

# GUIDELINES FOR AIR QUALITY

This WHO document on the *Guidelines for Air Quality* is the outcome of the WHO Expert Task Force meeting held in Geneva, Switzerland, in December 1997. It bases on the document entitled “Air Quality Guidelines for Europe” that was prepared by the WHO Regional Office for Europe and regional background papers.

**Note to the user:**

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## Foreword

Achievements in air quality management underlie increased economic and social welfare in many developing countries. Sound air quality management is also a proven way of enhancing public health, since air pollution is associated with increases in outpatient visits due to respiratory and cardiovascular diseases, in hospital admissions and in daily mortality. Recent estimates of the increase in daily mortality show that on a global scale 4-8% of premature deaths are due to exposure to particulate matter in the ambient and indoor environment. Moreover, around 20-30% of all respiratory diseases appear to be caused by ambient and indoor air pollution, with emphasis on the latter. It is suggested that without clean air, a sound economic development becomes virtually impossible and social conflicts inevitable.

Although enormous progress has been made in developing clean air implementation plans for urban areas, especially in developed countries, a substantial number of people living in urban areas - around 1.5 billion, or 25 per cent of the global population - are still exposed to enhanced concentrations of gaseous and particle compounds in the air they breathe. And the use of open fires for indoor cooking and heating currently exposes about 2 billion people to quite substantial concentrations of suspended particulate matter, 10-20 times higher than ambient concentrations according to the limited measurements available. Other sources of air pollution include industrial and vehicular emissions, as well as vegetation fires. Furthermore, the rate of population growth continues to increase and is likely to peak around the year 2000, leading to a doubling of the global population by the middle of the 21<sup>st</sup> century. Most population growth will occur in low-income countries and will stress already inadequate infrastructures and technical and financial capacities. In parallel, the process of urbanisation will continue, such that the proportion of the global population living in cities will increase from around 45% to around 62% by the year 2025, creating dense centres of anthropogenic emissions.

The primary aim of the WHO *Guidelines for Air Quality* is to protect public health from the effects of air pollution, and to eliminate or minimize exposure to hazardous pollutants. Air quality guidelines are set up to help governments derive legally enforceable air quality standards, and to guide the environmental health authorities and professionals who are trying to protect people from the harmful effects of environmental air pollution.

Agenda 21 states in Chapter 6 on human health and environmental pollution:

Nationally determined action programmes in this area, with international assistance, support and coordination where necessary, should include:

*(a) Urban air pollution:*

- (i) Develop appropriate pollution control technology on the basis of risk assessment and epidemiological research for the introduction of environmentally sound production processes and suitable safe mass transport.*
- (ii) Develop air pollution control capacities in large cities, emphasizing enforcement programmes and using monitoring networks, as appropriate.*

*(b) Indoor air pollution:*

*(i) Support research and develop programmes for applying prevention and control methods to reducing indoor air pollution, including the provision of economic incentives for the installation of appropriate technology.*

*(ii) Develop and implement health education campaigns, particularly in developing countries, to reduce the health impact of domestic use of biomass and coal.*

The WHO *Guidelines for Air Quality* should help to greatly reduce the burden of excess mortality and preventable disability suffered by the poor. It should also help counter potential health threats resulting from economic crises, unhealthy environments and risky behaviour. In this sense, the *Guidelines* contribute to meeting two of the key challenges that were highlighted in the 1999 World Health Report and, thus, they contribute to making health a fundamental human right.

Dr Richard Helmer  
Director  
Department of Protection of the Human Environment

## Preface

The risks posed to human health by air pollution have been evaluated since the 1950s and guideline values were derived in 1958. In 1987, the WHO Regional Office for Europe EURO published the *Air Quality Guidelines for Europe*. Since 1993, these guidelines have been revised and updated. In a recent Expert Task Force Meeting convened in December 1997 in Geneva, Switzerland, the Guidelines for Air Quality was extended to provide global coverage and applicability, and the issues of air quality assessment and control were addressed in more detail. The WHO *Guidelines for Air Quality* document is the outcome of the consensus deliberations of the WHO Expert Task Force.

