# OTHER PEOPLE'S TOBACCO SMOKE

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# 1. How Inadequate Ventilation may Impair Health and Well-Being

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In the space available all that I can hope to do is to present a physician's view of limited aspects of this very large topic. In doing so, I propose to exclude from discussion the effects of work exposure to industrial chemicals since for these there exist guidelines, regulations and standards (e.g. threshold limit values) which have evolved in the light of careful and systematic assessment of relevant exposure and health data. Provided that there is compliance with these regulations and guidelines, no significant adverse effects on health are to be expected from such exposure. I will, therefore, confine my remarks to the kinds of indoor air pollution that may be encountered in offices, hotels, public buildings, public transport, and homes.

## 1. 'Health' and 'Well-Being'

For many people, '*health*' is most easily understood not as a positive attribute but as a '*lack of disease*'. In other words, 'health' is a '*normal*' condition not a '*supranormal*' state, whereas disease is '*subnormal*', and constitutes a state in which health is impaired. Of course there are some rather bouncy individuals who behave as though they are fuller of health and energy than the rest of us. They can be quite tiresome and irritating. Indeed, I suspect they can adversely affect the health of others!

A doctor checks a human subject for evidence of ill health by firstly asking a series of questions about the various functions of his her body and then by physical examination to check the accuracy of the answers to those questions. The following is a typical check list:

Appetite
Maintenance of body weight
Bowel function
Micturition

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Breathing
Cardiac function
Muscular movements
Joints
Eyesight
Hearing
Taste
Smell
Touch
Nervous system
Sleeping
Skin condition
Libido
Sexual performance
Presence of abnormal lumps and bumps
Presence of abnormal bleeding or discharge, etc.

If all these functions are in good order, a person is deemed to be 'in good health'. If one or more of the functions is impaired, the doctor will endeavour to distinguish between minor ailments (transient or chronic, remediable or not); acute diseases (transient or recurrent, remediable or not, progressive or static, infectious or not, etc.); and chronic diseases (static or progressive, potentially fatal or not, etc.).

'*Well-being*' is a much more transient quality. A person who is not suffering from any condition that a doctor would class as a disease may nevertheless feel ill-at-ease, irritated, or uncomfortable. Thus a healthy person may be annoyed by noise, by bad or stale smells, by draughts, by stuffy or irritant atmospheres, or because he/she is continuously too hot or too cold, etc.

Whereas most of the problems associated with exposure to industrial chemicals are relevant to '*health*', some of those associated with non-industrial indoor air pollution are concerned solely with '*well-being*'.

Of course, the distinction between effects on health and effects on well-being is not absolute. There is overlap. Things which in the short-term merely temporarily interfere with well-being, might, if exposure is heavy and/or prolonged, constitute risks to health. This, for instance, could be true for exposure to relatively low-intensity noise of a particular wave length.

## 2. Expectation with regard to the quality of indoor air

Less than 25 years ago in Britain the air available for many people to breathe, both within their homes and outside them, was heavily polluted. Outside air was full of industrial pollutants and smoke from the burning of soft coal as a domestic fuel. Many people suffered from chronic bronchitis

and emphysema, which impaired their breathing making it difficult for them to expel air from their chests and causing them to produce and cough-up copious amounts of mucus each day. Superimposed on this, people so affected might once or twice a year, usually in February and November, develop febrile bronchitis because bacteria could flourish in their soggy lungs. As a child growing up in London, I thought that coughing and spitting were the hallmarks of normal masculine maturity! Even some people who lived mainly indoor lives suffered from the effects of air pollution. Foul, particle-laden air with a high sulphur dioxide content crept into houses under doors and through windows, and soft-coal burning domestic fires not infrequently added directly to the indoor air pollution when chimneys got blocked or the wind blew the wrong way. Many a non-smoking housewife suffered from chronic bronchitis, as I have described it above, in those days.

The picture I have just painted appears in stark contrast to the present day scene in Britain. So-called 'pea-souper fogs' and 'smogs' are things of the past. There is strict control of the burning of smoke-generating fuels and new cases of chronic bronchitis with the copious production of phlegm are nowadays only infrequently encountered.

