

**Towards Healthy Air in Dwellings in Europe**

**THADE Project**

**Report**

**INDOOR AIR QUALITY**

**Paolo Carrer**

Department of Occupational Health, University of Milan, Italy

**This project received financial support from the European Commission.**

<b>INTRODUCTION .....</b>	<b>3</b>
<b>SOURCES OF INDOOR AIR POLLUTANTS.....</b>	<b>3</b>
Building Components and Furnishings .....	3
Equipment.....	4
Human Activities.....	4
Sources Outside Building .....	4
<b>OVERVIEW OF INDOOR POLLUTANTS AND THEIR HEALTH EFFECTS.....</b>	<b>4</b>
Inorganic pollutants .....	5
Organic pollutants.....	7
Biocontaminants .....	11
Allergens in indoor air.....	13
<b>HEALTH EFFECTS RELATED TO INDOOR AIR QUALITY .....</b>	<b>15</b>
Building-related illness.....	15
Allergic diseases associated with exposure to indoor air pollution.....	15
Sick building syndrome .....	16
Individuals susceptible to IAQ effects.....	17
<b>REFERENCES .....</b>	<b>17</b>

## INTRODUCTION

Nowadays most people living in urban areas spend the greatest part of their time indoors, where concentrations of many air pollutants are higher than outdoors (Carrer et al., 1997). Pollutant levels inside our homes, offices, shopping malls, and schools may equal or exceed those outside. Concentrations of indoor air pollutants are often 2-5 times those found outdoors, and can occasionally reach 100 times the outdoor levels.

In fact, indoor environments have changed enormously with the introduction of soft furnishings, fitted carpets and mechanical air ventilation systems. The rate at which indoor air is exchanged for fresh air is now 10 times lower than it was 30 years ago, with a considerable increase both in humidity and in concentrations of indoor pollutants and airborne allergens.

Many factors affect indoor air quality. Outdoor pollutants (e.g. pollen, and traffic and factory emissions) enter buildings through open windows, ventilation system air intakes, and building leaks and cracks. These contaminants, along with those that arise inside the building (mould spores and chemical emissions from carpeting, wallpaper, furnishings, and cleaning products) concentrate in tightly sealed buildings with inadequate ventilation. Ventilation systems meant to bring in clean or filtered outdoor air to flush out 'used' indoor air, do not always function properly, either because of poor design or poor maintenance.

## SOURCES OF INDOOR AIR POLLUTANTS

The indoor environment in any kind of building, including dwellings, is a result of the interaction between building system (original design and later modifications in the structure and mechanical systems), construction techniques, contaminant sources (building materials and furnishings, moisture, processes and activities within the building), building occupants and outdoor sources. The most important sources of indoor pollutants are (Etkin and Vogt, 1996):

### **Building Components and Furnishings**

#### ***Locations that can produce dust or fibres***

- textured surfaces such as carpeting, curtains, and other textiles
- open shelving
- old or deteriorated furnishings
- materials containing damaged asbestos

#### ***Redecorating/remodelling/repair activities***

- emissions from new furnishings
- dust and fibres from demolition
- odours and volatile organic and inorganic compounds from paint, caulk, adhesives
- microbiological releases from demolition or remodelling activities

#### ***Unsanitary conditions and water damage***

- microbiological growth on or in soiled or water-damaged furnishings
- microbiological growth in areas of surface condensation
- standing water from clogged or poorly designed drains
- dry traps that allow the passage of sewer gas

#### ***Chemicals released from building components***

- furnishings emitting volatile organic compounds

#### ***Accidental events***

- spills of water or other liquids
- microbiological growth due to flooding or to leaks from roofs, piping
- fire damage (soot, PCBs from electrical equipment, odours)

## **Equipment**

### ***Equipment***

- emissions from office equipment (volatile organic compounds, ozone)
- supplies (solvents, toners, ammonia)

### ***HVAC system***

- dust or dirt in ductwork or other components
- microbiological growth in drip pans, humidifiers, ductwork, coils
- improper use of biocides, sealants, and/or cleaning compounds
- improper venting of combustion products

## **Human Activities**

### ***Personal activities***

- smoking
- body odour
- cosmetic odours
- cooking

### ***Activities***

- cleaning materials and procedures
- emissions from stored supplies or trash
- use of deodorisers and fragrances
- airborne dust or dirt (e.g., circulated by sweeping and vacuuming)

### ***Maintenance activities***

- microorganisms from improperly maintained cooling towers
- airborne dust or dirt
- volatile organic compounds from use of paint, caulk, adhesives, and other products
- pesticides from pest control activities
- emissions from stored supplies

## **Sources Outside Building**

### ***Contaminated outdoor air***

- pollen, dust, fungal spores
- industrial pollutants
- general vehicle exhaust

### ***Emissions from nearby sources***

- exhaust from vehicles on nearby roads or in parking lots, or garages
- odours from dumping sites
- re-entrained (drawn back into the building) exhaust from the building
- unsanitary debris near the outdoor air intake

### ***Soil gas***

- radon
- contaminants from previous uses of the site (e.g., landfills)
- pesticides

### ***Moisture or standing water promoting excess microbial growth***

- rooftops after rainfall
- crawl space

## **OVERVIEW OF INDOOR POLLUTANTS AND THEIR HEALTH EFFECTS**

This section concerns the toxicological properties of the major pollutants and their sources.

Most of these data are from Maroni et al., 1995.

## **Inorganic pollutants**

### **Carbon dioxide**

Carbon dioxide (CO<sub>2</sub>) is a colourless, odourless gas. Solely from his breath, man continuously emits amounts of carbon dioxide and water vapour. Gas-, kerosene- or wood-fuelled appliances can generally be considered the main sources of CO<sub>2</sub>, depending on how combustion exhausts find their way into the indoor atmosphere. In the absence of combustion sources, the indoor CO<sub>2</sub> concentration is considered a useful ventilation indicator.

### **Carbon monoxide**

Carbon monoxide (CO) is a colourless, odourless, and tasteless gas. It is a product of the incomplete combustion of carbon-containing materials, but is also produced by some industrial and biological processes. CO is widely generated indoors by unvented combustion appliances, particularly if they are operated in poorly ventilated rooms. Tobacco smoking is also an important source of indoor CO pollution.