The WHO *Guidelines for Air Quality* provides a basis for protecting public health from the adverse effects of environmental pollutants and for eliminating, or reducing to a minimum, contaminants that are known or likely hazards to human health and well-being. The *Guidelines* does so by providing background information and guidance to governments for making risk management decisions, particularly in setting standards. It also helps governments carry out local air quality control measures.

The WHO *Guidelines for Air Quality* values are levels of air pollution below which lifetime exposure, or exposure for a given averaging time, does not constitute a significant health risk. If these limits are exceeded in the short-term it does not mean that adverse effects automatically occur; however the risk of such effects increases. Although the *Guidelines for Air Quality* values are health- or environment-based levels, they are not standards *per se*. Air quality standards are air quality guidelines promulgated by governments, for which additional factors may be considered. For example, the prevailing exposure levels, the natural background contamination, environmental conditions such as temperature, humidity and altitude, and socio-economic factors.

When proceeding from the *Guidelines for Air Quality* to standards, policy options include such questions as what proportion of the general population, and which susceptible groups, should be protected. Several additional items must also be considered: the legal aspects; a definition of what constitutes adverse effects; a description of the population at risk; the exposure-response relationship; the characterisation of exposure; an assessment of risks and their acceptability; and the financial costs of air pollution controls and their benefits.

The *Air Quality Guideline* has been prepared as a practical response to the need for action with respect to air pollution at the local level, and for improved legislation, management and guidance at the national and regional levels. WHO will be pleased to see that these *Guidelines* are used widely. Continuing efforts will be made to improve its content and structure. It would be appreciated if users of the *Guidelines* would provide feedback and their own experiences. Please send your comments and suggestions on the WHO *Guidelines for Air Quality – Guideline document* directly to the Department of Protection of the Human Environment, Occupational and Environmental Health, World Health Organization, Geneva, Switzerland (Fax: +41 22-791 4123, e-mail: [schwelad@who.int](mailto:schwelad@who.int)).



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Prof. Dr Ursula Ackermann-Liebrich, University of Basel, Switzerland;  
Dr Amrit Aggarwal, National Environmental Engineering Research Institute, Nagpur, India;  
Mr Jonathan Bower, AEA Technology, Culham, United Kingdom;  
Dr Bingheng Chen, World Health Organization, Geneva, Switzerland;  
Dr Mostafa El-Desouky, Ministry of Health, Kuwait  
Dr Ruth Etzel, Centres for Disease Control and Prevention, Atlanta, GA, USA;  
Dr Hidekazu Fujimaki, National Institute for Environmental Studies, Ibaraki, Japan;  
Dr Kersten Gutschmidt, World Health Organization, Geneva, Switzerland;  
Dr Richard Helmer, World Health Organization, Geneva, Switzerland;  
Dr Michal Krzyzanowski, WHO European Centre for Environment & Health (ECEH), De Bilt, Netherlands;  
Dr Rolaf van Leeuwen, WHO European Centre for Environment & Health (ECEH), De Bilt, Netherlands;  
Mr Gerhard Leutert, Federal Office of Environment, Forests and Landscape, Bern, Switzerland;  
Professor Morton Lippmann, New York University Medical Centre, Tuxedo, NY, USA;  
Ms Angela Mathee, Eastern Metropolitan Substructure (Johannesburg), Sandton, South Africa; Dr Robert L. Maynard, Department of Health, London, United Kingdom;  
Professor Frank Murray, Murdoch University, Murdoch, Australia;  
Professor Mahmood Nasralla, National Research Centre, Dokki, Cairo, Egypt;  
Dr Roberto Romano, Pan American Health Organization/WHO Regional Office for the Americas, Washington, DC, USA;  
Dr Isabelle Romieu, Centres for Disease Control and Prevention, Atlanta, GA, USA;  
Dr Dieter Schwela, World Health Organization, Geneva, Switzerland;  
Professor Bernd Seifert, Institute for Water, Soil & Air Hygiene, Federal Environmental Agency Berlin, Germany;  
Dr Bimala Shrestha, WHO Representative’s Office, Kathmandu, Nepal;  
Professor Kirk Smith, University of California, Berkeley, CA, USA;  
Dr Yasmin von Schirnding, World Health Organization, Geneva, Switzerland;  
Professor Gerhard Winneke, Universität Düsseldorf, Germany;  
Dr Ruqiu Ye, National Environmental Protection Agency, Beijing, People’s Republic of China;  
Dr Maged Younes, World Health Organization, Geneva, Switzerland.