Despite this revolution in the quality of outside ambient air and reduction in the incidence of air-pollution-related disease, it is only during the last decade or so that indoor air pollution has become a major topic of interest and debate. Twenty years ago the term '*Sick Building Syndrome*' would have raised a giggle, but now it is generally accepted as a meaningful entity. Why should this be so?

I suggest that the main reason is that people's expectations, with regard to the thermal environment and quality of indoor air have greatly increased during the last two decades or so. Meanwhile, even for those living in cities, outside air has, except in relatively infrequent climatic conditions, ceased to be unpleasant to breathe. However, while these improvements in external air have been taking place, new building materials, furnishings and fabrics, which slowly release solvents and other unpleasant smelling chemicals into indoor air, have come increasingly into everyday use. Also, people have become used to higher indoor ambient temperatures which can only be maintained if heat is conserved by closing windows and by the use of ventilation systems which conserve heat, usually by recycling warm air. In comparison with the warm recirculating and sometimes slightly smelly indoor air, outside air has come to be regarded as having a positive quality of 'freshness'. I suspect that 'freshness' is, in reality, nothing more than an absence of smelly chemicals and a somewhat lower temperature.

When I was a child, I lived in a house in south east London of which only one small room was heated. On a cold winter's evening the entire family would gather there including my pipe-smoking father, a Scottish terrier with bad breath, and granny whose use of camphor in her wardrobe kept the

entire neighbourhood free from moths! The room was heated by an open fire and not all the smoke from this went straight up the chimney. Today I would describe the atmosphere in that room as foul, but then I knew no better, and I was so glad to be warm and not subjected to the cold dank air that pervaded other rooms in the house, that I never dreamed of complaining.

## 3. What is 'Sick Building Syndrome' (SBS)?

This term was originally applied to complaints by users of buildings where there were no obvious faults in the design or operation of ventilation systems. However, a better understanding of how ventilation systems should be designed and how their functioning should be tested has led to the realisation that poor design and/or inadequate ventilation is the main cause of SBS.

WHO (1984) listed 4 categories of symptoms as typical of SBS:-

- Sensory irritation of skin, dry mucous membranes in the upper airways, headache and abnormal taste.
- Odour.
- General symptoms such as fatigue, dizziness and nausea.
- Lower airway and gastro-intestinal symptoms.

This spectrum of symptoms and their slow build-up in persons working in 'sick' buildings is distinguishable from the complaints that can be elicited from workers in other buildings. In the latter case, symptoms such as discomfort, annoyance, eye and throat irritation, odour, sneezing and stuffy or runny nose are characteristically of acute onset, have obvious causes and are more transient. Characteristically, the symptoms of SBS quickly ameliorate when people leave an affected building.

The frequency of SBS varies widely in different countries and regions. In worst affected regions a high percentage of new and remodelled buildings is affected.

Two common features of affected buildings are that they are energy-efficient and constructed as airtight containers. Windows cannot be opened and people who work in such buildings resent not being able to control their own air environment. Also, common sense suggests that well-being actually requires a certain amount of variation within tolerable ranges in terms of air flow, temperature, and humidity, etc. (Gerlach, 1974). If all these qualities remain absolutely constant, even though they are within the tolerable range, the human spirit is somehow deadened. Despite much effort and many conferences, little progress has been made towards relating particular symptoms of SBS to exposure to specific indoor air pollutants. One of the reasons for this is that under conditions of poor ventilation, the levels of many different pollutants build up. Secondly, in the case of effects on health

due to micro-organisms, it is something of a matter of chance which particular organisms colonise the ducting, etc.

## 4. Which common indoor-air properties and pollutants are a cause for medical concern?

*Temperature and humidity* are clearly of great importance with regard to well-being. However, except perhaps for persons with existing health problems (e.g. asthma, other allergies, bronchitis, common colds, sinusitis, rheumatism, etc.) variations in temperature and/or humidity of the extent encountered in indoor air environments are not a matter for medical concern. Heat stroke may occur in industrial environments but is rarely encountered under ordinary air conditions (Shibolet *et al.*, 1976). Cold stress is generally regarded as presenting even less of a health hazard than heat stress. Whether a person feels hot or cold is heavily dependent on the ambient temperatures to which they are accustomed and what clothes they are wearing.