Like oxygen, CO is able to bind to hemoglobin so forming carboxyhemoglobin (COHb); however, CO is about 200 times as effective as oxygen in binding with hemoglobin. This means that when CO is present, hemoglobin will be less available to transport oxygen to the tissues. The health effects of CO exposure are generally discussed in terms of the per cent of COHb in the blood. The level of COHb is directly related to the CO concentration in the air, the duration of the exposure, and the activity level of the individual. As the CO concentration increases or decreases from this point, the COHb level will follow. Normally, metabolic processes in the body will result in a COHb level of 0.5% to 1.0%. Average COHb levels among non-smokers are 1.2%-1.5%. In cigarette smokers the level is about 3%-4% on average, but it may be as high as 10% in heavy smokers. CO can have detrimental effects on the heart and lungs, and on the central nervous system. Levels of 2.5% have been shown to aggravate symptoms in angina pectoris patients. At COHb levels of 10%, the major effects are cardiovascular and neurobehavioural.

### **Nitrogen dioxide**

Nitrogen dioxide (NO<sub>2</sub>) is a gas with a pungent, acrid odour. Generally, NO<sub>2</sub> is emitted from indoor combustion sources. These comprise tobacco smoke, gas appliances, kerosene heaters, woodstoves and fireplaces. Additionally, outdoor air can act as a source of indoor NO<sub>2</sub> pollution. It is an oxidising agent that is highly irritating to mucous membranes, and causes a wide variety of health effects.

Most studies demonstrate substantial changes in pulmonary function in normal healthy adults at or above NO<sub>2</sub> concentrations of 2 ppm. Asthmatics appear to be responsive at about 0.5 ppm, and subjective complaints have been reported at that level. NO<sub>2</sub> increases bronchial reactivity as measured by pharmacological bronchoconstrictor agents in normal and asthmatic subjects, even at levels that do not affect pulmonary function directly in the absence of a bronchoconstrictor. Epidemiological studies suggest that children who are exposed to combustion contaminants from gas stoves have higher rates of respiratory symptoms and illness than other children; NO<sub>2</sub> concentrations in these studies ranged from 0.005 ppm to about 0.3 ppm (WHO, 1987).

### **Particulate matter**

Airborne particulate matter (PM) is a composite of hundreds of different substances that exist as particles extraordinarily heterogeneous in terms of chemistry and size with a high degree of spatial and temporal variability.

The levels of indoor particle levels depend on outdoor and indoor concentrations. Outdoor combustion particles arise from industrial smokestack emissions, vehicle exhaust (from diesel and gasoline), heating exhaust (from coal, or wood), forest fires, and other open fires or incineration (e.g. yard waste and trash burning). The extent to which these outdoor-source particles affect a building's indoor air depends on the building's location, how close it is to the sources, the type of ventilation system in use, the proportion of outdoor air in the indoor air mixture, and the location of the air intakes. Indoor combustion particulate sources include heating appliances, dry-process photocopying machines, cooking appliances and tobacco smoke.

Recent epidemiological studies suggest that PM air pollution, at levels common to many urban and industrial areas, contributes to human morbidity and mortality for cardiorespiratory diseases (Carrer et al.,1999). The principal health effects associated with PM exposure include premature mortality, aggravation of respiratory and cardiovascular disease, changes in lung function and increased respiratory symptoms, changes to lung tissues and structure, and altered respiratory defence mechanisms. Numerous studies of hospital admissions reported positive associations between short-term concentrations of PM and hospital admissions for respiratory symptoms, respiratory diseases (e.g. asthmatic attack, chronic obstructive pulmonary disease and pneumonia) and cardiac diseases. Decreased lung function and increased respiratory symptoms are associated with increased PM concentrations in community epidemiological studies and controlled laboratory exposure studies of laboratory animals and humans. Particularly noteworthy is the observation of these PM-associated effects in children.

Epidemiological data indicate that several subpopulations are at special risk for PM exposure: children, individuals with respiratory diseases (e.g. chronic obstructive pulmonary disease, acute bronchitis, asthma, pneumonia) and cardiovascular disease (e.g. pneumonia), and the elderly.

In Europe, in a recent update and revision of the WHO Air Quality Guidelines no specific values are recommended for PM because there was no exposure level that could be judged a threshold (WHO, 1999). This indicates that a certain risk to health is related to all levels of PM exposure encountered in society. Differently, risk estimates for a number of acute and chronic health outcomes are provided for guidance in decision making-regarding risk assessment and reduction.

### **Asbestos fibres**

Asbestos is a generic term that applies to a group of impure hydrated silicate minerals that occur in various fibrous forms, which are incombustible and separable into filaments. Asbestos fibres belong to the mineral groups of amphibole (such as amosite, crocidolite, tremolite, anthophyllite, and actinolite) and serpentine (chrysotile). Because of its specific technical properties, asbestos has been used in an extremely wide variety of products (about 3000). In the future, legislative restrictions and the success in finding substitutes for asbestos in fibre-cement, brake linings, insulation and many other applications will most probably lead to a decline in asbestos consumption.

Indoor asbestos dust originates from insulation material sprayed on steelwork or ceilings (such material may become highly friable after some years), asbestos plasters, and low-weight insulation plates. Sometimes such materials have been used in direct or close contact with air-conditioning equipment. Even though some of these materials, such as spray asbestos, are no longer used, they are still found in many public buildings. Until the mid-1970s electric storage-heaters and some other electrical household equipment contained asbestos. One of the main applications of asbestos is as asbestos-cement; in this case the release of fibres into the general environment is minimised, since the fibres are essentially 'locked' in the cement matrix. Asbestos cement products, therefore, do not usually pose problems for indoor air

quality. Factors such as renovation and repair, maintenance, external vibrations and vandalism can considerably increase the emission of asbestos dust from indoor sources.

The most serious health effects from indoor asbestos exposure are lung cancer and mesothelioma (cancer of tissue of mesothelial origin).

### **Man-made mineral fibres**

The man-made mineral fibres (MMMMF) discussed in this document will be restricted largely to a subset known as man-made vitreous fibres (MMVF), which are fibres manufactured from glass, natural rock, or other minerals. They are classified according to their source material. Slag wool, rock wool, and glass wool or filaments are produced from slag, natural rock, and glass, respectively. While naturally occurring fibres are crystalline in structure, most man-made mineral fibres are amorphous silicates.

MMMMF are widely used as asbestos substitutes. Fibrous glass accounts for approximately 80% of MMMMF and is mainly used in acoustic and thermal insulation. Textile grades (5-10% of fibrous glass production) are used principally for the reinforcement of resinous materials and in textiles, such as draperies. Less than 1% of the production of glass fibre is in the form of fine fibres used in speciality applications, such as high efficiency filter paper and insulation for aircrafts. Mineral wool (rock wool/slag wool), which accounts for approximately 10-15% of MMMMF production in the USA, is used mainly in acoustic and thermal insulation. In Europe, glass wool and rock wool are produced in approximately equal volumes and are also used for thermal and acoustic insulation.

