

DEPARTMENT OF HEALTH

Committee on the Medical Effects of Air Pollutants

**Guidance on the Effects on Health of Indoor Air
Pollutants**

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Note

Every effort has been made to ensure that this report is free from error. The authors would, however, be grateful to readers who point out faults and also for constructive suggestions regarding material which might have been included.

**COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS
(COMEAP)**

**GUIDANCE ON THE EFFECTS ON HEALTH OF INDOOR AIR
POLLUTANTS**

Introduction

1. People spend more than 80% of their lives indoors. The indoor environment is not free of air pollutants: many pollutants generated outdoors penetrate indoors and to these are added pollutants produced by indoor sources. All buildings act as a filter for outdoor pollutants and thus the concentration of pollutants generated outdoors tends to be lower indoors as long as there are no additional domestic sources. This would apply, for example, to particles generated by diesel-engine vehicles. In this paper, only the home indoor environment is considered. It is appreciated that other indoor environments including adult workplaces are also important. Children may be exposed to air pollutants in nurseries and schools: these environments may form the subject for a later paper. The in-vehicle environment has not been considered.

2. Many studies have shown that the state of peoples' health is related to outdoor concentrations of air pollutants. An increase in hospital admissions for treatment of heart and lung disease and in the number of deaths occurring each day is seen when outdoor levels of pollutants rise (Department of Health, 1998). In addition to these effects of day-to-day changes in outdoor concentrations, it is known that long-term exposure to particles, an important pollutant, reduces life expectancy (Department of Health, 2001). Though these studies show an association between outdoor levels of air pollutants and ill health, they do not tell us where people experience their greatest exposure. As people spend much of their lives indoors, it is plausible that the largest part of a person's exposure, even to pollutants generated outdoors, actually occurs indoors. A considerable part of the increased exposure to pollutants experienced on days when outdoor levels are raised may be – in fact probably is – due to increased exposure indoors.

3. The Air Quality Strategy for England, Scotland, Wales and Northern Ireland aims to reduce outdoor concentrations of air pollutants by reducing the production of pollutants by outdoor sources (Department of the Environment, Transport and the Regions, 2000). Very significant reductions in emissions of air pollutants have occurred during the past twenty years: details of these reductions may be found in UK Emissions of Air Pollutants published in October 2003 (Dore et al 2003). The Strategy is based on Air Quality Standards that are designed to protect health. As outdoor sources of pollutants are reduced and outdoor concentrations fall, so will the contribution of outdoor sources to indoor concentrations be reduced.

4. The picture is complicated and it must be remembered that indoor sources of air pollutants that vent to the outdoor environment contribute to outdoor concentrations. Thus gas and solid fuel heating release oxides of nitrogen, particles, carbon dioxide, carbon monoxide, sulphur dioxide and organic substances via chimneys into the outdoor environment.

Purpose of this report

5. Members of COMEAP have often stressed the importance of indoor exposure to air pollutants and the desirability of providing advice on this. Requests for advice have also come from experts outside the Department of Health: Professor P. O' Sullivan (Former Chairman of the Building Regulations Advisory Committee (BRAC)) has raised the need for such guidance on several occasions.

6. We recognise that levels of some air pollutants found in at least some homes in the UK exceed those demonstrated by epidemiological and other study methods, to be associated with adverse effects on health. How long these effects actually persist cannot currently be predicted.

7. This report sets out the Committee's current views. Our thinking has been based extensively on work done by this Committee with respect to the effects of outdoor-generated air pollutants on health, on the work of the Expert Panel on Air Quality Standards (EPAQS) and on that of the World Health Organisation (WHO). The report is thus, in part, a compilation of widely accepted views: we take this opportunity to endorse selected views and to expand upon them in some areas.

8. The guidance is based on an understanding of sources of indoor air pollutants and on simple measures that people can take to ensure that concentrations of air pollutants are kept at a low level. This advice is supplemented by some quantitative (numerical) guidelines. These guidelines, which apply to only a few air pollutants, are not intended to be seen as standards, though we appreciate that there is a risk that this is how they will be interpreted. The guidelines we have developed are intended as an aid to three groups of people:

- (i) manufacturers of materials or devices that release pollutants into the domestic environment;
- (ii) architects and building-engineers who are interested in designing buildings with adequate levels of ventilation;
- (iii) individuals who are concerned about concentrations of air pollutants in their homes, who are prepared to pay for monitoring to be done and who need a benchmark with which to compare the results obtained. Landlords, building owners and building managers concerned to provide a high standard of indoor air quality should also find the guidelines useful.

9. We have provided, wherever possible, advice on how concentrations of pollutants may be monitored indoors. In several cases, the equipment needed is complex and expensive and likely to be beyond the means of private householders. In such cases, it might be asked whether recommending guidelines is worthwhile on the grounds that compliance cannot be tested. We think guidelines do have value and even in such instances, engineers and architects, for example, may be able to use the guidelines in designing appliances and buildings. It is hoped that the guidelines will play some part, for example, in the setting of ventilation standards.

10. This Committee has not attempted to recommend emission standards for devices that generate air pollutants indoors. This is the task of the Department of Trade and Industry and the Health and Safety Executive. The guidelines discussed in Section II may assist in this process.

Scope

11. Only the home indoor environment is considered in our guidance which is divided into two sections:

- I Advice on how to minimise the production of air pollutants indoors;
- II Guidelines for selected air pollutants in the indoor setting.

12. The guidance will not discuss the effects of indoor exposure to Environmental Tobacco Smoke (ETS), radon and pesticides as these substances do not fall within the remit of COMEAP and other agencies provide advice on these substances. Particles are also excluded because we feel it is not currently feasible to define a satisfactory guideline for them. Our reasoning behind this decision is presented in some detail in paragraphs 41 and 43.

Section I

ADVICE ON HOW TO MINIMISE THE PRODUCTION OF AIR POLLUTANTS INDOORS

13. Four factors control indoor concentrations of air pollutants:
- (i) the outdoor concentrations of the pollutants;
 - (ii) the extent of filtering imposed by the building on air passing from outdoors to indoors, indoor adsorption/desorption and chemical reactions;
 - (iii) the indoor sources of the pollutants;
 - (iv) the level of ventilation of the building.
14. It is obvious that in a poorly ventilated room the indoor concentrations of pollutants will be mainly determined by sources within the room. On the other hand, the level of pollutants in a room with no internal sources and open windows will be similar to or lower than that outdoors. It will be appreciated that indoor concentrations of air pollutants will vary with the season – windows being more likely to be open in summer than in winter. Heating and, perhaps, cooking appliances are likely to be used more often in winter than in summer.

What are the major indoor air pollutants?

Products of combustion

15. Combustion of fossil fuels (gas, oil, coal) and of other organic material (wood, peat, paper) is a chemical process that once begun is self-sustaining. The products of combustion depend on the conditions under which it takes place. With a plentiful supply of air, the main products are carbon dioxide, nitrogen dioxide and water. Particles may also be produced. Smoke, produced by wood and coal fires contains many particles of various sizes, but a luminous gas flame also generates particles: the

luminosity of the flame being due to the presence of very small, glowing carbon particles. These may be deposited and blackening of radiants, the fire-clay structures that glow when hot, and adjacent walls, is common when gas fires are poorly adjusted or not supplied with sufficient air. The air supply is critical: if sufficient oxygen is provided, the carbon of organic material will be largely converted to carbon dioxide; if the oxygen is limited, carbon monoxide will also be produced. High temperatures cause nitrogen, which makes up 80% of air, to react with oxygen, producing nitric oxide (NO) and nitrogen dioxide (NO₂).

16. These then, are the major pollutants generated by combustion: carbon dioxide and carbon monoxide, nitric oxide and nitrogen dioxide, particles and water. If sulphur is present in the fuel, sulphur dioxide is also produced. This was a major problem when coal was widely used for domestic heating. A wide range of other compounds are also produced in smaller quantities. These include polycyclic aromatic hydrocarbons (PAHs): a group of organic compounds that are known to be carcinogenic. Combustion can also produce measurable levels of formaldehyde, an irritant gas.

Volatile Organic Compounds (VOCs)

17. Fabrics, carpets, thermoplastic tiles and linoleum all have a characteristic smell when new. This is due to organic chemicals being released. Many thousands of individual organic compounds can be detected in the air both indoors and outdoors. Fortunately, most occur only at low concentrations. The individual organic compounds present in such materials include alcohols, aldehydes, ketones and many other organic species. Predicting the effects on health of raised concentrations of most of these compounds is impossible because of the lack of toxicological data. Measurements of Total Volatile Organic Compounds (TVOCs) are often made. The TVOC level has been used as an indicator of the likelihood of effects on health occurring, though its use for this purpose is declining. TVOC levels are still sometimes used as a general indicator of indoor air quality.

18. The Committee has previously considered the issue of volatile organic compounds in indoor air (Department of Health, 1998a). We concluded that there

was insufficient information to allow a precise assessment of the effects on health of the range of concentrations of TVOCs reported. However, we considered that, if concentrations exceeded the normal range of 0 to 1 mg/m³ (the 95th percentile of mean monthly concentrations in a study of Avon homes, Berry et al, 1996), and sensory or irritant symptoms were reported, then there would clearly be a case for looking at sources of VOCs in the home and for taking action to reduce exposure. This advice has been included in a leaflet on volatile organic compounds in indoor air (Institute for Environment and Health, 2000a). We consider it preferable to consider individual VOCs rather than ‘TVOCs’ and consider several examples (benzene, formaldehyde and other aldehydes) later in this document.

