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# Indoor air quality and health

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## Abstract

During the last two decades there has been increasing concern within the scientific community over the effects of indoor air quality on health. Changes in building design devised to improve energy efficiency have meant that modern homes and offices are frequently more airtight than older structures. Furthermore, advances in construction technology have caused a much greater use of synthetic building materials. Whilst these improvements have led to more comfortable buildings with lower running costs, they also provide indoor environments in which contaminants are readily produced and may build up to much higher concentrations than are found outside. This article reviews our current understanding of the relationship between indoor air pollution and health. Indoor pollutants can emanate from a range of sources. The health impacts from indoor exposure to combustion products from heating, cooking, and the smoking of tobacco are examined. Also discussed are the symptoms associated with pollutants emitted from building materials. Of particular importance might be substances known as volatile organic compounds (VOCs), which arise from sources including paints, varnishes, solvents, and preservatives. Furthermore, if the structure of a building begins to deteriorate, exposure to asbestos may be an important risk factor for the chronic respiratory disease mesothelioma. The health effects of inhaled biological particles can be significant, as a large variety of biological materials are present in indoor environments. Their role in inducing illness through immune mechanisms, infectious processes, and direct toxicity is considered. Outdoor sources can be the main contributors to indoor concentrations of some contaminants. Of particular significance is Radon, the radioactive gas that arises from outside, yet only presents a serious health risk when found inside buildings. Radon and its decay products are now recognised as important indoor pollutants, and their effects are explored. This review also considers the phenomenon that has become known as Sick Building Syndrome (SBS), where the occupants of certain affected buildings repeatedly describe a complex range of vague and often subjective health complaints. These are often attributed to poor air quality. However, many cases of SBS provide a valuable insight into the problems faced by investigators attempting to establish causality. We know much less about the health risks from indoor air pollution than we do about those attributable to the contamination of outdoor air. This imbalance must be redressed by the provision of adequate funding, and the development of a strong commitment to action within both the public and private sectors. It is clear that meeting the challenges and resolving the uncertainties associated with air quality problems in the indoor environment will be a considerable undertaking. © 1999 Elsevier Science Ltd. All rights reserved.

**Keywords:** Biological particles; Combustion pollution; Radon; Sick building syndrome; Volatile organic compounds

## 1. Introduction

Until recently, the health effects of indoor air pollution have received relatively little attention from the scientific community. Prior to the 1970s, problems with indoor air quality in residences and the non-industrial workplace were occasionally investigated, but the level of interest

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was low (Stolwijk, 1992). Even today, the bulk of public concern continues to be directed at the health impacts of outdoor pollution. Numerous studies suggest that most members of the public perceive the risks from poor quality outdoor air as being substantially higher than those from indoor contamination (LHEA, 1997). These perceptions are formed despite the fact that, in developed societies, many people pass most of their time indoors. For example, a recent investigation of time budgets amongst US residents found that, on average, individuals spent 88% of their day inside buildings, and 7% in a vehicle. Only 5% of participants' time was actually spent outside (Robinson and Nelson, 1995). Whilst this does not per se mean that indoor exposures will produce more harmful health effects, the evidence is that indoor concentrations of many pollutants are often higher than those typically encountered outside.

A preoccupation with the relationship between outdoor air quality and health is understandable. Particularly in more economically developed and rapidly developing countries, recent and unprecedented changes in lifestyles and environmental quality have meant that an increasing number of people are exposed to the contaminants of urban air (Lipfert, 1997). There is certainly evidence that, especially amongst more vulnerable members of society, outdoor pollutants may pose a real health risk (Burr, 1995). Furthermore, the frequently invisible nature of indoor air quality problems must be contrasted with the often only too obvious photochemical and particulate substances that characterise outdoor contamination.

Historically, problems with indoor air were unquestionably much more apparent than they are today. Soot found on the ceilings of prehistoric caves provides ample evidence of the high levels of pollution that was associated with inadequate ventilation of open fires (Spengler and Sexton, 1983). Whilst chimneys first began to appear in European homes in the late twelfth century, most large medieval houses still had a central hearth in the great hall, ventilated by a louvre in the roof. It was only during the sixteenth century that chimney stacks and fireplaces against walls came into general use (Brimblecombe, 1987; Burr, 1997). The blackened roof timbers in many buildings that predate these innovations bear testimony to the severe pollution problems that their inhabitants faced.

Today, concern over the health effects of poor quality indoor air is increasing. Despite the fact that the vast majority of buildings exhibit no immediately apparent problems, a wide spectrum of symptoms and illnesses are attributed to non-industrial indoor air pollution (Redlich et al., 1997). Indeed, problems associated with shortcomings in indoor air may be one of the most common environmental health issues most doctors now face (Seltzer, 1995).

Over recent decades, there have been many changes in the way buildings are constructed and operated. To some

extent, modifications in building design have been driven by the need for increased energy efficiency, largely brought about by higher fuel costs since the 1970s oil crisis (Jones, 1998). Modern homes and offices are much better insulated than was previously the case. Houses with ventilation rates as low as 0.2 to 0.3 air exchanges  $\text{h}^{-1}$  are now widespread (Platts-Mills et al., 1996). In older properties, particularly those with open fireplaces, ventilation rates above 1 air exchange  $\text{h}^{-1}$  are more common. Improved insulation has been accompanied by numerous other modifications to the management of indoor environments, and advances in construction technology have led to a much greater use of synthetic building materials (D'Amato et al., 1994). All these changes have undoubtedly meant that buildings are more comfortable. However, they have also provided an environment in which airborne contaminants are readily produced and may build up to substantially higher concentrations than are typically encountered outside (Teichman, 1995).

Indoor air pollutants emanate from a range of sources (Table 1). They are emitted by the fabric of buildings, and may also be a by-product of the activities that are undertaken within them. Sources can be broadly classified as being associated with the activities of building occupants and other biological sources, the combustion of substances for heating or fuel, and emissions from building materials. For some contaminants, infiltration from outside, either through water, air, or soil, can also be a significant source.

The concentration of a pollutant indoors depends on the relationship between the volume of air contained in the indoor space, the rate of production or release of the pollutant, the rate of removal of the pollutant from the air via reaction or settling, the rate of air exchange with the outside atmosphere, and the outdoor pollutant concentration (Maroni et al., 1995). However, actual human exposures are often difficult to quantify. This is largely because the behaviour and activity patterns of individuals can strongly affect their levels of exposure (Harrison, 1997). Results from the TEAM (Total Exposure Assessment Methodology) studies undertaken by the US Environmental Protection Agency (EPA) during the 1980s consistently show that personal exposures to many pollutants can markedly exceed those anticipated from concentrations in ambient air (Wallace, 1991a). This phenomenon has become known as the 'personal cloud effect' (Furtaw et al., 1996).

Problems in quantifying exposure also arise because many of the advanced technologies that have been developed for measuring outdoor pollution are not suitable for indoor use. In part, this is due to considerations of cost, size, and the amount of air they displace (Maroni et al., 1995). Although miniaturised measuring devices have been developed for indoors, they often only record average pollutant concentrations over several hours or days.

Table 1  
Major indoor pollutants and emission sources<sup>a</sup>

Pollutant	Major emission sources
Allergens	House dust, domestic animals, insects
Asbestos	Fire retardant materials, insulation
Carbon dioxide	Metabolic activity, combustion activities, motor vehicles in garages
Carbon monoxide	Fuel burning, boilers, stoves, gas or kerosene heaters, tobacco smoke
Formaldehyde	Particleboard, insulation, furnishings
Micro-organisms	People, animals, plants, air conditioning systems
Nitrogen dioxide	Outdoor air, fuel burning, motor vehicles in garages
Organic substances	Adhesives, solvents, building materials, volatilisation, combustion, paints, tobacco smoke
Ozone	Photochemical reactions
Particles	Re-suspension, tobacco smoke, combustion products
Polycyclic aromatic hydrocarbons	Fuel combustion, tobacco smoke
Pollens	Outdoor air, trees, grass, weeds, plants
Radon	Soil, building construction materials (concrete, stone)
Fungal spores	Soil, plants, foodstuffs, internal surfaces
Sulphur dioxide	Outdoor air, fuel combustion

<sup>a</sup>Taken from Spengler and Sexton (1983).

This can create uncertainties, especially for pollutants with health effects thought to be related to short-term extreme exposures. In the case of recording pollutants from sources such as micro-organisms an opposite problem can apply, whereby monitoring programmes must be operational for a very long time period to provide accurate estimates of exposure (Blomquist, 1994).

Because of the difficulties in assessing public exposure to indoor air pollution, coupled, perhaps, with the fact that the resurgence of scientific interest into the links between indoor air quality and health is still relatively recent, we know much less about the health risks from polluted indoor air than we do about those associated with outdoor contamination. Indeed much of what has been learnt about the health impacts of indoor air comes from studies of the outdoor environment. Exposure to indoor toxicants can potentially lead to a variety of adverse health outcomes (Bascom et al., 1995). The likelihood that an individual will become ill from the presence of a contaminant depends upon factors such as the individual's sensitivity to that contaminant, the contaminant concentration, the current state of their psychological and physical health, and the duration and frequency of exposure (Seltzer, 1997). Indoor air pollutants have the potential to cause transient morbidity, disability, disease, and even death in extreme cases (Berglund et al., 1992). Recent research into these health outcomes has involved human, animal, and in vitro studies (Maroni et al., 1995).

Human studies entail the observation and measurement of symptoms and other effects in individuals exposed to pollutants, either routinely (as in epidemiological studies) or in experimental circumstances. The main advantage of studies of routine situations is that the conditions under which exposure takes place are realistic,

although the power of these investigations is sometimes insufficient to signify the likely causality of associations. Experimental exposures have the advantage that exposure conditions and subject selection may be determined by the investigator. However, they are only suitable for studying slight, reversible, short-term effects in healthy or only moderately ill individuals.

Animal studies involve the assessment of the health effects of exposures in laboratory animals. Their main limitation is that it is necessary to extrapolate the findings to humans. Such extrapolations are fraught with difficulties, particularly as results are often based on much higher doses than would be commonly encountered in the indoor environment.

In in vitro research, the effects of pollutants on cell or organ cultures are examined. They have cost advantages over animal studies and are quick to undertake, but there are problems associated with the use of their findings to predict effects on whole organisms.

It is the aim of this review to draw together the results from these investigations and present a picture of our current understanding of the relationship between indoor air pollution and health. Most of the evidence presented will be taken from epidemiological studies. Animal and in vitro research has provided many important findings. However, the conditions of these studies may be rather removed from those that typically prevail when individuals are exposed to pollutants indoors. Major progress has recently been achieved in the field of developing guidelines for volatile organic compounds from bioassays (ECJRA, 1997), but their use for many substances is less well developed. Hence, this is not the place for a detailed consideration of their findings. Neither will research into the health impacts of occupational

exposures to pollutants be discussed. Such exposures are usually associated with high contaminant doses, and the health outcomes are often severe. Hence, the aetiology and mechanisms of industrial exposures are sufficiently different from those commonly encountered in the domestic or office environment to warrant consideration elsewhere.

In this account, pollutants are considered according to whether they arise from biological or non-biological sources. Of course, dividing them in this way provides a simplistic picture of true patterns of exposure; in reality individuals are often concurrently exposed to a wide variety of contaminants from a range of different sources. In some situations, they may react with each other, leading to patterns and levels of exposure that are rather different from the apparent sum of their parts. However, the use of a simplified division is necessarily adopted for the sake of clarity.

Also considered in this text is the phenomenon that has become known as ‘sick building syndrome’ (SBS), where the occupants of some buildings repeatedly describe a complex range of vague and often subjective health complaints (Horvath, 1997). Since the early 1970s, numerous outbreaks of SBS have been reported. These have usually been in offices, but sometimes schools, hospitals, homes for the elderly, or apartments have been involved. The symptoms of SBS are often relatively minor, non-specific, and common amongst the general population, but are more frequent amongst the occupants of ‘sick’ buildings (Lahtinen et al., 1998). Despite their minor nature, SBS symptoms may have a greater impact on public health and cost to the economy than some major diseases due to widespread absenteeism and lowered productivity amongst affected workers (Wallace, 1997). For example, it has been estimated that the annual ‘cost’ of headaches amongst the employees of the US Environmental Protection Agency may be as high as \$2 million (Wallace, 1997).

Sick building syndrome is normally distinguished from the term ‘building related illness’ (BRI) (Ryan and Morrow, 1992). Whilst BRI has a known aetiology, many studies of SBS have failed to determine any specific factor that may explain the health complaints (Lahtinen et al., 1998). Outbreaks of SBS have been known to occur in older, naturally ventilated buildings. However they are more common in modern energy-efficient ‘airtight’ buildings, in particular those served by mechanical heating, ventilation, and air-conditioning (HVAC) systems (Redlich et al., 1997). This observation has led many to conclude that SBS must in some way be associated with indoor air quality problems.

2. Pollution from non-biological sources

It is important to note that outdoor sources may be the main contributors to indoor concentrations of a number of non-biological pollutants commonly found in indoor air. This is especially the case for contamination in buildings situated in urban areas and close to industrial zones or streets with heavy traffic. The factors which determine the contribution of outdoor pollution to indoor air quality include the type of ventilation in use (natural or forced), the ventilation rate (air changes per hour), and the nature of the contaminants in question (Wanner, 1993). The major outdoor sources of important indoor air pollutants are given in Table 2. The USA EPA TEAM studies have shown that reactive gasses such as ozone tend to occur at lower concentrations indoors than outdoors because they react rapidly with indoor surfaces (Wallace, 1987). Non-reactive gases may accumulate indoors and exposures there may be greater than outside.

Where meaningful, typical indoor–outdoor concentration ratios of pollutants are provided. However, to discuss the full impact of outdoor pollutants on indoor air

Table 2  
Outdoor sources of major indoor air pollutants

Pollutant	Percentage of emissions associated with industry <sup>a</sup>	Percentage of emissions associated with transport <sup>a</sup>
Benzene	32	65
Carbon monoxide (CO)	3	90
Lead (Pb)	31	60
Oxides of nitrogen (NO <sub>x</sub> )	38	49
Particulates (PM <sub>10</sub> )	56	25
Sulphur dioxide (SO <sub>2</sub> )	90	2
Volatile organic compounds (VOCs)	52	34
Ozone (O <sub>3</sub> )	Arises from atmospheric chemical reactions	

<sup>a</sup>Figures based on UK estimates (DOE, 1997).

quality would require a detailed consideration of the chemistry and processes that operate within the outdoor environment. This is not the place for such a discussion, although the health effects of one outdoor pollutant, radon, are considered. Although radon arises from outside, it merits consideration as it only presents a significant risk to health when found inside buildings.

Heating and cooking are often essential indoor activities but may produce smoke and gasses that present a problem of disposal. This difficulty is greatest in colder climates, where it is necessary to retain heat at the same time as removing combustion by-products (Burr, 1997).

A wide range of pollutants are associated with combustion. In the past, a high reliance on wood and coal for the production of heat meant that the indoor pollution profile was dominated by smoke from these sources (Wallace, 1996). In some countries, especially those of eastern Europe and China, the use of these fuels still predominates (Lan et al., 1993). In much of western Europe and the USA, coal and wood have largely been replaced by natural gas and electricity (Lambert, 1997), and approximately one-half of all American homes now use gas for cooking and heating (Hines et al., 1993). If properly vented to the outside, most appliances are typically not major contributors to indoor air quality problems. However, the use of unvented appliances or those that are malfunctioning or improperly installed can generate severe problems (Nagda et al., 1996).

Cigarette smoking is an important source of indoor combustion related pollution. Tobacco smoke is an aerosol containing several thousand substances that occur as particles, vapours, and gases. Exposure to cigarette smoke has been associated with a wide range of acute and chronic health impacts. Although the prevalence of smoking in western societies is much lower than it was in the 1950s and 1960s, tobacco smoke still contributes a significant proportion of the total indoor pollutant dose for many individuals.

Materials that comprise the fabric of buildings are another important source of non-biological pollutants. A large number of the chemical compounds which are found in indoor air originate from paints, varnishes, solvents, and wood preservatives used in buildings (ECJRC, 1997). Furthermore, deteriorating materials that comprise the fabric of a building can become friable and release contaminants into the air. Pollutants from these sources are often difficult to quantify because they are present in relatively low concentrations, and their sources are diffuse (Wanner, 1993). An additional complication arises where levels of contamination are mediated by climatic factors such as temperature and relative humidity. Hence, possible health risks of contaminants from building materials depend very much on the nature and concentration of the pollutants involved.

The following sections consider the typical concentrations and important health effects of the range of non-biological pollutants from the various sources that are most commonly encountered in the indoor environment.

### 2.1. Asbestos

Asbestos is a generic term that applies to a group of impure hydrated silicate minerals which occur in various forms, and are incombustible and separable into filaments (Maroni et al., 1995). Until recently, asbestos was widely used by the building industry for the production of many different products. It was particularly valued for its electrical and thermal insulating properties, and was heavily utilised in pipe and boiler insulation, cement-board, thermal tiles, paint, and wallpaper (Bigonon et al., 1989). Concern over the health effects of asbestos exposure has led to legislation being introduced to prohibit its use in many countries. Hence, most asbestos-related air quality problems are associated with the management of existing asbestos installations.

Accurate assessment of exposure to asbestos is particularly difficult because most airborne fibres are too small to be counted by optical microscopy, and hence scanning and transmission electron microscopy must be used to analyse samples (Hines et al., 1993). Because of this, relatively little information on typical indoor concentrations is available (Gaensler, 1992). In one of the few studies that have been undertaken, Lee et al. (1992) examined exposure to airborne asbestos in 315 public, commercial, residential, school, and university buildings in the USA. The average concentration of asbestos was 0.02 structures  $\text{ml}^{-1}$  of air, whilst the average concentration of fibres greater than 5  $\mu\text{m}$  in length was 0.00013 fibres  $\text{ml}^{-1}$ . However, it is important to note that, in 48% of samples, no asbestos was detected. In general, asbestos-containing materials within buildings that are in good repair are unlikely to lead to exposure of occupants to concentrations higher than ambient levels. A recent literature review commissioned by the US EPA cost 4 million dollars and suggested that well-maintained asbestos in public buildings posed little risk to office workers (Hines et al., 1993).

Acute exposure to asbestos can cause skin irritation (Spengler and Sexton, 1983). However, the most serious health effects from asbestos are lung cancer, mesothelioma (cancer involving a proliferation of mesothelial cells) and asbestosis (a slowly developing but lethal fibrosis of the lung) (McDonald, 1991). Recent data suggest that the fibres must remain in the respiratory tract for approximately one year to produce effects, and those with a diameter of less than 1  $\mu\text{m}$  and a length greater than 5–10  $\mu\text{m}$  are believed to be particularly dangerous. There is generally a latent period of 20–50 yr between first exposure to asbestos, and the clinical manifestation of tumours (Doll and Peto, 1985).

Whilst the relationship between asbestos exposure and the incidence of mesothelioma and asbestosis is well documented, clarifying the relationship with lung cancer is complicated, and is still a controversial area (Huncharek, 1994). It is accepted that exposure to asbestos and tobacco smoke from active smoking has a marked synergistic effect. It is important to note that the majority of population risk from asbestos arises from industrial exposures. However, cases of disease associated with non-industrial exposures do occur. For example, Roggli and Longo (1991) highlight the instance of a teacher's aid with pleural mesothelioma who was for many years exposed to asbestos-containing acoustic plaster in a school building.