It seems likely that the main source of emissions of MMMMF (mainly glass fibres) in indoor air is insulation in public buildings or homes. Although quantitative data are not available, emissions are probably highest shortly after installation or following disturbance of the insulation.

Fibrous glass and rock wool fibres (mainly those greater than 4.5-5  $\mu\text{m}$  in diameter) cause mechanical irritation of the skin characterised by a fine, punctuate, itching erythema, which often disappears with continued exposure. Until recently, reports of eye irritation in populations exposed occupationally to MMMMF were restricted to a few isolated cases in the early literature. While carcinogenic effects have been suspected in cases of high occupational exposure, no such studies have been conducted under non-occupational circumstances.

### **Organic pollutants**

In the last decade hundreds of organic compounds have been identified in indoor air. Although most occur at concentrations that are several orders of magnitude below known effect levels, there is appropriate concern about their effect on human health. Some of the compounds are genotoxic and many exhibit toxic, irritant and/or odorant properties.

### **Volatile organic compounds**

The volatile organic compound (VOC) category has been defined by a boiling-point range with a lower limit between 50°C and 100°C and an upper limit between 240°C and 260°C. Volatile organic compounds are ubiquitous in the indoor environment. The number of VOCs detected in indoor air is usually higher than in outdoor air and has been continuously increasing over the past decade. Over 900 VOCs have been identified thus far (EPA, 1989).

Various materials commonly found in schools emit many VOCs. Emission strength varies considerably among sources and also varies with building conditions. Furnishings used in schools, including desks and other work surfaces, open shelving, cabinets, upholstered sofas and chairs, and modular partitions, can produce numerous VOCs, particularly formaldehyde. The irritating emissions from these pressed-wood products come mainly from the resins,

adhesives, and glues. The furnishings usually emit the highest contaminant levels during the first few months after manufacture and installation, and emissions then drop off rapidly. Indoor concentrations may fluctuate widely with temperature, humidity, occupant activities, and ventilation changes. Interior treatments, paints, polyurethane, coatings, sealants, polishes, and cleaners, can also emit VOCs. Dry-cleaned fabrics can emit harmful concentrations of tetrachloroethylene (also known as perchloroethylene or PERC). Polyurethane foam found in cushions and upholstered furniture often emits enough toluene di-isocyanate (TDI) to cause respiratory difficulties in sensitive persons. Studies have shown significant emissions of phenol from upholstered furnishings. However, most of these decrease significantly over time. Furnishings with textured fabric surfaces, particularly upholstered furniture, draperies, and modular partitions, can adsorb or attract VOCs emitted from other sources such as carpeting, paints, copying fluids, and environmental tobacco smoke (ETS). The furnishings then become secondary sources of these contaminants and significantly affect indoor concentrations under certain conditions. Higher temperatures tend to increase emissions from primary and secondary sources and thus increase VOC concentrations.

*Office Equipment* - Most schools contain computer terminals (or video display terminals), photocopying machines, laser printers, electric typewriters, and fax machines. Office equipment emits numerous contaminants, placing an additional burden on the already poor IAQ in many buildings. Equipment type and the processes involved, as well as usage, determine emission potential. The equipment that may present emission problems are: dry-process copying machines, which emit hydrocarbons, respirable suspended particulates, and ozone; wet-process photocopying machines, which emit aliphatic hydrocarbons, other VOCs, and ozone; laser printers and other computer printers, which emit hydrocarbons and ozone; and computer terminals, fax machines, and other electrical equipment, which emit ozone and VOCs. Besides machine emissions during operation, paper documents produced by fax machines, laser printers, and photocopiers can also emit VOCs.

*Floor Coverings* - Carpeting, tiles, and other materials used as floor coverings give off VOCs. The emissions from some carpets may be high initially, and they tend to diminish rapidly, depending on carpet type. These emissions account for odour problems associated with new carpets, and researchers have also linked the chemical to complaints about headache, runny eyes, mucous membrane irritation, dizziness, neurological symptoms, and fatigue after carpet installation. Floor covering adhesives can emit significant VOCs, particularly since they are often used to cover large surface areas. Emissions from adhesives are highest during application and for some time thereafter. Adhesives used to anchor carpeting and floor tiles can still emit significant VOCs as long as one week after application, and lower VOC emissions can continue for at least two weeks. Vinyl floor tiles also emit VOCs. The principal emissions from hardwood floors appear to be from urea-formaldehyde or polyurethane coatings applied to the surface, although adhesives used to glue the parquet to the sub-floor may also contribute to the level of emission. Many emissions have been traced to coatings, which emit high VOC concentrations while still wet and for a few days after application.

*Chemical Emissions from Maintenance Activities* - Regular building maintenance can help prevent IAQ contamination, but cleaning may itself create problems by adding pollutants to the air. Most cleaning materials, air fresheners, and pesticides emit numerous VOCs and other contaminants, creating problems for building occupants and for maintenance or janitorial workers. While workers applying cleaning products face the greatest danger, these products can also affect building occupants. Emissions from cleaning products can linger long after they have been applied, and can affect occupants in areas served by the same ventilation system.

*Cleaning Products* - The chemicals that allow cleaning products to remove dirt, grease, and grime, and to disinfect, are often toxic and irritating to humans. While their toxicity increases at higher concentrations, and thus concerns workers handling the compounds, exposure to

lower concentrations in indoor air can also cause irritation and, potentially, more serious health effects.

*Air Fresheners* - Despite their name, air fresheners rarely do anything to fresh the air or break down any offensive odour. Usually, air fresheners function by: interfering with the ability to smell by deadening nerve endings; coating the nasal passages with an oil film; and disguising one odour with a stronger one. The ingredients in air fresheners themselves can add harmful VOCs to indoor air.

*Outdoor environment* - All buildings exhibit a more or less pronounced exchange between outdoor and indoor air, hence, outdoor air cannot be neglected as a source of contaminants in indoor air. This contribution may be especially important under certain meteorological conditions (high pollution episodes). In mechanically ventilated buildings, polluted outdoor air may contribute to indoor air pollution because of malfunctioning of the ventilation system or an unfavourable location of the air inlet (e.g. close to parking garages or loading docks).