19. Adhesives used in making wood-composite boards give off formaldehyde. New sheets of board give off formaldehyde – the rate at which the gas is produced declines as the board ages. Formaldehyde is also released if boards are sawn or abraded. Other organic compounds are produced by solvents and liquid adhesives: these include substances with pleasant smells which have effects on the central nervous system that can lead to addiction. Long-term exposure to organic solvents can damage the nervous system though exposure to much higher concentrations than are generally found indoors is needed to produce such effects. Organic compounds are also released from consumer products such as polishes, cleaning agents and sprays.

20. Some organic compounds that are found in both indoor and outdoor air are carcinogenic. These include benzene, 1,3-butadiene and PAHs. Some of the latter occur as gases or vapours, others occur in association with particles. High concentrations of PAHs can be found in charred organic material, for example on grilled meat and they are also released into indoor air during cooking.

Environmental tobacco smoke (ETS)

21. Perhaps the most important and avoidable pollutant found in many houses is tobacco smoke. ETS is a complex mixture of particles and thousands of volatile compounds. Particles, carbon monoxide and the oxides of nitrogen are present in high concentrations in smoke emitted from smouldering tobacco and exhaled by smokers. Irritant compounds including the aldehydes mentioned above and acrolein

are present, as are carcinogenic substances including many species of PAH compounds and benzene. ETS is a special case indoor air pollutant; it is avoidable and only contaminates the houses of those who smoke. Advice on smoking has been provided by the Department of Health. A detailed series of reviews on the topic have recently been published. (Strachan and Cook, 1997; Anderson and Cook, 1997; Cook and Strachan, 1997; Strachan and Cook, 1998a; Strachan and Cook, 1998b; Strachan and Cook, 1998c; Cook and Strachan, 1998; Coultas 1998; Cook *et al*, 1998; Cook and Strachan, 1999).

Sources of indoor air pollutants

22. There are many indoor sources of air pollutants. These include equipment for heating and cooking in which some form of fossil fuel is burnt. Thus gas fires, gas boilers and gas cookers all produce pollutants, as do their solid or liquid fuel powered equivalents. Pollutants are also generated during cooking by the effects of heat on organic material: the fumes produced by heating fat or frying being common examples. Pollutants are produced in this way irrespective of the source of heat. Less obvious sources include new carpets and furnishings and wood-composite boards, e.g. medium density fibre board (MDF) and “particle board” used in furniture and in kitchen fittings. The pollutants produced are discussed below. Another important source is paint with an organic base. People should also be aware that the use of household cleaning products and air fresheners will contribute to levels of indoor air pollution. The integral garage is often a source of indoor air pollution and this can be very dangerous if a vehicle’s engine is allowed to idle in the garage: especially petrol engines as they produce carbon monoxide that can leak into the house. Evaporation of fuel from vehicles and storage cans also generates pollutants including benzene. Solvents, paints containing organic solvents, polishes, glues and oils are often stored in integral garages and these too, act as sources of indoor air pollutants. Accidental spillages, including petrol and cleaning fluids for example, can also cause short-term increases in concentrations of organic compounds. DIY and hobby-activity may also release solvents.

Does exposure to indoor air pollution damage health?

23. Modern studies of the effects of air pollutants on health began because of serious episodes of outdoor air pollution including, for example, that occurring in London in 1952. Since then, methods to detect associations between outdoor levels of air pollutants and ill health have developed and effects continue to be detected at the comparatively low levels of air pollution found outdoors in the UK today. Detailed reports on the effects of outdoor air pollution on health have been published by this Committee and previously by the Advisory Group on the Medical Aspects of Air Pollution Episodes (Department of Health, 1991; Department of Health, 1992; Department of Health, 1993; Department of Health, 1995a; Department of Health, 1995b and 1995c). These reports show that concentrations of air pollutants that frequently occur indoors can have an adverse effect on health. How large the effects of indoor exposure may be is less easy to say - the findings of associations between outdoor concentrations of pollutants and effects on health cannot easily be extended to predict the scale of effects occurring as a result of indoor exposure. Studies of associations between outdoor concentrations of pollutants and effects on health are also likely to reflect the effects of indoor exposure to pollutants that are generally monitored outdoors.

24. Other evidence on the effects of air pollutants on health comes from studies involving the carefully controlled exposure of volunteers to pollutants. These studies show that for some pollutants, concentrations that occur indoors can affect health. More details of these studies are given below. These volunteer studies tell us about the effects of concentrations of pollutants that do not cause deaths or hospital admissions, but, rather, produce modest changes in a person's physiology. Indices of lung function for example, are frequently monitored. It is notable that these studies generally do not detect effects at such low concentrations as do the epidemiological studies mentioned above. This may be because volunteers tend to be healthy adults: few studies are carried out employing elderly or very ill subjects or children. However, for some pollutants greater confidence in an association between pollutant concentrations and effects on health can be gained from volunteer studies than from epidemiological studies. Volunteer studies have been used as a basis for setting air quality standards for some pollutants. The limitations of such an approach are

recognised: such studies provide us with information on the effects of short-term exposure or on changes that affect only a minority of people.

25. A comparatively small number of studies have been specifically designed to look at the effects on health of indoor exposure to air pollutants. Such studies are difficult because measurement of concentrations of air pollutants may involve installation of complex monitoring equipment and this is seldom feasible, although passive samplers (e.g. Palmes tubes for NO₂) can be used, depending on the objectives of the study. In addition, the number of individuals taking part tends to be limited and the power of the studies to detect effects at low concentrations tends to be low in comparison with the methods used to study the effects of outdoor concentrations. However, a number of important studies have been undertaken. A few are discussed below and a more detailed discussion may be found in a report published by the Institute for Environment and Health (Institute for Environment and Health, 1996). More detailed accounts of carbon monoxide and particles may be found in other reports from the Institute. (Institute for Environment and Health, 1998, Institute for Environment and Health, 2000b).

26. Epidemiological studies of the effects of long-term exposure to low concentrations of carcinogens in indoor and outdoor air are difficult to undertake, as very large long-term studies would be needed to reveal an effect. Studies of exposure to the much higher concentrations that used to be encountered in occupational settings, however, show clear effects. Mechanistic work designed to investigate how carcinogenic substances such as benzene or PAH compounds act on cells to produce cancer, show that even at very low levels of exposure, no guarantee of complete safety can be given. At the low concentrations found outdoors today in the UK, risks from these compounds are certainly very low. Concentrations found indoors can exceed those found outdoors but, again, the risks are likely to be low. This is discussed in more detail below.

Minimising production of indoor air pollutants

27. This is discussed in terms of sources of pollutants.

Heating devices connected to chimneys, fires and boilers

28. The general principles: minimise emissions and maintain good ventilation apply. Well adjusted and regulated devices release comparatively small amounts of pollutants into the indoor environment. Periodic adjustment and servicing is, however, essential and all devices should be fitted and maintained in accordance with the manufacturer's instructions. Signs of malfunctioning should be sought. In the case of gas fires, where unsatisfactory combustion can lead to dangerous amounts of carbon monoxide being produced, the tell tale signs: luminous yellow flames, blackening of radiants and walls close to devices, are important. Boilers are less easy to examine and coke-fired boilers can be particularly dangerous if not properly adjusted. All these devices require venting via chimneys and stacks. That these should be free of obstructions is obvious and maintenance engineers should check for this. Any signs of ill health, including headaches, nausea, unusual sleepiness when a device is in use should be an immediate and urgent warning to have the device – be it a fire, a boiler or water heater – checked by a service engineer. Although carbon monoxide detectors can provide a warning, they should never be seen as a substitute for regular maintenance work.

Cookers

29. Gas cookers generate air pollutants and high concentrations of oxides of nitrogen have been regularly recorded, not only in kitchens but also in other rooms including bedrooms, when these devices are in use (Institute for Environment and Health, 1996). These concentrations can exceed standards set for outdoor concentrations of nitrogen dioxide and cannot be regarded as harmless to health¹. Signs of less than ideal combustion, including luminous flames should also be looked for. It is clearly an advantage to have a cooker that is fitted with a hood that extracts cooking fumes directly to the outside, causing fresh air to be drawn into the kitchen through passive ventilators in other parts of the house. Hoods of two types are available: extractor hoods, which collect pollutants and vent to the outdoor environment, and recirculating hoods that rely on filters to absorb pollutants and odorous compounds. The latter type do not remove gaseous pollutants such as

nitrogen dioxide. Current Building Regulations require pollutant extraction from kitchens via either an extractor hood or some other device such as an extractor fan fitted near the cooker. These regulations are comparatively recent and older properties often lack such devices. Opening a window during cooking is advisable in that it can reduce concentrations of pollutants in the kitchen. However, an open window in the kitchen can lead to an increase in the concentration of pollutants in other rooms unless the door separating the kitchen from the remainder of the property is closed. On balance, the reduction in concentrations of pollutants produced by opening a kitchen window outweighs the possible shunting of pollutants into other rooms. Gas cookers should not, of course, be used as a means of warming kitchens.

Unflued natural gas and liquid petroleum gas heaters (cabinet heaters)

30. Any unflued device that burns fuel in what may be an inadequately ventilated room is potentially dangerous. Manufacturers are aware of this and safety devices including cutouts that respond to carbon monoxide levels are fitted to some devices. Some newer devices are fitted with catalytic converters that convert emitted pollutants into harmless compounds. All such devices should only be installed and operated in accordance with the manufacturers' instructions. Safety devices can themselves fail and regular servicing is necessary. Ventilation is important: users should ensure that these devices are not used in small unventilated rooms. Paraffin heaters are uncommon today but can be dangerous if not functioning properly. Servicing is, again, necessary, particularly for devices that may not have been used for some time.

