid radon; catalytic oxidation of CO to CO_2 in kerosene burners
Air cleaning: Purification of indoor air by gas adsorbers, air filters, and electrostatic precipitators	Particulate matter; combustion by-products; biological agents (particles)	Residential air cleaners to control tobacco smoke or wood smoke; ultraviolet irradiation to decontaminate ventilation air; formaldehyde sorbant filters
Behavioral adjustment: Reduction in human exposure through modification of behavior patterns; facilitated by consumer education, product labeling, building design, warning devices, and legal liability	Organic substances; combustion by-products; tobacco smoke	Smoke-free zones; architectural design of interior space; certification of formaldehyde concentrations for home purchases

eye discomfort in the concentration range 0.1 to 0.4 ppm (*68*), while residential exposures as low as 0.02 ppm were associated with tearing and eye irritation. The fact that in-home responses occur at lower exposures may reflect a broader spectrum of sensitivity in the population at large, increased sensitivity due to prolonged low-level exposures, adaptation of volunteers in the chamber studies to elevated formaldehyde concentrations, or effects of other irritants present in indoor air. At concentrations of 10 to 100 ppm, formaldehyde exposures can cause lower respiratory irritation and pulmonary edema.

Formaldehyde effects on the nervous system are not well understood, although psychological and neurophysical effects have been reported (*10, 70*). The results are difficult to interpret, but suggest that formaldehyde levels of ~1 ppm can affect the central nervous system. Effects include subtle changes such as short-term memory loss, increased anxiety, and slight changes in adaptation to darkness.

A variety of other organic contaminants have also been identified in indoor environments. People emit bioeffluents such as acetone, butyric acid, ethyl and methyl alcohol, and other acids and alcohols. Combustion of wood, kerosene, and tobacco produces polycyclic aromatic hydrocarbons. Application of pesticides can release chlorinated hydrocarbons or organophosphate compounds. Personal care products, cleaning materials, paints, lacquers, and varnishes generate chlorinated compounds, acetone, ammonia, toluene, and benzene. Health risks associated with indoor exposures to this diverse group of chemicals have not been investigated adequately.

Asbestos fibers. Because of the widespread use of asbestos-containing products in ceiling tiles, floor tiles, pipe insulation, spackling compounds, concrete, and acoustical and thermal insulating material, there is a large potential for public exposure (*71, 72*). Acute exposures to asbestos and glass fibers cause severe skin irritation. A variety of neoplastic diseases, with latency periods of 15 or more years, have been associated with asbestos exposures. Increases in lung cancers, pleural and peritoneal mesotheliomas, and gastrointestinal tract cancers have been linked to occupational exposures (*73–75*). Several studies have shown increased mesothelioma rates among persons living near asbestos-production facilities and shipyards and among family members living with asbestos-exposed workers (*76–79*).

Airborne fiber concentrations determined by NIOSH standard methods range from zero during normal activities outdoors and in schools, offices, and dormitories to more than 100 fibers per milliliter during disrupting contact maintenance to ceilings (*76, 80, 81*). Unless the material is extremely friable, which is rare, asbestos exposures are likely to be episodic and therefore difficult to evaluate by random monitoring. Electron microscopic examination of air samples revealed values ranging from nearly zero outdoors in many U.S. cities to about 2000 ng/m^3 in a New Jersey school during custodial activities (*72, 80, 81*). Current asbestos standards include an Occupational Safety and Health Administration (OSHA) standard of 2 fibers per milliliter (*73, 82*) and a recommended NIOSH limit of 0.1 fiber per milliliter (*83*).

Although few measurements of air concentrations are available, it is clear that houses, schools, and office buildings can become contaminated. Risks to the general public are unknown since only exposure-effect data from occupational settings are available. However, the possible synergism with other contaminants, particularly cigarette smoke, and the severity of the potential health consequences justify steps to limit exposure to asbestos.