## 2.2. Carbon dioxide

Carbon dioxide ( $\text{CO}_2$ ) is a colourless, odourless gas. Humans continuously exhale  $\text{CO}_2$  formed in the body during metabolic processes and, where fuel is not being burnt, these emissions comprise the greatest contribution to indoor concentrations (Wanner, 1993). Carbon dioxide is also the main combustion product from gas, kerosene, and wood or coal fuelled appliances and these can represent significant sources when in operation (Moriske et al., 1996). Typical indoor  $\text{CO}_2$  concentrations range between 700 and 2000 ppm (approximately  $3657 \text{ mg m}^{-3}$ ) but can exceed 3000 ppm ( $5486 \text{ mg m}^{-3}$ ) during the use of unvented appliances (Arashidani et al., 1996).

Carbon dioxide is a simple asphyxiant, and can also act as a respiratory irritant (Maroni et al., 1995). However, although indoor to outdoor ratios of the gas are typically in the range 1–3 for most environments, exposure to an extremely high  $\text{CO}_2$  concentration (above 30,000 ppm or  $54860 \text{ mg m}^{-3}$ ) is required before significant health problems are likely (NASA, 1973). At moderate concentrations,  $\text{CO}_2$  can cause feelings of stuffiness and discomfort, an effect noted by Pettenkofer as far back as 1858 (Pettenkofer, 1858). Respiration can be slightly affected at levels above 15,000 ppm ( $27430 \text{ mg m}^{-3}$ ). Exposures above 30,000 ppm can lead to headaches, dizziness, and nausea (Schwarzberg, 1993). Yang et al. (1997) found that these concentrations also affect perception of motion. This may be because  $\text{CO}_2$  has been shown to moderate the activity of cells within the visual cortex.

## 2.3. Carbon monoxide

Carbon monoxide (CO) is a toxic odourless gas produced by the incomplete combustion of fuel (IEH, 1996). Water heaters, gas or coal heaters, and gas stoves are all indoor sources of CO (Gold, 1992). High indoor levels of CO can also result from the entry of outdoor vehicle exhausts into the ventilation system of a building. This can be a particular risk in buildings that have delivery areas where vehicles are parked with their motors run-

ning. In addition, tobacco smoking is an important source of transitory indoor CO pollution (Sterling, 1991), and the burning of charcoal briquettes and the use of gasoline-powered electrical generators during disruptions to electrical services can present temporary problems (Houck and Hampson, 1997). Another source of CO is methylene chloride, a substance used as a paint stripper. It is metabolised within the body to form CO, and its use can lead to a significant dose in even well ventilated rooms (Gold, 1992).

In the absence of emission sources, concentrations of CO in indoor environments are generally lower than those outdoors. In a US Environmental Protection Agency (EPA) survey of the personal CO exposures of 1000 non-smoking adults, residential exposures were generally low, ranging from 2 to 4 ppm (approximately  $2.3\text{--}4.7 \text{ } \mu\text{g m}^{-3}$ ) (Akland et al., 1985). Where gas stoves are in operation, hourly concentrations of CO are generally around 6 ppm ( $6.9 \text{ } \mu\text{g m}^{-3}$ ), and do not often exceed 12 ppm ( $13.8 \text{ } \mu\text{g m}^{-3}$ ) (Samet et al., 1987).

The toxic properties of CO are largely associated with its high affinity for oxygen-carrying proteins such as haemoglobin and myoglobin (Coulton and Lambert, 1991). Because its affinity for haemoglobin is approximately 200 times greater than that of oxygen, CO displaces oxygen, forming carboxyhaemoglobin (COHb), lowering the oxygen carrying capacity of the blood, and producing a left shift in the oxyhaemoglobin dissociation curve (Roughton and Darling, 1994). Carbon monoxide can also interfere with oxygen diffusion into cellular mitochondria, and interfere with intracellular oxidation (Gold, 1992).

The health effects of exposure to CO are generally described relative to COHb levels (Madany, 1992). In non-smoking individuals unexposed to environmental CO, blood COHb levels are usually around 0.5% (Lambert, 1997). Various symptoms of neuropsychological impairment have been associated with acute low-level exposures. Amitai et al. (1998) found that subjects exposed to CO from residential stoves for up to 2.5 h showed declines in their learning and planning abilities, as well as a drop in their attention and concentration spans. Chronic exposure (at 10–30% COHb) often produces symptoms that are easily misdiagnosed or overlooked, such as headache, fatigue, dizziness, and nausea (Stewart et al., 1970). There is evidence from animal studies that some foetal damage may occur from maternal exposure to CO at these levels (Longo, 1977).

Carbon monoxide poisoning has its most toxic acute effects on the organs with high oxygen requirements; the heart and brain. Hence, individuals with ischaemic heart disease are at particularly high risk (USEPA, 1991). At moderate concentrations of CO, adverse cardiovascular effects may be observed amongst susceptible individuals (Dahms et al., 1993). Amongst 20 non-smoking men with ischaemic heart disease, Lambert (1994) found that the

Table 3  
Acute health effects from formaldehyde exposure

Formaldehyde concentration (ppm)	Observed health effects
< 0.05	None reported
0.05–1.5	Neurophysiologic effects
0.05–1.0	Odour threshold limit
0.01–2.0	Irritation of eyes
0.10–25	Irritation of upper airway
5–30	Irritation of lower airway and pulmonary effects
50–100	Pulmonary edema, inflammation, pneumonia
> 100	Coma, death

Sources: Hines et al. (1993).

probability of occurrence of an episode of myocardial ischemia was 2.1 times higher at COHb levels of 2% relative to those below 1%.

Although non-reversible impairment is relatively rarely associated with CO exposure, the greatest danger of serious CO poisoning comes from faulty combustion appliances, or those with blocked or malfunctioning external vents (Howell et al., 1997). In such situations, COHb levels of between 50 and 60% can result in fainting and convulsions, whilst higher exposures can lead to coma and death. In England and Wales, there are on average around 60 deaths annually associated with accidental CO poisoning (Burr, 1997), and similar rates have been observed in the USA (Cobb and Etzel, 1991). Individuals who survive acute CO poisoning may still exhibit neurological and psychological symptoms many weeks or months after exposure, particularly if a period of unconsciousness has occurred (Choi, 1983).

#### 2.4. Formaldehyde

Formaldehyde is the most widespread aldehyde found in the environment. Although it is a volatile compound, it is not detected by the gas chromatographic methods commonly applied to VOC analysis, and hence is often considered separately (Maroni et al., 1995).

At normal room temperatures, formaldehyde is a colourless gas with a pungent odour. The primary sources of formaldehyde are building materials such as particle-board, medium-density fibreboard, plywood, resins, adhesives, and carpeting (Hines et al., 1993). It is also used in the manufacture of urea formaldehyde foam insulation (UFFI) which is injected into wall cavities to supplement the insulation in existing buildings. However, because of health concerns, UFFI is little used nowadays.

As with all volatile organic compounds, the concentration of formaldehyde within a given indoor space will be very dependent upon the presence of important emission sources. The background concentration of formaldehyde

in outdoor air is generally lower than 0.1 ppm (Maroni et al., 1995). Indoors, the rate of emission of formaldehyde varies according to conditions of temperature and humidity. Indoor formaldehyde concentrations usually exceed those observed outdoors. In an early study of 23 Danish homes by Anderson et al. (1975), the average formaldehyde concentration was 0.5 ppm ( $0.6 \text{ mg m}^{-3}$ ), with a range from 0.07 to 1.9 ppm ( $0.08\text{--}2.28 \text{ mg m}^{-3}$ ). Similar findings have subsequently been reported in Germany by Prescher and Jander (1987), in Finland by Niemala et al. (1985) and in the USA by Breysse (1984).

Adverse health effects from formaldehyde exposure may arise from inhalation, or direct contact. A range of acute health impacts have been attributed to the substance (Table 3). Exposure to concentrations of less than 1 ppm ( $1.2 \text{ mg m}^{-3}$ ) may result in sneezing, coughing, and minor eye irritation, although these symptoms often rapidly subside after the start of the exposure (Koeck et al., 1997). Numerous studies show that formaldehyde vapour is also an irritant of the skin (Eberlein-König et al., 1998) and the respiratory tract (Bardana and Montanaro, 1991).

There is conclusive evidence that formaldehyde is an animal carcinogen (Morgan, 1997). In the 1980s, a number of occupational research projects were carried out to address the potential carcinogenicity of formaldehyde in humans (e.g. Wong, 1983). All subjects were workers exposed to high concentrations of formaldehyde, but none of the studies found strong evidence of a cancer risk. One of the few pieces of evidence of a risk to humans from typical indoor exposures comes from the work of Vaughan et al. (1986) who reported a significant correlation between formaldehyde exposure and nasopharyngeal cancer in mobile home residents.

#### 2.5. Nitrogen dioxide

Nitrogen dioxide ( $\text{NO}_2$ ) is a water-soluble red to brown gas with a pungent acrid odour. It is formed from

the combination of nitrogen and oxygen during combustion at high temperatures (Maroni et al., 1995). Hence, the production of  $\text{NO}_2$  is particularly associated with the operation of gas appliances, kerosene heaters, and wood-burning stoves, as well as the smoking of cigarettes. Additionally, outdoor air can act as an important source for indoor  $\text{NO}_2$  pollution (Chan et al., 1990) although, in many areas of the USA and Europe, ambient outdoor  $\text{NO}_2$  levels are relatively low.

There have been numerous studies of indoor concentrations of  $\text{NO}_2$ . In the absence of emission sources, levels generally correlate well with those observed outdoors (Monn et al., 1998). In homes with gas cooking stoves in Albuquerque, New Mexico, Lambert et al. (1993a) found that 2-week average  $\text{NO}_2$  levels were 21 ppb ( $39 \mu\text{g m}^{-3}$ ) in bedrooms and 34 ppb ( $63 \mu\text{g m}^{-3}$ ) in kitchens. In comparison, bedroom concentrations in homes using electric stoves averaged just 7 ppb ( $13 \mu\text{g m}^{-3}$ ). On average, normal use of a vented gas cooking range adds 25 ppb ( $47 \mu\text{g m}^{-3}$ ) of  $\text{NO}_2$  to the background concentration in a home (Samet et al., 1987). In homes with unvented kerosene space heaters, 1-week average concentrations exceeding 45 ppb ( $84 \mu\text{g m}^{-3}$ ) have been observed (Leaderer et al., 1986). One-week average levels of greater than 50 ppb ( $94 \mu\text{g m}^{-3}$ ) have been reported in homes with unvented gas space heaters (Ryan et al., 1989).

Transient peak concentrations of  $\text{NO}_2$  during the operation of appliances can greatly exceed average measurements. Whilst cooking with a gas range, peak levels in the kitchen may be as high as 400–1000 ppb ( $752\text{--}1880 \mu\text{g m}^{-3}$ ) (Spengler et al., 1981). As individuals operating gas ranges often stay close to the appliance, personal exposures may be even greater.

Nitrogen dioxide is an oxidising agent that can be very irritating to the mucous membranes of the lung (Spengler, 1993). It is highly soluble in water, and a large proportion of inhaled  $\text{NO}_2$  is removed in the respiratory tract (Lambert, 1997). It is thought to combine with water in the lungs to form nitric acid ( $\text{HNO}_3$ ) and may react with lipids and proteins to form nitrite anions and hydrogen ions (Postlethwait and Bidani, 1990). Nitrogen dioxide within the airways is also converted into vapour phase nitrous acid ( $\text{HONO}$ ) via heterogeneous reactions involving water vapour, invoking the formation of free oxygen radicals and lipo-peroxidation (Spicer et al., 1993). Whilst it has been suggested that oxidant injury is the principal mechanism by which  $\text{NO}_2$  damages the lung, substantial uncertainty remains (Lambert, 1997).

Evidence from experimental research suggests that exposure to  $\text{NO}_2$  may increase respiratory infections, and adversely affect lung function (Frampton et al., 1991). However, there is rather little evidence from epidemiological studies that exposure to  $\text{NO}_2$  has deleterious health effects amongst the majority of the population.

Two important projects have discovered possible associations. In Italy, Viegi et al. (1992) found the use of bottled gas for cooking was associated with increased reporting of cough and phlegm in males. In England, Jarvis et al. (1996) found that a general population sample of females who reported they used mainly gas for cooking were more likely to report respiratory symptoms in the 12 months prior to survey. These women were also found to have reduced lung function and increased airway obstruction. No effects were observed amongst males, suggesting women may be more susceptible to  $\text{NO}_2$ , or are exposed to higher concentrations.

There is evidence that the health effects of  $\text{NO}_2$  may be greater amongst certain vulnerable population subgroups, such as children and asthmatics (Li et al., 1994). In the Harvard Six Cities Study, involving 10,106 children aged 6–10 yr, serious illness in infancy was more common amongst infants from homes with gas cooking (Ware et al., 1984). Recent meta-analyses suggest that the risk associated with  $\text{NO}_2$  exposure is probably only significantly increased for children aged over 2 yr (Hasselblad et al., 1992). In Australia, Pilotto et al. (1997) monitored  $\text{NO}_2$  exposures amongst 388 children aged between 6 and 11 yr. They found hourly peak levels of 80 ppb ( $150 \mu\text{g m}^{-3}$ ) and above were associated with significant increases in the reporting of sore throats, colds, and absences from school, and they concluded that it was important to consider short-term peak exposures. However, in another study, Samet et al. (1993) found no association between personal exposures and symptoms amongst infants, and Brunekreff et al. (1990) failed to find an association between  $\text{NO}_2$  and the pulmonary function of a sample of children.

Amongst asthmatic women and children, Goldstein et al. (1988) examined the relationship between mean 48 h  $\text{NO}_2$  concentrations in the subjects' kitchens, and their spirometric lung function. They found that exposure to  $\text{NO}_2$  levels ranging between 300 and 800 ppb ( $564$  and  $1504 \mu\text{g m}^{-3}$ ) was associated with a reduction in lung capacity (measured by FEV) of the order of 10%. Salome et al. (1996) also found that the experimental exposure of 600 ppb ( $1128 \mu\text{g m}^{-3}$ ) of  $\text{NO}_2$  over a period of one hour was associated with a slight increase in airway hyperresponsiveness amongst a sample of 20 asthmatics.

Exposure to  $\text{NO}_2$  may act as a trigger for asthma in one of two ways (Jones, 1997). One possibility is that the pollutant causes a direct effect on the lungs by inflicting toxic damage. Another is that it may irritate and sensitise the lungs, making individuals more susceptible to allergic response upon contact with indoor allergens. Evidence for the sensitisation mechanism comes from a study by Tunnicliffe et al. (1994); in asthmatics previously exposed to 400 ppb ( $752 \mu\text{g m}^{-3}$ )  $\text{NO}_2$  for a period of 1 h, lung function dropped by 19% after the inhalation of house dust mite allergen. This was compared to a reduction of just 14% in those exposed only to air.



## 2.6. Sulphur dioxide

Sulphur dioxide (SO<sub>2</sub>) is produced by the oxidation of sulphur impurities during the burning of coal and other fuels that contain sulphur (Burr, 1997). It is a colourless gas with a strong pungent odour that can be detected at about 0.5 ppm (0.9 mg m<sup>-3</sup>). It is readily soluble in water and can be oxidised within airborne water droplets (Maroni et al., 1995). Sulphur dioxide levels are generally lower indoors than outdoors, and indoor/outdoor concentration ratios between 0.1 and 0.6 are commonly observed in buildings without indoor sources. As a result of reductions in emissions, annual mean levels of ambient SO<sub>2</sub> in most major cities in Europe and the USA are below 20 ppb (52 µg m<sup>-3</sup>). However indoor SO<sub>2</sub> concentrations can be higher for homes with kerosene heaters and poorly vented gas and coal appliances. Leaderer et al. (1993) recorded average concentrations of 30 ppb (78 µg m<sup>-3</sup>) in a study of 33 homes with kerosene space heaters in Connecticut, USA. Mean values of 57 ppb (149 µg m<sup>-3</sup>) have been measured in homes equipped with both kerosene heaters and gas stoves (Leaderer et al., 1984).

From a health effects viewpoint, two substances are important; the SO<sub>2</sub> itself, and the acid aerosols that may result from its oxidation in the atmosphere. Absorption of SO<sub>2</sub> in the mucous membranes of the nose and upper respiratory tract occurs as a result of its aqueous solubility. The deposition pattern of acid aerosols within the respiratory tract will be dependent upon the size distribution of the droplets and the level of humidity indoors (Maroni et al., 1995).

Whilst evidence from animal experiments and occupational exposures suggests that exposure to extreme concentrations of SO<sub>2</sub> and acid aerosols can precipitate an acute reduction in lung function (Islam and Ulmer, 1979), there are relatively few indications of short-term health impacts associated with typical indoor concentrations. However, long term exposures to indoor SO<sub>2</sub> may be associated with elevated reporting of chronic respiratory complaints. In the UK, Burr et al. (1981) found that residents of South Wales who had open coal fires were more likely than others to suffer from breathlessness and wheezing. They speculated that a high death rate from respiratory conditions amongst miners wives may have been associated with exposure to SO<sub>2</sub> from the burning of concessionary coal, although particles may have also played a part. Recent studies in China, where the domestic burning of coal is still widespread, have also associated exposure to SO<sub>2</sub> with impaired lung function, and a range of other respiratory symptoms (Qin et al., 1993; Jin et al., 1993).

## 2.7. Radon

Radon is an inert radioactive gas that arises directly from the decay of radium-226 contained in various min-

Table 4  
The uranium-238 decay series

Nuclide	Half-life	Decay particle
U-238	4.5 × 10 <sup>9</sup> yr	Alpha
Th-234	24 d	Beta
Pa-234	1.2 min	Beta
U-234	2.5 × 10 <sup>5</sup> yr	Alpha
Th-230	8 × 10 <sup>4</sup> yr	Alpha
Ra-226	1620 yr	Alpha
Radon-222	3.8 d	Alpha
Po-218	3 min	Alpha
Pb-214	27 min	Beta
Bi-214	20 min	Beta
Po-214	160 µ	Alpha
Pb-210	22 yr	Beta
Bi-210	5 d	Beta
Po-210	138 d	Alpha
Pb-206	Stable	-

Source: Lyman (1997).

erals (Lyman, 1997). It has a half-life of 3.82 d. As radon undergoes further radioactive decay, it produces a series of short-lived radioisotopes, known as radon daughters or progeny (Table 4). Radon itself is inert and causes little damage as most of it is exhaled in the breath. However, the progeny, Po-218 and Po-214 are electrically charged and can be inhaled either directly or through their attachment to airborne particles (Cohen, 1998). Once inhaled, they tend to remain in the lungs, where they may eventually cause cancer (Polpong and Bovornkitti, 1998). As activities such as the smoking of cigarettes can lead to considerably elevated levels of airborne particles, smokers are at particular risk from the inhalation of radon progeny (Hampson et al., 1998). Indeed, the US EPA has estimated that the cancer risk from radon for smokers is as much as 20 times the risk for individuals who have never smoked (US EPA, 1992).

In the past, contamination of air by radon and the subsequent exposure to radon daughters were believed to be a problem only for uranium and phosphate miners. However, it has recently been recognised that homes and buildings far away from uranium or phosphate mines can exhibit high concentrations of radon. As a consequence, radon and radon progeny are now recognised as important indoor pollutants (Létourneau, 1997).