31. All the devices mentioned above produce water vapour and ventilation is needed to prevent condensation and dampness occurring. Damp conditions encourage the growth of fungal moulds and sensitisation to spores produced by such moulds is a well recognised exacerbating agent of asthma. Particles (spores) and volatile compounds (fungal metabolites) can also be released from such growths. Water vapour can be released into the indoor environment in many ways. Drying washing indoors is an obvious example: if this is done, care should be taken to ensure that the room in use is adequately ventilated.

¹ The Committee has recently published a statement 'Assessment of the Health Implications of Concentrations of Nitrogen Dioxide and Carbon Monoxide Indoors – Advice to HSE and DTI' <http://www.advisorybodies.doh.gov.uk/comeap/statementsreports/no2coindoors.htm>

Sources of organic pollutants

32. Little can be done to reduce the release of organic pollutants by adhesives that are found in carpets, particle-board and other furnishings and fittings by individual householders once these items have been installed. Given that this is the case, ventilation must be provided to keep concentrations as low as possible. Opening windows whilst using household cleaning products or painting indoors, using water-based rather than solvent-based paints, cutting particle-board outdoors rather than indoors and increasing ventilation to allow new particle-board furniture and carpets to 'air' when first installed are all obvious steps that can be taken. Significant efforts are being made by manufacturers to reduce emissions from such products.

Leakage of pollutants from integral garages into homes

33. Building regulations call for a fireproof door (with 30 minutes fire resistance) connecting an integral garage with a house. Solvents, especially petrol, should not be stored in integral garages and under no circumstances should vehicle engines be allowed to idle whilst in the garage. These may appear to be obvious precautions but they are often ignored. It is sometimes thought that because petrol engine vehicles are fitted with catalytic converters, their exhaust is not dangerous. This is not true – during the catalyst warming-up period little reduction in the amount of carbon monoxide emitted is achieved. A vehicle idling in an enclosed space is always potentially dangerous. Cars and lawnmowers, which are habitually kept in garages, also give off organic compounds when not in use. Fire doors provide only a limited barrier to the slow diffusion of such compounds from an integral garage into the main part of a house.

34. The need for adequate ventilation has been stressed in the preceding paragraphs. Ventilation by means of properly functioning chimneys and by opening windows is the key to maintaining good indoor air quality. Ensuring that airbricks are not obstructed and that trickle ventilators are open is important. Dampness is reduced by ventilation and this is also important. Some newer properties are less well ventilated than older properties: a lower level of ventilation reduces heating needs, but unless the production of pollutants is reduced by other means, a lower level of ventilation may also mean poorer indoor air quality. Future building design may well

seek to lower heating needs, in order to reduce emission of CO₂ to the atmosphere, by providing better insulation and, much less satisfactorily, by reducing ventilation. If this is the case, very careful attention will need to be paid to reducing indoor sources of air pollution.

Section II

GUIDELINES FOR SELECTED AIR POLLUTANTS IN THE INDOOR SETTING.

Are there standards for indoor air pollution?

35. When indoor air pollutants are discussed, the question of whether standards should be set often arises. Air Quality Standards play an important part in developing policies to deal with outdoor air pollution and it is easy to understand that they might be thought of as likely to be useful in dealing with indoor air pollution. This perception is strengthened by the fact that workplace exposure to many of the pollutants encountered in homes is regulated by means of standards set in the UK by the Health and Safety Executive (Health and Safety Executive, 2001). The proposition that outdoor air quality standards should simply be adopted “for use indoors” is often advanced; that occupational standards should be used is suggested less often.

36. Much discussion revolves around what is meant by a “standard”. In the outdoor air field, the word “standard” conveys more than an acceptable concentration. It includes the assumption that an appropriate method for measuring concentrations exists, that monitoring will be undertaken to an acceptable standard of accuracy and, often, that policies can be developed to reduce levels of pollutants towards the standard. The picture is actually more complicated than this and, in the UK, Air Quality Objectives derived from the health-based standards are used in policy development. The objectives take into account the costs of reducing levels of air pollutants and weigh these costs against quantified benefits to health that are predicted to occur as levels of air pollutants fall. Standards and objectives imply a capability for monitoring and a will to enforce, reflected by policies for reductions in levels.

37. Outdoor air can be adequately monitored at a limited number of sites in cities because pollutant concentrations tend to even out in the mixing process that goes on outdoors. If we are interested in concentrations at roadsides in general, monitoring at just a few roadsides may well produce useful data. If we are interested in rural

concentrations of ozone, then monitoring at a few rural sites provides adequate data. Policies to control levels are then introduced on a national scale.

38. The problem of indoor air is different. People across a city are not exposed to the same levels of indoor air pollutants; and it is the concentrations to which people are exposed in their own homes that concern us. Monitoring each and every home is, of course, impracticable; restricting people's activities in their own homes is not possible and thus the concept of a standard that can be monitored and enforced is less applicable indoors than outdoors.

Development of guidelines for individual indoor air pollutants

39. Before suggesting guidelines for a limited number of indoor air pollutants we will explain how these pollutants have been selected and why some pollutants upon which much attention is focused with regard to outdoor air have been excluded. Pollutants produced indoors in sufficient quantities to raise concentrations to such levels as to cause, on occasion, harm to health have been identified in the report by the IEH (Institute for Environment and Health, 1996). Much information has been collected on levels of these pollutants in UK homes by studies undertaken by the Building Research Establishment (Berry, 1996). The literature on indoor air pollution is extensive, but the pollutants listed below are always identified as of concern. It will be seen that the list is short: it may be possible to add more substances to the list at a later date. Many other compounds occur in indoor air. Some, such as carbon dioxide, have little effect on health and others including sulphur dioxide and ozone generally occur at such low concentrations in houses that from a health viewpoint, they may be ignored. A wide range of organic compounds also occur, but generally at low concentration and data on their effects on health, if any, is insufficient to allow guidelines to be recommended. This point has been made, above, in our discussion of Volatile Organic Compounds.

40. In producing guidelines for indoor air quality we have not returned to the original literature on the compounds considered. We do not believe that this is necessary as this literature has been reviewed and assessed by a number of expert

groups including the UK Expert Panel on Air Quality Standards (EPAQS) and the World Health Organization (WHO).

Particles

41. One notable omission from our list of guidelines is particles. Essentially all of our current understanding of the effects of low concentrations of particles on public health is derived from population-scale epidemiological studies of the outdoor ambient aerosol. We explained above why we think that the findings of these studies cannot be used to predict the effects of indoor concentrations of particles on health, nor as a basis for suggesting guidelines.

42. Particles are generated indoors by a number of processes. Simply moving about throws up dust, sweeping and vacuum cleaning may cause temporary increases in particle concentrations. Burning organic materials: coal, wood, tobacco etc. all generate particles, as does cooking.

43. It is not currently feasible to define a satisfactory guideline for indoor concentrations of particles. This may be difficult to reconcile with the fact that the UK itself has an outdoor air quality standard for particles measured as PM₁₀ of 50 µg/m³ (24-hour average concentration) and objectives of 23 and 20 µg/m³ (annual average concentration) for London and other locations in England respectively. The Air Quality Objectives are set on the basis of a trade off between the benefits to health of specified reductions in particle concentrations and the costs of delivering these reductions. This approach is feasible in the case of outdoor particles because of the exceptional weight of evidence regarding the effects of outdoor concentrations of particles on health. We have no such database regarding indoor concentrations and it is known that the indoor aerosol may differ significantly in source, chemical composition and size distribution from the outdoor aerosol. This is not always the case and there is evidence to suggest that in some locations the majority of the indoor aerosol may originate outdoors. These considerations have persuaded us that an attempt to suggest a guideline for indoor concentrations of particles would be inappropriate despite the acknowledged fact that indoor concentrations of particles may exceed those found outdoors.

Nitrogen dioxide

44. Nitrogen dioxide (NO₂) is produced as a result of the oxidation of nitrogen that occurs at high temperatures. Burning coal, wood, tobacco, oil and gas all generate nitrogen dioxide. Nitric oxide (NO) is produced firstly and then this is itself oxidised to produce the dioxide. Nitrogen dioxide is an irritant substance, an oxidant and produces inflammation and oedema of the lungs if inhaled in high concentrations (Department of Health, 1993). Concentrations encountered indoors are not sufficiently high as to produce serious acute effects but are sufficient to cause a reduction in lung function and an increase in response to allergens that can cause narrowing of the airways in some sensitive individuals.

45. Three strands of evidence relating to the possible effects of indoor exposure to NO₂ are worth considering in some detail.

Evidence relating to the increased likelihood of respiratory infection in children living in homes where gas is used for cooking and heating.