Depending on the pathway of the air contaminant from emission to the moment it starts inducing negative health effects, exposure to VOCs can result in both acute and chronic health effects (Bruinen de Bruin, 1998). VOCs can cause irritation of the eyes and respiratory tract and sensitisation reactions that involve the eyes, skin, and the respiratory tract. Symptoms of VOC exposure could include fatigue, headache, drowsiness, dizziness, weakness, blurred vision, skin irritation, irritation of the eyes and respiratory tract; there is some evidence that VOCs can provoke some of the symptoms typical of the Sick building syndrome (SBS). At higher concentrations, many of these chemicals have been shown to be potent narcotics and cause depression of the central nervous system, and liver and kidney damage. Many of the VOCs are known human carcinogens (benzene) or animal carcinogens (carbon tetrachloride, chloroform, trichloroethylene, tetrachloroethylene, and p-dichlorobenzene). VOCs such as 1,1,1-trichloroethane, styrene, and pinene are mutagens and possible carcinogens. Although there are few risk assessments available for VOCs in indoor air, VOCs appear likely to pose a significant cancer risk (EPA, 1989).

## **Formaldehyde**

Formaldehyde is the simplest and most common aldehyde found in the environment. It is perhaps the most important single indoor air pollutant because of its wide occurrence and its strong irritation potential.

The major sources of formaldehyde affecting human beings are in the indoor environment. They include cigarette smoke and other combustion sources, and urea-formaldehyde resins that are used in large quantities as glues in the manufacturing of wooden products such as particleboard and plywood. Formaldehyde may also be used in urea formaldehyde foam insulation. Formaldehyde can be released over long periods of time, even years, at a slowly decreasing rate. Indoor concentrations are influenced by temperature, humidity, ventilation rate, age of the building, product usage, presence of combustion sources, and the smoking habits of occupants.

Formaldehyde has a pungent odour and has irritating properties causing discomfort. The symptoms displayed after short-term exposure to formaldehyde are: irritation of eyes, nose and throat, together with exposure-dependent discomfort, lachrymation, sneezing, coughing, nausea and dyspnoea. Children have been reported to be more sensitive. A number of reports also show that formaldehyde gas exposure causes direct irritation of the respirable tract. Because of absorption in the upper respiratory tract, higher concentrations of formaldehyde are required to stimulate bronchial receptors than those needed to cause sensory irritation. A number of studies point to formaldehyde as a potential factor predisposing certain groups, particularly children, to respiratory tract infections.

No sufficiently well controlled scientific studies have been made that establish definitely that formaldehyde gas is able *per se* to cause a respiratory tract allergy. Clinical reports describe

tests for respiratory sensitivity to formaldehyde gas in which allergic reactions were elicited, but their interpretation is uncertain. There may be susceptible groups or genetic differences in the population. Occupational studies indicate that 1-2% of the population exposed to high concentrations may develop asthma.

### **Environmental tobacco smoke**

Environmental tobacco smoke (ETS) is the single largest contributor to indoor particulate concentrations in office buildings, residences, and other public buildings where tobacco smoking occurs. In some cases, the particulates attributable to ETS account for 50%-90% of the total particulate concentration. Inhaling ETS is sometimes referred to as 'passive smoking'. ETS consists of:

- Mainstream smoke (inhaled and exhaled by the smoker).
- Sidestream smoke (from the end of the cigarette between puffs).
- Vapour-phase components (diffused through the cigarette paper).

Burning cigarettes emit numerous airborne contaminants, including particulates in sidestream smoke and exhaled mainstream smoke. Approximately 85% of passive smoke exposure comes from sidestream smoke and 15% from mainstream smoke. Sidestream and mainstream smoke components are similar in number, but vary in composition. Some ETS components are more prevalent in sidestream smoke. For example, carbon monoxide is 2.5 times higher in sidestream smoke than in main

stream smoke. Overall particulate matter is up to three times more concentrated in sidestream smoke. Therefore, many toxic and carcinogenic compounds are at higher levels in sidestream smoke, though dilution by room air significantly reduces the concentrations inhaled by non-smokers compared with those inhaled by smokers.

Inhaling these particles and other ETS constituents can cause various health effects that have been reviewed by the USA Environmental Protection Agency (EPA, 1992) and Jaakkola (Jaakkola, 2000). The widespread exposure to ETS presents a serious and substantial health impact, and concerning respiratory effects in children there is evidence that:

- ETS exposure is causally associated with an increased risk of lower respiratory tract infections such as bronchitis and pneumonia.
- ETS exposure is causally associated with additional episodes and increased severity of symptoms in children with asthma.
- ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.
- ETS exposure is causally associated with increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract irritation, and a small but significant reduction in lung function.

Several studies support the conclusion that ETS causes lung cancer (EPA, 1992; Jaakkola, 2000). Childhood ETS exposure may influence the development of lung cancer in adult life, since childhood may represent a more susceptible age period, and because childhood exposure adds to the total cumulative exposure.

Many people do not tolerate tobacco smoke very well. Non smokers exposed to smoke often complain of intolerance to its odour, eye irritation, nasal symptoms, cough and headache.

### **Pesticides**

Pesticides, applied by exterminators or building maintenance staff, can introduce harmful contaminants into indoor spaces. The substances that make the pesticides fatal to rats, mice, insects, and other pests are also toxic to humans. Exposure to chemical pesticides can have serious health effects, ranging from mucous membrane irritation to systemic toxic effects, depending on the exposure concentration. These chemicals generally compose only about 0.5%-5% of the volume of ingredients in the pesticide solutions, and are known as the 'active'

ingredients because they are what kills the pests.

The 'inert' ingredients of pesticide solutions, such as xylene, n-decane, 1,1,1-trichloroethane, mesitylene, methyl ethyl benzene, cumene, and kerosene, dissolve the active ingredients and allow for better dispersion on application. But these 'inert' ingredients can have their own adverse health effects.

## **Biocontaminants**

Biological agents are natural and ubiquitous components of the earth's ecosystems. In the last few decades biocontaminants and their by-products have increased in many indoor environments. Biocontaminants that can contribute to IAQ problems include bacteria, fungi and fungal spores, viruses, algae, parasites (free-living amoebae), pet dander allergen, dust mite allergens, plant pollen and insect pest allergens. Microbial growth and other biocontaminants in indoor environments have been associated with human health effects, including allergic and irritant responses, infectious diseases, respiratory problems, and hypersensitivity reactions. Factors affecting indoor biocontaminant proliferation include:

- Sources of microorganisms: outdoor air, air handling systems, humidification systems, building materials and furnishings, occupants, pets, and houseplants.
- Water sources: roof and plumbing leaks, water migration, condensation, houseplants, humidifiers, human occupants, and aquariums.
- Nutrient (or food) sources: dust, dirt, food, water, house plants, detritus (dead plant tissue), building materials, and furnishing surfaces.
- Temperature: indoor ambient and surface temperatures in the range most favourable to the growth of many micro-organisms.