Radon formed in rocks and soils is released into the surrounding air. Typical rates of radon release from soils throughout the world range from about 0.0002 to 0.07 becquerels (Bq) m<sup>-3</sup> s<sup>-1</sup>. Production rates from any soil are very dependent upon the geological characteristics of the soil itself and its underlying geological strata (Lévesque et al., 1997). Porous soils overlaying uranium rich alum shales, granites and pegmatites are a particularly high risk for radon, while gas-impermeable soils

consisting of fine sand, silt, and moist clay present a low risk (IARC, 1988).

Outdoors, radon emanating from the ground is quickly dispersed, and concentrations never reach levels that may be a threat to health. However, inside confined areas, low rates of air exchange can result in a build-up of radon and its daughters to concentrations tens of thousands of times higher than those observed outside (Wanner, 1993). Radon concentrations within a building depend very much on both the concentration of radon in the soil surrounding the structure, and the presence of entry points that allow the gas to infiltrate from outside (Jedrychowski et al., 1995). Some of the common entry points of radon into buildings include foundation joints, cracks in floors and walls, drains and piping, electrical penetrations, and cellars with earth floors (Nielson et al., 1997).

In the USA, Nero et al. (1986) summarised the results of 19 studies of indoor radon concentrations, covering 552 single-family homes. They found the mean indoor concentration was  $56 \text{ Bq m}^{-3}$ . As part of the more recent US National Residential Radon Survey, Marcinowski et al. (1994) estimated an annual average radon concentration of  $46.3 \text{ Bq m}^{-3}$  in US homes. They also calculated that around 6% of homes had radon levels greater than the US EPA action level for mitigation of  $148 \text{ Bq m}^{-3}$ . Outside the USA, Albering et al. (1996) found a much higher average concentration of  $116 \text{ Bq m}^{-3}$  in 116 homes in the township of Visé in a radon prone area in Belgium. In Italy, Bochicchio et al. (1996) reported an average concentration of  $75 \text{ Bq m}^{-3}$  in a sample of 4866 dwellings, and observed concentrations exceeding  $600 \text{ Bq m}^{-3}$  in 0.2% of homes. Yun et al. (1998) recently undertook one of the relatively few studies of radon concentrations in the office environment. In 94 Hong Kong office buildings they recorded radon concentrations similar to those that have been observed in domestic situations, with a mean of  $51 \text{ Bq m}^{-3}$ .

Radon exposure has been linked to lung carcinogenesis in both human and animal studies. It has also been associated with the development of acute myeloid and acute lymphoblastic leukaemia. However, the estimation of health risks from residential radon is extremely complex, and encompasses many uncertainties. Studies of smoking and non-smoking uranium miners indicate that radon is a substantial risk factor for lung cancer at high concentrations. Based on data on dose–response relationships amongst miners, it is estimated that between 5 and 15% of lung cancer deaths might be associated with exposure to residential radon (Steindorf et al., 1995). The relevance of data from mines to the lower-exposure home environment is often questioned (Lubin et al., 1997). Nevertheless, a meta-analysis of eight epidemiological studies undertaken by Lubin and Boice (1997) found that the dose–response curve associated with domestic radon exposures was remarkably similar to that observed amongst miners.

Ecological (geographical) study designs have been adopted by a number of recent epidemiological investigations into the health risks associated with non-industrial radon exposures. Lucie (1989) reported positive county level correlations between radon exposure and mortality from acute myeloid leukaemia in the UK, and Henshaw et al. (1990) found that mean radon levels in 15 counties were significantly associated with the incidence of childhood cancers and, specifically, all leukaemias. However, these reports have been met with considerable criticism because ecological designs can suffer from serious limitations (Wolff, 1991). In particular, the effects of migration are often difficult to account for, information on potential confounding variables can be unavailable, and estimates of exposure for populations of large areas may differ greatly from actual individual doses. Recent more refined ecological analyses, such as that undertaken by Etherington et al. (1996), have reported no association between indoor radon exposure and the occurrence of cancer.

An alternative to ecological analyses is the case-control study design, where radon exposures amongst individuals with cancer are compared to those of control subjects free from the disease. Most case-control studies have reported a small, but significant, association between radon exposure and lung cancer mortality (Pershagen et al., 1994). For example, in a recent examination of over 4000 individuals in Sweden, Lagarde et al. (1997) estimated that there is an excess relative risk of contracting lung cancer of between 0.15 and 0.20 per  $100 \text{ Bq m}^{-3}$  increase in radon exposure.

## 2.8. Respirable particles

Smoke from the burning of wood and fossil fuels produces an extremely complex mixture of pollutants, both in physical and chemical characteristics, and toxicological properties (Lambert, 1997). One of the primary constituents of the smoke are respirable particles (Cooper, 1980). These are aerosols that are of a small enough diameter to enter and remain in the lung. Many are around  $6\text{--}7 \mu\text{m}$ , or less in diameter (Martonen et al., 1992). The particulate matter comprises a mix of organic and inorganic substances including aromatic hydrocarbon compounds, trace metals, nitrates, and sulphates (Maroni et al., 1995). In developing countries, exposure to smoke is arguably the greatest indoor pollution problem, as in many buildings the burning of wood, charcoal, crop residues, or animal dung is often undertaken without adequate ventilation (Gold, 1992). For example, in India, Pandey et al. (1989) found that airborne particle concentrations, measured during cooking, were as high as  $21,000 \mu\text{g m}^{-3}$ . Such figures are not typical of indoor exposures in the developed world.

In most developed societies, a movement away from the use of coal and wood to provide heat has meant that

indoor particulate pollution associated with combustion is rather lower than in the past. However, the popularity of woodburning stoves has increased in both Europe and the USA in recent years (Samet, 1990). Whilst particulate pollution from modern airtight stoves is much lower than was previously common with open fires, problems can still occur during start-up, stoking, and reloading.

In the absence of significant indoor sources, indoor to outdoor ratios of respirable particles are generally slightly below unity (Wallace, 1996; Janssen et al., 1998). However, measurement techniques routinely used in reported studies are often not able to differentiate combustion related particles from those from other sources (Maroni et al., 1995). Given the heterogeneous nature of particulate material, this makes it impossible to formulate a direct relationship between exposure to specific compounds in the aerosol and health effects.

As part of the Harvard Six-City Study, Neas et al. (1994) recorded mean annual  $PM_{2.5}$  (particles less than  $2.5\ \mu m$  in diameter) concentrations in the order of  $17\ \mu g\ m^{-3}$  in a sample of 470 non-smoking homes. The presence of a smoker can greatly add to indoor concentrations, and is discussed elsewhere in this review. In another sample of homes, Traynor et al. (1986) reported indoor concentrations of respirable particles slightly above background (up to  $30\ \mu g\ m^{-3}$ ) during the use of airtight woodstoves and substantially higher with non-airtight stoves ( $200\text{--}1900\ \mu g\ m^{-3}$ ). When not in operation, homes with woodburning stoves have on average about  $4\ \mu g\ m^{-3}$  higher indoor particulate concentrations than homes without the appliances.

It is important to note that personal exposures to particles are often higher than ambient indoor concentrations. This is particularly the case during the daytime. For example, Clayton et al. (1993) observed that daytime mean personal  $PM_{10}$  (particles less than  $10\ \mu m$  in diameter) exposure in a sample of individuals was more than 50% higher than either indoor or outdoor levels. Similar findings have recently been reported by Janssen et al. (1998).

In general, the health impacts of outdoor exposures to respirable particles are rather better studied than those associated with indoor exposures (see Ostro and Chestnut, 1998). Hence, the most extensive data on the health effects from exposure to particles are derived from epidemiological studies of outdoor air. Given that total exposure to particles is greater indoors than outside by virtue of the time spent indoors, a good deal of the apparent effects of outdoor particles probably occurs due to exposure indoors. Whilst *in vitro* and animal experiments suggest that emissions from coal or woodburning stoves are mutagenic, epidemiological evidence from studies in the indoor environment is limited to a relatively small number of reports (Marbury, 1991).

Irritant effects from a range of inhaled particles may result in airway constriction. There is evidence that

woodsmoke may be associated with respiratory illness, particularly amongst vulnerable groups such as children and those with pre-existing chronic respiratory disease. Honickey et al. (1985) examined respiratory symptoms amongst 62 children in Michigan, USA. They found 84% of children in homes with woodburning stoves recounted at least one severe symptom, compared to only 3% of children in homes without a stove. As part of the Six-City Study, Dockery et al. (1993) found an odds ratio of 1.32 (95% CI 0.99–1.76) for respiratory illness in households with woodburning stoves in comparison with those using other sources of heating. Koenig et al. (1993) reported that infants exposed to woodsmoke were more likely to recount asthma symptoms, and Abbey et al. (1998) observed a reduction in lung function in non-smokers exposed to high concentrations of indoor particles over a period of 20 yr.

Polycyclic aromatic hydrocarbons (PAHs) in woodsmoke are of particular concern because of their carcinogenic potential. These fat-soluble compounds are produced as a result of incomplete combustion, and include a large number of organic molecules which contain two or more benzene rings (Maroni et al., 1995). Once airborne, PAH compounds can be absorbed onto particles and inhaled into the lungs. It is thought that their carcinogenic properties arise as a result of their metabolism within the body (Sisovic et al., 1996). Evidence for this comes from studies such as that undertaken by Mumford et al. (1995). Amongst residents of Xuan Wei county, China, they found that urinary concentrations of PAH metabolites were highest in villages with correspondingly elevated lung cancer death rates.

## 2.9. Tobacco smoke

Although the health effects of smoking have been recognised for a number of decades, it is only relatively recently that concerns have been directed towards the inhalation of environmental tobacco smoke (ETS) by the non-smoking population. Tobacco smoke is an aerosol containing several thousand substances that are distributed as particles, vapours, and gasses (Gold, 1992). Environmental tobacco smoke is possibly one of the most important indoor air pollutants in homes and offices (Wanner, 1993). This is because a substantial proportion of the population are exposed to high concentrations of ETS on a regular basis. In 1990, it was estimated that approximately 50 million US citizens (corresponding to 26% of the adult population) were smokers (Rando et al., 1997), and around 70% of all children may be living in homes with at least one parent who smokes (Weiss, 1986).

The constituents of ETS are commonly sub-divided into those associated with sidestream and mainstream smoke. Mainstream (MS) smoke is that which is directly exhaled from the smoker, whilst sidestream smoke (SS) is emitted from the smouldering tobacco between puffs

Table 5  
Emission factors for mainstream and sidestream smoke

Substance	Mainstream ( $\mu\text{g}$ per cigarette)	SS/MS ratio	Calculated sidestream ( $\mu\text{g}$ per cigarette) <sup>a</sup>
Carbon dioxide	10,000–80,000	8.1	81,000–640,000
Carbon monoxide	500–26,000	2.5	1200–65,000
Oxides of nitrogen	16–600	4.7–5.8	80–3500
Ammonia	10–130	44–73	400–9500
Hydrogen cyanide	280–550	0.17–0.37	48–203
Formaldehyde	20–90	51	1000–4600
Acrolein	10–140	12	100–1700
<i>N</i> -nitrosodimethylamine	0.004–0.18	10–830	0.04–149
Nicotine	60–2300	2.6–3.3	160–7600
Total particulate	100–40,000	1.3–1.9	130–76,000
Phenol	20–150	2.6	52–390
Catechol	40–280	0.7	28–196
Naphthalene	2.8	16	45
Benzo(a)pyrene	0.008–0.04	2.7–3.4	0.02–0.14
Aniline	0.10–1.20	30	3–36
2-Naphthylamine	0.004–0.027	39	0.02–1.1
4-Aminobiphenyl	0.002–0.005	31	0.06–0.16
<i>N</i> -nitrosonornicotine	0.2–3.7	1–5	0.02–18

Source: Rando et al. (1997).

<sup>a</sup>Calculated from ratio of SS to MS.

(Maroni et al., 1995). As information has become known about mainstream and sidestream smoke generation and its chemical composition, many irritants and carcinogens have been identified (Rando et al., 1997). By inhaling ETS, non-smokers are exposed to most of the toxins inhaled by active smokers, as well as some additional substances. For example, *N*-nitrosodimethylamine, a potent animal carcinogen, is emitted in quantities 20–100 times higher in SS than in MS smoke (Guerin et al., 1992), and a 'passive' smoker at a distance of 50 cm from a cigarette may inhale more than 10 times the amount of carbonyl compounds actively taken up by the smoker (Schlitt and Knöppel, 1989).

Some of the more common compounds found in ETS are outlined in Table 5. However, the exact composition of the smoke is known to be very dependent upon the type of tobacco being consumed, its packing density, the composition of the wrapping paper, and the puffing rate of the smoker (Hines et al., 1993).

Because of the complex nature of ETS, measurements of respirable particles are often used as indicators of indoor concentrations. Reported cigarette smoke particle sizes have varied in the range from 0.1 to 1.5  $\mu\text{m}$  (Chen et al., 1990). Spengler et al. (1981) observed respirable particulate concentrations in 80 homes over several weeks. They found that a smoker of one pack a day contributed around 20  $\mu\text{g m}^{-3}$  to 24 h indoor concentrations. However, because cigarettes are not uniformly smoked throughout the day, the authors concluded that short-term

particulate concentrations of 500–1000  $\mu\text{g m}^{-3}$  were likely when cigarettes were actually ignited. Over a period of 3 months, Leaderer et al. (1990) investigated indoor aerosol mass concentrations in a sample of almost 400 houses. They found that homes with smokers exhibited mass concentrations that were approximately three times higher than those observed in non-smoking residences.

As well as being an important pollutant of the domestic environment, ETS can be a serious problem in the workplace. In a report on tobacco smoke discomfort published in the USA (Anonymous, 1992) 43.5% of non-smoking employees recounted at least some discomfort from ETS at their place of work. Occupational groups particularly at risk from ETS include those such as bartenders and waiters who work in a traditionally smoky environment. Lambert et al. (1993b) recorded a median respirable concentration of 53.2  $\mu\text{g m}^{-3}$  in a study of 7 restaurants, whilst a median concentration of 355  $\mu\text{g m}^{-3}$  has been noted in a billiard lounge (Rando et al., 1997).

The most common acute health effects associated with exposure to ETS are eye, nose, and throat irritation (Maroni et al., 1995). For some people, eye tearing can be so intense as to be incapacitating. Smoke also contains substances that can activate the immune system, and approximately half of allergy prone individuals react to various extracts of tobacco leaf or smoke in skin tests (Maroni et al., 1995).

Exposure to ETS has been particularly associated with the exacerbation of symptoms in asthmatics (Jones,

1998). In Italy, parental smoking and the self-reported prevalence of asthma symptoms was examined amongst 3239 children by Forastiere et al. (1992). They found that the prevalence of asthma symptoms was significantly elevated in children whose mother smoked. Similarly, Gortmaker et al. (1982) found the prevalence of severe asthma was 2.2% amongst a sample of children with a smoking parent, compared to only 1.1% amongst children whose parents did not smoke. There is some evidence that exposure to ETS may be important in actually inducing the onset of asthma in very young children. On the Isle of Wight, UK, Arshad et al. (1992) reported an odds ratio for asthma at 12 months of 3.33 (95% CI 0.8–14.6) if one parent smoked, compared to 11 (95% CI 2.5–48.2) if both were smokers.

Strong evidence is available for establishing a link between acute childhood lower respiratory tract illnesses and exposure to ETS at home (Somerville et al., 1988). Furthermore, passive smoking may have a significant effect on the level and growth of lung function in children (NRC, 1986) and the onset of chronic obstructive pulmonary disease in later life. Smoke-exposed children also have increased rates of hospitalisation; as far back as 1974, Harlap and Davies observed elevated rates of hospital admission for bronchitis, pneumonia, and bronchiolitis amongst the infants of smokers (Harlap and Davies, 1974). An important current field of research is investigating whether the greatest effects of environmental tobacco smoke on the lung are established pre-birth, or are associated with exposures during the first few years of life (Gold, 1992).

The strongest evidence for the carcinogenic effects of ETS comes from research into cases of lung cancer. Based on the results from a case-control study of 191 individuals in the USA, Janerich et al. (1990) concluded that approximately 17% of lung cancers amongst non-smokers can be attributed to exposure to ETS during early life. The risks associated with exposure appear to be particularly high amongst women; a multi-centre national study of lung cancer in lifetime non-smokers found an increased cancer risk of approximately 30% in women whose husbands smoked (Rando et al., 1997). Although it is difficult to determine causality, there is evidence that cancer in sites other than the lung may be associated with passive smoking. Fukuda and Shibata (1990) discovered an association between ETS exposure and cancer of the sinus, and a relationship with the incidence of brain tumours has also been reported (Ryan et al., 1992).

## 2.10. Volatile organic compounds

Any chemical compound that contains at least one carbon and a hydrogen atom in its molecular structure is referred to as an organic compound. Organic compounds are further classified into various categories which include volatile organic compounds (VOCs), semi-volatile

organic compounds (SVOCs) and non-volatile organic compounds (NVOCs). Volatile organic compounds are defined as having a boiling point that ranges between 50°C and 260°C (Maroni et al., 1995). Their low boiling point means that they will readily off-gas vapours into indoor air. However, the fact that materials containing them exhibit the desirable characteristics of good insulation properties, economy, fire-resistance, and ease of installation, means that their use in construction projects is widespread (Burton, 1997).

Table 6 lists sources of common VOCs found indoors. Out of a total of more than 900 chemical and biological substances that have been identified in indoor air, more than 350 VOCs have been recorded at concentrations exceeding 1 ppb (Brooks et al., 1991). The typical concentrations of some of the most frequently encountered compounds in the home are summarised by Table 7.

Indoor concentrations of VOCs have been well quantified; they are mostly considerably below the odour threshold but often exceed outdoor levels by up to 5 times (Wallace, 1991a). One of the earliest studies was undertaken by Mølhave (1979). He targeted 29 chemicals in 14 office buildings in Denmark. Most VOCs identified were alkylbenzenes and had concentrations in the range of 0.03–2.8  $\mu\text{g m}^{-3}$ . Shah and Singh (1988) found that levels of the majority of a sample of 66 indoor VOCs ranged from 0.4 to 4  $\mu\text{g m}^{-3}$  and Brown et al. (1994) reported that concentrations of most VOCs they studied were below 5  $\mu\text{g m}^{-3}$ .

In any given environment, the concentration of individual VOCs will be very variable and depend upon the presence or absence of an extremely wide range of potential emission sources. Hence, many publications report levels of 'Total Volatile Organic Compounds' (TVOCs) rather than individual values, although this of little help in determining the toxicological properties of substances (ECJRC, 1997). A large study of TVOC concentrations in a sample of 179 UK homes found that the mean concentration of all readings in the rooms measured was 200–500  $\mu\text{g m}^{-3}$  (Brown and Crump, 1996). The maximum recorded concentration was 11,401  $\mu\text{g m}^{-3}$  in a living room. Mean concentrations were highest in main bedrooms, and lowest in second bedrooms. Similar findings have been reported by research in Denmark (Wolkoff et al., 1991), Germany, (Adlkofer et al., 1993), Sweden (Norbäck et al., 1993), and the USA (Hartwell, 1987).