- (i) In 1992, Hasselblad *et al* published an important and influential meta-analysis that combined the findings of eleven studies that had looked at the relationship between indoor exposure to nitrogen dioxide and respiratory illness in children (Hasselblad *et al*, 1992). The analysis showed that children exposed to a long-term increase in concentration of 30 µg/m³ (15 parts per billion (ppb)) NO₂, relative to a less exposed group, experienced a 20% increase in their risk of developing respiratory illness. This increment in long-term concentration of NO₂ can be produced by the regular use of a gas cooker. A review undertaken by the IEH pointed out, however, that other studies not included in Hasselblad's analysis had produced inconsistent results (Institute for Environment and Health, 1996). Samet *et al* (1993) in a detailed review of the evidence found no association between respiratory illness in infants and neonates and indoor concentrations of NO₂.
- (ii) Recent studies carried out in the UK have shown an association between the use of gas for cooking and heating and a reduction in lung

function in women suffering from asthma (Jarvis *et al*, 1996; Jarvis *et al*, 1998). Studies from California have shown that lung function levels amongst children aged 9 – 16 were reduced in communities with raised NO₂ concentrations (Peters *et al*, 1999). Development of lung function, when studied using a longitudinal approach, was found to be reduced in these children (Gauderman *et al*, 2000, Gauderman *et al*, 2002). A study undertaken in Switzerland (Ackermann-Lieblich *et al*, 1997, Schindler *et al*, 1998) supported an association between NO₂ exposure and reduction in lung function in adults. Further studies have also suggested that long-term exposure to NO₂ impaired respiratory health (Braun-Fahrlander *et al*, 1997, Shima and Adachi, 2000, McConnell *et al*, 1999, Zemp *et al*, 1999, Forsberg *et al*, 1997).

- (iii) The third strand of evidence relates to the possible effect of NO₂ on the response to allergens amongst specifically sensitised individuals. Volunteer studies have shown that exposure to NO₂ leads to an enhanced response to allergens (Tunnicliffe *et al*, 1994). There is currently little evidence to suggest that exposure to NO₂ increases the likelihood of initial sensitisation to allergens (Institute for Environment and Health, 1996). Recent work by Chauhan *et al*, (2003 a,b) has shown that exposure to nitrogen dioxide increases the likelihood of viral infections in children and increases the bronchoconstrictor response seen in asthmatic subjects so infected. This is an important finding as it is known that such infections play an important role in triggering attacks of wheezing in young children.

46. All the above evidence was reviewed in detail by IEH (Institute for Environment and Health, 1996) and detailed reviews have been published: Samet *et al* (1997a, b) Samet and Utell (1990), Pilotto and Douglas (1992). A recent review, focussing on specific Guidelines, has been published by WHO (World Health Organization, 2003, World Health Organization, 2004).

47. Both WHO and EPAQS adopted similar approaches in setting, respectively, a guideline and a standard for NO₂. The effects of NO₂ on indices of lung function were selected as an endpoint. Volunteer studies examining the effect of NO₂ on lung

function were reviewed in detail by the Advisory Group on the Medical Aspects of Air Pollution Episodes in 1993. The passages setting out the justification for the WHO guideline and EPAQS's recommended standard are reproduced below:

WHO Air Quality Guideline for NO₂

“Despite the large number of acute human controlled exposure studies on humans, several of which used multiple concentrations, there is no evidence for a clearly defined concentration- response relationship for NO₂ exposure. For acute exposures, only very high concentrations (1,990 µg/m³, >1,000 ppb) affect healthy people. Asthmatics and patients with chronic obstructive pulmonary disease are clearly more susceptible to acute changes in lung function, airway responsiveness, and respiratory symptoms. Given the small changes in lung function (<5% drop in FEV₁² between air and NO₂ exposure) and changes in airway responsiveness reported in several studies, a range of 200 to 300 ppb (375 to 565 µg/m³) is a clear lowest-observed-effect level. A 50% margin of safety is proposed because of the reported statistically significant increase in response to a bronchoconstrictor (i.e. increased airway responsiveness) with exposure to 190µg/m³ and a meta analysis suggesting changes in airway responsiveness below 365 µg/m³ (200 ppb). (The significance of the response at 190 µg/m³ [100 ppb] has been questioned on the basis of an inappropriate statistical analysis.)

Based on these human clinical data, a 1-hour guideline of 200 µg/m³ (100 ppb) is proposed. At double this recommended guideline (400 µg/m³, 200 ppb), there is evidence to suggest possible small effects in the pulmonary function of asthmatics. Should the asthmatic be exposed either simultaneously or sequentially to NO₂ and an aeroallergen, the risk of an exaggerated response to the allergen is increased. At 50% of the suggested guideline (i.e. about 100 µg/m³, 50 ppb), there have been no studies of acute response in 1 hour.

Although there is no particular study or set of studies that clearly support selection of a specific value for an annual average guideline, the database nevertheless indicates a need to protect the public from chronic NO₂ exposures. For example, indoor air studies with a strong NO₂ source (e.g. gas stoves) suggest that an increment of about 30 µg/m³ (15 ppb), (2-week average) is associated with a 20% increase in lower respiratory illness in children 5 to 12 years of age. However, the affected children had a pattern of indoor exposure that included peak exposures higher than those

typically encountered outdoors. Thus, they cannot be readily extrapolated quantitatively to the outdoor situation. Outdoor epidemiological studies have found qualitative evidence of ambient NO₂ exposures being associated with increased respiratory symptoms and lung function decreases in children (most clearly suggestive at annual average NO₂ concentrations of 50-70 µg/m³ or higher and consistent with findings from indoor studies), although they do not provide clear exposure-response information for NO₂. In these epidemiological studies, NO₂ has appeared to be a good indicator of the pollutant mixture. Furthermore, animal toxicological studies show that prolonged exposures can cause decreases in lung host defence and changes in lung structure. On these grounds, it is proposed that a long-term guideline for NO₂ be established. Selecting a well supported value based on the studies reviewed has not been possible, but it has been noted that a prior review conducted for the IPCS EHC (Environmental Health Criteria) document on NO₂ recommended an annual value of 40 µg/m³ (20 ppb). In the absence of support for an alternative value, this figure is recognized as an air quality guideline.”

EPAQS Justification of an Air Quality Standard for Nitrogen dioxide

“The Panel first considered whether there is evidence of a threshold concentration at which adverse health effects of short-term exposure to nitrogen dioxide cannot be detected. We concluded that experimental studies of healthy human beings have shown a threshold above 2000 ppb (400µg/m³). People with asthma are clearly more sensitive to the effects of the gas. However, the balance of evidence from experimental inhalation studies suggests that adverse health effects are unlikely to occur in subjects with asthma below a threshold of about 200 ppb.

In contrast to these findings, which are based on studies taking place over limited time periods, usually less than two hours, epidemiological studies suggest that adverse health effects may occur at lower concentrations averaged over longer periods of time. These observations based on levels of nitrogen dioxide recorded at fixed monitoring sites rather than personal exposure measurements and have involved exposures usually of several days or even more prolonged periods. In association with this, the health outcomes in such studies are related to concentrations of nitrogen dioxide measured one or two days earlier. Therefore, the Panel have taken the view that there may be a lower threshold than is indicated by the short-term experimental studies, possibly because of a cumulative effect.

² FEV₁ is the volume of air expired during the first second of a maximal or ‘forced’ expiration.

The Panel discussed the averaging time over which the proposed Standard should be measured and concluded that, since effects on health on people with asthma were detectable within an hour of exposure, in experimental studies, commencing an hourly averaging period would be appropriate. In the best judgement of the Panel, ambient outdoor concentrations of nitrogen dioxide in the United Kingdom do not rise to levels at which fit, healthy people will experience adverse effects. However, in order to protect susceptible people, especially those with asthma and other chronic lung diseases, from acute effects we concluded that a figure below 200 ppb, the apparent experimental threshold, would be appropriate. Since the epidemiological studies suggest acute effects below this threshold, we decided that a margin of safety should be applied and recommend a Standard for nitrogen dioxide of 150 ppb ($300\mu\text{g}/\text{m}^3$) measured over one hour.”

48. It will be seen that two expert groups reviewing essentially the same data came to slightly different conclusions regarding a short-term standard or guideline for NO₂. The WHO figure is 100 ppb and the EPAQS figure is 150 ppb, both expressed as hourly averages. In terms of likely impacts on health at the concentrations recommended, the difference is slight and reflects a difference in interpretation of effects recorded at low concentrations and in the margin of safety that was deemed desirable.

49. *On the basis of the above data, we suggest that a figure of 150 ppb ($300\mu\text{g}/\text{m}^3$) (1-hour average concentration) be adopted for use as an indoor air quality guideline.* In recommending this figure, we are aware that it may frequently be exceeded in houses that use gas as a fuel for cooking. The guideline includes a margin of safety and it should be recalled that it was derived from an analysis of studies that considered effects on individuals of greater than average sensitivity to NO₂. Exceedence of the guideline should not be taken as meaning that damage to health is inevitably associated with gas cookers. Indeed, for the majority of people, this seems very unlikely. However, a minority of individuals exposed to NO₂ indoors will be at risk of adverse effects as the guideline is exceeded.

50. In the case of NO₂, we have been able to adopt the concentration chosen by EPAQS as a guideline because the basis of the standard was the effect of NO₂ on lung function, studied in volunteers. These data apply as closely to the indoor environment as to the outdoor environment.

51. The case for a long term standard for NO₂ in outdoor air was considered by EPAQS:

“Adherence to the proposed hourly Standard is likely to protect vulnerable people, such as those with asthma, from acute health effects. However, taking account of the epidemiological studies, indoor and outdoor, and the longer exposures that these have entailed, the Panel have concluded that a longer-term Standard is also desirable in order to protect against possible cumulative effects on the health of the population. We believe that the evidence to date is insufficient to decide on an appropriate figure and instead we recommend a strategy of reduction in the annual average concentration of nitrogen dioxide in our towns and cities, in order to reduce the magnitude of any such effects. Since much new epidemiological evidence on the health effects of nitrogen dioxide will become available over the next few years, we recommend that a long-term Standard is reconsidered within the next three years.”