If the relative humidity is allowed to exceed 70%, people tend to feel sweaty and in the long run may suffer as a consequence of fungal growth and increased contamination of ambient air with potentially allergenic fungal spores. By contrast, if the relative humidity falls to below 20%, noses, eyes and throats start to feel dry and uncomfortable.

From a medical viewpoint, *radon and its radioactive daughters* are potentially the most serious indoor air pollutants. The fact that they are odourless, non-irritant and without acute toxicity renders them an insidious hazard to health. The main health risk from them is lung cancer. According to a recently published survey conducted in the UK (Clarke and Southwood, 1989), radon and its daughters are the greatest single source of exposure of the population to ionising radiation. The source of the radon is from rocks such as granite, on which houses are built and of which some are constructed. It may also be contained in ground-water. Ambient indoor levels are apt to be increased by heating and ventilation systems which heat up and circulate air which has seeped into basements from the ground and then circulate it and recirculate it through the other parts of the house. Levels of exposure vary widely from home to home but, based on data for uranium miners, Japanese atom bomb survivors and laboratory animal studies, it is estimated that about 6% of the present annual incidence of lung cancer in Great Britain is attributable to exposure to radon, that is to say, some 2,500 cases per annum (see Clarke and Southwood, 1989). Further consideration of radon is given in Chapters 5 and 10.

Whether or not *cooking and heating fumes* pose a health problem depends very much on climate as well as on the amount of indoor space and ventilation. The colder the climate, the smaller the space, the poorer the ventilation, the worse the potential health problems. When cooking is

carried out in the open air or when the windows of indoor kitchens can be left open, the potential for health effects attributable to cooking and heating fumes is minimal. The same is true when cooking is by electricity. The spectrum of pollutants of main interest varies with the type of fuel and with the efficiency of its combustion. The spectrum includes carbon monoxide (CO), carbon dioxide ($CO_2$), oxides of nitrogen, particularly nitrogen dioxide ($NO_2$), sulphur dioxide ($SO_2$), aldehydes, particles, and pyrolysis products such as polycyclic aromatic hydrocarbons (PAH). These individual pollutants are discussed in Chapter 10 so that it is only necessary here to consider the medical consequences of their presence in indoor air.

*Carbon monoxide* is a product of incomplete combustion of all carbonaceous fuels. It is odourless and non-irritant but nevertheless potentially acutely toxic because of its affinity for haemoglobin which leads to a reduction in the oxygen-carrying capacity of the blood. In turn this may lead to dizziness, blurred vision and rapid breathing when the concentration of carboxyhaemoglobin in the blood is sufficiently high.

In the short-term and on an intermittent basis, most people can inhale enough carbon monoxide to reduce the oxygen carrying capacity of the blood by 10% or more without ill-effect. However, people with reduced circulatory reserve (e.g. because of existing heart disease) may, in theory at least, be temporarily compromised by heavy exposure to carbon monoxide such that their exercise tolerance is reduced and they develop anginal pain after less exercise than they are normally capable of. The hard evidence that this happens under realistic conditions of indoor air pollution is, however, lacking. In any case, once exposure to carbon monoxide ceases, it is fairly rapidly lost from the body and the blood returns to normal. Under conditions of prolonged exposure to carbon monoxide an equilibrium is reached wherein a proportion of the haemoglobin is continuously unavailable for the purposes of carrying oxygen. In response to this the body increases its production of red blood cells and haemoglobin in much the same way as it does when people climb from sea level to altitudes where the oxygen pressure is reduced. Research aimed at finding an association between chronic exposure to carbon monoxide and increased risk of atherosclerosis or coronary heart disease has given only negative or equivocal results. Also, concern that exposure to carbon monoxide at the levels present in indoor air might be a cause of reduced birthweight or of birth defects has proved to be unfounded.

It has to be recognised, of course, that in conditions of very bad ventilation, the use of some types of indoor heating and cooking appliances can lead to such high levels of carbon monoxide in a room that death occurs. Also, lethal concentrations of carbon monoxide can be built up by running a car engine in a garage with the doors closed.