The growing significance of biocontaminants in indoor environments over the last few decades can be attributed to the following:

- Building envelopes have become tighter.
- Less outdoor air is used in heating, ventilating and air conditioning (HVAC) systems.
- Microorganisms shed indoors by humans are not readily diluted by mixing with outdoor air.
- Energy conservation measures have contributed to the build-up of moisture in indoor environments, which facilitates the growth of microorganisms.
- Neglected maintenance programs have contributed to the build-up of dirt and debris (potential nutrients for microorganisms) in HVAC systems.

The four major categories of biological particles which affect indoor air quality are viruses, bacteria including actinomycetes, fungi, including moulds and yeast, mites and their faeces, and dander from pets and other furred animals.

## **Viruses**

Some viral illnesses such as the common cold and measles may be transmitted via indoor air, e.g. measles in schools and rates of viral infection in buildings with HVAC systems with recirculating air may be higher than in naturally ventilated buildings. However, it is generally held that person-to-person transmission is the principal cause of outbreaks of most viral diseases, e.g. mumps.

## **Bacteria**

In indoor air, the main sources of bacterial aerosols are usually humans and animals, but disturbing previously settled dust may also create bacterial aerosols. Furthermore, humidifiers and drainage fans in HVAC systems are potential sources of airborne bacteria.

Together with fungi, bacteria (including their antigens and endotoxins) in humidifiers are implicated in humidifier fever, a disease with elements of both toxic and allergic

manifestations. Infrequently, bacteria in buildings may also be the cause of extrinsic allergic alveolitis in occupants. Bacteria in the air of houses or offices reported to have caused extrinsic allergic alveolitis among occupants are *Bacillus subtilis* and *Pseudomonas aeruginosa*, and thermophilic actinomycetes.

Occasionally, bacteria that cause important infectious diseases and that are present in droplet nuclei from individuals shedding pathogenic agents, e.g. *Mycobacterium tuberculosis*, can be rapidly dispersed throughout an enclosed environment via a recirculation system. Of course, other medically important bacteria such as *Legionella* may also be transmitted in aerosols and can cause respiratory problems. This Gram-negative bacterium causes two types of disease, Legionnaires' disease and Pontiac fever. Legionnaires' disease is a serious type of pneumonia, which takes its name from a serious outbreak at a meeting of the American Legion. The disease has a considerable mortality rate and appears mostly as sporadic or hyperendemic cases. Epidemics are rare. The frequency of sporadic cases is estimated to be 2% of all hospitalised pneumonias in the United Kingdom, and 10% of community-acquired pneumonias in France and Germany (WHO, 1990). Less than 5% of those exposed appear to develop the illness; in 10-15% of these the illness is fatal.

Pontiac fever, named after an outbreak in 1968 in Pontiac, USA, is caused by a number of the *Legionella* species, is milder and appears as a non-pneumonic fever. The illness resolves spontaneously in 2-5 days. No fatal cases have been reported in relation to Pontiac fever. This disease mainly appears as epidemics, and 95% of those exposed to aerosols will become ill. However, the incidence of Pontiac fever in the general population is unknown and reports of sporadic cases are unlikely to be made even if they are recognised (WHO, 1990).

### **Mould/Fungi**

Mould growth in any building is in itself undesirable and uncontrollable, but in addition to being a potential health hazard it is an indicator that conditions of relative humidity may also be favourable for yeast and bacteria. Although most investigations have shown that far fewer respiratory patients with suspected allergy react to moulds than to house dust mites and animal dander, mould allergy among atopic children is frequent and may be severe.

Fungi can be the cause of individual cases of rhinitis, asthma, extrinsic allergic alveolitis and atopic allergic dermatitis, and have been invoked as a cause of humidifier fever, and may have a wider role in respiratory health via non-allergic mechanisms. Such mechanisms may involve mycotoxins in inhaled mould spores. Wall lipopolysaccharide fractions of Gram-negative bacteria are also toxic, and provoke symptoms very similar to those of humidifier fever.

Among the approximately 100,000 known species of fungi, those of prime interest in indoor environments are moulds belonging to the Deuteromycetes or *Fungi imperfecti*, but a few species in the Mucorales, Ascomycetes and wood-rotting Basidiomycetes, and some yeast are also of relevance.

The majority of fungi are saprophytic, utilising dead organic material for food. Providing temperature and moisture conditions are met, many species can utilise a wide range of organic materials. This ranges from plant or animal remains to materials such as cellulose, paint or stored products. The majority of the fungi in indoor environments grow at 10-35°C. The water content of these materials is the most critical factor in the development of fungi.

Unless scrupulously clean, indoor environments offer a wide variety of substrates for growth. Damp, non-living organic material can be quickly colonised. Condensation is the principal source of the moisture needed for the growth of fungi on the internal surfaces of domestic dwellings. Besides superficial condensation, interstitial condensation within porous building materials such as concrete, brick and plaster, may provide a reservoir allowing fungal growth to continue in circumstances under which the surface would otherwise dry out. Interior dampness problems are usually related to construction faults, such as inadequate insulation or 'cold-bridging', in combination with inadequate ventilation and/or the pattern of usage of

these houses. Condensation and mould problems may also be encountered in 'tight' houses built to conserve energy, particularly when measures to prevent excessive generation of moisture are not taken e.g. in cooking or laundering.

A large number of species can be found in indoor air. The most common are likely to belong to the genera *Cladosporium*, *Penicillium*, *Alternaria* and *Aspergillus*. In addition to being implicated in cases of atopic allergic dermatitis their causative role in individual cases of respiratory allergy is well-known, their overall significance in respiratory health is still debated. Epidemiological studies have observed a strong association between reported dampness and mould in houses and respiratory symptoms in children (Brunekreef et al., 1989; Platt et al., 1989) and adults (Dales et al., 1991a) occupying the houses.

Mycotoxins are produced by a wide range of moulds and are located in hyphae and spores, often being present in particularly high concentrations in the latter. It is well established that ingested mycotoxins can cause illness and death in humans and animals.

Although there is no definitive proof in humans, it appears distinctly possible that inhalation of high concentrations of mould spores containing these toxins may deleteriously affect macrophage functions such as phagocytosis of living and non-living particles in the lung, and therefore affect respiratory health. The effects on the immune system could also compromise health by reducing resistance to other microorganisms, perhaps resulting in chronic health problems.