It is important to note that average VOC concentrations may give an inaccurate indication of personal exposures (Rodes, 1991). This is because individuals are often situated close to emission sources, and may also be subjected to pollutants emitted from substances on their person. As an illustration of this, Wallace (1991a) reported the results of measurements of personal exposure to 25 VOCs among 51 Los Angeles Residents. Whilst ambient indoor maxima ranged from 10 to 100  $\mu\text{g m}^{-3}$ ,

Table 6  
Sources of common volatile organic compounds in indoor air

Sources	Examples of typical contaminants
Consumer and commercial products	Aliphatic hydrocarbons ( <i>n</i> -decane, branched alkanes), aromatic hydrocarbons (toluene, xylenes), halogenated hydrocarbons (methylene chloride), alcohols, ketones (acetone, methyl ethyl ketone), aldehydes (formaldehyde), esters (alkyl ethoxylate), ethers (glycol ethers), terpenes (limonene, alpha-pinene).
Paints and associated supplies	Aliphatic hydrocarbons ( <i>n</i> -hexane, <i>n</i> -heptane), aromatic hydrocarbons (toluene), halogenated hydrocarbons (methylene chloride, propylene dichloride), alcohols, ketones (methyl ethyl ketone), esters (ethyl acetate), ethers (methyl ether, ethyle ether, butyl ether).
Adhesives	Aliphatic hydrocarbons (hexane, heptane), aromatic hydrocarbons, halogenated hydrocarbons, alcohols, amines, ketones (acetone, methyl ethyl ketone), esters (vinyl acetate), ethers.
Furnishings and clothing	Aromatic hydrocarbons (styrene, brominated aromatics), halogenated hydrocarbons (vinyl chloride), aldehydes (formaldehyde), ethers, esters.
Building materials	Aliphatic hydrocarbons ( <i>n</i> -decane, <i>n</i> -dodecane), aromatic hydrocarbons (toluene, styrene, ethylbenzene), halogenated hydrocarbons (vinyl-chloride), aldehydes (formaldehyde), ketones (acetone, butanone), ethers, esters (urethane, ethylacetate).
Combustion appliances	Aliphatic hydrocarbons (propane, butane, isobutane), aldehydes (acetaldehyde, acrolein).
Potable water	Halogenated hydrocarbons (1,1,1-trichloroethane, chloroform, trichloroethane).

Source: Maroni et al., 1995.

Table 7  
Concentration ( $\mu\text{g m}^{-3}$ ) and distribution of selected VOCs found in indoor air

Pollutant	Concentration				Mean
	Percentile				
	10th	50th	90th	98th	
Benzene	2	10	20	30	10
Toluene	30	65	150	250	80
<i>n</i> -Decane	3	10	50	90	20
Limonene	2	15	70		30
<i>o</i> -Xylene	3	5	10		10
1,1,1-Trichloroethane	2	5	20		10
<i>p</i> -Dichlorobenzene	1	5	20		
1,2,4-Trimethylbenzene		5	20		10
<i>m</i> - and <i>p</i> -Xylene	10	20	40		20
Undecane	3	5	25		10
1,3,5-Trimethylbenzene		2	5		5
Dichloroethane		< 10	< 10	600	
Trichloroethane	1	5	20	30	

Source: IEH, 1996.

personal maxima were generally between 100 to 1000  $\mu\text{g m}^{-3}$ . VOC concentrations may be much higher than typical ambient levels in newly constructed buildings, or those in which building work or decoration has recently taken

place. This is because many VOCs will off-gas a significant proportion of their volume in a relatively short time, and hence their concentrations will decline rapidly and exponentially. Because of this, builders and decorators may receive particularly high doses (Wieslander et al.,

1997). For example, Wallace et al. (1991), found that breath concentrations of decane in a subject being studied increased by a factor of 100 (from 2.9–290  $\mu\text{g m}^{-3}$ ) after they were engaged in painting and the use of solvents.

Exposure to VOCs can result in both acute and chronic health effects. It is possible that asthmatics and other individuals with prior respiratory complaints may be particularly susceptible to low-dose VOC exposures. In a recent study, Norbäck et al. (1995) reported a positive association between levels of VOCs and the prevalence of nocturnal breathlessness amongst 88 Swedish asthmatics aged between 20–45 yr. However, most information on VOC toxicity has been established from animal and experimental studies at high concentrations, as levels in the majority of indoor environments are well below those required to demonstrate measurable health impacts.

At high concentrations, many VOCs are potent narcotics, and can depress the central nervous system (Maroni et al., 1995). Exposures can also lead to irritation of the eyes and respiratory tract, and cause sensitisation reactions involving the eyes, skin, and lungs. Møhlave (1991) reported complaints of unpleasant mucous membrane irritation amongst individuals exposed to a mix of 22 VOCs at a concentration of 8  $\mu\text{g m}^{-3}$ . Because of the similarity of these symptoms, exposure to VOCs has frequently been attributed as a cause of sick building syndrome, discussed later in this text. To back this up, a number of studies have reported a strong association between mucous membrane irritation, central nervous system symptoms, and total exposure to VOCs amongst office workers (Hodgson et al., 1991).

At extreme concentrations, some VOCs may result in impaired neurobehavioral function (Burton, 1997). In an experimental study, Otto et al. (1992) noted that subjects exposed to a concentration of 22 VOCs at 25  $\mu\text{g m}^{-3}$ , reported symptoms of headache, drowsiness, fatigue, and confusion. At concentrations as high as 188  $\mu\text{g m}^{-3}$ , VOCs such as toluene may cause symptoms of lethargy, dizziness, and confusion. These may progress to coma, convulsions, and possibly death at levels in excess of 35,000  $\mu\text{g m}^{-3}$  (Sandmeyer, 1982). However, these concentrations have never been recorded in the non-industrial environment.

Exposure to high concentrations of several VOCs commonly found in indoor air have been associated with cancers in laboratory animals. Based on laboratory evidence, Wallace (1991b) estimated that typical concentrations of seven VOCs exceeded the  $1 \times 10^{-6}$  risk of cancer by at least a factor of 10. These include benzene, vinylidene chloride, *p*-dichlorobenzene, chloroform, ethylene dibromide, methylene chloride and carbon tetrachloride. However, it should be noted that many risk calculations involve an assumption of a linear relationship of the dose–response curve when extrapolating from

high to low exposures, and hence they are subject to much uncertainty (Stolwijk, 1991).

One recent theory proposes that the products of chemical reactions involving VOCs may be more important than direct exposure to the VOCs themselves (Wolkoff et al. 1997). This proposition is based on the fact that the majority of sick building syndrome studies have recorded concentrations of VOCs at considerably lower levels than those required to induce symptoms. Whilst there is an assumption here that sick building syndrome must be associated with exposure to VOCs, there is increasing evidence that many chemical reactions take place on surfaces and in air in the indoor environment (Reiss et al., 1995). Results from epidemiological research suggests that reactions between indoor ozone and VOCs may produce irritant substances that could cause SBS symptoms (Groes et al., 1996). Reactions involving nitrogen dioxide (Grosjean et al., 1992), and particles (Schneider et al., 1994) may also be important.

### 3. Pollution from biological sources

Whilst discussions of indoor air pollution frequently concentrate on chemical pollutants, the health effects of inhaled biological particles should not be overlooked, as a large variety of biological material is present in indoor environments (Montanaro, 1997). Biological agents can cause disease through atopic mechanisms, infectious processes, or direct toxicity. House dust in carpets, on sofas, and in air ducts is a major source of a range biological allergens (Lewis et al., 1994). The growth of moulds is not only aesthetically unpleasant, but can pose serious health problems, and some indoor environments provide ideal conditions for the maintenance of populations of harmful viruses and bacteria. The health effects of these agents are discussed below.

#### 3.1. Indoor biological allergens

Table 8 summarises the main biological allergens found in indoor air. Probably the best studied source of indoor allergens is the house dust mite. In temperate climates, ten species of mites can be found from the genera *Dermatophagoides*, *Euroglyphus*, *Malayoglyphus*, *Hirstia*, and *Sturnophagoides* (D'Amato et al., 1994). *D. pteronyssinus* and *E. Maynei* mites appear to comprise around 95% of all mites found in a typical house (Hart and Whitehead, 1990). In tropical climates the mite *Blomia tropicalis* is also commonly found. Undoubtedly the most researched mite is the common house dust mite *D. pteronyssinus*. They measure 250–350  $\mu\text{m}$  in size, and grow from egg to adult in around 25 d (D'Amato et al., 1994). Most mite populations prefer constant temperatures of around 25°C and a relative humidity of between 70 and 80% (Arlain et al., 1990; Salerno et al., 1992). They

Table 8  
Common indoor allergenic agents

Source	Genus	Species	Allergen
Dust-mite	<i>Dermatophagoides</i>	<i>pteronysinus</i>	<i>Der p</i>
	<i>Dermatophagoides</i>	<i>farinae</i>	<i>Der f</i>
	<i>Euroglyphus</i>	<i>maynei</i>	<i>Eur m</i>
	<i>Hirstia</i>	<i>domicola</i>	<i>Hir d</i>
	<i>Lepidoglyphus</i>	<i>destructor</i>	<i>Lep d</i>
	<i>Malayoglyphus</i>	<i>intermedius</i>	<i>Mal I</i>
	<i>Malayoglyphus</i>	<i>carmelitus</i>	<i>Mal C</i>
	<i>Sturnophagoides</i>	<i>brasiliensis</i>	<i>Stu b</i>
Cat	<i>Felis</i>	<i>domesticus</i>	<i>Fel d</i>
Dog	<i>Canis</i>	<i>familiaris</i>	<i>Can f</i>
Rodent	<i>Mus</i>	<i>musculus</i>	<i>Mus m</i>
	<i>Rattus</i>	<i>norvegicus</i>	<i>Rat n</i>
Cockroach	<i>Blattella</i>	<i>germanica</i>	<i>Bla g</i>
	<i>Periplanetta</i>	<i>americana</i>	<i>Per a</i>
Fungi	<i>Alternaria</i>	<i>alternata</i>	<i>Alt a</i>
	<i>Aspergillus</i>	<i>fumigatus</i>	<i>Asp f</i>
	<i>Cladosporium</i>	<i>herbarium</i>	<i>Cla h</i>

do not survive well in cool, dry conditions. Mites inhabit a range of soft furnishings, including sofas, fabrics, carpets, sheets, duvets, pillows, and mattresses.

The droppings from the mite are a primary source of indoor antigens (Kaliner and White, 1994). Mite faeces are encased in a coating of intestinal enzymes including a protein which is a strong allergen. Although over 10 different antigens have been characterised for *D. pteronyssinus* so far, the first to be identified, *Der p* 1, is the most commonly reported.

There may be up to 100,000 mite faecal particles, ranging between 10 and 40  $\mu\text{m}$  in diameter, in a gram of house dust (Platts-Mills et al., 1991). The size of the particles, similar to that of pollen grains, means they do not remain airborne for long, although concentrations in disturbed rooms can be more than 1000 times higher than those observed in undisturbed environments (Kalra et al., 1990). A concentration of mite allergen above 2  $\mu\text{g Der p 1 g}^{-1}$  (equivalent to 100 mites) of dust appears to represent a significant factor for mite sensitisation (WHO, 1995), and 10  $\mu\text{g Der p 1 g}^{-1}$  increases the risk of triggering an acute or severe asthma attack in mite allergic individuals (IEH, 1996).

Typical *Der p* 1 concentrations are below 5  $\mu\text{g g}^{-1}$  of dust (Verhoeff, 1994). However, the actual concentration of mite antigen in a home will strongly depend upon a range of factors, and is particularly influenced by climatic variables. This is illustrated by the work of Friedman et al. (1992). They reported mean concentrations of *Der p* 1 and *Der f* 1 as being 3.98 and 8.17  $\mu\text{g g}^{-1}$  of dust in a sample of 15 New England homes during the month of June. In September the corresponding mean concentrations had risen to 21.43 and 14.53  $\mu\text{g g}^{-1}$ .

Given that inhalation is the main route of exposure for the majority of allergens in indoor air, it is unsurprising that most research has concentrated on the effects on atopic (allergic) respiratory diseases, and in particular asthma. Over 70 yr ago, Ancona (1923) made the first documented observation that mites may be a cause of respiratory symptoms. Today, the evidence suggests that exposure to airborne mite allergen will exacerbate symptoms in up to 85% of asthmatics (Platts-Mills and Carter, 1997). Studies of the effects of dust mite allergen exposure are numerous. Recently, Björnsson et al. (1995) reported that Swedish subjects inhabiting homes with large mite populations were more likely to report nocturnal breathlessness and other asthma-related symptoms. Peat et al. (1994) also found an association between exposure to dust mite *Der p* 1 and wheezing in the previous year amongst Australian children.

As well as being a major trigger of attacks of asthma, there is some evidence that, particularly amongst infants, exposure to mite allergen may induce the onset of the condition in previously healthy individuals (Tariq et al., 1998). In an important investigation, Arshad et al. (1992) studied the role of early exposure to dust-mite allergens in the development of allergic disorders amongst infants on the Isle of Wight. One hundred and twenty infants were randomly allocated to prophylactic and control groups. In the prophylactic group, the infants' bedrooms and living rooms were treated to control mite populations. In the control group, there were no interventions. After 12 months, the odds ratio for asthma was 4.13 (95% CI 1.1–15.5) amongst the control group. After 24 months, the excess number of cases amongst the control group had fallen, but still remained (Hide et al., 1994).

Like dust mites, cockroaches have been associated with manifestations of symptoms in individuals with allergic asthma, and up to 60% of asthmatics test positive to cockroach allergen in the USA (Kuster, 1996). Although cockroaches are generally indigenous to warm tropical climates, some species are able to thrive elsewhere due to the presence of central heating. The most commonly found cockroach in homes in Europe is the German Cockroach, *Blattella Germanica*, from which at least 5 antigens have been isolated (*Bla g* 1–5). In the USA, the American Cockroach, *Periplaneta americana* is ubiquitous. Sources of cockroach allergens have been identified in body parts, as well as faecal extracts (Musmand et al., 1995).

Van Wijnen et al. (1997) studied concentrations of *Bla g* 1 allergen in 46 homes in Amsterdam. It was detected in over 44% of homes, with mean concentrations ranging between 1.3 and 11  $\text{ng g}^{-1}$  of dust. The highest level recorded in a single sample was 3899  $\text{ng g}^{-1}$ . In general, concentrations were elevated in rooms with textile floor coverings. Similar findings were made in the USA by Rosenstreich et al. (1997). They measured concentrations of the allergen in dust taken from 476 homes situated in



various inner-city locations. Interestingly, they found that asthmatic children allergic to cockroaches were three times more likely to be hospitalised for their asthma if they lived in a home with a large cockroach population. The authors concluded that the problems of cockroach sensitisation might be particularly severe amongst the residents of poor quality inner-city housing, as these homes provide an ideal environment for cockroach populations.

Domestic cats and dogs are a further important source of allergens in indoor air. This is particularly so in colder climates, which are less favourable to dust mites. Cat allergen (*Fel d*) is found in saliva, skin and dander. Particles of the primary cat allergen, *Fel d* 1, are often small, and hence remain airborne for many hours (Luczynska et al., 1990). *Fel d* 1 concentrations in domestic dust can exceed  $10 \mu\text{g g}^{-1}$  and airborne levels can vary between 2 and  $20 \text{ ng m}^{-3}$  (Luczynska, 1994). As with cat allergens, dog allergens are mostly found in saliva and dander. The major dog allergen has been termed *Can f* 1 (Montanaro, 1997). Concentrations of *Can f* 1 in dust are often similar to *Fel d* 1, although *Fel d* 1 can remain airborne longer due to its smaller particle size (Puerta et al., 1997).

Because *Fel d* and *Can f* are readily transported by becoming attached to clothing, exposure to these allergens can even be a problem in public places such as schools, trains, and hospitals, where pets are not allowed. Custovic et al. (1998) recently examined concentrations of mite, cockroach, dog, and cat allergens in a sample of 14 British hospitals. Although levels of mite and cockroach allergen were low, high concentrations of *Fel d* 1 (Mean  $22.9 \mu\text{g g}^{-1}$ , range 4.5–58) and *Can f* 1 (Mean  $21.6 \mu\text{g g}^{-1}$ , range 4–63) were found in upholstered chairs. Out of a total of 10 sampling days, airborne *Can f* 1 was detected on every occasion (range 0.12–0.56  $\text{ng m}^{-3}$ ), whilst airborne *Fel d* 1 was recorded on 7 d (range 0.09–0.22  $\text{ng m}^{-3}$ ).

Recently, it has been proposed that concentrations of dog and cat allergens over  $8 \mu\text{g g}^{-1}$  in house dust represent a threshold for allergic sensitisation (D'Amato et al., 1994). Because of the generally small particle size, dog and cat allergen readily enters the lung and can produce a rapid and severe asthmatic response. For example, Pollart et al. (1991) found 30 out of 188 asthmatic patients admitted to a hospital emergency department were allergic to cats, compared to only 1 out of 202 control asthmatics who did not require emergency treatment.

### 3.2. Fungi, bacteria and viruses

Microorganisms are an important form of biological pollution in the indoor environment. A large number of species of fungi and bacteria are found indoors, where they are associated with the presence of organic matter (e.g. wall coatings, wood, foodstuffs) (IEH, 1996). The

outdoor air is one of the major sources of fungi and bacteria in indoor environments, particularly during the summer and autumn (Wanner et al., 1993). It is also well documented that high levels of humidity favour fungal growth (Sterling and Lewis, 1998). They are frequently found in homes that contain damp conditions, especially those with structural faults, or basements and under-floor crawl spaces (Montanaro, 1997).

A major difficulty to the sampling of viable airborne fungi is the large variability with time, even over short periods (IEH, 1996). A particular problem arises as temporal variations in airborne concentrations are often much greater in magnitude than variations between homes (Flannigan and Miller, 1994). Despite these difficulties, indoor concentrations of bacteria and fungi have been relatively well studied.

In the UK, Hunter et al. (1996) used air filtration to monitor 163 homes for the presence of fungi and bacteria over the period November 1990 to December 1992. The geometric mean count was 234 colony forming units  $\text{CFU m}^{-3}$  air for fungi, and 365.6  $\text{CFU m}^{-3}$  air for bacteria. In a more intensive study of 35 of the houses, mean counts were 912 and 818  $\text{CFU m}^{-3}$  air for fungi in living rooms and bedrooms respectively, compared to 917 and 933  $\text{CFU m}^{-3}$  air for bacteria. *Penicillium* was the most frequently isolated fungus, found in 53% of samples. It was followed by *Cladosporium* and *Aspergillus*. The dominant bacteria were *Bacillus*, followed by *Staphylococcus* and *Micrococcus*. In Norway, Dotterud et al. (1995) found that *Penicillium* was the most common microfungus in homes and schools, followed by *Aspergillus*, *Cladosporium*, and *Mucor*. In a study in Japan, Takahashi (1997) reported that concentrations of fungal CFU ranged from between 13 to 3750  $\text{CFU m}^{-3}$  in samples of air from indoor environments. They also found that concentrations were strongly correlated with indoor temperature and relative humidity, as well as outdoor climatic factors. Macher et al. (1991) measured rather less extreme spore concentrations, of the order of 198  $\text{CFU m}^{-3}$ , in an apartment in the USA.