Most (85 percent or more) of the asbestos in use is immobilized in strong binding material (*84*). However, if this material is deliberately or accidentally disrupted, asbestos fibers can be released. Because the material can be found in easily accessible places and in ventilation systems, there is a potential for fiber release as a result of maintenance, renovation, negligence, or vandalism. In 1978, the U.S. Environmntal Protection Agency (EPA) banned spray-on application of asbestos-containing substances, except those in which the asbestos fibers are encapsulated with a bituminous or resinous binder during spraying to reduce friability after drying (*85*).

Controls

Workable and effective control strategies must be based on an understanding of several pertinent factors. First, contaminant characteristics need to be assessed, including: concentrations, reac-

tivity, physical state, and particle size, if applicable. Second, emission source configurations should be taken into account. Are discharges continuous or intermittent, are they point or area releases, and do they originate primarily indoors or outdoors? Third, the nature of exposure response relations must be considered. Are individuals to be protected from long-term exposures to low concentrations or periodic short-term exposures to peak concentrations? Finally, the type of indoor enclosure is important. Some ameliorating measures are more suited to private residences than to public buildings, or to new, as opposed to existing, structures.

Identified indoor pollution control methods fall into five general categories: ventilation; source removal or substitution; source modification; air purification; and behavioral adjustments to reduce exposures (avoidance). These classifications are not mutually exclusive and effectual strategies might use combinations, such as ventilation, source removal, and behavioral changes to reduce nonvoluntary exposures to tobacco smoke. Table 2 summarizes the applicability of control methods to important indoor air contaminants.

Public Policy Issues

Realization that indoor air pollution may represent a public health hazard presents policy-makers with familiar questions: Is the problem serious enough to warrant official intervention and, if so, what public actions are most appropriate? Healthful indoor air quality—what it is and how to achieve it—is not yet well understood, and scientific data on exposures and associated health effects are lacking. Nevertheless, the mounting evidence of elevated indoor contaminant levels suggests that government efforts to safeguard citizens' health and safety may be justified.

Government response so far has been piecemeal and complaint-oriented. In some cases, such as asbestos, many different federal agencies have jurisdiction over portions of the problem. For other pollutants, like microorganisms, combustion by-products, and organic emissions from building materials, responsibility is not clearly defined. No overall strategy exists to provide a coordinated, well-managed approach to ensure adequate indoor air quality.

Historically, government measures to redress environmental pollution have been taken when identified pollutant hazards receive abundant attention in the media and sufficient public awareness is generated. With the possible exception of antismoking groups, no organized constituency has developed to champion the cause of clean air inside buildings. The absence of a group lobbying for healthful indoor air quality removes much of the political urgency normally associated with environmental problems. As results of ongoing research become available, it is likely that public pressure for official intervention will build and legislators will have more incentive to take action.

A more structured and institutionalized approach is advocated by many professionals involved with this issue. Suggestions have included: coordinated interagency research (*86*); consolidation of federal responsibility within one agency (*6, 87, 88*); amendment of the Clean Air Act (*6, 11*); and granting of authority to states to regulate indoor air quality (*89*). A bill (HR-6323) was introduced in Congress during 1982 to appropriate funds for the Environmental Protection Agency to study the problem and recommend remedial actions.

As Sexton and Repetto (*90*) pointed out, it is essential to realize that a fundamental difference exists between indoor and outdoor air. Outdoor air is a "public good" in the sense that members of a community breathe basically the same ambient air. No rational individual would undertake the task of cleaning up the air over Boston, for example, since his or her share of the benefits would be much smaller than the costs. Nor would voluntary cooperation suffice, since those who refused to contribute could not be excluded from enjoying the benefits of reduced pollution. Similarly, in the absence of regulations or legal liability, no pollution source would spend enough on abatement, due to the difficulty of collecting from beneficiaries. The rationale for government regulation of outdoor air pollution has focused on the issue that those who suffer the effects are not compensated, nor is their interest in cleaner air readily effective in influencing polluters.