*Carbon dioxide* is not normally listed as a pollutant; however, at high concentrations it can cause headaches, loss of judgement, hyperventilation,

and even death. Chronic effects on health have not been described. The main sources of carbon dioxide are the exhaled breath of humans and animals and the fumes of cookers and heaters burning carbonaceous fuels.

*Oxides of nitrogen*, and particularly *nitrogen dioxide*, are irritant to the eyes and nose and can damage lung tissue. The damage, which is due to the oxidant properties of the gas, occurs primarily in the vicinity of the terminal and respiratory bronchioles and consists of metaplastic changes in airway epithelium at these sites, increased susceptibility to respiratory infections, and impairment of lung function because of the destruction of alveolar walls. Repeated heavy exposure to nitrogen dioxide such as occurs in some occupations (e.g. shot-firing in mines), can result in centrilobular emphysema with destruction of elastic fibres and loss of elastic lung recoil. Particularly at risk are individuals suffering from the genetically determined disorder known as $\alpha_1$-antitrypsin deficiency.

There is considerable evidence that exposure to oxides of nitrogen derived from gas cookers, oil-fired heaters and wood fires, under ordinary home circumstances, can be sufficient to produce adverse effects on health. Thus, Melia *et al.* (1979) found that boys living in Scottish homes with gas cooking have an 18% higher prevalence of respiratory illnesses than boys living in comparable homes with electric cooking. This difference was, statistically, highly significant (p less than 0.01) after correction for age, sex, social class, and number of cigarettes smoked within the dwelling. Other studies have shown that exposure to nitrogen dioxide-containing emissions from gas stoves adversely affect lung function in children (Speizer *et al.*, 1980; Samet *et al.*, 1987).

*Formaldehyde* and other *aldehydes* are among the products of incomplete combustion. Thus they are present in fumes from coal, wood and oil-fired heaters, bonfires and tobacco smoke. However, as mentioned in Chapter 10, polyurethane house insulation and other building materials are also important sources of formaldehyde. Aldehydes are extremely irritant to the mucous membranes of the upper respiratory tract and are lachrymatory. Formaldehyde is a mutagen and prolonged exposure to high concentrations has been shown to cause nasal cancers in laboratory animals (Griesemer *et al.*, 1982). Acetaldehyde has also been found to be carcinogenic in laboratory animals:

In rodents, nasal cancers arise out of a background of pre-existing irritation and necrosis of the nasal epithelium. There is no evidence that tumours arise in the absence of pre-existing severe tissue damage of these kinds. The hope therefore is that exposure to levels of formaldehyde lower than those that produce nasal cancers in rodents and lower than those associated with any evidence of chronic damage to nasal epithelium in man, is wholly without cancer risk for man. So far, the results of numerous epidemiological studies that have been carried out regarding people exposed to formaldehyde at work (e.g. pathologists, embalmers) suggest that this

hope is fulfilled. This being so, there would appear to be no serious health effects from exposure to the relatively low ambient levels of formaldehyde encountered in indoor air environments. The same is, hopefully, true for acetaldehyde and other aldehydes, although in this case there are no epidemiological data to back up the hope.

Nowadays, *sulphur dioxide* in indoor air is generally not a source of major concern in developed countries. However, it still may be so in parts of the world where dwellings are not adequately ventilated and where sulphur-containing fuels are used without appropriate control.

The concentration of *respirable particles*, including fibres in indoor air environments, is extremely variable both with regard to size and chemical composition. Large particles tend to be deposited in the nose, nasopharynx and larynx whereas smaller particles may be taken into the lungs.

The extent to which particles enter the lungs from ambient air depends *inter alia* on their size. The aerodynamic properties of a spherical particle can be expressed in terms of its diameter, but those of fibres and irregularly shaped particles are more complicated. A long thin fibre aligned at an angle of 90 degrees to the airstream behaves like a sphere with a diameter equal to the length of the fibre, and is deposited high up in the respiratory tract. A fibre aligned longitudinally with the airstream in which it is suspended behaves like a spherical particle of the same diameter and density as the fibre and tends to be co-deposited with such particles. For this reason long thin fibres can penetrate deeply into the lungs and reach sites which only small spherical particles normally reach.