Lastly, fungi produce a complex mixture of volatiles that are frequently evident as 'mouldy smells'. The volatiles are mixtures of alcohols, ester, aldehydes, various hydrocarbons and aromatics, and a large number have been identified. Some individuals do not react to these, some become nauseous and others may be quite ill.

Mites and dander from pets and other furred animals are discussed in the next paragraph.

## **Allergens in indoor air**

House dust mites, pets, insects, plants, moulds and chemical agents in the indoor environment are important causes of allergic diseases (Lowenstein et al., 1986; Platts-Mills & De Weck, 1988; Infante-Rivard, 1993; Carrer et al., 2000). House dust mites and their debris and excrements that contain the allergens are normally found in the home in beds, mattresses, pillows, carpets and furniture stuffing, but they have also been found in office environments. Domestic animals such as cats, dogs, birds and rodents may cause allergic asthma and rhinoconjunctivitis. The exposure usually occurs in homes, but also in schools and kindergartens where domestic animals are kept as pets or for education; moreover cat and dog owners can carry allergens to public areas in their clothes. Outdoor allergens such as pollens and moulds may penetrate into the indoor environment through open windows, doors or ventilation systems.

### **Dust mites**

*Dermatophagoides pteronyssinus* and *D. farinae* are the most important mite species. They are prevalent in climates where winters are humid and mild. Humidity and temperature are the most critical factors in the survival and development of house dust mite populations: for *D. pteronyssinus* the optimum conditions are 25°C and a relative humidity of 70-80%.

The major mite allergens are now well characterised and purified, in particular Der p I for the *D. pteronyssinus* and Der f I for the *D. farinae*; they are faecal allergens. Skin scales represent the principal food source of house dust mites, primarily from man. (Platts-Mills and De Weck, 1988).

Mite allergen levels in house dust do not correlate well with mite numbers in house dust; dead

or degraded mite bodies retain their allergenic properties. The dust mite allergens may become airborne during indoor activity. These allergens are normally found in homes: high allergen values are found in mattresses and significantly higher allergen values are found in carpeted living rooms and bedrooms than in uncarpeted living rooms and bedrooms. It is important to note that dust mite allergens can also be found in other environments, such as offices and schools.

### **Dander from furred animals**

Domestic animals such as cats, dogs, birds and rodents may cause allergic asthma and rhinoconjunctivitis. Prevalence studies of hypersensitivity to cat allergens in unselected populations have shown a frequency of positive skin reactions of around 20%. The prevalence of dog allergy in unselected populations is reported to be 4-15%. The allergens derived from pets are mostly associated with dander, hair, saliva and/or urine.

Dander is the most important source of cat allergens; the major allergen released by the cat, named Fel d I, has been isolated, characterised and standardised (Anderson and Baer, 1981). This allergen is released in indoor environments with dander and shed hair (Lowenstein et al., 1986). The allergen becomes airborne, presumably after it dries and flakes off the fur, on particles from 1 to 10 µm in diameter, mostly on 5 µm particles. The indication that airborne Fel d I is associated with small particles which remain airborne for long periods, may explain the distinctive rapid onset of asthma or rhinitis in patients allergic to cats entering a house with a cat (Luczynska et al., 1990). In houses where at least one cat is present, allergen concentrations were found to range from 250 to 1,140 ng/m<sup>3</sup> air. The concentration of Fel d I in house dust from houses without cats is from below the limit of detection to 100,000 ng/g dust, in houses where cats were present the concentrations ranged from usually more than 8 ng Fel d I/g dust to around 300,000 ng/g (Lowenstein et al., 1986; Wood et al., 1988; Luczynka et al., 1990).

The major allergen from the dog, Can f I, has also been isolated, characterised and standardised (Schou et al., 1991). The concentrations of dog allergens found in house dust vary widely. In house dust sampled in houses without dogs, concentrations of 110-82,500 IU/g have been found, and in houses where dogs are present concentrations of 1,100-585,000 IU/g have been reported (Wood et al., 1988). Dog allergens have also been detected in mattress dust. In houses where dogs are present, dust samples contain generally more than 10 ng Can f I/g dust.

The level of pet-derived allergenic material in homes where pets are kept is also determined by the thoroughness of cleaning in the building, in particular the removal of allergenic material produced in the house. The level is independent of environmental factors such as humidity, temperature, and the quality of the building.

Exposure can occur also in other places, because cat and dog owners can carry allergens in their clothes to other environments, especially public areas (Custovic et al., 1994; Custovic et al., 1996). Curtains, mattresses, sofas and soft toys have been reported to be the most important reservoirs of dog and cat allergens (Munir et al., 1995).

### **Insects**

Shed skin scales, dried secretions and faecal particles from insects may cause allergic asthma and rhinoconjunctivitis. In particular, cockroaches are an important source of allergens in USA homes with poor sanitary conditions (Chapman, 1993). The allergens from the cockroaches, Bla g 1 and 2, have been characterised and standardised (Custovic et al., 1996).

### **Microbial allergens**

A wide range of microorganisms, including thermophilic actinomycetes, moulds, bacteria, amoebae and nematodes, has been described as sources of offending allergens. Persistent

damp areas, particularly bathrooms and basements, may support abundant mould growths indoors, but also water seepage in building material causing damp ceilings, walls, carpeting and furniture may provide favourable conditions for the growth of moulds. Furthermore, draught-proofed ('tight') buildings may offer ideal conditions for mould growth when indoor humidity is high and moisture condensation on cold areas or on walls or windows occurs (Maroni et al., 1995).

Contaminated humidifiers in homes, industrial and non-industrial buildings, and cars can generate aerosols loaded with micro-organisms and debris of micro-organisms; they have been associated with allergic asthma, humidifier fever and extrinsic allergic alveolitis.

It has to be pointed out that non-immune specific inflammation is more important than allergic reactions in the respiratory health effects induced by inhaled microbial agents, but these effects may be stronger and occur more frequently in atopics and asthmatics.

### **Green plants**

Green plants indoors should be considered potential allergen sources and should be investigated with allergological tests; examples are *Ficus benjamina* (weeping fig) and *Spathiphyllum floribundum* (spathe flower).

### **Chemical agents**

Various indoor chemical pollutants have been shown to be related to asthma, including nitrogen dioxide from gas cooking (Hasselbald et al., 1992), environmental tobacco smoke (Willers et al., 1991), formaldehyde and VOCs (Norback et al., 1995). There are some indications that emission of formaldehyde and VOCs from newly painted indoor surfaces may cause asthma-like symptoms (Wieslander et al., 1997). Also some chemical constituents of floor cleaning materials have been recognised as a possible cause of asthma in indoor environments (i.e. colophony based products such as pine oil and tall oil, and benzalkonium chloride) (McCoach et al., 1999).