The situation is quite different for some indoor environments, especially private residences. Both the costs and benefits of pollution control are internalized within households. If occupants foul the air in their own home, they are forced to breathe it. If they attempt to improve its quality by increasing ventilation or installing air-cleaning devices, they bear the costs and enjoy the benefits. For some contaminants, such as tobacco smoke, odorants, and water vapor, benefits are readily recognizable through improvements in perceptible air quality and reduction of corrosion, soiling, and molds. For pollutants that are harmful to human health, but below perception thresholds, benefits will include reduced health risks due to lower exposures.

Creation of a regulatory framework for indoor air quality poses special policy issues which bear directly on choices about appropriate public responses (*90*). Certain aspects of nonworkplace environments are now subject to government ordinances, including residential and commercial building codes, health regulations, safety rules, and fire ordinances. However, this form of intervention is not necessarily optimal or even desirable. Although there are similarities between indoor and outdoor air, the complex set of regulations comprising the Clean Air Act should not automatically serve as a guide for indoor control strategies. Setting strict indoor air quality standards would almost certainly be expensive because of the costs associated with monitoring and regulating approximately 100 million buildings in the United States. Perhaps the most serious impediments to the regulatory approach are public antipathy toward this form of intervention and problems associated with enforcement. The diversity of nonoccupational indoor environments needs to be considered before practical and cost-effective control strategies can be designed and implemented.

It must also be taken into account that households are already making decisions about their own air quality. Promulgation of regulations might or might not improve those decisions. To determine the appropriateness of regulatory intervention it is necessary to do more than compile information about pollutant concentrations, human exposures, and associated health hazards. It is equally important to obtain information about individual perceptions of indoor air quality, public awareness of health risks, and the extent to which better information influences consumer choices. Commonsense precautions by building occupants may well prove to be the most effective and least expensive control measures. For this reason, the effectiveness of public information programs in promoting behavioral adjustments that will reduce personal exposures to air pollution needs to be evaluated (for instance, voluntary segregation of smokers and nonsmokers inside buildings).

In the development of effective public policy, the responsibilities of different sectors in society that have a stake in this issue should be emphasized. There

are important distinctions between the responsibilities of individuals, building designers, contractors, operators and owners, professional organizations, product and material manufacturers, and government. Major responsibilities that should be formally recognized are summarized in Table 3.

Table 3. Responsibilities for healthful indoor environments.

Individuals
Maintain and properly use products and appliances
Exercise direct discretionary control of ventilation in most residential and some occupational circumstances
Building owners or managers
Operate and maintain a balanced ventilation system in compliance with building ventilation codes
Use zone ventilation or local exhaust for indoor contaminant sources
Properly use cleaning solvents, paints, varnishes, herbicides, insecticides, furnishings, and insulation
Architects, developers, contractors
Adopt protection of indoor air quality as a design objective
Design ventilation systems to comply with new ASHRAE standard 62-1981
Provide for separation of occupants and indoor pollutant sources
Elimination or containment of potential sources
Manufacturers
Test, certify, and label products that are potential air pollution sources
Conduct research on potential health and comfort effects resulting from normal use or possible misuse of products
Substitute less harmful products and materials, if necessary
Government
Ensure healthfulness of indoor environments built, maintained, supervised, or financed through public funding
Ensure compliance with building ventilation codes and acceptable indoor air quality throughout the occupied life of a building
Sponsor research to assess indoor concentrations, health and comfort effects, control and policy options
Establish model or mandatory guidelines, codes, ordinances, or performance standards to protect the public
Provide information and assistance to state and local governments
Advise the public on safety of products, construction materials and practices, availability of monitoring equipment, and performance of HVAC systems

Recommendations

The issue of indoor air pollution and its effect on public health is complex. Yet because society has been slow to recognize the importance of healthful indoor air quality, the information we have is fragmented, anecdotal, and often contradictory. As with other environmental contaminants, strategies for reducing risk to exposed populations must be based on defensible exposure-response relations. In this way, the cost-effectiveness of various control options can be evaluated on the basis of reductions in population exposures. Table 4 shows the important components of a comprehensive evaluation of indoor air pollution and summarizes our current knowledge.