Exposure to airborne bacteria and fungi is associated with a number of well-defined diseases, as well as various less well-defined symptoms (Peat et al., 1998). Table 9 summarises some of the main reported health impacts. Although many studies use indoor dampness as a surrogate measure of microorganism concentrations, some direct measurements have been attempted. In the UK, Platt et al. (1989) examined the relationship between fungal spore counts and self-reported symptoms amongst the occupants of almost 600 homes. They found higher numbers of  $\text{CFU m}^{-3}$  were associated with an elevated prevalence of reported wheeze and fever in children, and high blood pressure, and breathlessness in adults. Also in the UK, Potter et al. (1991) detected allergic responses to nine different fungal allergens in a survey of 2000 patients with allergic respiratory disease. In Sweden, Wickman

Table 9

Diseases and disease syndromes associated with exposure to bacteria and fungi

Disease/Syndrome	Examples of causal organisms cited
Rhinitis (and other upper respiratory symptoms)	<i>Alternaria</i> , <i>Cladosporium</i> , <i>Epicoccum</i>
Asthma	Various aspergilli and penicillia, <i>Alternaria</i> , <i>Cladosporium</i> , <i>Mucor</i> , <i>Stachybotrys</i> , <i>Serpula</i> (dry rot)
Humidifier fever	Gram-negative bacteria and their lipopolysaccharide endotoxins, Actinomycetes and fungi
Extrinsic allergic alveolitis	<i>Cladosporium</i> , <i>Sporobolomyces</i> , <i>Aureobasidium</i> , <i>Acremonium</i> , <i>Rhodotorula</i> , <i>Trichosporon</i> , <i>Serpula</i> , <i>Penicillium</i> , <i>Bacillus</i>
Atopic dermatitis	<i>Alternaria</i> , <i>Aspergillus</i> , <i>Cladosporium</i>

Source: IEH, 1996.

et al. (1992) found that *Penicillium*, *Alternaria*, and *Cladosporium* moulds were more common in homes of children with allergies, and Neas et al. (1996) reported that morning lung function was inversely associated with *Epicoccum* and *Cladosporium* spore concentrations amongst a panel of 108 children. Contrary to many findings, Verhoff et al. (1994) were unable to find any relationship between microbial contamination and the directly measured pulmonary function of a sample of children living in 60 homes in the Netherlands. Although levels of CFU were higher in dust from mattresses in damp homes, they were not associated with objective measures of lung function amongst the infants.

Interestingly, the most recent research on moulds and fungi suggests that allergic reactions may not be the most important factor in the development of respiratory symptoms associated with exposure to spores (Howden-Chapman et al., 1996). Mycotoxins are toxic compounds produced naturally by many fungi (Hendry and Cole, 1993). They induce a wide range of acute and chronic systemic effects that cannot be attributed to fungal growth within the host. Samson et al. (1994) provide evidence that mycotoxins emitted by fungi are readily absorbed through the membranes of the respiratory tract, and it could be that their presence in the lung affects the immune system, precipitating the onset of symptoms.

Most bacterial and viral infections that spread within buildings are transmitted from human to human by airborne droplets (Ayars, 1997). More often than not, the building is an 'innocent bystander' and plays no role in harbouring populations of infectious agents, other than by providing a living environment for infected individuals. However, there are examples of serious bacterial and viral infections where the building itself can act as a potential reservoir (Burrell, 1991).

Probably the best-known infection associated with the indoor environment is Legionnaire's disease. It is caused by the bacteria *Legionella pneumophila*. It was first recognised at a legionnaire's convention in Philadelphia in

1976. One hundred and eighty four legionnaires attending the symposium developed symptoms of the disease, and 29 died (Fraser et al., 1977). The disease has an incubation period of 2–12 d, and an attack rate of 1–7%. Typical symptoms include malaise and headache, followed by dry cough, chest pain, diarrhoea, and altered mental status (Ayars, 1997). Since that epidemic, numerous other outbreaks and a host of other *Legionella* species have been identified (Hanrahan et al., 1987).

*Legionella* bacteria thrive in a warm and humid environment, and a common denominator in most outbreaks is a source of water in or around the building. Typical reservoirs of the bacteria include air humidifiers, air conditioning cooling towers, warm water supplies, shower heads and plumbing systems (Hines et al., 1993). The mechanisms by which *Legionella* reach the lungs were, until recently, thought to be via the inhalation of aerosols. However, recent evidence suggests that many cases may be associated with the aspiration of potable water (Yu, 1993).

An influenza illness known as Pontiac fever has also been identified, and a number of outbreaks have been documented. It is caused by *Legionella* organisms, but is more benign than Legionnaire's disease (Glick et al., 1978). Q fever is another bacterial infection that can spread in buildings independent of humans (Ayars, 1997), and the bacterium that causes tuberculosis can also be transmitted via air-management systems in closed environments. One of the most studied examples occurred in 1965 aboard a US naval vessel when 7 crew members developed tuberculosis that was spread via the ship's ventilation system (Houk et al., 1968).

Although outbreaks of bacterial infections are more commonly associated with indoor environments, there are also a number of serious viral diseases that can be harboured within buildings. Included are the often deadly but rare viruses that produce haemorrhagic fevers such as the Marburg virus, Ebola virus, and Lassa fever (Ayars, 1997). Many of these agents appear to be spread by aerosolised animal products, particularly urine, and

Table 10  
Common symptoms of sick building syndrome

- 
- Headache and nausea
  - Nasal congestion (runny/stuffy nose, sinus congestion, sneezing)
  - Chest congestion (wheezing, shortness of breath, chest tightness)
  - Eye problems (dry, itching, tearing, or sore eyes, blurry vision, burning eyes, problems with contact lenses)
  - Throat problems (sore throat, hoarseness, dry throat)
  - Fatigue (unusual tiredness, sleepiness, or drowsiness)
  - Chills and fever
  - Muscle pain (aching muscles or joints, pain or stiffness in upper back, pain or stiffness in lower back, pain or numbness in shoulder/neck, pain or numbness in hands or wrists)
  - Neurological symptoms (difficulty remembering or concentrating, feeling depressed, tension, or nervousness)
  - Dizziness
  - Dry skin
- 

Source: Wallace (1997).

hence may pose particular problems in buildings with rodent infestations.

#### 4. Sick building syndrome

Over the past two decades, numerous field studies on indoor air quality and the sick building syndrome (SBS) have been conducted, mostly in office environments. The symptoms of SBS are usually non-specific, and are often somewhat particular to the building being occupied by the workers (Table 10). Amongst employees, symptoms often worsen during working hours, and lessen or disappear after leaving the building. It may be that SBS does not warrant separate attention from the rest of the discussion of indoor air pollutants, as it is unlikely that its mechanisms are much different from those of some well defined pollution problems. However, the study of cases of SBS provides a valuable insight into the problems faced by investigators attempting to find evidence of causal relationships between any indoor air pollution and health.

For a time, exposure to VOCs was thought to be the major cause of SBS. This view was based on the results of experimental chamber studies in which persons exposed to mixtures of VOCs exhibited symptoms commonly associated with SBS (Mølhave et al., 1986). Some epidemiological studies have also supported the hypothesis that VOCs may be important. In a longitudinal study of a 'sick' library building, Berglund et al. (1990) found an association between the reporting of SBS symptoms and temporal variations in VOC concentrations. In a cross-sectional study of SBS symptoms amongst 147 office workers, Hodgson et al. (1991) found that VOC concentrations in the breathing zone of the building occupants were good predictors of mucous membrane irritation, and central nervous system complaints. However, despite these findings, a large number of studies have been un-

able to find any association between VOC exposures and SBS outbreaks. Indeed, one recent large-scale project actually reported a negative relationship (Sundell et al., 1993). Therefore it appears that, in many cases, VOCs may not be primarily to blame.

Some attention has recently been focused on the role that building ventilation systems may play in SBS (Bourbeau et al., 1997). There is much debate as to whether ventilation may actually be a cure or cause of SBS. Certainly, hot stuffy air is a common complaint in SBS studies, suggesting that efficient ventilation is desirable (Wallace, 1997). However, the exact role of building ventilation in this phenomenon is not well understood, and the results from studies that have been undertaken are often conflicting (Mendell, 1994). Although many previous reports have found reduced symptom prevalence to be associated with increased outside air ventilation (e.g. Nagda et al., 1991; Sundell et al., 1993) a more recent double-blind experiment by Jaakkola et al. (1994) found no significant difference in symptom prevalence amongst the occupants of buildings with ventilation rates between 13 and 42 cfm  $p^{-1}$  of outside air.

In an important meta-analysis of six previous studies, Mendell and Smith (1990) examined the relationship between symptom reporting and ventilation provision amongst office workers. Compared with those in naturally ventilated buildings, workers in air-conditioned offices consistently reported an increased prevalence of work related headache (OR = 1.3–3.1), lethargy (OR = 1.4–5.1), and upper respiratory/mucus membrane symptoms (OR = 1.3–4.8). Interestingly, the provision of mechanical ventilation without air conditioning was not associated with higher symptom prevalence.

The fact that some ventilation systems may themselves contribute to the concentrations of indoor pollutants is a possible explanation for the finding of Mendell and Smith (1990). Sources might include bacterial contamination, fungal mycotoxins, antiseptic agents and pesticides,

or VOCs from air filtration systems or maintenance ducts. Harrison et al. (1987) found that, compared with the inhabitants of 8 naturally ventilated buildings, the occupants of 19 buildings with mechanical ventilation reported significantly higher frequencies of eye and nose irritation, headaches, attacks of lethargy, and dry skin. However, a number of more recent studies (e.g., Sundell, 1994) have failed to confirm these findings, and whilst recent research by Vincent et al. (1997) did find some evidence of an association between the mode of building ventilation and SBS symptoms amongst Parisian office workers, the authors concluded that ventilation only explained a relatively small proportion of non-specific symptoms.

Despite the voluminous research undertaken over the last two decades to investigate the reasons for SBS outbreaks, it has been estimated that no specific cause has been identified in over 75% of cases (Rothman and Weintraub, 1995). This may, in part, be due to methodological considerations. One problem is that currently available pollutant sampling techniques are costly and suffer from some technical limitations. This has often meant that only relatively limited data on the actual concentrations of pollutants that may be important in SBS have been available (Bardana, 1997). This is particularly the case for studies of VOCs, where many investigations have measured concentrations of only a defined subset of a limited number of compounds. Potentially important VOCs such as aldehydes and carboxylic acids of low molecular weight are rarely analysed quantitatively (Wolkoff et al., 1997).

Another methodological limitation that has hampered many studies is the lack of a standardised system for the reporting and diagnosis of symptoms. Numerous different questionnaires have been used to elicit information on SBS symptoms, meaning that measures of symptom prevalence are often not comparable between studies. Furthermore, the lack of standards for objective findings or diagnostic criteria, coupled with the non-specific nature of many symptoms, may lead to the potential introduction of bias by respondents who already have strong beliefs concerning the origin of their illness (Burton, 1997).

Whilst perceived sensory irritation is often a primary determinant in the assessment of the quality of indoor air, there is an increasing recognition that any stimulus–response may be greatly influenced by the complex environment surrounding the exposure, which can include the social context or the perceiver's mental state (Dalton, 1996). To illustrate this, Dalton et al. (1997) exposed three groups of individuals to 800 ppm of acetone for a 20 min period. Before the exposure, the groups were given different information about the consequences of long-term exposure to the substance. The study found that subjects given a positive characterisation of the consequences of exposure perceived signifi-

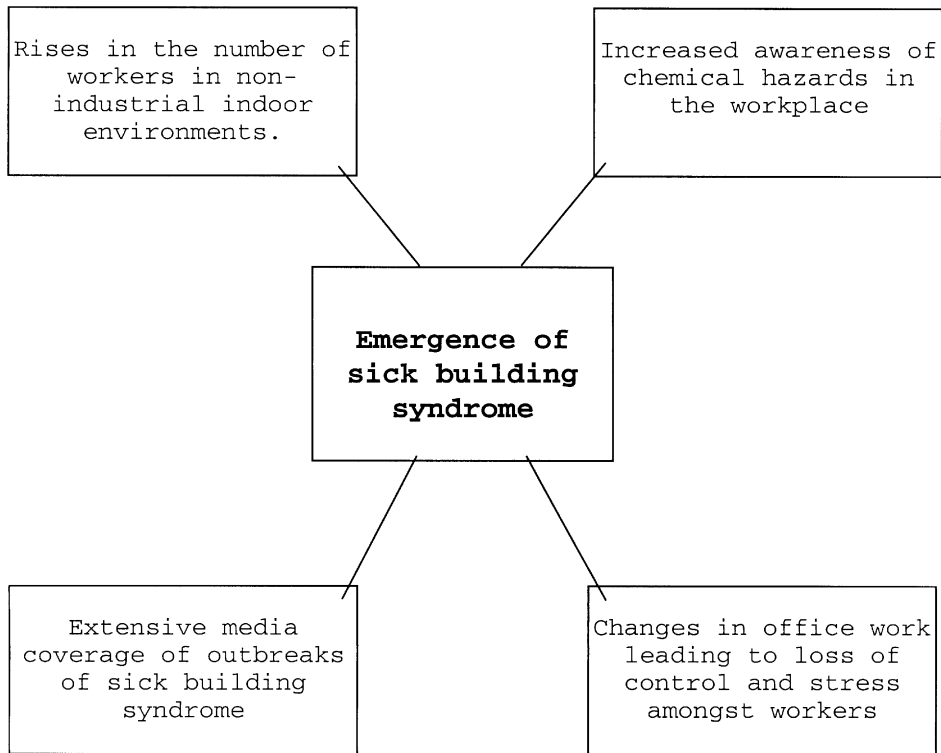
cantly less odour and irritation during exposure than did subjects given a negative or neutral characterisation ( $R^2 = 0.72$ ,  $p < 0.001$ ).

Whilst a reliance on the self-reporting of symptoms for the assessment of indoor air quality might be problematic, there are uncertainties in formulating more objective measures. For example, Hempel-Jorgensen et al. (1998) examined the relationship between self-reported symptoms, cytological changes, and conjunctival hyperaemia (redness) in the eyes of subjects exposed to mixtures of VOCs. Whilst perceived irritation and hyperaemia did increase with exposure, cytological changes in samples of eye fluid were unrelated to both the level of exposure and the reporting of perceived irritation.

Methodological issues aside, it may be that the likely causal mechanisms that underlie most outbreaks of SBS have not yet been correctly identified. The theory discussed earlier that reactions involving VOCs and substances such as ozone may be more important than direct exposure to the VOCs themselves could be significant (Wolkoff et al., 1997). If reactive mechanisms do prove to be one cause SBS, the prudent siting of electrostatic office equipment (such as photocopiers, and laser printers) that produce these substances may be particularly significant in preventing outbreaks (Wolkoff et al., 1997).

Another possibility that has been suggested is that the symptoms of SBS may, in some part, be associated with the syndrome of multiple chemical sensitivity (MCS). Proponents of MCS believe that symptoms may be produced by exposure to many chemically distinct substances at very low doses (Nethercott, 1996). The concept of MCS has largely evolved out of concern over food allergies during the 1960s (Shorter, 1997), and recent research by Meggs et al. (1996) suggests that around one-third of US citizens consider themselves to be suffering from MCS. The existence of MCS could explain why outbreaks of SBS have been recorded in buildings with extremely low concentrations of individual pollutants. However, MCS is not generally recognised by traditional medicine and a number of recent studies have failed to identify any clinical mechanism that might underlie it (Wolf, 1996). For example, Simon et al. (1993) were unable to detect any immunological differences between 34 control subjects and a sample of 41 patients reporting MCS. Interestingly, the authors did, however, find that MCS patients were more likely to show symptoms of anxiety and depression. Hence it has been argued that MCS may have a much stronger psychological than clinical basis (Shorter, 1997).

The debate over MCS highlights the importance that psychological factors may play in SBS. The symptoms commonly associated with SBS also characterise the diagnostic criteria for generalised anxiety disorder and panic disorder. Furthermore, individual characteristics and non-toxicological job related factors often appear to be important correlates of SBS (Burton, 1997).



Source: Adapted from Letz (1990)

Fig. 1. Possible socio-psychological factors responsible for the recent emergence of sick building syndrome.

Research on occupational stress has repeatedly shown that factors such as work overload, role ambiguity, and low status can be important in determining the health and well-being of employees (Kivimäki, 1996). In their study of the significance of psychosocial factors on the prevalence of SBS symptoms amongst 450 Swedish office workers, Eriksson and Höög (1993) found that the combination of a high workload, a perception of low control of the personal environment and workload, and lack of support from supervisors generated an increased risk of symptom reporting. In an extensive study of 4900 office employees in the USA, Wallace et al. (1993) also found that a heavy workload and conflicting demands in work were associated with headaches, eye irritations, dizziness, and nasal and chest discomfort. Amongst women, the researchers found that low educational status was also significant. Letz (1990) has produced a theoretical framework that explains how psychological mechanisms may explain the observed increases in the number of outbreaks of SBS in the last two decades. It is outlined in Fig. 1.

It seems, then, that SBS probably has a multi-factorial aetiology, in which chemical, physical, biological, and

psychosocial factors all interact to produce symptoms and discomfort (Baker, 1989). Psychosocial factors may act as modifiers of the responses of individuals to chemical, physical, and biological challenges in the indoor environment (Lahtinen et al. 1998). It seems probable that work stress functions as a mediating factor around environmental symptoms, possibly by altering the body's sensitivity to perceived physical demands and toxic exposures (Hedge et al., 1992). However, despite the multitude of theories that have been proposed, the exact processes of SBS are still unknown, and many uncertainties remain.

## 5. Discussion

Differences in factors such as climatic conditions, lifestyles, and construction habits mean that indoor environments vary from region to region, and country to country. However, despite this variation, many industrialised societies are increasingly facing a growing number of very similar indoor air quality problems. Although considerable advances have been made in our knowledge and understanding of the sources of indoor air pollution,

progress on the definition of the effects on human health and welfare has been on a much smaller scale (Stolwijk, 1992).

There is undoubtedly a need for more high-quality research to be undertaken to investigate further the links between indoor air quality and health. At present, projects concerning indoor air receive only around one tenth of the funding allocated to those studying the health effects of outdoor pollution (Seltzer, 1995). Given the evidence for the health impacts of indoor pollutants, this imbalance clearly needs to be addressed. Future research must fully acknowledge the complex nature of the indoor environment. Indoor air quality is the result of an intricate series of interactions involving many indoor and outdoor ventilation, microbiological, toxicological, and physical systems. Their interdependence may lead to the instigation of a multitude of synergistic processes. Hence the current paradigm, of categorising air quality problems according to single independent fields of expertise, will probably not provide solutions to most of unanswered questions.

Protocols must be developed and validated for analytical techniques that can consider, not just individual classes of pollutants, but a whole range of combinations and mixtures. It must be recognised that research limited to knowledge of a single class of pollutant may produce interesting results in a laboratory situation, but is unlikely to explain accurately the human health problems that will result from several interacting contaminants (Seltzer, 1995). All of the required advances will demand more refined methods for measuring and characterising pollutant emissions and concentrations. At present, techniques for measuring the complex combinations of pollutants that exist in many indoor environments are not well developed. In the future, it is important that this information is available in a useable form that will facilitate study, and aid the development of pollution mitigation strategies (Farland, 1991).

There is also a need for research to refine our understanding and measurement of health impacts. A lack of knowledge has led to particular difficulties in identifying many of the causes of sick building syndrome. Studies of sick buildings also highlight the need to consider mechanisms that lie outside the traditional toxicological dose–response relationships. Among the factors that should be encompassed in the development of future methodologies are the impact of environmental variables such as temperature and lighting, as well as psychological indicators that include job dissatisfaction, controllability, and stress.