52. We note that a WHO group (The Scientific Advisory Committee set up to advise the EC Clean Air for Europe project) has recently supported the retention of the WHO annual average guideline for NO₂ of 40 µg/m³ (20 ppb) (WHO 2004). This decision was based on a detailed review of recent evidence some of which has been outlined, above. It is also noted that there has been disagreement as to the extent by which associations between outdoor concentrations of NO₂ and effects on health represent effects of NO₂ *per se*. The point has been made that outdoor NO₂ concentrations could be acting as an index of exposure to the complex mixture of pollutants generated by motor vehicles. However, the WHO group felt that, on balance, the evidence relating the possible effects of exposure to raised long-term average concentrations of NO₂ was sufficiently strong to support the annual average guideline. It will be understood that if there was debate and disagreement regarding the adoption of the guideline for use outdoors, there is likely to be similar or greater disagreement regarding its adoption indoors where NO₂ levels would not be expected to act as an indicator of exposure to pollutants generated by motor vehicles.

53. *However, we conclude that the figure of 40 µg/m³, 20 ppb (annual average concentration) should be adopted as a provisional guideline for long-term average concentrations of NO₂ indoors.* Long-term average concentrations can be monitored more easily than short-term average concentrations: diffusion tubes are suitable for the former but not for the latter. We recommend that diffusion tubes should be used

for comparing long-term indoor concentrations of NO₂ with our provisional guideline. Diffusion tubes are small plastic tubes containing an absorbent and reactive material. Tubes designed to absorb NO₂ are left in place for a fortnight, collected and analysed in a laboratory. We note that meeting the short-term (1 hour average) guideline should reduce long-term average concentrations.

Nitric oxide

54. Nitric oxide (NO) is produced with nitrogen dioxide when nitrogen in the air reacts with oxygen at high temperatures (see above). Nitric oxide is, remarkably, an important substance in the body and acts as a short-range messenger or transmitter substance. One of its most important roles is the mediation of relaxation of muscle cells in the walls of blood vessels. NO has been used to induce dilatation of lung blood vessels in babies with pulmonary hypertension. NO is also released by inflammatory processes in the airways. NO, NO₂ and other oxides of nitrogen are sometimes grouped together as NO_x. Until recently, little attention had been paid to the possible effects on health of exposure to ambient concentrations of nitric oxide: early work (see MAAPE report for references) showed that NO was significantly less active than NO₂ as an airway irritant. Work on animal models has shown that long-term exposure to concentrations of NO that can occur indoors, though generally for shorter periods than considered in the animal work, can produce changes in the lung. The changes are suggestive of early emphysema: this is interesting, as NO₂ at high concentrations, has been used as an agent for inducing emphysema in animal models.

55. We note that HSE have recently recommended that levels of nitric oxide should be kept below 1000 ppb in workplaces (Health and Safety Executive, 2003). It is possible for this level to be exceeded indoors. We have not previously considered nitric oxide in detail but plan to do so and incorporate our views in a future edition of this guidance. *We recommend that this should be kept under review.*

Carbon monoxide

56. Carbon monoxide is an important indoor air pollutant that is known to cause accidental deaths and severe damage to health in the UK. These effects only occur when concentrations rise above those found generally in UK homes. The picture is thus very different from that of NO₂ and in the case of carbon monoxide, the risk comes only from malfunctioning or unflued appliances which burn fossil fuels. Studies undertaken by BRE and reported by IEH show that carbon monoxide concentrations in UK houses are generally low though comparatively high peak concentrations can occur when unflued appliances such as gas cookers are in use. (Institute for Environment and Health, 1998).

Summary table of typical background carbon monoxide levels in mg/m³ (ppm) in UK homes (table adapted from Table 2.4 of Institute for Environment and Health report on Indoor Air Quality in the Home, 1998).

Study reference	Measurement	CO levels	Comments
Cox & Whichelow (1985) UK	CO detected and measured by a portable Ecolyzer in the living room 168 subjects	0-48.1 (0-42)	Higher levels recorded in homes with combustion appliances, lower levels in homes with non-CO-generating heating appliances
Wiech & Raw (1995) UK	CO detected and measured by Dräger passive diffusion tubes in the kitchen, living room and bedroom 40 households each with at least one asthmatic patient	All readings below 3.4 (3)	Installation of MVHR ³ had no effect on CO levels in the kitchen but was related to small reductions in CO levels in the living room and bedroom
Ross (1996) UK	Weekly average measurements by electrochemical detector and Dräger passive diffusion tubes 14 homes in the UK	CO levels [*] , electrochemical detector Kitchen 0.3-2.7 (0.3-2.4) Living room 0.2-2.5 (0.2-2.2) Bedroom 0.5-2.1 (0.4-1.8)	Differences in measurements due to differences in instrument precision and accuracy

*: Levels measured in Home 1 not included

³ MVHR: Mechanical Ventilation with Heat Recovery

57. In comparison with other air pollutants, the toxicological effects of carbon monoxide are well understood though the mechanisms underlying some effects, particularly those on the central nervous system, remain obscure (Maynard and Waller, 1999). Recent work has shown that, as in the case of NO, carbon monoxide is a naturally occurring short-range transmitter substance in the body. The major toxicological effects are mediated by the capacity of carbon monoxide to compete with oxygen for binding to haemoglobin and to reduce the capacity of haemoglobin to release such oxygen molecules as are transported to the tissues. This combination of effects leads to a reduction in oxygen availability and is particularly damaging to the brain and heart. The effect of carbon monoxide exposure may be expressed in terms of the amount of haemoglobin that is bound by carbon monoxide, i.e. the percentage of haemoglobin that is present as carboxyhaemoglobin. The following table taken from the EPAQS report on carbon monoxide (Department of the Environment, 1994a) shows the effects on health expected at defined carboxyhaemoglobin concentrations.

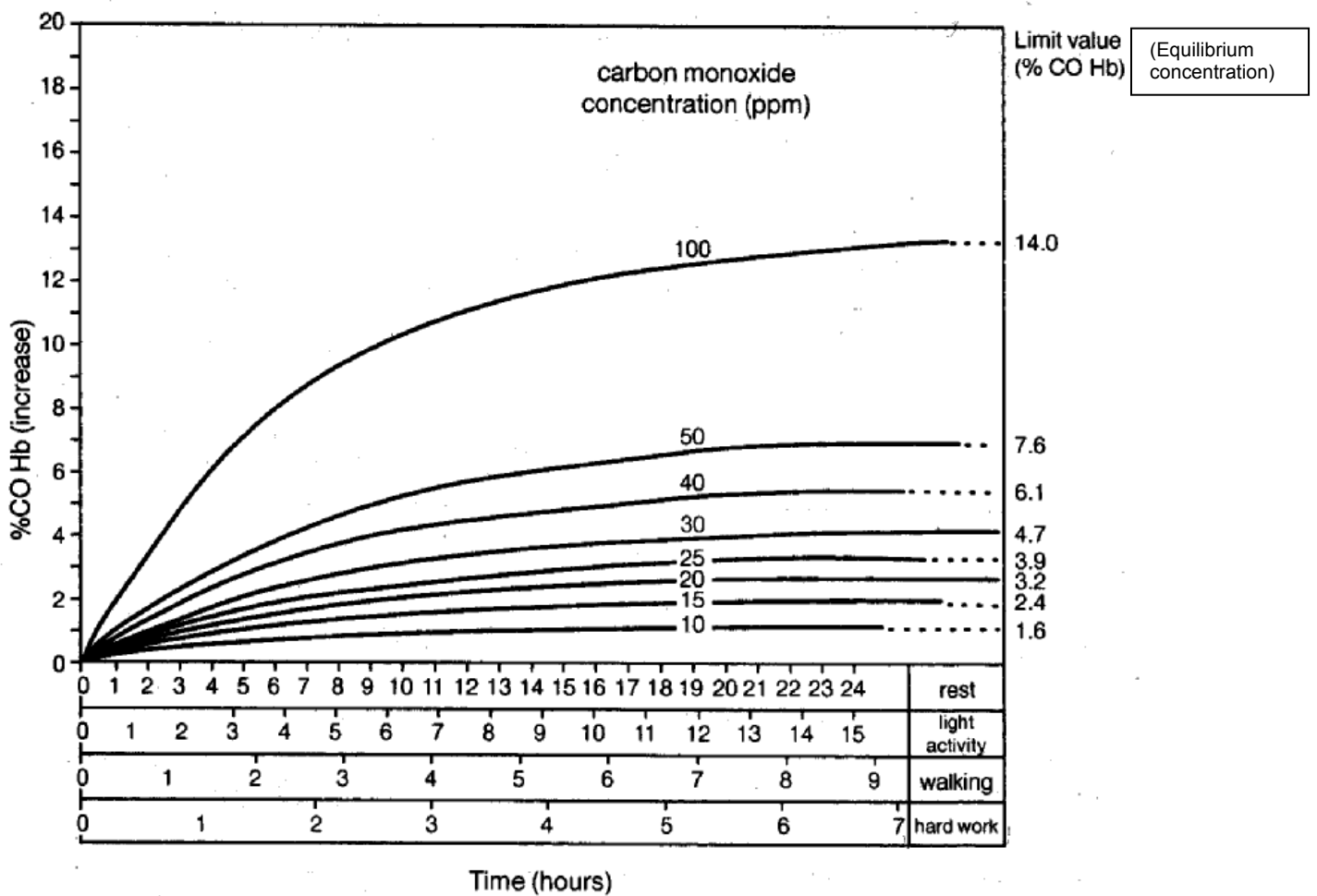
Human health effects of exposure to carbon monoxide

Blood carboxyhaemoglobin levels (%)	Observed health effects
2.5-4.0	Decreased short-term maximal exercise duration in young healthy men
2.7-5.2	Decreased exercise duration due to increased chest pain (angina) in patients with ischaemic heart disease
2.0-20.0	Equivocal effects on visual perception, audition, motor and sensorimotor performance, vigilance and other measures of neurobehavioral performance
4.0-33.0	Decreased maximal oxygen consumption with short-term strenuous exercise in young healthy men
20-30	Throbbing headache
30-50	Dizziness, nausea, weakness, collapse
over 50	Unconsciousness and death

58. Carbon monoxide competes with oxygen in a simple way for binding to haemoglobin. Exposure to a constant concentration of carbon monoxide causes the carboxyhaemoglobin (COHb) concentration to increase and reach an equilibrium

level. This is important. It is often thought that long-term exposure to a low concentration of carbon monoxide causes the COHb concentration to continue to increase: the expectation being that it would eventually reach 100%. This is quite untrue. The following graph shows the equilibrium concentrations of COHb that are reached on exposure to different concentrations of carbon monoxide.