Whether or not particles (including fibres) taken into the lungs are harmful to health depends firstly on the number of particles inhaled, secondly on where particles are deposited, thirdly on whether the inhaled particles are toxicologically active or inert, and soluble or insoluble in body fluids, and fourthly on whether they are inhaled alone or along with other particles or gases which are toxicologically active.

Provided that the particles are not too big, not too numerous, not toxicologically active, and are deposited on airway epithelium as distinct from alveolar epithelium, they are relatively easily and efficiently removed from the lungs, either via what is called the ciliary escalator or by phagocytosis by lung macrophages. The airways are lined by cells which have tiny hairs (cilia) which 'wave' particles upwards towards and through the larynx. Once through the larynx they are swallowed and cease to be of any toxicological importance. Removal of inhaled particles is a silent continuous process. If irritant gases are inhaled or the air is so heavily contaminated with particles that the rate of deposition exceeds the rate of elimination, there may also be increased production of mucus by glands within the airway walls, and to a lesser extent by mucus-producing cells in the surface epithelium lining airways. In this case the particles travel upwards on the mucus-escalator along with variable amounts of mucus. If

mucus production is heavy then its expulsion from the lungs may be aided by intermittent coughing.

Smaller particles which are gobbled up by lung macrophages may leave the lung within the macrophages as they take a free ride on the upward escalator. Alternatively, the macrophages may find their way into blood vessels and carry their burden of engulfed particles away from the lungs in the blood-stream. Macrophages are equipped with enzymes capable of destroying many of the chemicals of which particles consist. Macrophages that have picked up indestructible particles tend to get lodged in local lymph nodes where their presence does little or no harm.

By these various means the healthy lung can rid itself of huge numbers of inhaled particles each day. However, in certain circumstances things go wrong. The waving movements of cilia can be paralysed temporarily by poisonous gases. In practice this is probably not as serious a problem as was once thought. More serious is the fact that chronic exposure to irritants can destroy cilia and lead to the replacement of ciliated cells by flat, so-called, squamous cells. When this happens the capacity of the lung for clearing itself of foreign matter is embarrassed and chest disease is much more likely to ensue.

Problems also arise if macrophages accumulate in the lungs because of excessive exposure to particles or because they are poisoned by the particles which they have engulfed or by gases taken in to the lungs along with the particles. Such macrophages eventually die and release the proteolytic enzymes which they normally use to destroy engulfed particles. These released enzymes destroy lung tissue and thereby cause the condition known as emphysema. The lungs of genetically-normal individuals are equipped to neutralise proteolytic enzymes released from macrophages in this way. However, a minority of the population (i.e. those suffering from $\alpha_1$-antitrypsin deficiency) is genetically less able to neutralise proteolytic enzymes and these people are especially prone to develop emphysema as a consequence of inhaling particles into their lungs.

It is now widely accepted that the main reason why *asbestos* fibres are dangerous to health is the fact that they can behave aerodynamically like much smaller particles. As a consequence they end up in peripheral parts of the respiratory tree where there is no effective way of getting rid of them. A long thin asbestos fibre is much too large to be engulfed by a single macrophage. Although chemically relatively inert, the prolonged residence of asbestos fibres starts a lung reaction which can lead to fibrosis (asbestosis) or to two different forms of cancer: cancer of the lung itself and cancer of the pleura (mesothelioma). There is some evidence that asbestotic fibrosis predisposes to lung cancer, but it is by no means certain that asbestos-related lung cancer is necessarily preceded by pulmonary fibrosis. The extent to which the variable chemical composition of asbestos fibres is implicated in their fibrogenicity and carcinogenicity continues to be a topic

for debate. However, the importance of their physical shape is not disputed. For this reason there is a fear that other insoluble or poorly soluble fibres of the same long, thin dimensions, such as glass fibres, might prove similarly fibrogenic and carcinogenic.

Second to radon, the contamination of indoor air by asbestos fibres is potentially the most serious problem as far as cancer risk is concerned. Fortunately, the seriousness of this risk is now widely appreciated and in developed countries, at least, the casual use of asbestos in building materials and paints for fireproofing and for heat insulation has virtually ceased. However, many older houses, offices, and other buildings still retain asbestos in their construction particularly in the lagging around water pipes. Little danger attaches to this unless the lagging is disturbed, disintegrates or is carelessly removed.