Exposure to various indoor air pollutants, namely nitrogen dioxide, ozone, formaldehyde, environmental tobacco smoke, and VOCs, can also impair specific immune responses and bring about subsequent immune-mediated pulmonary inflammation (Gilmour et al., 1996).

### **Outdoor allergens**

Outdoor allergens, such as pollens and moulds may penetrate into the indoor environments through open windows, doors or ventilation systems. Mould allergens are mostly found outdoors, however they may penetrate into the indoor environments vehiculated by pollens.

## **HEALTH EFFECTS RELATED TO INDOOR AIR QUALITY**

### **Building-related illness**

Building-related illness (BRI) is a term referring to illness brought on by exposure to the building air, where symptoms of diagnosable illness are identified (e.g. certain allergies or infections) and can be directly attributed to environmental agents in the air. Legionnaire's disease and hypersensitivity pneumonitis are examples of BRI that can have serious, even life-threatening consequences.

### **Allergic diseases associated with exposure to indoor air pollution**

Various reports have linked indoor allergens with one or more of the following allergic

manifestations among the occupants (Maroni et al., 1995):

- Rhinitis, with 'hay fever' symptoms such as nasal congestion, runny nose, sneezing, conjunctivitis and lachrymation.
- Asthma, with symptoms which include wheeze, tightness of the chest and shortness of breath.
- Extrinsic allergic alveolitis (hypersensitivity pneumonitis), with acute pneumonia-like bouts of fever, cough, tightness of the chest and lung infiltration, or chronic development of cough, shortness of breath and infiltration of lungs.
- Humidifier fever, with symptoms including fever, chills, muscle ache and malaise, but no obvious respiratory effects.

Rhinoconjunctivitis and asthma may be caused by exposure to pollutants acting either as allergens or irritants; immunological specific IgE reaction to an airborne allergen is a major component of these diseases, but non-specific hypersensitivity is also important for diseases occurring on exposure to irritants in the indoor air.

The main symptoms of allergic rhinoconjunctivitis are itching of the eye and/or the nose, sneezing, watery nasal secretion and some stuffiness of the nose; the severity of symptoms varies in relation to the level of exposure to the allergen and to certain irritants. Allergic rhinoconjunctivitis is especially prevalent among children and young adults. Individuals often suffer from both allergic rhinoconjunctivitis and allergic asthma.

Asthma is characterised by reversible narrowing of the lower airways and airway inflammation. Pulmonary function during an attack shows an obstructive pattern, in serious cases together with reduced ventilation capacity. Asthma may cause death. The costs of medical care are also considerable in terms of hospital admissions, medication, and lost workdays.

Extrinsic allergic alveolitis, also called hypersensitivity pneumonitis, is characterised by recurrent bouts of pneumonitis or milder attacks of breathlessness and flu-like symptoms; studies of the pulmonary function during an acute episode usually show a restrictive pattern with a decreased oxygen diffusion capacity. The disease consists of an inflammatory reaction in the alveoli and bronchioles, involving circulating antibodies and a cell-mediated immunological response to an allergen. The disease has in a few cases been associated with exposure to indoor air pollutants in homes and offices, most frequently with a humidifier contaminated with bacteria, fungi or protozoans.

Allergic asthma and extrinsic allergic alveolitis resolve with cessation of exposure to the allergen; continued exposure in sensitised persons may result in permanent lung damage and death from pulmonary function failure.

Humidifier fever is a flu-like illness associated with breathlessness, where X-ray abnormalities are usually absent. The disease normally occurs among persons exposed to humidification systems contaminated with microbial growth. The symptoms typically occur 4-8 hours after exposure on the first day back at work after a weekend, but resolve within 24 hours. Despite continuous exposure the disease does not recur until after the next weekend. Even though pulmonary functional changes are seen during attacks of humidifier fever, the disease does not lead to permanent lung damage. The exact cause of this disease is not known. In particular, it is not clear if and how the immunological system is involved; likely the disease is caused by deposition of immuno-complexes in tissues.

### **Sick building syndrome**

The term sick building syndrome (SBS) is used to describe cases in which building occupants

experience acute health and comfort effects that are apparently linked to the time they spend in the building, but in which no specific illness or cause can be identified (Maroni et al., 1995). The complaints may be localised in a particular room or zone or may be widespread throughout the building. Many different symptoms have been associated with SBS, including respiratory complaints, irritation, and fatigue. Symptoms commonly attributed to IAQ problems include: headache, fatigue, shortness of breath, sinus congestion, cough, sneezing, eye, nose, and throat irritation, skin irritation, dizziness, nausea. Odours are often associated with a perception of poor air quality, whether or not they cause symptoms.

Environmental stressors such as improper lighting, noise, vibration, overcrowding, ergonomic stressors, and job-related psychosocial problems (such as job stress) can produce symptoms that are similar to those associated with poor air quality.

A small percentage of the population may be sensitive to a number of chemicals in indoor air, each of which may occur at very low concentrations. The existence of this condition, which is known as multiple chemical sensitivity (MCS), is a matter of considerable controversy. MCS is not currently recognised by the major medical organisations, but medical opinion is divided, and further research is needed.

### **Individuals susceptible to IAQ effects**

Groups that may be particularly susceptible to the effects of indoor air contaminants include, but are not limited to:

- . allergic or asthmatic individuals;
- . people with respiratory disease;
- . people with a suppressed immune system;
- . contact lens wearers.

Some other groups are particularly vulnerable to exposure to certain pollutants or pollutant mixtures. For example, people with heart disease may be more affected by exposure to lower levels of carbon monoxide than healthy individuals. Children exposed to environmental tobacco smoke have been shown to be at a higher risk of respiratory illnesses and those exposed to nitrogen dioxide have been shown to be at higher risk of respiratory infections.

Symptoms that are limited to just a few persons can also occur when the pollutant is confined to their area. In other cases, complaints may be widespread. Reactions can differ by degree, but can also differ by type in different people.

### **REFERENCES**

Anderson MC, Baer H. Allergenicity active components of cat allergen extracts. *J Immunol* 1981; 127: 972-5.

Bruinen de Bruin Y. Comparative study of the strategies for human exposure assessment to benzene levels in ambient air. Environmental and Occupational Health Group, Wageningen Agricultural University, 1998: 9-15 (Thesis).