Several major issues are involved in decisions concerning the need for public action to deal with indoor air pollution. First, the role of government may depend on the degree of "publicness" of a particular building. The rationale for government intervention is stronger for public buildings (such as hospitals) than private residences. Government responsibility may be different for occupational and nonoccupational settings and for existing and planned buildings.

Second, dissimilarities between indoor emission sources may be important. Many contaminants in indoor environments (tobacco smoke, combustion by-products) result from human activities, while others (radon, formaldehyde) are less dependent on the activity patterns of occupants. Behavioral adjustment (such as prohibiting smoking in public places or using hood ventilation during cooking) may be the most effective and inexpensive way to control pollutants arising from discretionary actions. For pollutants not directly related to human activities, such as those emitted by soil, tap water, building materials, and furnishings, stricter building codes, simple air-cleaning devices, or sealants might be required.

Third, some indoor pollutants, such as tobacco smoke, are perceptible to most people, and individual actions to reduce personal exposures may be predicated on sensory stimuli. Other pollutants are

Table 4. Components of comprehensive evaluation of indoor air pollution and state of current knowledge.

Emission sources
Chamber studies done for several sources
Few measurements under dynamic conditions
Few studies of emission rates during normal use
Lack of information about distribution of sources within population
Dilution
Understanding of basic components affecting air-exchange rates
Measurement techniques available
Site-specific models developed, but more general application problematic
Only limited information available on distribution of air-exchange rates in existing buildings
Mixing inside buildings without mechanical ventilation systems not well understood
Indoor concentrations
Survey-type data collected for some pollutants
Applicability of survey data to entire building stock unknown
Dilution and mechanical filtration typically assumed to be first-order determinants of concentrations
Chemical and physical interactions, as well as removal rates, not well defined
Little known about variations in both removal and penetration rate
Human activity patterns
General features of population activity patterns known
Insufficient information about variations in activity patterns with age, sex, socioeconomic class, employment status, location, and season
Exposures
Relatively few studies of personal exposures to air pollution
Limited data indicate poor correlation between outdoor concentrations and personal exposures
Lack of suitable instrumentation limits application of personal exposure studies
Distribution of exposures across the population and effects of energy conservation on indoor exposures not known
Health effects
Irritant, toxic, mutagenic, and carcinogenic effects noted for many indoor contaminants
Additional information needed on central nervous system effects
Epidemiologic evidence of adverse health effects available for some pollutants
Data on dose-response relation accumulated for a few pollutants
Numbers, characteristics, and distribution of chemically sensitive individuals not known
Information lacking on health effects of long-term, integrated exposures compared to short-term, peak exposures

below perception thresholds (radon, microorganisms, asbestos, CO, NO_2, and so on). Consideration of voluntary and nonvoluntary risks is important for policy decisions. Development of simple warning devices could provide individuals with information on which to base decisions about the appropriateness of remedial measures. Such actions could reduce the need for government intervention to mitigate public health risks.

Fourth, it is necessary to specify whether building occupants are to be protected from chronic exposures to low levels of pollution or short-term peak exposures. If long-term exposures are considered important, then reductions in total human exposures to air pollution should be the goal. If short-term exposures are critical, then efforts should be focused on identifying peak concentrations and protecting the population at risk (for instance, limiting CO exposures for building occupants who use gas-cooking stoves for space heating).

Fifth, policy-makers must balance the benefits of energy conservation measures (for instance, reduced ambient pollution from fossil-fueled power plants) against the costs of deteriorating indoor air quality. Determining the essential components of healthful indoor air is a fundamental part of this process.

Sixth, if it is decided that public intervention is needed, a regulatory approach should not automatically be adopted. Policy alternatives such as economic incentives, better definition of legal rights and liabilities, public information programs, and expanded administrative efforts based on existing legislation might be more appropriate for control of indoor environmental hazards.