It is most unlikely that anyone would be sufficiently exposed to asbestos fibres in indoor air environments to be at risk of developing asbestosis. However, cases of mesothelioma have been seen in persons no more than lightly exposed to the inhalation of asbestos fibres, so that a risk of this form of cancer and also of lung cancer from asbestos fibres in indoor air cannot be dismissed. (For further discussion of asbestos and man-made mineral fibres see Chapter 10).

*Polycyclic aromatic hydrocarbons (PAH)* are present in particles generated during the pyrolysis of almost any organic material. Thus, PAH are present in bonfire smoke, smoke particles in cooking fumes, fumes from kerosene heaters, and in tobacco smoke. Several PAH have been shown to be carcinogenic and their inhalation may well increase the risk of lung cancer in man. In the past, in cities where people were exposed to air that was heavily polluted with smoke particles, PAH have been blamed for the higher cancer risk. The levels of PAH in indoor air, except under conditions of very heavy pollution by smoke particles (i.e. cooking, smoking, heating fumes, or tobacco smoke), are unlikely to constitute more than a negligible risk of lung cancer.

Samet *et al.* (1987) list numerous *volatile organic chemicals* which may contaminate indoor air along with the sources from which they are derived. Consideration is given to these in Chapter 10.

Oxygen can be converted to *ozone* by any source of ultraviolet light or electrical discharge. Copying machines found in most offices and in an increasing number of homes are a significant source of ozone. In terms of concentration required to produce toxic effects, ozone is more than 10 times as toxic as nitrogen dioxide. Exposure to a low concentration (e.g. 0.7 parts per million) for a period of two hours is enough to cause significant impairment of gaseous exchange in the lungs — probably because of oedema of alveolar walls (Young *et al.*, 1964). The symptoms of excessive exposure to ozone are irritation of the respiratory tract, headache, tightness of the chest and wheezing (Challen *et al.*, 1958).

By contrast with the effects due to most of the chemicals considered above, adverse effects on health from *bacteria*, *fungal spores*, and *allergens* may be frequent and sometimes serious.

The truth of the matter is that at the present time we do not really know how much illness is caused by the presence of these agents in indoor air pollution. Prior to the advent of air-conditioning systems, it was well known that people living in dirty, damp conditions were more prone to a variety of respiratory infections and rheumatic disorders than people living in dry, warm conditions. Also, it was clear that atopic individuals were at greater risk of developing hay-fever, allergic rhinitis and asthma because of their exposure to the excreta of house dust mites which live on flakes of skin shed continuously by humans.

The introduction of circulating ducted air systems has magnified old problems and introduced new ones (Robertson, 1988). If, because of bad design, improper use and/or inadequate inspection and maintenance, moisture, fungi, bacteria, protozoa, rodents or birds etc. are allowed to collect and multiply in air ducting and filter pads, etc., then dangerous dusts and living bacteria may be widely circulated in high concentrations to the occupants of buildings. Some of the serious consequences of this are listed in Table 1.

Humidifier fever is a typical example. This form of hypersensitivity pneumonitis is caused by antigenic proteins in cells of the amoeba, *Acanthamoeba*, which can thrive in the warm stagnant water of badly maintained humidifier reservoirs.

**Table 1 Examples of diseases caused by bacteria, fungi and allergens, etc.**

| | |
|---|---|
| *Traditional diseases:* | |
| Coughs and colds and other infectious diseases | |
| Tuberculosis | |
| Allergic symptoms | — House mite |
| — hay fever | — Fungal spores |
| — rhinitis | — Various other dusts containing allergenic proteins, etc., cotton dust, bird feather dust |
| — asthma | |
| *Diseases that have been newly described or magnified by badly designed and or badly maintained air conditioning systems:* | |
| Humidifier fever | — Acanthamoeba |
| | — Thermoactinomyces |
| | — Micropolyspora species |
| Legionnaires' | — Legionella pneumophila |
| Lung cancer | — Radon |

## OTHER PEOPLE'S TOBACCO SMOKE

An increasing problem in recent years has been that not only persons working within buildings but even persons walking the streets outside them can be at risk from the bacterium which causes the potentially fatal lung condition Legionnaires' disease (i.e. *Legionella pneumophila*). This disease is caused by breathing deeply into the lungs very small water droplets containing the bacterium. Such infected droplets can arise from water cooling towers associated with air-conditioning plant, from the spray arising from hot water taps and from bathroom showers.