As the health effects of indoor air pollution become better understood, the legal and regulatory aspects of the issue will require careful consideration. Nowadays, indoor air quality and pollution are becoming concerns not just of scientists, but also of the legal community.

The legal community's focus is on efforts to control the quality of indoor air through the passage of legislation and the development of regulations, as well as to impose liability upon those who are allegedly responsible for the indoor environment (Tokarz and Potterfield 1994). It is important to remember that we live in an increasingly litigious society, and the number of claims from workers who feel their health has been damaged by the contamination of their surroundings is increasing.

With the introduction of the US Clean Air Act in 1970, the US EPA was faced with the task of setting national ambient air quality standards for a set of common outdoor air pollutants. No consideration of the health risks of indoor contamination was included. The Act was substantially revised in 1990, but still does not provide the EPA with clear authority to address indoor pollution. Complicating the public-policy issue is the shortage of solid data. Within the United Kingdom, the National Environmental Protection Plan, published in 1996, identifies indoor air quality as a key area for action (DOE, 1996), but provides no mandatory regulations. Indeed, outside the Scandinavian countries, Holland, and Germany, policy on indoor air quality issues is generally poorly developed in Europe as a whole (Harrison, 1997).

The introduction of new legislation may go some way towards reducing levels of contamination. However, it is important to remember that simple interventions by home-owners and building operators can also have major impacts. Measures such as the avoidance of smoking indoors, the deployment of de-humidifiers in damp environments, and the use of natural ventilation are important. Here, education will be critical. Good education programmes are the key. The US Master Home Environmentalist Program (MHEP) is an example of a public education programme designed to reduce exposures amongst homeowners by providing personal contact with experts in the home (Roberts and Dickey, 1995). Volunteers operating the scheme provide free home assessments and follow-ups to families wishing to reduce their exposure to indoor air pollutants, house dust, and chemicals. Programmes such as this may be one of the most efficient ways to teach control methods to motivated individuals.

It might be necessary to re-evaluate the way in which buildings are designed and constructed in the future. Whilst problems with existing homes and offices are often difficult to overcome, architects and builders may need to be educated so as to create less contaminated structures. Simple measures, such as the selection of building materials and indoor fittings on the principle that the emission of pollutants such as VOCs and formaldehyde should be as low as reasonably achievable may prove beneficial. More radical ideas, such as a change of attitude with respect to the energy efficiency of buildings may also be required. Increased fuel costs could be offset against potential reductions in the considerable financial

burden from the treatment of both acute and chronic indoor pollution related diseases. In these areas, a policy shift from a reactive to a proactive mode with respect to building regulations may be required. However, improved data on the links between building design and health will have to precede the development and enforcement of any new construction legislation.

Meeting the challenges that are posed by indoor air pollution will take some time. A strong commitment will be required within both the public and private sectors, and the provision of adequate funding will be essential. In the meantime, we must manage our indoor environments using the best scientific advice available. Our accumulating knowledge of the sources, exposures, and health impacts of indoor pollutants will need to be put to good use to ensure that future indoor environments provide the healthiest possible conditions.

## References

- Abbey, D.E., Burchette, R.J., Knutsen, S.F., McDonnell, W.F., Lebowitz, M.D., Enright, P.L., 1998. Long-term particulate and other air pollutants and lung function in non-smokers. *American Journal of Respiratory Critical Care Medicine* 158 (1), 289–298.
- Adlkofer, F., Angerer, J., Ruppert, T., Scherer, G., Tricker, A.R., 1993. Determination of benzene exposure from occupational and environmental sources. In: (Anon) *Volatile Organic Compounds in the Environment*. Indoor Air International, Rotherfleet, pp. 511–518.
- Akland, G.G., Hartwell, T.D., Johnson, T.R., Whitmore, R.W., 1985. Measuring human exposure to carbon monoxide in Washington, D.C., and Denver, Colorado, during the winter of 1982–1983. *Environmental Science and Technology* 19 (10), 911–918.
- Albering, H.J., Hoogewerff, J.A., Kleinjans, J.C., 1996. Survey of  $^{222}\text{Rn}$  concentrations in dwellings and soils in the Dutch Belgian border region. *Health Physics* 70 (1), 64–69.
- Amitai, Y., Zlotogorski, Z., Golan-Katzav, V., Wexler, A., Gross, D., 1998. Neuropsychological impairment from acute low-level exposure to carbon monoxide. *Archives of Neurology* 55 (6), 845–848.
- Ancona, G., 1923. Asma epidemica da pediculoides ventricosus. *Polislinico Sez Med* 30 (1), 45–70.
- Anderson, I., Lundquist, G.R., Mølhave, L., 1975. Indoor air pollution due to chipboard used as a construction material. *Atmospheric Environment* 9, 1121–1127.
- Anonymous, 1992. Discomfort from environmental tobacco smoke among employees at worksites with minimal smoking restrictions. *U.S. Morbidity and Mortality Weekly Report* 41 (20), 351–354.
- Arashidani, K., Yoshikawa, M., Kawamoto, T., Matsuno, K., Kayama, F., Kodama, Y., 1996. Indoor pollution from heating. *Industrial Health* 34 (3), 205–215.
- Arlain, L.G., Rapp, C.M., Ahmed, S.G., 1990. Development of *Dermatophagoides pteronyssinus*. *Journal of Medical Entomology* 27 (6), 1035–1040.
- Arshad, S.H., Matthews, S., Gant, C., Hide, D.W., 1992. Effect of allergen avoidance on development of allergic disorders in infancy. *Lancet* 339, 1493–1497.
- Ayars, G.H., 1997. Biological agents and indoor air pollution. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 11–60.
- Baker, D.B., 1989. Social and organisational factors in office building-associated illness. *Occupational Medicine* 4 (4), 607–624.
- Bardana, E.J., 1997. Sick building syndrome — a wolf in sheep's clothing. *Annals of Allergy and Asthma Immunology* 79 (4), 283–293.
- Bardana, E.J., Montanaro, A., 1991. Formaldehyde: an analysis of its respiratory, cutaneous, and immunologic effects. *Annals of Allergy* 66 (6), 441–452.
- Bascom, R., Kesavanathan, J., Swift, D.L., 1995. Human susceptibility to indoor contaminants. *Occupational Medicine* 10 (1), 119–132.
- Berglund, B., Brunekreef, B., Knoppel, H., Lindvall, T., Maroni, M., Mølhave, L., Skov, P., 1992. Effects of indoor air pollution on human health. *Indoor Air* 2 (1), 2–25.
- Berglund, B., Jöhanesson, I., Lindvall, T.H., Lundin, L., 1990. A longitudinal study of perceived air quality and comfort in a sick library building. In: *Proceedings of the Fifth International Conference on Indoor Air Quality and Climate*, Vol. 3. Canada Mortgage and Housing Corporation, Ottawa, Canada.
- Bigonon, J., Peto, J., Saracci, R. (Eds.), 1989. *Non-occupational Exposure to Mineral Fibres*. International Agency for Research on Cancer, Lyon.
- Björnsson, E., Nörback, D., Janson, C., 1995. Asthmatic symptoms and indoor levels of micro-organisms and house dust mites. *Clinical and Experimental Allergy* 25 (5), 423–431.
- Blomquist, G., 1994. Sampling of biological particles. *Analyst* 119 (1), 53–56.
- Bochicchio, F., Campos-Venuti, G., Nuccetelli, C., Piermattei, S., Risica, S., Tommasino, L., Torri, G., 1996. Results of the representative Italian national survey on radon indoors. *Health Physics* 71 (5), 741–748.
- Bourbeau, J., Brisson, C., Allaire, S., 1997. Prevalence of the sick building syndrome symptoms in office workers before and six months and three years after being exposed to a building with an improved ventilation system. *Occupational and Environmental Medicine* 54 (1), 49–53.
- Breyse, P.A., 1984. Formaldehyde levels and accompanying symptoms associated with individuals residing in over 1000 conventional and mobile homes in the state of Washington. *Indoor Air* 3, 403–408.
- Brimblecombe, P., 1987. *The Big Smoke: a History of Air Pollution in London Since Medieval Times*. Routledge, London.
- Brooks, B.O., Utter, G.M., DeBroy, J.A., Schimke, R.D., 1991. Indoor air pollution: an edifice complex. *Clinical Toxicology* 29 (3), 315–374.
- Brown, S.K., Sim, M.R., Abramson, N.J., Gray, C.N., 1994. Concentrations of volatile organic compounds in indoor air—a review. *Indoor Air* 4 (1), 123–134.
- Brown, V.M., Crump, D.R., 1996. Volatile organic compounds. In: Berry, R.W., Brown, V.M., Coward, S.K.D. et al. (Eds.), *Indoor Air Quality in Homes, the Building Research Establishment Indoor Environment Study*, part 1. Construction Research Communications, London.

- Brunekreef, B., Houthuijs, D., Dijkstra, L., Boleij, J.S., 1990. Indoor nitrogen dioxide exposure and children's pulmonary function. *Journal of the Air and Waste Management Association* 40 (9), 1252–1256.
- Burr, M.L., 1997. Health effects of indoor combustion products. *Journal of the Royal Society of Health* 117 (6), 348–350.
- Burr, M.L., 1995. Pollution: does it cause asthma? *Archives of Disorders in Childhood* 72 (5), 377–387.
- Burr, M.L., St-Leger, A.S., Yarnell, J.W.G., 1981. Wheezing, dampness, and coal fires. *Community Medicine* 3 (3), 205–209.
- Burrell, R., 1991. Microbiological agents as health risks in indoor air. *Environmental Health Perspectives* 95 (11), 29–34.
- Burton, B.T., 1997. Volatile organic compounds. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 127–153.
- Chan, C.C., Yanagisawa, Y., Spengler, J.D., 1990. Personal and indoor/outdoor nitrogen dioxide exposure assessments of 23 homes in Taiwan. *Toxicology and Industrial Health* 6 (1), 173–182.
- Chen, B.T., Nanenji, J., Yeh, H.C., Mauderly, J.L., Cuddihy, R.G., 1990. Physical characterisation of cigarette-smoke aerosol generated from a walton smoke machine. *Aerosol Science and Technology* 12 (2), 364.
- Choi, I.S., 1983. Delayed neurological sequelae in carbon monoxide intoxication. *Archives of Neurology* 40 (7), 433–435.
- Clayton, C.A., Perritt, R.L., Pellizzari, E.D., Thomas, K.W., Whitmore, R.W., Özkaynak, H., Spengler, J.D., Wallace, L.A., 1993. Particle total exposure assessment methodology (PTEAM) study: distributions of aerosol and elemental concentrations in personal, indoor, and outdoor air samples in a Southern California community. *Journal of Exposure Analysis and Environmental Epidemiology* 3 (2), 227–250.
- Cobb, N., Etzel, R.A., 1991. Unintentional carbon-monoxide related deaths in the United States, 1979 through 1988. *Journal of the American Medical Association* 266 (5), 659–663.
- Cohen, B.S., 1998. Deposition of charged particles on lung airways. *Health Physics* 74 (5), 554–560.
- Cooper, J.A., 1980. Environmental impact of residential wood combustion emissions and its implications. *Journal of the Air Pollution Control Association* 30, 855–861.
- Coultas, D.B., Lambert, W.E., 1991. Carbon monoxide. In: Sanet, J.M., Spengler, J.D. (Eds.), *Indoor Air Pollution: A Health Perspective*. Johns Hopkins University Press, Baltimore, MD, pp. 187–208.
- Custovic, A., Fletcher, A., Pickering, C.A., 1998. Domestic allergens in public places III: house dust mite, cat, dog and cockroach allergens in British hospitals. *Clinical and Experimental Allergy* 28 (1), 53–59.
- Dalton, P., 1996. Odor perception and beliefs about risk. *Chemical Senses* 21, 447–458.
- Dalton, P., Wysocki, C.J., Brody, M.J., Lawley, H.J., 1997. The influence of cognitive bias on the perceived odor, irritation, and health symptoms from chemical exposure. *International Archives of Occupational and Environmental Health* 69, 407–417.
- D'Amato, G., Liccardi, G., D'Amato, M., 1994. Environment and the development of respiratory allergy. II: Indoors. *Monaldi Archive of Chest Disorders* 49 (5), 412–420.
- Dahms, T.E., Younis, L.T., Wiens, R.D., Zarnegar, S., Byers, S.L., Chaitman, B.R., 1993. Effects of carbon monoxide exposure in patients with documented cardiac arrhythmias. *Journal of the American College of Cardiology* 21 (2), 442–450.
- Dockery, D.W., Pope, C.A., Xu, X., Spengler, J.D., Ware, J.H., Martha, E.F., Ferris, B.G., Speizer, F.E., 1993. An association between air pollution and mortality in six U.S. cities. *The New England Journal of Medicine* 329 (24), 1753–1759.
- DOE (Department of the Environment), 1997. The United Kingdom National Air Quality Strategy. H.M. Stationary Office, London.
- DOE (Department of the Environment), 1996. The United Kingdom National Environmental Health Action Plan. H.M. Stationary Office, London.
- Doll, R., Peto, J., 1985. Asbestos: Effects on Health of Exposure to Asbestos. H.M. Stationary Office, London.
- Dotterud, L.K., Vorland, L.H., Falk, E.S., 1995. Viable fungi in indoor air in homes and schools in the Sor-Varanger community during winter. *Paediatric Allergy and Immunology* 6 (4), 181–186.
- Eberlein-König, B., Przybilla, B., Kühnl, P., Pechak, J., Gebefügi, I., Klenschmidt, J., Ring, J., 1998. Influence of airborne nitrogen dioxide or formaldehyde on parameters of skin function and cellular activation in patients with atopic eczema and control subjects. *Journal of Allergy and Clinical Immunology* 101 (1), 141–143.
- ECJRC (European Commission Joint Research Centre), 1997. Total volatile organic compounds (TVOC) in indoor air quality investigations. European Commission, Luxembourg.
- Eriksson, N., Höög, J., 1993. The office illness project in northern Sweden, the significance of psychosocial factors for the prevalence of the 'sick building-syndrome'. A case reference study. In: *Proceedings of the Sixth International Conference on Indoor Air Quality and Climate*, Vol. 1. Helsinki, Finland, pp. 333–337.
- Etherington, D.J., Pheby, D.F., Bray, F.I., 1996. An ecological study of cancer incidence and radon levels in south west England. *European Journal of Cancer* 32 (7), 1189–1197.
- Farland, W.H., 1991. Future directions and research needs. *Environmental Health Perspectives* 95, 131–133.
- Flannigan, B., Miller, J.D., 1994. Health implications of fungi in indoor environments—an overview. In: Sampson, R.A., Verhoeff, A.P., Flannigan, B., Flannigan, M.E., Adan, O., Hoekstra, E. (Eds.), *Health Implications of Fungi in Indoor Environments*. Elsevier, Amsterdam.
- Forastiere, F., Corbo, G.M., Michelozzi, P., Pistelli, R., Agabiti, N., Brancato, G., Ciappi, G., Perucci, C.A., 1992. Effects of environment and passive smoking on the respiratory health of children. *International Journal of Epidemiology* 21 (1), 66–73.
- Frampton, M.W., Morrow, P.E., Cox, C., Gibb, F.R., Speers, D.M., Utell, M.J., 1991. Effects of nitrogen dioxide exposure on pulmonary function and airway reactivity in normal humans. *American Review of Respiratory Disorders* 143 (3), 522–527.
- Fraser, D.W., Tsai, T.R., Orestein, W., 1977. Legionnaire's disease: description of an epidemic of pneumonia. *New England Journal of Medicine* 297 (22), 1187–1197.
- Friedman, F.M., Friedman, H.M., O'Connor, G.T., 1992. Prevalence of dust mite allergens in homes and workplaces of the Upper Connecticut river valley of New England. *Allergy Proceedings* 13, 259–262.