Uptake of carbon monoxide by blood



(Department of the Environment, 1994a)

59. Note, for example, that at a concentration of carbon monoxide of 100 ppm, the equilibrium concentration of COHb is 14%. The diagram also makes clear that the increase in COHb concentration that takes place on exposure to carbon monoxide occurs more rapidly the faster one breathes. The diagram shows that if an individual breathes a concentration of carbon monoxide of 100 ppm then a COHb concentration of just over 10% is achieved after 3 hours of hard physical work but only after about 11 hours at rest. This understanding of how carbon monoxide is taken up by the blood and of the relationship between COHb levels and health effects is of great assistance in suggesting a guideline for carbon monoxide concentrations in indoor air.

60. A review of recent studies has suggested that prolonged exposure to concentrations of carbon monoxide that produce few symptoms and no clinical signs of acute poisoning, may produce effects on the central nervous system (Townsend and Maynard 2002a; Townsend and Maynard 2002b). The exact mechanism by which such effects are produced remains incompletely understood, but there is increasing recognition that they may occur as a result of prolonged exposure to levels of carbon monoxide that produce only fairly minor symptoms. Malfunctioning heating devices burning fossil fuel can produce such levels of carbon monoxide. Failure to recognise such effects can lead to prolonged and worsening exposure and thus to serious injury and death. The dangers of carbon monoxide exposure have been explained in a recent letter sent by the Chief Medical Officer and Chief Nursing Officer to all general practitioners and community nurses in the UK (Department of Health, 2002).

61. Some evidence on the possible effects of exposure to carbon monoxide has recently been acquired from epidemiological studies of associations between outdoor concentrations of pollutants and ill health. These studies suggest an association between peak daily concentrations of carbon monoxide and the occurrence of heart attacks and admissions to hospital for treatment of heart attacks (Renton A, 1997; Lanes S, 1985; Cox DR, 1992; Morgenstern H and Thomas D, 1993; Zmirou D *et al*, 1998, Anderson HR *et al*, 1997). The studies are difficult to interpret and it is possible that effects caused by exposure to other pollutants, including particles, are incorrectly attributed to exposure to carbon monoxide. These studies were considered in relation to the effects on outdoor air pollutants by the Committee in 1998

(Department of Health, 1998): it was concluded that the evidence base was insufficient to allow the effects of outdoor exposure to carbon monoxide, if any, to be quantified.

62. Both WHO and EPAQS have set guidelines and standards for carbon monoxide concentrations in outdoor air (World Health Organization, 2000; Department of the Environment, 1994a). It is noted, in passing, that the WHO guidelines should be seen as being applicable to indoor air though they are more generally regarded as being specifically applicable to outdoor air. The same evidence was considered by both groups of experts and identical conclusions were reached. Very similar conclusions have been reached by expert groups in other countries including the United States (US Environmental Protection Agency, 2000). These conclusions, based on studies of the relationship between exposure to carbon monoxide and COHb concentrations and between COHb concentrations and effects on health, are as applicable to the indoor as to the outdoor environment and we recommend that the same concentrations and averaging times be used for guidelines for indoor air quality. *Our recommended guidelines are:*

<i>15 minute averaging time:</i>	<i>90 ppm (100 mg/m³)</i>
<i>30 minute averaging time:</i>	<i>50 ppm (60 mg/m³)</i>
<i>1 hour averaging time:</i>	<i>25 ppm (30 mg/m³)</i>
<i>8 hour averaging time:</i>	<i>10 ppm (10 mg/m³)</i>

63. These combinations of concentrations and averaging times have been set to prevent the COHb concentration rising above about 2%. This is likely to be protective and to incorporate a margin of safety for all individuals at rest. Some studies in volunteers suffering from a reduced blood supply to the heart (coronary artery disease) have shown that exposure to a concentration of carbon monoxide designed to raise the COHb concentration rapidly to about 2%, can reduce the period of vigorous exercise needed to induce chest pain (angina) (Anderson EW *et al* 1973; Kleinman MT *et al* 1989; Allred EN *et al* 1989). These observations call into question the concept of a threshold of effect of carbon monoxide. A threshold is likely to exist in all healthy individuals and whilst this might well not be identical from subject to subject, the guidelines recommended above incorporate a margin of safety. In people suffering from coronary artery disease, individual thresholds are

also likely to exist, albeit at lower levels of COHb than in healthy subjects. Though our understanding of the toxicological properties of carbon monoxide is greater than that of other air pollutants and though the guidelines proposed above include a margin of safety, this margin may be narrower than for some other pollutants.

Formaldehyde

64. Formaldehyde is an irritant gas with a characteristic odour. It is a small molecule, the simplest of the large family of aldehydes and a strongly irritant substance. A detailed review of ambient concentrations of formaldehyde, indoors and out and of the health effects associated with exposure has been provided by the IEH review: Indoor Air Quality in the Home (Institute for Environment and Health, 1996). This review provides tables of measurements of concentrations of formaldehyde in houses of different ages and types, and also of concentrations found in mobile or prefabricated homes. Concentrations found indoors are often higher than those found outdoors. Outdoor concentrations range from about 0.001 mg/m³ to 0.02 mg/m³: a figure of 0.01 mg/m³ is often quoted. In the indoor environment, the most significant source of formaldehyde is likely to be pressed wood products containing urea-formaldehyde resins, especially medium-density fibreboard and particleboard. In the 1970s, the widespread use of 'foam-in-place' urea-formaldehyde cavity insulation also provided a significant source. Tobacco smoke is a potent source of formaldehyde. The table shown below is reproduced from the WHO Air Quality Guidelines published in 2000 (World Health Organization, 2000).

Average exposure concentrations to formaldehyde and contribution of various atmospheric environments to average exposure to formaldehyde

Source	Concentration (mg/m ³)	Exposure (mg/day)
Ambient air (10% of time; 2 m ³ day)	0.001-0.02	0.002-0.04
Indoor air		
Home (65% of time; 10 m ³ /day)		
- Conventional	0.03-0.06	0.3-0.6
- Mobile home	0.1	1.0
- Environmental tobacco smoke	0.05-0.35	0.5-3.5
Workplace (25% of time 8 m ³ /day)		
- without occupational exposure ^a	0.03-0.06	0.2-0.5
- with occupational exposure	1.0	8.0
- environmental tobacco smoke	0.05-0.35	0.4-2.8
Smoking (20 cigarettes/day)	60-130	0.9-2.0 ^b

^a Assuming the normal formaldehyde concentration in conventional buildings

^b Total amount of formaldehyde in smoke from 20 cigarettes

Acute health effects observed at different formaldehyde levels in mg/m³ (ppm)
(from Samet *et al*, 1998)

Reported effects	Concentration of formaldehyde	
None reported	0.00 – 0.06	(0.0 – 0.5)
Neurophysiologic effects ^a	0.06 – 1.8	(0.05 – 1.5)
Odour threshold	0.06 – 1.2	(0.05 – 1.0)
Eye irritation ^b	0.01 – 2.4	(0.01 – 2.0)
Upper airway irritation	0.12 – 30.0	(0.10 – 25.0)
Lower airway and pulmonary effects	6.0 – 36.0	(5.0 – 30.0)
Pulmonary oedema, inflammation, pneumonia	60 – 120	(50 – 100)
Death	> 120	(> 100)

^a: Determination of optical chronaxy⁴, electroencephalography, and sensitivity of dark-adapted eyes to light

^b: At 0.01 mg/m³ (0.01 ppm) other possibly synergistically acting pollutants were also present

⁴ Optical chronaxy: the duration of exposure needed to allow detection of illumination at twice the minimum level that can be detected.