Recently Holst (1988) reported that people who keep pet birds in the home have a 6.7 times higher risk of developing lung cancer than those who do not, after taking smoking into account. The only plausible explanation of this finding, which needs to be confirmed, is that the increased risk stems from the liberation into the atmosphere of excess allergens and dust particles from the birds' cages. These cause pathological fibrotic changes which, in turn, predispose to the development of cancer.

Of all sources of non-industrial internal air pollution, environmental tobacco smoke (ETS) has received the most attention because of its visibility, characteristic odour and irritant properties (Chapter 3).

With the exception of nicotine and certain tobacco odours, the spectrum of chemicals in ETS is similar to that produced by the incomplete combustion (pyrolysis) of other carbonaceous materials. In the past when houses were heated by open coal or wood-burning fires and when chimneys were wont to get blocked, ETS would often have been no more than a minor contributor to the particulate content of ambient air. But under conditions of central heating combined with inadequate ventilation, ETS can contribute more particles than any other source except cooking fumes. Only under conditions of heavy smoking, combined with very poor ventilation, is the particle density in ambient air likely to have any measurable effect on health. However, the aldehydes in ETS, particularly for a person down-wind from a smouldering cigarette, can be unpleasantly irritant and lachrymatory under conditions of moderately poor ventilation. Non-smokers exposed to ETS absorb nicotine and excrete a metabolite of nicotine, namely cotinine, in their urine. However, the level of these chemicals found in exposed non- smokers is to be seen more as a triumph for analytical chemistry than as any indication that the health of such people is being damaged! The levels of carbon monoxide found in the blood of non-smokers who have been exposed to ETS in smoky rooms is measurably raised, but not to levels as high as those frequently found in persons (e.g. traffic wardens) exposed to outside air where there is heavy pollution from vehicular exhaust fumes.

ETS and the smoke inhaled by active smokers differ in many ways (see Chapter 2). Nonetheless, it may be helpful to bear in mind that the intake of tobacco smoke constituents, such as particles, nicotine, and carbon monoxide from ETS rarely exceeds the equivalent of the active smoking of

a small fraction of one cigarette per day. It is reasonable to deduce, therefore, that any risk to health from ETS must be immeasurably small.

The most persuasive evidence of a health risk from exposure to ETS is derived from studies on school children. The children of parents who smoke at home have repeatedly been reported to have a higher incidence of respiratory infections and days away from school than the children of non-smoking parents. However, these studies are beset with problems. Differences in social class, exposure to cooking and heating fumes, levels of pollution in outside air, in the location of the dwelling, distance from main roads, and other variables can act as confounders and need to be controlled for. Not all studies of school children have given positive results, and there is no evidence that increased incidence of respiratory symptoms in young children leads to adverse effects on respiratory function or disease incidence later in life.

Many attempts to demonstrate an effect of ETS exposure on respiratory illness and/or lung function in adults have given negative or no more than equivocal results. Positive results presented in a much quoted paper by White and Froeb (1980) were both surprising and implausible and the paper has been seriously criticised on methodological grounds.

Aronow (1978) reported reduced exercise tolerance and increased heart rate in patients with angina pectoris when they were exposed to ETS in sufficient concentration to raise their mean carboxyhaemoglobin level from 1.3% to 1.8%. He suggested that these effects were due to absorbed nicotine. However, the US Surgeon General (1979) considered this to be unlikely and that the physiological changes seen were more probably due to stress following anxiety or aggravation induced by the smoke-filled room.