Brunekreef B, Dockery DW, Speizer FE et al. Home dampness and respiratory morbidity in children. *Am Rev Resp Dis* 1989; 140: 1363-7.

Burney PGJ. Evidence for an increase in atopic disease and possible causes. *Clin Exp Allergy* 1993; 23: 484-92.

Carrer P, Alcini D, Cavallo D, Lovato L, Vercelli F, Visigalli F, Bollini D, Ghalandar R, Maroni M. Daily personal exposure to air pollutants of office workers in Milano. *Proceedings*

of Healthy Buildings/IAQ '97, JE Woods, DT Grimsrud, N Boschi (eds.), Washington 1997; 2: 249-54.

Carrer P, Cavallo D, Maroni M, Foà V. Airborne particles and human health: state of the knowledge and research perspectives. *Eur J Oncol*, 1999; 4, 5: 545-51.

Carrer P, Maroni M, Alcini D, Cavallo D. Allergens in indoor air: environmental assessment and health effects. *The Science of Total Environment*, 2000, in press.

Chapman MD. Cockroach allergens: a common cause of asthma in North American cities. *Insights in Allergy* 1993; 8: 1-8.

Custovic A, Taggart SCO, Woodcock A. House dust mite and cat allergen in different indoor environments. *Clin Exp Allergy* 1994; 24: 1164-8.

Custovic A, Green R, Taggart SCO, Smith A, Pickering CAC, Chapman MD, Woodcock A. Domestic allergens in public places II: dog (Can f 1) and cockroach (Bla g 2) allergens in dust and mite, cat, dog and cockroach allergens in the air in public buildings. *Clin Exp Allergy* 1996; 26: 1246-52.

Dales RE, Zwanenburg H, Burnett R, Franklin CA. Adverse health effects in adults exposed to home dampness and mold. *Am Rev Resp Dis* 1991; 143: 505-9.

Environmental Health Protection Agency (EPA). Air quality criteria for particulate matter and sulfur oxides. vol. I, II and III. Report N. EPA-600/8-82-029, 1982.

Environmental Health Protection Agency (EPA). Report to Congress on Indoor Air Quality. vol. II, Assessment and control of indoor air pollution. Report N. EPA/400/1-89-001C, 1989.

Environmental Health Protection Agency (EPA). Respiratory Health Effects of Passive Smoking: Lung Cancer and other Disorders. Report N. EPA/600/6-90-006F, 1992.

Etkin DS, Vogt C. Indoor air quality in schools. Cutter Information Corporation, 1996.

Gilmour MI, Park P, Selgrade MJ. Increased immune and inflammatory responses to dust mite antigen in rats exposed to 5 ppm NO<sub>2</sub>. *Fundam Applied Toxicol* 1996; 31(1): 65-70.

Hasselblad V, Eddy DM, Kotchmar DJ. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J Air Waste Manage Assoc* 1992; 42: 662-71.

Infante-Rivard C. Childhood asthma and indoor environmental risk factors. *Am J Epidemiol* 1993; 137: 834-44.

Jaakkola MS. Environmental tobacco smoke and respiratory diseases. In: Annesi-Maesano I, Gulsvik A, Viegi G (eds). *Respiratory epidemiology in Europe. European Respiratory Monograph 2000; Monograph 15, vol. 5: 322-83.*

Lowenstein H, Gravesen S, Larsen L, Lind P, Shwartz B. Indoor allergens. *J Allergy Clin Immunol* 1986; 78: 1035-9.

Luczynska CM, Li Y, Chapman MD, Platts-Mills TAE. Airborne concentrations and particle

size distribution of allergen derived from domestic cats (*Felis domesticus*). Measurements using cascade impactor, liquid impinger and a two-site monoclonal antibody assay for Fel-d-1. *Am Rev Respir Dis* 1990; 141: 361-7.

Maroni M, Seifert B, Lindvall T (eds). *Indoor Air Quality. A comprehensive reference book.* Elsevier, 1995.

McCoach JS, Robertson AS, Burge PS. Floor cleaning materials as a cause of occupational asthma. *Proceedings of Indoor Air '99*, Raw G, Aizlewood C, Warren P (eds.), Edinburgh 1999; vol.5: 459-64.

Munir AKM, Einarsson R, Dreborg SKG. Mite (Der p I, Der f I), cat (FEL d I) and dog (Can f 1) allergens in dust from Swedish day-care centres. *Clin Exp Allergy* 1995; 25: 119-26.

Norback D, Bjornsson E, Janson C, Widstrom J, Boman G. Asthmatic symptoms and volatile organic compounds, formaldehyde, and carbon dioxide in dwellings. *Occup Environ Med* 1995; 52: 388-95.

Platts-Mills TAE, De Weck AL. Dust mite allergens and asthma: a worldwide problem. *International Workshop Report Bull World Health Org* 1988; 66: 769-80.

Platt SD, Martin CJ, Hunt SM, Lewis CW. Damp housing, mould growth and symptomatic health state. *Br Med J* 1989; 298: 1673-8.

Schou C, Svendsen UG, Lowenstein H. Purification and characterization of the major dog allergen, can-f-1. *Clin Exp Allergy* 1991; 21: 321-8.

Viegi G, Carrozzi L, Paoletti P et al. Effect of some indoor environmental factors on respiratory symptoms and lung function in a sample of young nonsmokers in North Italy. *Aerobiologia* 1991; 7: 152-9.

World Health Organisation (WHO). *Air Quality Guidelines for Europe.* WHO Regional Publications, European Series N. 23, WHO Regional Office for Europe, 1987.

World Health Organisation (WHO). *Epidemiology, prevention and control of Legionellosis.* Bull. WHO 1990; 68: 155-62.

World Health Organisation (WHO): *Air Quality Guidelines for Europe.* WHO Regional Publications, European Series 1999. World Health Organisation, Regional Office for Europe, Copenhagen, in press. Internet address: <http://www.who.dk>

Wieslander G, Norback D, Bjornsson E, Janson C, Boman G. Asthma and the indoor environment: the significance of emission of formaldehyde and volatile organic compounds from newly painted indoor surfaces. *Int Arch Occup Environ Health* 1997; 69: 115-24.

Willers S, Svenomius E, Skarping G. Passive smoking and childhood asthma. *Allergy* 1991; 46: 330-4.

Wood R, Eggleston PA, Lind P, Ingemann L, Shwartz B, Gravesen S, Therry D, Wheeler B, Adkinson NF. Antigenic analysis of house dust samples. *Am Rev Respir Dis* 1988; 137: 358-63.