65. The capacity to detect the odour of formaldehyde varies widely from person to person. WHO's Air Quality Guidelines suggest that at a concentration of 0.03 mg/m³ about 10% of people can detect formaldehyde, at 0.6 mg/m³, 90% of people can detect it. It is generally accepted that nose and throat irritation begins on exposure to a concentration of about 0.1 mg/m³. The evidence base that supports the above estimates of effects at different concentrations of formaldehyde is extensive and has been reviewed in detail in the IEH report (Institute for Environment and Health, 1996). These studies include epidemiological studies of people in homes with low indoor concentrations of formaldehyde and also volunteer studies of the effects of short-term exposure to specified concentrations. As expected with an irritant gas that can cause marked bronchoconstriction at high concentrations, a reduction in indices of lung function has been observed on exposure to low concentrations. One epidemiological study by Krzyzanowski *et al* (1990) looked specifically at effects in children and reported that long-term exposure to concentrations as low as 0.036 mg/m³ produced a significant reduction in peak flow⁵ in about 10% of subjects. Children with asthma were more affected than children without asthma: this would be expected in the case of a respiratory irritant. No threshold of effect of formaldehyde on indices of lung function was detected.

66. Many studies have concentrated on looking for health effects in homes with known sources of formaldehyde, for example, homes in which urea-formaldehyde cavity insulation has been installed. Some of these studies examined the change in symptoms produced by removal of the insulation (Broder *et al*, 1988). The general conclusion was that urea-formaldehyde cavity insulation could produce levels of formaldehyde that induced minor symptoms though more severe effects were occasionally observed in the period immediately following the installation of the insulation.

67. The sensitivity of the respiratory system to formaldehyde varies significantly from subject to subject. WHO's Air Quality Guidelines concluded that lung function of healthy non-smokers and asthmatics exposed to formaldehyde at levels up to 3.7 mg/m³ was generally unaffected and quoted work by Sauder *et al* (1987) in support of this.

⁵ Peak Flow: the maximum rate at which air can be expired from the lungs, expressed in litres/minute. Used as a means of monitoring airway resistance and thus constriction of the airways.

68. Formaldehyde is recognised as a probable human carcinogen: in 1995 the WHO's International Agency for Research on Cancer (IARC) (World Health Organization, 1995) classified formaldehyde as a probable human carcinogen: Group 2A. Formaldehyde is certainly carcinogenic in rats but uncertainty about its carcinogenicity in man, even at high exposure levels, remains. Partanen concluded after a review of 32 studies, that the evidence was about sufficient for the conclusion that at least substantial occupational formaldehyde exposures are carcinogenic and that the risk was probably greatest for the nasal cavities and nasopharynx, but not the lung (Partanen, 1993).

69. If it is accepted that formaldehyde is at least likely to be a human carcinogen at high exposure concentrations, it remains to discuss possible carcinogenic effects at the much lower concentrations found indoors in most UK homes. The WHO Expert Group that reviewed formaldehyde for the revised Air Quality Guidelines (World Health Organization, 2000) noted that the dose-response curve for nasal cancer in rats was markedly non-linear at low concentrations – the response being clear at 16.7 mg/m³ but being disproportionately reduced at low concentrations. It was also noted that the dose-response curves for neoplastic changes, cell turnover, DNA cross-links and for hyper-proliferation of the nasal epithelium were nearly identical. This concordance led the expert group to think that hyper-proliferation induced by cytotoxicity played a significant role in the causation of nasal tumours by formaldehyde in rats, and led them to conclude that as long as exposure to formaldehyde was kept below a level likely to damage the nasal epithelium, the risk of carcinogenicity would remain very low – if any existed.

70. The conclusion of WHO's expert group:

“Thus, if the respiratory tract is not repeatedly damaged, exposure of humans to low, noncytotoxic concentrations of formaldehyde can be assumed to be associated with a negligible cancer risk”

has been accepted by us (Department of Health, 2000). WHO's air quality guideline for formaldehyde, expressed as a 30-minute average concentration is 0.1 mg/m³ set on the basis that this was identified by a WHO expert group as the lowest level associated with nose and throat irritation in humans. WHO experts accepted that some individuals could detect lower concentrations of formaldehyde by odour. The WHO expert group added that since this is over one order of magnitude lower than a

presumed threshold for cytotoxic damage to the nasal mucosa, this guideline value represents an exposure level at which there is a negligible risk of upper respiratory tract cancer in humans (World Health Organization, 2000). In comparison with factors applied by EPAQS when setting outdoor air quality standards for the known genotoxic carcinogens benzene, 1,3-butadiene and the polycyclic aromatic hydrocarbons, this is a small safety factor. However, the evidence relating to the carcinogenic effects of exposure to low concentrations of formaldehyde is not conclusive and we feel that a guideline of 0.1 mg/m^3 is acceptable.

71. *A guideline of 0.1 mg/m^3 with an averaging time of 30 minutes is recommended. It is not possible to extrapolate satisfactorily from this short averaging time to a longer one.* Simply reducing the concentration for longer averaging times to keep the concentration-time product constant, does not seem appropriate for a substance where avoidance of irritation is the desired effect.

72. We recommend that the evidence base on the effects of indoor exposure to formaldehyde be kept under review and that advances in the understanding of the mechanisms by which formaldehyde produces cancer be monitored.

Other aldehydes found in indoor air

73. There may be other aldehydes present in indoor air as well, but these are generally not as strong irritants as formaldehyde. A review of the health effects of several aldehydes is available on our website: (<http://www.advisorybodies.doh.gov.uk/comeap/>).

Benzene

74. Benzene is a genotoxic human carcinogen that is found in greater concentrations indoors than outdoors if smokers occupy the indoor environment. Tobacco smoke and the permeation of air from integral garages are the main sources of benzene indoors. Concentrations in homes without smokers are generally low though may exceed those found outdoors and may be significantly increased if the indoor environment connects with an integral garage. A WHO report on Indoor Air Quality (World Health Organization, 1989) found that the 10th, 50th, 90th and 98th percentiles of concentrations of benzene in “typical homes” were 2, 10, 20 and

30 $\mu\text{g}/\text{m}^3$, respectively⁶. A study of homes in Avon (UK) undertaken by the Building Research Establishment and published in 1996 (Brown and Crump, 1996) looked at 173 homes and monitored TVOCs in addition to benzene and toluene. The mean benzene concentration for most rooms was around 8 $\mu\text{g}/\text{m}^3$. In homes with at least one smoker, significantly higher concentrations of benzene were recorded: the highest living room concentration being 106 $\mu\text{g}/\text{m}^3$, the highest bedroom concentration being 78 $\mu\text{g}/\text{m}^3$. It was discovered that in homes without smokers the most important sources of indoor benzene lay outdoors but that indoor concentrations were typically 1-1.6 times those recorded outdoors. A summary of the findings is presented in the table below.

Summary of benzene measurements ($\mu\text{g}/\text{m}^3$) in homes in Avon (Table taken from BRE report, Berry *et al*, 1996)

Room	Mean of monthly readings in each house						All readings					
	n	Mean	SD	RSD(%)	Min	Max	n	Mean	SD	RSD (%)	Min	Max
Main bedrooms	173	8	4	58	2	2	1504	8	6	86	0	78
Living rooms	173	8	6	59	2	46	1503	8	8	95	0	106
Kitchens	6	6	2	35	3	8	51	6	4	62	0	17
Bathrooms	6	6	3	44	3	9	51	6	5	76	0	20
Second bedrooms	20	5	2	49	2	11	109	5	4	72	0	21
Outside	13	5	1	29	3	8	125	5	3	65	0	16

Percentiles of mean benzene concentrations in homes in the Avon area of the UK were as follows:

Percentiles of mean benzene concentrations in homes in Avon (table taken from the BRE report, Berry *et al*, 1996)

Room	Percentile values ($\mu\text{g}/\text{m}^3$)			
	10%	50%	75%	95%
Main bedroom	4	7	9	14
Living room	4	7	9	15

75. A detailed account of the findings of international studies of indoor concentrations of benzene and other VOCs can be found in the IEH report (Institute for Environment and Health, 1996).

76. The only health effect of concern at indoor (or ambient outdoor) concentrations of benzene is carcinogenicity: the main effect of concern is the induction of leukaemia. The evidence relating to the capacity of benzene to induce

⁶ 10% of homes had concentrations of less than 2 $\mu\text{g}/\text{m}^3$ and 98% of homes had concentrations of less than 30 $\mu\text{g}/\text{m}^3$.

leukaemia on exposure to high concentrations was reviewed by EPAQS in 1994 (Department of the Environment, 1994b). This review concluded that none of the studies of occupational exposure to benzene that were considered had demonstrated a statistically significant increased risk of leukaemia as a result of occupational exposure to the equivalent of a working lifetime exposure to less than 500 ppb ($1620\mu\text{g}/\text{m}^3$). EPAQS noted that, as benzene is a recognised genotoxic human carcinogen, no completely safe level of exposure could be identified, but argued that by applying a series of uncertainty or safety factors, an air quality standard that would provide a high level of safety could be derived. Two factors, each of 10, were applied: the first to take account of whole life as compared with working life exposures and the second to take some account of possible variations in individual sensitivity to the carcinogenic effects of benzene. Thus, an air quality standard of 5 ppb ($16.3\mu\text{g}/\text{m}^3$) expressed as an annual average concentration was derived. A rider was added to the effect that levels of genotoxic carcinogens such as benzene should be as low as possible and a target of 1 ppb (based not on toxicological reasoning, but rather on an appreciation that this would be a feasible figure) was recommended.