Hirayama (1981), in a prospective study of Japanese women, claimed to have found a significantly higher risk of lung cancer in non-smoking women married to smokers than in non-smoking women married to non-smokers. This publication has been followed by similar studies in other countries. Most of the later studies were case-control studies which relied for information both about the case's and spouse's smoking habits on retrospective questionnaire data. Furthermore, in most of the studies the accuracy of diagnosis of lung cancer in the so-called cases had not been checked by pathological assessment. Since in women it is particularly easy to confuse a primary adenocarcinoma of the lung with a lung metastasis from a primary adenocarcinoma arising in another tissue (e.g. breast, ovary, colon), one can easily see that the data used in many of these studies are rather fragile. A majority of the studies gave results that suggested that the risk of developing lung cancer was slightly but not significantly greater in the non-smoking wives of smokers; however, a few suggested the opposite. By combining the results of several different studies and undertaking a so-called meta-analysis, Wald *et al.* (1986) claimed to have found a significantly positive risk ratio of 1.35:1.0. However, the propriety of the application of

meta-analysis to such disparate data is highly questionable.

Furthermore, there is evidence that a systematic bias influences the findings in many or all of the studies in question. It is known from measurements of salivary cotinine that a proportion of people who claim to be non-smokers are actually smokers (Lee, 1987). It is also known, by comparing the answers to questionnaires completed by the same individual but several years apart, that a proportion of people who claim never to have been active smokers are, in fact, ex-smokers (Lee, 1987). There are many social reasons why people lie or give inaccurate answers to questions about their indulgences and, as anti-smoking campaigners press home their attack, the social reasons for people saying they do not smoke when in fact they do, are increasing. It is a fact that smokers tend to marry smokers and non-smokers tend to marry non-smokers. This fact combined with the fact that some women who develop lung cancer are misclassified as non-smokers can explain most or all of the 1.35:1.0 relative risk found by Wald in his meta-analysis. The reasons for this are given in detail by Peter Lee in his recent book (Lee, 1988).

Thus we are left in the case of ETS with the conclusion that under conditions of inadequate ventilation, other people's smoke can be irritant and annoying. It is possible that it can increase the incidence of respiratory infections in children. However, there is no adequate evidence that it causes any of the serious diseases that have been associated with active smoking (i.e. heart disease, emphysema, lung cancer).

## 5. Conclusions

The adverse effects of indoor air pollution have always been with us. They can be broadly separated into effects on health and effects on well-being. Health effects used to stem mainly from damp, cold, dirty conditions associated with overcrowding and excessive pollution of internal air by outdoor pollutants, cooking and heating fumes, house dust, asbestos fibres and fungal spores. Radon as a serious contributor to lung cancer risk always existed in houses built on or constructed with radon- seeping rocks such as granite.

During the last several decades, ducted air heating and conditioning systems have been increasingly used both in public buildings and private dwellings. If these are not properly designed or maintained, so that radon, bacteria, fungal spores and other allergens are distributed in high concentrations within indoor air environments, then they can introduce new health risks or magnify traditional ones. Outbreaks of humidifier fever and Legionnaires' disease, and a risk of lung cancer from exposure to radon, are examples of this.

The present day high level of concern about the possible dangers of exposure to environmental tobacco smoke stems neither from any solid

evidence of serious health risk nor from evidence that any risk is increasing. On the contrary, its origins lie, firstly, in the fact that people's expectations of being able to breathe fresh clean air have increased, particularly because the quality of external air has been greatly improved. Secondly, irritancy from other people's tobacco smoke is easy to identify because of its characteristic odour. Thirdly, the proportion of people who do not smoke and whose well-being is disturbed by ETS is increasing. Fourthly, anti-smoking campaigners have been purposefully exaggerating the possible health danger from ETS exposure as a means of putting pressure on people to give up smoking.

In this day and age there is no need for anyone in developed countries to be inconvenienced by dangerous or unpleasant indoor air pollution and it is not unreasonable to use irritation by ETS as a marker of inadequate ventilation. However, the most sensible response to complaints of irritation is not to ban smoking but to check and improve the ventilation system. If smoking is banned and nothing else is done, then any adverse health effects from indoor air pollution will persist. Furthermore, if, following a smoking ban, ventilation standards are reduced (i.e. because ETS as a marker of inadequate ventilation is no longer available), then risks to health from less obvious pollutants will actually increase.

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