- Fukuda, K., Shibata, A., 1990. Exposure–response relationships between woodworking, smoking, or passive smoking, and squamous cell neoplasms of the maxillary sinus. *Cancer Causes and Control* 1 (2), 165–168.
- Furtaw, E.J., Pandian, M.D., Nelson, D.R., Behar, J.V., 1996. Modelling indoor air concentrations near emission sources in imperfectly mixed rooms. *Journal of the Air and Waste Management Association* 46 (9), 861–868.
- Gaensler, E.A., 1992. Asbestos exposure in buildings. *Clinics in Chest Medicine* 13 (2), 231–242.
- Glick, T.H., Gregg, M., Berman, B., 1978. Pontiac fever: an epidemic of unknown etiology in a health department. I. Clinical and epidemiological aspects. *American Journal of Epidemiology* 107 (2), 149–160.
- Gold, D.R., 1992. Indoor air pollution. *Clinics in Chest Medicine* 13 (2), 215–229.
- Goldstein, I.F., Andrews, L.R., Hartel, D., 1988. Assessment of human exposure to nitrogen dioxide, carbon monoxide, and respirable particles in New York inner-city residences. *Atmospheric Environment* 22 (10), 2127–2139.
- Gortmaker, S.L., Walker, D.K., Jacobs, F.H., Ruch-Ross, H., 1982. Parental smoking and the risk of childhood asthma. *American Journal of Public Health* 72 (6), 574–579.
- Groes, L., Pejtersen, J., Valbjørn, O., 1996. Perceptions and symptoms as a function of indoor environmental factors and building characteristics in office buildings. In: *Proceedings of the Sixth International Conference on Indoor Air Quality and Climate*, Vol. 4. Nagoya, Japan, pp. 237–242.
- Grosjean, D., Williams, E.L., Seinfeld, J.H., 1992. Atmospheric oxidation of selected terpenes and related carbonyls: gas-phase carbonyl products. *Environmental Science and Technology* 26 (8), 1526–1533.
- Guerin, M.R., Jenkins, R.A., Tomkins, B.A., 1992. *The Chemistry of Environmental Tobacco Smoke: Composition and Measurement*. Lewis Publishers, Chelsea, MI.
- Hampson, S.E., Andrews, J.A., Lee, M.E., Foster, L.S., Glasgow, R.E., Lichtenstein, E., 1998. Lay understanding of synergistic risk: the case of radon and cigarette smoking. *Risk Analysis* 18 (3), 343–350.
- Hanrahan, J.P., Morse, D.L., Scharf, V.B., 1987. A community hospital outbreak of legionellosis. Transmission by potable hot water. *American Journal of Epidemiology* 125 (4), 639–649.
- Harlap, S., Davies, A.M., 1974. Infant admissions to hospital and maternal smoking. *Lancet* 1 (857), 529–532.
- Harrison, J., Pickering, A.C., Finnegan, M.J., Austick, P.K.C., 1987. The sick building syndrome—further prevalence studies and investigations of possible causes. In: *Proceedings of the Fourth International Conference on Indoor Air Quality and Climate*. Institute for Water, Soil, and Air Hygiene, Berlin, pp. 487–491.
- Harrison, P.T.C., 1997. Health impacts of indoor air pollution. *Chemistry and Industry* 17, 677–681.
- Hart, B.L., Whitehead, L., 1990. Ecology of house dust mites in Oxfordshire. *Clinical and Experimental Allergy* 20, 203–209.
- Hartwell, T.D., Pellizzari, E.D., Perritt, R.L., 1987. Results from the Total Exposure Assessment Methodology (TEAM) study in selected communities in Northern and Southern California. *Atmospheric Environment* 21, 1995–2004.
- Hasselblad, V., Eddy, D.M., Kotchmar, D.J., 1992. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *Journal of the Air and Waste Management Association* 42 (5), 662–671.
- Hedge, A., Erickson, W.A., Rubin, G., 1992. Effects of personal and occupational factors on sick building syndrome reports in air-conditioned offices. In: Quick, J.C., Murphy, L.R., Hurrell, J.J. (Eds.), *Work and Well-being: Assessment and Interventions for Occupational Mental Health*. American Psychological Association, Washington, DC, pp. 286–298.
- Hempel-Jorgensen, A., Kjaergaard, S.K., Molhave, L., 1998. Cytological changes and conjunctival hyperemia in relation to sensory eye irritation. *International Archives of Occupational and Environmental Health* 71 (4), 225–235.
- Hendry, K.M., Cole, E.C., 1993. A review of mycotoxins in indoor air. *Journal of Toxicology and Environmental Health* 38 (2), 183–198.
- Henshaw, D.L., Eatough, J.P., Richardson, R.B., 1990. Radon as a causative factor in induction of myeloid leukaemia and other cancers. *Lancet* 335, 1008–1012.
- Hide, D.W., Matthews, S., Matthews, L., Stevens, M., Ridout, S., Twiselton, R., Gant, C., Arshad, S.H., 1994. Effect of allergen avoidance in infancy on allergic manifestations at age 2 years. *Journal of Allergy and Clinical Immunology* 93 (5), 842–846.
- Hines, A.L., Ghosh, T.K., Loyalka, S.K., Warder, R.C. (Eds.), 1993. *Indoor Air – Quality and Control*. Prentice-Hall, Englewood Cliffs, NJ.
- Hodgson, M.J., Frohlinger, J., Permar, E., Tidwell, C., Traven, N.D., Olenchok, S.A., Karpf, M., 1991. Symptoms and micro-environmental measures in non-problem buildings. *Journal of Occupational Medicine* 33 (4), 527–533.
- Honickey, R.E., Osborne, J.S., Akpom, C.A., 1985. Symptoms of respiratory illness in young children and the use of wood-burning stoves for indoor heating. *Pediatrics* 75 (3), 587–593.
- Horvath, E.P., 1997. Building-related illness and sick building syndrome: from the specific to the vague. *Cleveland Clinical Journal of Medicine* 64 (6), 303–309.
- Houck, P.M., Hampson, N.B., 1997. Epidemic carbon monoxide poisoning following a winter storm. *Journal of Emergency Medicine* 15 (4), 469–473.
- Houk, V.N., Baker, J.H., Sorensen, K., Kent, D.C., 1968. The epidemiology of tuberculosis infection in a closed environment. *Archives of Environmental Health* 16 (1), 26–35.
- Howden-Chapman, P., Isaacs, N., Crane, J., Chapman, R., 1996. Housing and health: the relationship between research and policy. *International Journal of Environmental Health Research* 6 (2), 173–185.
- Howell, J., Keiffer, M.P., Berger, L.R., 1997. Carbon monoxide hazards in rural Alaskan homes. *Alaskan Medicine* 39 (1), 8–11.
- Huncharek, M., 1994. Asbestos and cancer: epidemiological and public health controversies. *Cancer Investigations* 12 (2), 214–222.
- Hunter, C.A., Hull, A.V., Higham, D.F., Grimes, C.P., Lea, R.G., 1996. Fungi and bacteria. In: Berry, R.W., Brown, V.M., Coward, S.K.D. et al. (Eds.), *Indoor Air Quality in Homes, the Building Research Establishment Indoor Environment Study*, part 1. Construction Research Communications, London.

- IARC (International Agency for Research on Cancer), 1988. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 43: Man-made Mineral Fibres and Radon. International Agency for Research on Cancer, Lyon.
- IEH (Institute for Environment and Health), 1996. IEH assessment on indoor air quality in the home. Institute for Environment and Health, Leicester, UK.
- Islam, M.S., Ulmer, W.T., 1979. Threshold concentrations of SO<sub>2</sub> for patients with oversensitivity of the bronchial system. *Wissenschaft und Umwelt* 1 (1), 41–47.
- Jaakkola, J.J.K., Tuomaala, P., Seppänen, O., 1994. Air recirculation and sick building syndrome: a blinded crossover trial. *American Journal of Public Health* 84, 422–428.
- Janerich, D.T., Thompson, W.D., Varela, L.R., 1990. Lung cancer and exposure to tobacco smoke in the household. *New England Journal of Medicine* 323 (10), 632–636.
- Janssen, N.A., Hoek, G., Brunekreef, B., Harssema, H., Mensink, I., Zuidhof, A., 1998. Personal sampling of particles in adults: relation among personal, indoor, and outdoor air concentrations. *American Journal of Epidemiology* 147 (6), 537–547.
- Jarvis, D., Chinn, S., Luczynska, C., Burney, P., 1996. Association of respiratory symptoms and lung function in young adults with the use of domestic gas appliances. *Lancet* 1 (8999), 426–431.
- Jedrychowski, W., Flak, E., Wesolowski, J., Liu, K.S., 1995. Relation between residential radon concentrations and housing characteristics. The Cracow Study. *Central European Journal of Public Health* 3 (3), 150–160.
- Jin, H., Zheng, M., Mao, Y., Wan, H., Hang, Y., 1993. The effect of indoor pollution on human health. In: Jantunen, M., Kalliokoski, P., Kukkonen, E., Saarela, K., Seppänen, A., Vuorelma, H. (Eds.), *Proceedings of the Sixth International Conference on Indoor Air Quality and Climate*. Helsinki, Finland, pp. 477–482.
- Jones, A.P., 1998. Asthma and domestic air quality. *Social Science and Medicine* 47 (6), 755–764.
- Jones, A.P., 1997. The main causative factors of asthma, available information on population exposure to those factors, and current measures of control. In: *Asthma in Europe*. European Federation of Asthma and Allergy Associations, Holland, pp. 207–301.
- Kaliner, M.A., White, M.V., 1994. Asthma: causes and treatment. *Complementary Therapy* 20 (11), 645–650.
- Kalra, S., Owen, S.J., Hepworth, J., Woodcock, A., 1990. Airborne house dust mite antigen after vacuum cleaning (Letter). *Lancet* 336 (8712), 449.
- Kivimäki, M., 1996. Stress and Personality Factors. In: *Specifications of the Role of Test Anxiety, Private Self-Consciousness, Type A Behaviour Pattern, and Self-Esteem in Relationship Between Stressors and Stress Reactions*. Finnish Institute of Occupational Medicine, Helsinki.
- Koeck, M., Pichler-Semmelrock, F.P., Schlacher, R., 1997. Formaldehyde – study of indoor air pollution in Austria. *Central European Journal of Public Health* 5 (3), 127–130.
- Koenig, J.Q., Larson, T.V., Hamley, Q.S., Rebolledo, V., Dummer, K., Checkoway, H., Wang, S.Z., Lin, D., Pierson, W.E., 1993. Pulmonary lung function in children associated with fine particulate matter. *Environmental Research* 63 (1), 26–38.
- Kuster, P.A., 1996. Reducing the risk of house dust mite and cockroach allergen exposure in inner-city children with asthma. *Pediatric Nursing* 22, 297–299.
- Lagarde, F., Pershagen, G., Akerblom, G., Axelsson, O., Båverfält, U., Damberg, L., Enflo, A., Svartengren, M., Swedje-mark, G.A., 1997. Residential radon and lung cancer in Sweden: risk analysis accounting for random error in the exposure assessment. *Health Physics* 72 (2), 269–276.
- Lahtinen, M., Huhtanen, P., Reijula, K., 1998. Sick building syndrome and psychosocial factors – a literature review. *Indoor Air (Suppl 4)*, 71–80.
- Lambert, W.E., 1994. Urban exposures to carbon monoxide and myocardial ischemia in men with ischemic heart disease. Ph.D. Thesis, University of California.
- Lambert, W.E., 1997. Combustion pollution in indoor environments. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 83–103.
- Lambert, W.E., Samet, J.M., Hunt, W.C., Skipper, B.J., Schwab, M., Spengler, J.D., 1993a. Nitrogen dioxide and respiratory illness in infants – part II: assessment of exposure to nitrogen dioxide (research report 58). Health Effects Institute, Cambridge, MA.
- Lambert, W.E., Samet, J.M., Spengler, J.D., 1993b. Environmental tobacco smoke concentrations in no-smoking and smoking sections of restaurants. *American Journal of Public Health* 83 (9), 1339–1341.
- Lan, Q., Chen, W., Chen, H., He, X.Z., 1993. Risk factors for lung cancer in non-smokers in Xuanwei County of China. *Bio-medical and Environmental Science* 6 (2), 112–118.
- Leaderer, B.P., Stowe, M., Li, R., Sullivan, J., Koutrakis, P., Wolfson, M., Wilson, W., 1993. Residential levels of particle and vapor phase acid associated with combustion sources. In: Jantunen, M., Kalliokoski, P., Kukkonen, E., Saarela, K., Seppänen, A., Vuorelma, H. (Eds.), *Proceedings of the Sixth International Conference on Indoor Air Quality and Climate*. Helsinki, Finland, pp. 147–152.
- Leaderer, B.P., Koutrakis, P., Briggs, S.L.K., Rizzuto, J., 1990. Measurement of toxic and related air pollutants. In: *Proceedings of the EPA/Air and Waste Management Association International Symposium (VIP-17)*, United States Environmental Protection Agency, Washington, DC, pp. 567.
- Leaderer, B.P., Zragraniski, R.T., Berwick, M., Stolwijk, J.A.J., 1986. Assessment of exposure to indoor air contaminants from combustion sources: methodology and application. *American Journal of Epidemiology* 124 (2), 275–289.
- Leaderer, B.P., Stolwijk, J.A.J., Zragraniski, R.T., Qing-Shan, M., 1984. A field study of indoor air contaminant levels associated with unvented combustion sources. In: *Proceedings of the 77th Annual Meeting of the Air Pollution Control Association*, San Francisco, CA.
- Lee, R.J., Van Orden, D.R., Corn, M., Crump, K.S., 1992. Exposure to airborne asbestos in buildings. *Regulatory Toxicology and Pharmacology* 16 (1), 93–107.
- Letz, G.A., 1990. Sick building syndrome: acute illness among office workers – the role of building ventilation, airborne contaminants, and work stress. *Allergy Proceedings* 11 (3), 109–116.
- Lévesque, B., Gauvin, D., McGregor, R., Martel, R., Gingras, S., Dontigny, A., Walker, W.B., Lajoie, P., Létourneau, E., 1997. Radon in residences: influences of geological and housing characteristics. *Health Physics* 72 (6), 907–914.

- Lewis, R.G., Fortmann, R.C., Camann, D.E., 1994. Evaluation of methods for monitoring the potential exposure of small children to pesticides in the residential environment. *Archives of Environmental and Contamination Toxicology* 26 (1), 37–46.
- LHEA (London Health Education Authority), 1997. What people think about air pollution, their health in general, and asthma in particular. Health Education Authority, London.
- Li, Y., Powers, T.E., Roth, H.D., 1994. Random effects linear regression meta-analysis models with application to nitrogen dioxide health effects studies. *Journal of the Air and Waste Management Association* 44 (3), 261–270.
- Lipfert, F.W., 1997. Air pollution and human health: perspectives for the '90s and beyond. *Risk Analysis* 17 (3), 137–146.
- Longo, L.D., 1977. The biological effects of carbon monoxide on the pregnant woman, fetus, and new-born infant. *American Journal of Obstetrics and Gynaecology* 129 (1), 69–103.
- Lubin, J.H., Boice, J.D., 1997. Lung cancer risk from residential radon: meta-analysis of eight epidemiological studies. *Journal of the National Cancer Institute* 89 (1), 49–57.
- Lubin, J.H., Tomásek, L., Edling, C., Hornung, R.W., Howe, G., Kunz, E., Kusiak, R.A., Morrison, H.I., Radford, E.P., Samet, J.M., Tirmarche, M., Woodward, A., Yao, S.X., 1997. Estimating lung cancer mortality from residential radon using data for low exposures of miners. *Radiation Research* 147 (2), 126–134.
- Lucie, N.P., 1989. Radon and leukaemia. *Lancet* 2 (8654), 99–100.
- Luczynska, C.M., 1994. Risk factors for indoor allergen exposure. *Respiratory Medicine* 88 (10), 723–729.
- Luczynska, C.M., Li, Y., Chapman, M.D., Platts-Mills, T.A.E., 1990. Airborne concentrations and particle size distribution of allergen derived from domestic cats: measurements using a cascade impactor, liquid impinger and a two-site monoclonal antibody assay for Fel d I. *American Review of Respiratory Disorders* 141 (2), 361–367.
- Lyman, G.H., 1997. Radon. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 83–103.
- Macher, J.M., Huang, F.Y., Flores, M., 1991. A two-year study of microbiological indoor air quality in a new apartment. *Archives of Environmental Health* 46 (1), 25–29.
- Madany, I.M., 1992. Carboxyhemoglobin levels in blood donors in Bahrain. *Science of the Total Environment* 116 (1), 53–58.
- Marbury, M.C., 1991. Wood Smoke. In: Samet, J.M., Spengler, J.D. (Eds.), *Indoor Air Pollution: A Health Perspective*. John Hopkins University Press, Baltimore, MD, pp. 209–222.
- Marcinowski, F., Lucas, R.M., Yeager, W.M., 1994. National and regional distributions of airborne radon concentrations in US homes. *Health Physics* 66 (6), 699–706.
- Maroni, M., Seifert, B., Lindvall, T. (Eds.), 1995. *Indoor Air Quality – a Comprehensive Reference Book*. Elsevier, Amsterdam.
- Martonen, T.B., Katz, I., Fuels, K., Hickey, A.J., 1992. Use of analytically defined estimates of aerosol respirable fraction to predict lung deposition. *Pharmaceutical Research* 9 (12), 1634–1639.
- McDonald, J.C., 1991. An epidemiological view of asbestos in buildings. *Toxicology and Industrial Health* 7 (5–6), 187–193.
- Meggs, W.J., Dunn, K.A., Bloch, R.M., Goodman, P.E., Davidoff, A.L., 1996. Prevalence and nature of allergy and chemical sensitivity in a general population. *Archives of Environmental Health* 51 (4), 275–282.
- Mendell, M.J., Fine, L., 1994. Building ventilation and symptoms – where do we go from here? (editorial). *American Journal of Public Health* 84 (3), 346–348.
- Mendell, M.J., Smith, A.H., 1990. Consistent pattern of elevated symptoms in air-conditioned office buildings: a reanalysis of epidemiologic studies. *American Journal of Public Health* 80 (10), 1193–1199.
- Mølhave, L., 1991. Indoor climate, air pollution, and human comfort. *Journal of Exposure Analysis and Environmental Epidemiology* 1 (1), 63–81.
- Mølhave, L., 1979. Indoor air pollution due to building materials. In: *Proceedings of the First International Indoor Climate Symposium*, Copenhagen, Denmark, p. 89.
- Mølhave, L., Bach, B., Pedersen, O.F., 1986. Human reactions to low concentrations of volatile organic compounds. *Environment International* 12 (1–4), 167–175.
- Monn, C., Brändli, O., Schindler, C., Ackermann-Liebrich, U., Leuenberger, P., 1998. Personal exposure to nitrogen dioxide in Switzerland: SAPALDIA team Swiss study on air pollution and lung diseases in adults. *Science of the Total Environment* 215 (3), 243–251.
- Montanaro, A., 1997. Indoor allergens: description and assessment of health risks. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 201–214.
- Morgan, K.T., 1997. A brief review of formaldehyde carcinogenesis in relation to rat nasal pathology and human health risk assessment. *Toxicologic Pathology* 25 (3), 291–307.
- Moriske, H.J., Drews, M., Ebert, G., Menk, G., Scheller, C., Schöndube, M., Konieczny, L., 1996. Indoor air pollution by different heating systems: coal burning, open fireplace and central heating. *Toxicology Letters* 88 (1–3), 349–354.
- Mumford, J.L., Li, X., Hu, F., Lu, X.B., Chuang, J.C., 1995. Human exposure and dosimetry of polycyclic aromatic hydrocarbons in urine from Xuan Wei, China with high lung cancer mortality associated with exposure to unvented coal smoke. *Carcinogenesis* 16 (12), 3031–3036.
- Musmand, J.J., Horner, W.E., Lopez, M.M., Lehrer, S.B., 1995. Identification of important allergens in German cockroach extracts by sodium dodecylsulphate-polyacrylamide gel electrophoresis and Western blot analysis. *Journal of Allergy and Clinical Immunology* 95, 877–885.
- Nagda, N.L., Koontz, M.D., Billick, I.H., Leslie, N.P., Behrens, D.W., 1996. Causes and consequences of backdrafting of vented gas appliances. *Journal of the Air and Waste Management Association* 46 (9), 838–846.
- NASA (National Aeronautics and Space Administration), 1973. *Bioastronautics Data Book – SP 3006*. NASA, Washington, D.C.
- Neas, L.M., Dockery, D.W., Burge, H., Koutrakis, P., Speizer, F.E., 1996. Fungus spores, air pollutants, and other determinants of peak expiratory flow rate in children. *American Journal of Epidemiology* 143 (8), 797–807.
- Neas, L.M., Dockery, D.W., Ware, J.H., Spengler, J.D., Ferris, B.G., Speizer, F.E., 1994. Concentration of indoor particulate matter as a determinant of respiratory health in children. *American Journal of Epidemiology* 139 (11), 1088–1099.