77. It seems to us that this outdoor standard would apply equally well indoors. We note that a long averaging time was recommended. This was to take account of the generally accepted view that it is the extent of cumulative exposure to genotoxic carcinogens that controls the risk of cancer. We are aware of the argument that peak exposures to benzene also play a part in controlling the likelihood or risk of cancer. The mechanistic reasoning is appealing: overloading of defence systems and the possible metabolic shunting of carcinogens down pathways that lead to metabolic activation rather than deactivation, could make short duration exposure to very high concentrations disproportionately dangerous. Whilst recognising the force of this argument, we feel that the peak concentrations encountered in occupational settings are unlikely – indeed impossible – in the domestic indoor air setting. Occupational exposures years ago ran, on occasion, to concentrations of tens of parts per million – such concentrations are unheard of in the domestic indoor environment. It may also be argued that if such peaks were necessary to induce cancer, the approach adopted by EPAQS was particularly cautious.

78. The above considerations lead us to think 5ppb ($16.3\mu\text{g}/\text{m}^3$) would be a satisfactory figure for a guideline for indoor concentrations of benzene. However, we

note that the National Air Quality Strategy, in accordance with the latest EC Directive dealing with benzene, has established $5\mu\text{g}/\text{m}^3$ (1.6ppb) as an Air Quality objective. To avoid confusion we have adopted this value as a indoor air guideline.

79. Having made this recommendation, we point out that short-term measurements of benzene indoors often report concentrations greater than 1.6ppb. The effects of such concentrations on health cannot be estimated – only in terms of the long-term average concentration have we a basis for establishing a level likely to be associated with only very small or negligible risks.

80. It may be asked why we have not estimated the risk to health posed by exposure to indoor concentrations of benzene in numerical terms, i.e. why have we not undertaken a quantitative risk assessment. Our reasons are two-fold:

- (i) a quantitative risk assessment (QRA) approach does not allow definition of a guideline unless some acceptable level of risk can be established. In some countries this has been done and estimated lifetime risks of less than 1 in 1 million (1×10^{-6}) are regarded as negligible (*de minimus*) and thus acceptable. No such decision has been taken regarding risks from carcinogenic air pollutants (indoors or out) or other exposures to carcinogens in the UK;
- (ii) we are not persuaded of the accuracy of the mathematical models used to calculate risks attendant on exposure to low concentrations of genotoxic carcinogens. In holding this opinion, we draw on advice of the Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment and on the published conclusions of that committee (Department of Health, 2004).

Polycyclic aromatic hydrocarbon (PAH) compounds

81. Polycyclic Aromatic Hydrocarbon (PAH) compounds are compounds in which the molecules are made up of several, often four or more, benzene rings. Amongst the PAH compounds are notable carcinogens including some of the first compounds to be demonstrated to be carcinogenic in animal models.

82. PAH compounds are produced as a result of incomplete combustion of organic material. Such materials include tobacco, coal, wood, petrol, diesel oil and food heated during cooking processes. As would be expected from such a range of sources, PAH compounds are ubiquitous indoors and out. There are also many industrial sources of PAH compounds and occupational exposure in the old coal gas industry (coking processes) and in aluminium smelting, can lead to cancer if high exposures occur. Modern occupational standards closely regulate such exposures.

83. PAH compounds occur both in the gas phase (as vapours) and adsorbed or condensed onto the surfaces of airborne particles. Individual PAH compounds – and there are many such compounds – vary in their volatility and thus in the extent of their partitioning between the gas phase and the solid phase. Many PAH compounds have been investigated toxicologically and amongst those studied are some of the most potent and best recognised genotoxic carcinogens known.

84. In 1999, EPAQS recommended an outdoor air quality standard for PAH compounds using benzo[a]pyrene as an indicator compound (Department of the Environment, Transport and the Regions, 1999). In essence, the same approach was adopted by the Panel as in the case of benzene, though instead of identifying as a starting point a level of occupational exposure at which no effects had been observed, a level at which effects had been observed was defined. The occupational exposures used as a basis to derive the standard took place in aluminium smelting works and the Panel confirmed by examining data on the composition of the mixture of PAH compounds measured during occupational exposure that this was broadly similar to that found in outdoor air. They also considered the comparative carcinogenicity of the major PAH compounds found in the occupational studies and in ambient air and concluded that benzo[a]pyrene made an approximately equivalent contribution to the total carcinogenicity of both mixtures. This led the Panel to their view that benzo[a]pyrene could be reliably used as an index or marker compound.

85. EPAQS argued as follows: a lifetime occupational exposure to a mixture of PAH compounds characterised by a concentration of benzo[a]pyrene of 0.25-2.5 $\mu\text{g}/\text{m}^3$ was identified as being associated with a 50% increase in the risk of lung cancer. Lung cancer was identified as the effect of greatest concern: evidence relating to bladder cancer was regarded as less well developed. Taking 0.25 $\mu\text{g}/\text{m}^3$ as a

starting point, the Panel applied three factors each of 10. These were to account and adjust for: (i) the fact that the starting point was a *lowest observed adverse effect level* rather than a *no observed effect level*; (ii) the difference between a working lifetime exposure and a whole life exposure; and, (iii) to allow for individual variation in susceptibility to the carcinogenic effects of PAH compounds. Application of these factors led to a standard of 0.25 ng/m³ expressed as an annual average concentration. As regards measurement of PAH compounds to investigate compliance with the standard, it was agreed that the total benzo[a]pyrene concentration (in gas phase and particle/solid phase) should be measured. The Panel commented that any risks from exposures below the recommended standard are (likely to be) minimal.

86. We are not aware of data on concentrations of PAH compounds in homes in the UK. Furthermore, we are not certain that benzo[a]pyrene makes the same contribution to the total carcinogenicity of the indoor mixture as it does to the outdoor mixture of PAH compounds. We therefore recommend that, as an interim measure, the outdoor standard recommended by EPAQS be adopted as a provisional indoor guideline. We note that EPAQS will be re-examining the outdoor standard in the light of new data concerning the PAH compound di-benzo[a,l]pyrene. We recommend that the PAH profile of indoor air be studied and that our provisional guideline be reviewed in the light of greater knowledge.

The monitoring of pollutants in the home

87. We note that having provided information on the types and sources of pollutants found in indoor environments, readers will be interested in the type of equipment available to them for monitoring levels of pollutants in, for example, their homes.

88. Monitoring of every pollutant described in this guidance document may not be possible for a variety of reasons. Detailed advice on assessment of indoor air quality can be found in a recent report published by the Building Research Establishment (Crump *et al*, 2002). As mentioned earlier, in some instances the complexity or the expense of equipment required for monitoring may make monitoring impractical to anyone other than specialists in the field of air pollutant monitoring. In these situations, it would be sensible reduce the emissions of the pollutant and seek advice from a company which specialises in monitoring the pollutant of concern.

89. The table below shows the type of equipment available for monitoring some of the pollutants found in indoor environments:

Types of equipment available for the monitoring of pollutants found in indoor environments

Pollutant	High concentration alarms available	Monitoring for compliance with guidelines	
		Simple/Cheap	Sophisticated/Expensive
NO ₂		✓ (for annual guideline)	✓ (for 1-h guideline)
CO	✓		✓
Formaldehyde			✓
Benzene		✓	
PAH			✓

Conclusions

90. In suggesting guidelines for indoor air quality, we have tried to identify levels of pollutants which, if not exceeded, would offer a significant level of safety to those exposed to them. It is accepted that complete safety cannot be guaranteed in the case of carcinogens such as benzene and PAH compounds, but in the cases of nitrogen dioxide and carbon monoxide, for example, we believe that it is possible to suggest a level that can be regarded as acceptably safe.

91. It is, however, true to say that there is a school of thought that draws heavily on time-series epidemiological studies and which argues that all levels of even such pollutants as nitrogen dioxide and carbon monoxide pose some level of risk for everybody. For the great majority, at low concentrations, the risk is small and might sensibly be ignored. However, for some very susceptible individuals, even very low concentrations may pose a significant risk. It may be this risk to susceptible individuals that is manifested in the findings of time-series epidemiological studies. We have tried to combine an appreciation of the scientific evidence with a clear perception of the need for practical advice, which, if followed, will ensure that the great majority of people are not exposed to unacceptable risk.

92. WHO has faced this problem in developing its Air Quality Guidelines for Europe (World Health Organization, 2000). It is made clear by WHO that the

guidelines indicate a level of exposure, which, in the great majority of people, would not be expected to cause significant harm to health. It is further explained that no guarantee of safety for “*the most sensitive individual*” is provided. Another and more subtle point should also be noted in respect of WHO’s Air Quality Guidelines and indeed, with all such guidelines and standards: the guideline identifies a level of pollution at which for the great majority of people significant effects on health are not expected. This certainly does not imply that significant effects will appear as soon as the guideline or standard is exceeded. Many guidelines and standards include a margin of safety and thus, only very few people would be expected to experience adverse effects on health as levels of pollutants rise a little above the guideline or standard. This is important and places in context media headlines that report numbers of exceedences of guidelines and standards but not the extent of those exceedences or their likely consequences.

93. The following table summarises our recommendations for guideline values.

COMEAP recommendations for guideline values for pollutants found in indoor air.

Pollutant	Concentration	Averaging time
Nitrogen dioxide	150 ppb (300 µg/m ³)	1 hour average
	20 ppb (40 µg/m ³)	Annual average*
Carbon monoxide	90 ppm (100 mg/m ³)	15 minutes
	50 ppm (60 mg/m ³)	30 minutes
	25 ppm (30 mg/m ³)	1 hour
	10 ppm (10 mg/m ³)	8 hours
Formaldehyde	0.1 mg/m ³ (0.1 ppm)	30 minutes
Benzene	1.6 ppb (5.0 µg/m ³)	Annual average
Benzo[a]pyrene	0.25 ng/m ³	Annual average*

* Provisional Guidelines

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