Because data from several studies indicate indoor exposures to some pollutants may represent significant health risks, official efforts to define the magnitude and extent of public health consequences seem justified. However, the future of public efforts to deal with indoor air pollution in nonoccupational environments is uncertain. Adequate funding is not currently available. Given the present regulatory climate of limited government intrusion and increasing reliance on free-market economics, it seems unlikely that programs focusing on indoor air pollution will be initiated in the near future. We believe that an overall strategy should be developed to ensure a coordinated, well-managed investigation of indoor air pollution exposures and their health consequences. In combination with efforts to define the problem, control options and policy alternatives should be evaluated.

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Cost-Effective Priorities for Cancer Prevention

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Summary. Faced with limited resources, the United States must set priorities for research to identify preventable causes of cancer. A quantitative approach to priority setting, based on principles of decision analysis and cost-effectiveness analysis, can offer guidance in this process. An illustrative application of such a model suggests that the National Institutes of Health–supported clinical trial of dietary β-carotene offers a greater expected reduction in cancer mortality per research dollar than carcinogen bioassays of high-volume industrial chemicals such as *p*-dichlorobenzene. National research priorities should reflect the relative cost-effectiveness of such investments.

Environmental factors are responsible for 80 to 90 percent of cancer deaths in the United States (*1, 2*). This conclusion, which once aroused considerable controversy, is now generally accepted, provided that the "environment" is broadly defined to include not only industrial chemicals and pollution, but also diet, reproductive behavior, and other elements of life-style and culture, as well as such natural phenomena as infectious agents and nonionizing radiation. Doll and Peto have placed the contribution to U.S. cancer mortality of occupational and environmental exposures to industrial chemicals at less than 5 percent, including 2 percent due to asbestos (*2*).

Growing hope during the 1970's that cancer could be controlled in large part by detecting and eliminating carcinogens has been tempered during the 1980's by the sober realization that preventing cancer will not be simple. Epidemiologic data firmly support the proposition that most cancers are preventable in principle, but current understanding leaves us far short of being able to prevent most cancer in fact. The challenge of the coming decades will be to identify the specific agents that cause or prevent cancer and, after identifying them, to develop and implement interventions to alter human exposure to them.

The problem of identifying carcinogens in the environment seems formidable enough when attention is focused on the 70,000 or so industrial chemicals in production. The cost of testing this inventory of chemicals, let alone the thousands of new chemicals entering production each year, would be huge. Even if financial cost were not a constraint, the limited supply of toxicologists and laboratories would constrain the volume of long-term bioassays.

Epidemiologic insights should, however, lead us to examine the priority-setting problem in a broader framework. If industrial chemicals other than asbestos account for 3 percent of cancer deaths, the prospect of saving 12,000 lives each year (out of 400,000 cancer deaths) should encourage us to discover the key industrial agents and control exposure to them. But if 35 percent of cancer deaths are related to diet (*2*), efforts to discover dietary factors in cancer might deserve an even greater claim on resources.

Toxicologic studies of industrial chemicals and epidemiologic studies of dietary agents are, in general, funded from different budgets, and might seem not to be in competition for the same limited resources. For the society as a whole, however, it is imperative to ask how best to spend resources in the general domain of cancer prevention. Priorities need to be set among alternative research strategies for detecting carcinogenic and anticarcinogenic agents, and such priority setting should encompass the full range of environmental factors (broadly defined) in cancer prevention.

This article illustrates a quantitative approach to priority setting, based on principles of cost-effectiveness and decision analysis. It also shows how the approach may be used to compare the cost-effectiveness of toxicologic studies of industrial chemicals and prospective trials of dietary constituents. The industrial chemical examined is *p*-dichlorobenzene, the active ingredient in mothballs. The cost-effectiveness of a randomized prospective trial of dietary β-carotene, a close relative of vitamin A, is also assessed. This comparison and other considerations lead to policy implications regarding the optimal use of resources in investigating the cancer-related effects of environmental agents.

Uncertainty is inherent in this kind of prospective analysis, and the attempt to quantitate this uncertainty may make some readers uncomfortable. However, policy decisions must and will be made in the face of uncertainty, and analysis

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