- Nero, A.V., Schwehr, M.B., Nazaroff, W.W., Revzan, K.L., 1986. Distribution of airborne radon-222 concentrations in US homes. *Science* 234 (4779), 992–997.
- Nethercott, J.R., 1996. Whither multiple chemical sensitivities?. *American Journal of Contact Dermatology* 7 (4), 199–201.
- Nielson, K.K., Rogers, V.C., Holt, R.B., Pugh, T.D., Grondzik, W.A., de Meijer, R.J., 1997. Radon penetration of concrete slab cracks, joints, pipe penetrations, and sealants. *Health Physics* 73 (4), 668–678.
- Niemala, R., Vaino, H., 1985. Formaldehyde exposure in work and the general environment. *Scandinavian Journal of Work and Environmental Health* 7 (1), 95–100.
- Norbäck, D., Björnsson, E., Janson, C., Widstrom, J., Boman, G., 1995. Asthma and the indoor environment: the significance of emission of formaldehyde and volatile organic compounds from newly painted indoor surfaces. *Occupational and Environmental Medicine* 52 (6), 388–395.
- Norbäck, D., Björnsson, E., Widström, J., 1993. Asthma symptoms in relation to volatile organic compounds (VOCs) and bacteria in dwellings. In: (Anon) *Volatile Organic Compounds in the Environment*. Indoor Air International, Rotherfleet, pp. 377–386.
- NRC (National Research Council), 1986. *Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects*. National Academy Press, Washington, DC.
- Ostro, B., Chestnut, L., 1998. Assessing the health benefits of reducing particulate matter air pollution in the United States. *Environmental Research* 76 (2), 94–106.
- Otto, D., Hundell, H., House, D., Mølhave, L., Counts, W., 1992. Exposure of humans to a volatile organic mixture. I. Behavioural assessment. *Archives of Environmental Health* 47 (1), 23–30.
- Pandey, M.R., Smith, K.R., Boleij, J.S.M., 1989. Indoor air pollution in developing countries and acute respiratory infection in children. *Lancet* 1 (8635), 427–429.
- Peat, J.K., Dickerson, J., Li, J., 1998. Effects of damp and mould in the home on respiratory health: a review of the literature. *Allergy* 53, 120–128.
- Peat, J.K., Tovey, E., Gray, E.J., Mellis, C.M., Woolcock, A.J., 1994. Asthma severity and morbidity in a population sample of Sydney schoolchildren. *Australian and New Zealand Journal of Medicine* 24 (3), 270–276.
- Pershagen, G., Akerblom, G., Axelsson, O., Clavenson, B., Damber, L., Desai, G., Enflo, A., Lagarde, F., Mellander, H., Svartengren, M., Swedjemarm, G.A., 1994. Residential radon exposure and lung cancer in Sweden. *New England Journal of Medicine* 330 (3), 159–164.
- Pettenkofer, M.S., 1858. *Über den luftwechsel in Wohngebäuden*. Cotta'sche Buchhandlung, Munich.
- Pilotto, L.S., Douglas, R.M., Attewell, R.G., Wilson, S.R., 1997. Respiratory effects associated with indoor nitrogen dioxide exposure in children. *International Journal of Epidemiology* 26 (4), 788–796.
- Platt, S.D., Martin, C.J., Hunt, S.M., Lewis, C.W., 1989. Damp housing, mould growth, and symptomatic health state. *British Medical Journal* 298, 1673–1678.
- Platts-Mills, T.A.E., Carter, M.C., 1997. Asthma and indoor exposure to allergens. *New England Journal of Medicine* 336 (19), 1382–1384.
- Platts-Mills, T.A.E., Woodfolk, J.A., Chapman, M.D., Heymann, P.W., 1996. Changing concepts of allergic disease: the attempt to keep up with real changes in lifestyles. *Journal of Allergy and Clinical Immunology* 98 (Suppl), S297–S306.
- Platts-Mills, T.A.E., Ward, G.W., Sporik, R., Gelber, L.E., Chapman, M.D., Heymann, P.W., 1991. Epidemiology of the relationship between exposure to indoor allergens and asthma. *International Archives of Allergy and Applied Immunology* 94 (1–4), 339–345.
- Pollart, S.M., Smith, T.F., Morris, E.C., Gelber, L.E., Platts-Mills, T.A.E., Chapman, M.D., 1991. Environmental exposure to cockroach allergen: analysis with monoclonal antibody based enzyme immunoassays. *Journal of Allergy and Clinical Immunology* 87 (2), 505–510.
- Polpong, P., Bovornkitti, S., 1998. Indoor Radon. *Journal of the Medical Association of Thailand* 81 (1), 47–57.
- Postlethwait, E.M., Bidani, A., 1990. Reactive uptake governs the pulmonary air space removal of inhaled nitrogen dioxide. *Journal of Applied Physiology* 68 (2), 594–603.
- Potter, P.C., Juritz, J., Little, F., McCaldin, M., Dowdle, E.B., 1991. Clustering of fungal-allergen specific IgE antibody responses in allergic subjects. *Annals of Allergy* 66 (2), 149–153.
- Prescher, K.E., Jander, K., 1987. Formaldehyde in indoor air. *Bundesgesundheitsblatt* 30, 273–278.
- Puerta, L., Fernandez-Caldas, E., Lockey, R.F., 1997. Indoor allergens: detection and environmental control measures. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 215–229.
- Qin, Y.H., Zhang, X.M., Jin, H.Z., Liu, Y.Q., Fan, D.L., Cao, Z.J., 1993. Effects of indoor air pollution on respiratory illness of school children. In: Jantunen, M., Kalliokoski, P., Kukkonen, E., Saarela, K., Seppänen, A., Vuorelma, H. (Eds.), *Proceedings of the Sixth International Conference on Indoor Air Quality and Climate*. Helsinki, Finland, pp. 477–482.
- Rando, R.J., Simlote, P., Salvaggio, J.E., Lehrer, S.B., 1997. Environmental tobacco smoke: measurement and health effects of involuntary smoking. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 61–82.
- Redlich, C.A., Sparer, J., Cullen, M.R., 1997. Sick-building syndrome. *Lancet* 349 (9057), 1013–1016.
- Reiss, R., Ryan, P.B., Koutrakis, P., Tibbetts, S.J., 1995. Ozone reactive chemistry on interior latex paint. *Environmental Science and Technology* 29 (8), 1906–1912.
- Roberts, J.W., Dickey, P., 1995. Exposure of children to pollutants in house dust and indoor air. *Reviews of Environmental Contamination and Toxicology* 143 (1), 59–78.
- Robinson, J., Nelson, W.C., 1995. *National Human Activity Pattern Survey Data Base*. United States Environmental Protection Agency, Research Triangle Park, NC.
- Rodes, C.E., Kamens, R.M., Wiener, R.M., 1991. The significance and characteristics of the personal activity cloud on exposure assessments for indoor contaminants. *Indoor Air* 2, 123–145.
- Roggli, V.L., Longo, W.E., 1991. Mineral fibre content of lung tissue in patients with environmental exposures: household contacts vs. building occupants. *Annals of the New York Academy of Science* 643, 511–518.
- Rosenstreich, D.L., Eggleston, P., Kattan, M., 1997. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity amongst inner-city children with asthma. *New England Journal of Medicine* 336, 1356–1363.

- Rothman, A.L., Weintraub, M.I., 1995. The sick building syndrome and mass hysteria. *Neurologic Clinics* 13 (2), 405–412.
- Roughton, F.J.W., Darling, R.C., 1994. The effect of carbon monoxide on the oxyhemoglobin dissociation curve. *American Journal of Physiology* 141 (1), 17–31.
- Ryan, C.M., Morrow, L.A., 1992. Dysfunctional buildings or dysfunctional people: an examination of the sick building syndrome and allied disorders. *Journal of Consulting and Clinical Psychology* 60 (2), 220–240.
- Ryan, P.B., Lee, M.W., North, B., McMichael, A.J., 1992. Risk factors for tumours of the brain and meninges: results from the Adelaide adult brain tumour study. *International Journal of Cancer* 51 (1), 20–27.
- Ryan, P.B., Hemphill, C.P., Billick, I.H., Nagda, N.L., Koontz, M.D., Fortmann, R.C., 1989. Estimation of nitrogen dioxide concentrations in homes equipped with unvented gas space heaters. *Environment International* 15 (1–6), 551–556.
- Salerno, M.S.R., Huss, K., Huss, E.W., 1992. Allergen avoidance in the treatment of dust-mite allergy and asthma. *Nursing Practitioner* 17 (1), 53–65.
- Salome, C.M., Brown, N.J., Marks, G.B., Woolcock, A.J., Johnson, G.M., Nancarrow, P.C., Quigley, S., Tio, J., 1996. Effect of nitrogen dioxide and other combustion products on asthmatic subjects in a home-like environment. *European Respiratory Journal* 9 (5), 910–918.
- Samet, J.M., 1990. Environmental controls and lung disease. *American Review of Respiratory Disorders* 142 (6), 915–939.
- Samet, J.M., Lambert, W.E., Skipper, B.J., Cushing, A.H., Hunt, W.C., Young, S.A., McLaren, L.C., Schwab, M., Spengler, J.D., 1993. Nitrogen dioxide and respiratory illnesses in infants. *American Review of Respiratory Disorders* 148 (8), 1258–1265.
- Samet, J.M., Marbury, M.C., Spengler, J.D., 1987. Health effects and sources of indoor air pollution: part 1. *American Review of Respiratory Disorders* 136 (6), 1486–1508.
- Samson, R.A., Flannigan, B., Flannigan, M.E., Verhoeff, A.P., Adan, O.C.G., Hoekstra, E.S., 1994. Health indicators of fungi in indoor environment. In: *Air Quality Monographs*, Vol. 2. Elsevier, Amsterdam.
- Sandmeyer, E.E., 1982. Aromatic hydrocarbons. In: Clayton, G.D., Clayton, F.E. (Eds.), *Patty's Industrial Hygiene and Toxicology*, Vol. 2, 3rd Edition. Wiley, New York, pp. 3253–3431.
- Schlitt, H., Knöppel, H., 1989. Carbonyl compounds in mainstream and sidestream tobacco smoke. In: Bieva, C.J., Courtois, Y., Govaerts, M. (Eds.), *Present and Future of Indoor Air Quality*. Excerpta Medica, Amsterdam, pp. 197–206.
- Schneider, T., Bohgard, M., Gudmundsson, A., 1994. A semi-empirical model for particle deposition onto facial skin and eyes. Role of air currents and electric fields. *Journal of Aerosol Science* 25 (3), 583–593.
- Schwarzberg, M.N., 1993. Carbon dioxide level as migraine threshold factor: hypothesis and possible solutions. *Medical Hypotheses* 41 (1), 35–36.
- Seltzer, J.M., 1997. Sources, concentrations, and assessment of indoor pollution. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 11–60.
- Seltzer, J.M., 1995. Effects of the indoor environment on health. *Occupational Medicine* 10 (1), 26–45.
- Shah, J.J., Singh, H.B., 1988. Distribution of volatile organic compounds in outdoor and indoor air. *Environmental Science and Technology* 22 (12), 1381–1388.
- Shorter, E., 1997. Multiple chemical sensitivity: pseudodisease in historical perspective. *Scandinavian Journal of Work and Environmental Health* 23 (Suppl. 3), 35–42.
- Simon, G.E., Daniell, W., Stockbridge, H., Claypoole, K., Rosenstock, L., 1993. Immunologic, psychological, and neuropsychological factors in multiple chemical sensitivity. A controlled study. *Annals of Internal Medicine* 119 (2), 97–103.
- Sisovic, E., Fugas, M., Sega, K., 1996. Assessment of human inhalation exposure to polycyclic aromatic hydrocarbons. *Journal of Exposure Analysis and Environmental Epidemiology* 6 (4), 439–447.
- Somerville, S.M., Rona, R.J., Chinn, S., 1988. Passive smoking and respiratory conditions in primary school children. *Journal of Epidemiology and Community Health* 42 (2), 105–110.
- Spengler, J.D., 1993. Nitrogen dioxide and respiratory illnesses in infants. *American Review of Respiratory Disorders* 148 (5), 1258–1265.
- Spengler, J.D., Sexton, K., 1983. Indoor air pollution: a public health perspective. *Science* 221 (4605), 9–17.
- Spengler, J.D., Dockery, D.W., Turner, W.A., Wolfson, J.M., Ferris, B.G., 1981. Long-term measurements of respirable particles, sulphates, and particulates inside and outside homes. *Atmospheric Environment* 15 (1), 23–30.
- Spicer, C.W., Kenny, D.V., Ward, G.F., Billick, I.H., 1993. Transformations, lifetimes, and sources of NO<sub>2</sub>, HONO, and HNO<sub>3</sub> in indoor environments. *Journal of the Air and Waste Management Association* 43 (11), 1479–1485.
- Steindorf, K., Lubin, J., Wichmann, H.E., Becher, H., 1995. Lung cancer deaths attributable to indoor radon exposure in West Germany. *International Journal of Epidemiology* 24 (3), 485–492.
- Sterling, D.A., Lewis, R.D., 1998. Pollen and fungal spores indoor and outdoor of mobile homes. *Annals of Allergy and Asthma Immunology* 80 (3), 279–285.
- Sterling, T.D., 1991. Concentrations of nicotine, RSP, CO and CO<sub>2</sub> in non-smoking areas of offices ventilated by air recirculated from smoking designated areas. *American Industrial Hygiene Association Journal* 52 (10), 564–565.
- Stewart, R.D., Peterson, J.E., Baretta, E.D., Bachand, R.T., Hosko, M.J., Herrmann, A.A., 1970. Experimental human exposure to carbon monoxide. *Archives of Environmental Health* 21 (2), 154–164.
- Stolwijk, J.A., 1992. Risk assessment of acute health and comfort effects of indoor air pollution. *Annals of the New York Academy of Sciences* 641, 56–62.
- Stolwijk, J.A., 1991. Assessment of population exposure and carcinogenic risk posed by volatile organic compounds in indoor air. *Risk Analysis* 10 (1), 49–57.
- Sundell, J., 1994. On the association between building ventilation characteristics, some indoor environmental exposures, some allergic manifestations, and subjective symptom reports. *Indoor Air* (Suppl. 2), 1.
- Sundell, J., Andersson, B., Andersson, K., Lindvall, T., 1993. Volatile organic compounds in ventilating air in buildings at different sampling points in the buildings and the relationship with the prevalence of occupant symptoms. *Indoor Air* 2 (1), 82–93.

- Takahashi, T., 1997. Airborne fungal colony-forming units in outdoor and indoor environments in Yokohama. Japan. *Mycopathologia* 139 (1), 23–33.
- Tariq, S.M., Matthews, S.M., Hakim, E.A., Stevens, M., Arshad, S.H., Hide, D.W., 1998. The prevalence of and risk factors for atopy in early childhood: a whole population birth cohort study. *Journal of Allergy and Clinical Immunology* 101 (5), 587–593.
- Teichman, K.Y., 1995. Indoor air quality: research needs. *Occupational Medicine* 10 (1), 217–227.
- Tokarz, A.P., Potterfield, P.M., 1994. Regulatory and legal aspects of indoor air quality and pollution. *Indoor Air Pollution* 14 (3), 679–691.
- Traynor, G.W., Apte, M.G., Carruthers, A.R., 1986. *Indoor Air Pollution Due to Emission from Woodburning Stoves*. Lawrence Berkeley Laboratories, Berkeley, CA.
- Tunnicliffe, W.S., Burge, P.S., Ayres, J.G., 1994. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 344 (8939–8940), 1733–1736.
- US EPA (United States Environmental Protection Agency), 1992. Technical support document for the 1992 citizen's guide to radon (EPA 400-R-92-011). US Environmental Protection Agency, Washington, D.C.
- US EPA (United States Environmental Protection Agency), 1991. *Air Quality Criteria for Carbon Monoxide*. US Environmental Protection Agency, Washington, D.C.
- Vaughan, T.L., Strader, C., Davis, S., Daling, J.R., 1986. Formaldehyde and cancers of the pharynx, sinus, and nasal cavity: II. Residential exposures. *International Journal of Cancer* 38 (5), 685–688.
- Verhoeff, A.P., 1994. Home Dampness, Fungi and House Dust Mites, and Respiratory Symptoms in Children. Posen & Looijen b.v., Wageningen, Germany.
- Viegi, G., Carrozzi, L., Paoletti, P., Vellutini, M., DiViggiano, E., Baldacci, S., Modena, P., Pedreschi, M., Mammini, U., di-Pede, C., 1992. Effects of the home environment on respiratory symptoms of a general population sample in middle Italy. *Archives of Environmental Health* 47 (1), 64–70.
- Vincent, D., Annesi, I., Festy, B., Lambrozo, J., 1997. Ventilation system, indoor air quality, and health outcomes in Parisian modern office workers. *Environmental Research* 75 (2), 100–112.
- van Wijnen, J.H., Verhoeff, A.P., Mulder-Folkerts, D.K.F., Brachel, H.J.H., Schou, C., 1997. Cockroach allergen in house dust. *Allergy* 52 (4), 460–464.
- Wallace, L.A., 1991a. Personal exposure to 25 volatile organic compounds. EPA's 1987 team study in Los Angeles. California. *Toxicology and Industrial Health* 7 (5–6), 203–208.
- Wallace, L.A., 1991b. Comparison of risks from outdoor and indoor exposure to toxic chemicals. *Environmental Health Perspectives* 95 (1), 7–13.
- Wallace, L.A., 1996. Indoor particles: a review. *Journal of the Air and Waste Management Association* 46 (2), 98–126.
- Wallace, L.A., 1997. Sick building syndrome. In: Bardana, E.J., Montanaro, A. (Eds.), *Indoor Air Pollution and Health*. Marcel Dekker, New York, pp. 83–103.
- Wallace, L.A., Nelson, C.J., Highsmith, R., Duntzman, G., 1993. Association of personal and workplace characteristics with health, comfort, and odor: a survey of 3948 office workers in three buildings. *Indoor Air* 3 (1), 193–205.
- Wallace, L.A., Nelson, W.C., Ziegenfus, R., Pellizzari, E., 1991. The Los Angeles TEAM study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. *Journal of Exposure Analysis and Environmental Epidemiology* 1 (2), 37–72.
- Wallace, L.A., 1987. The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis. US Environmental Protection Agency, Washington, DC.
- Wanner, H.U., 1993. Sources of pollutants in indoor air. *IARC Scientific Publications* 109, 19–30.
- Ware, J.H., Dockery, D.W., Spiro, A., Speizer, F.E., Ferris, B.G., 1984. Passive smoking, gas cooking, and respiratory health of children living in six cities. *American Review of Respiratory Disorders* 129 (3), 366–374.
- Weiss, S.T., 1986. Passive smoking and lung cancer: what is the risk? *American Review of Respiratory Disorders* 133 (1), 1–3.
- WHO (World Health Organisation), 1995. *Global Strategy of Asthma Management and Prevention*. World Health Organisation, Geneva.
- Wickman, M., Gravesen, S., Nordvall, S.L., Pershagen, G., Sundell, J., 1992. Indoor viable dust-bound microfungi in relation to residential characteristics, living habitats, and symptoms in atopic and control children. *Journal of Allergy and Clinical Immunology* 89 (3), 752–759.
- Wieslander, G., Norbäck, D., Björnsson, E., Janson, C., Boman, G., 1997. Asthma and the indoor environment: the significance of emission of formaldehyde and volatile organic compounds from newly painted indoor surfaces. *International Archives of Occupational and Environmental Health* 69 (2), 115–124.
- Wolf, C., 1996. Multiple chemical sensitivity (MCS) – the so-called chemical multiple hypersensitivity. *Versicherungsmedizin* 48 (5), 175–178.
- Wolff, S.P., 1991. Leukaemia risks and radon. *Nature* 352 (6333), 288.
- Wolkoff, P., Clausen, P.A., Jensen, B., Nielsen, G.D., Wilkins, C.K., 1997. Are we measuring the relevant indoor pollutants? *Indoor Air* 7 (1), 92–106.
- Wolkoff, P., Clausen, P.A., Neilsen, P.A., Mølhave, L., 1991. The Danish twin apartment study; part 1: formaldehyde and long-term VOC measurements. *Indoor Air* 4, 478–490.
- Wong, O., 1983. A epidemiologic mortality study of a cohort of chemical workers potentially exposed to formaldehyde, with a discussion on SMR and PMR. In: Gibson, J.E. (Ed.), *Formaldehyde Toxicity*. Hemisphere Publishing Corporation, New York, NY.
- Yang, Y., Sun, C., Sun, M., 1997. The effect of moderately increased CO<sub>2</sub> concentration on perception of coherent motion. *Investigative Ophthalmology and Visual Science* 38 (4), 1786.
- Yu, V.L., 1993. Could aspiration be the major mode of transmission for *Legionella*? *American Journal of Medicine* 95 (1), 13–15.
- Yun, K.N., Young, E.C., Stokes, M.J., Tang, K.K., 1998. Radon properties in offices. *Health Physics* 75 (2), 159–164.