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## Executive Summary

### 1. *Introduction*

Air pollution is a major environmental health problem, affecting developed and developing countries around the world. Increasing amounts of potentially harmful gases and particles are being emitted into the atmosphere on a global scale, resulting in damage to human health and the environment. It is damaging the resources needed for the long-term sustainable development of the planet.

There are three broad sources of air pollution from human activities: Stationary sources, Mobile sources, and Indoor sources. In developing countries, indoor air pollution from using open fires for cooking and heating may be a serious problem. It has been estimated that in developing countries, about 1.9 million people die annually due to exposure to high concentrations of suspended particulate matter in the indoor air environment of rural areas, while the excess mortality due to suspended particulate matter and sulphur dioxide in the ambient air amounts to about 500 000 people annually. Although the indoor air database is weak due to the scarcity of monitoring results, these estimates indicate that a serious indoor air problem may exist in developing countries.

Air pollutants are usually classified into suspended particulate matter (dusts, fumes, mists, smokes), gaseous pollutants (gases and vapours) and odours. Current techniques used to measure the mass concentration of particles in air make use of size-specific sampling devices. Thus the mass of particles less than 10  $\mu\text{m}$  diameter may be determined ( $\text{PM}_{10}$ ) as an index of the mass concentration of particles that can penetrate into the human thorax. The mass concentration of particles of less than 2.5  $\mu\text{m}$  diameter ( $\text{PM}_{2.5}$ ) is a means of measuring the total gravimetric concentration of several chemically distinct classes of particles that are emitted into, or formed within, the ambient air as very small particles.

Fine and coarse particles generally have distinct sources and formation mechanisms, although there may be some overlap. Biological material such as bacteria, pollen and spores may also be found in the coarse mode. Fine and coarse particles typically exhibit different behaviour in the atmosphere and these differences must be taken into consideration when interpreting central-site monitored values, as well as the behaviour of particles after they penetrate homes and buildings, where people spend most of their time. Fine accumulation-mode particles typically have longer atmospheric lifetimes (days to weeks) than coarse particles, and tend to be more uniformly dispersed across an urban area or large geographic region. Larger particles generally deposit more rapidly than small particles; as a result, total coarse particle mass is less uniform in concentration across a region than that of fine particles.

This publication is focused on those gases and particulate matter that have been accepted as posing a threat to health. The relative health threat of different pollutant gases and particles varies with their concentrations over time and distance, implying that the effects of air pollutants on health may vary from country-to-country. Consequently, careful monitoring of the concentrations of polluting gases, as well as of the particle size distribution, concentration and composition, is needed before an acceptable estimate of the effects can be produced. The picture is further complicated because some pollutant combinations act in an additive manner and some perhaps synergistically.

WHO's air quality guidelines were first published as *Air Quality Guidelines for Europe* in 1987 (WHO 1987). Since 1993 the *Air Quality Guidelines for Europe* has been revised and updated, incorporating a review of the literature published since 1987 (WHO 1999a). Also, the following additional compounds were considered in the review procedure: 1,3 butadiene, environmental tobacco smoke (ETS), fluoride, man-made vitreous fibres and platinum. Parallel to the review of the air quality guidelines for Europe, the Environmental Health Criteria series of the International Programme on Chemical Safety has continued and the health risks of more than 120 chemical compound and mixtures were assessed between 1987 and 1998.

The WHO *Air Quality Guidelines for Europe* (WHO 1987) were based on evidence from the epidemiological and toxicological literature published in Europe and North America. They did not consider the effects of exposure to the different ambient air particle concentrations in developing countries, as well as the different conditions in these countries. However, these guidelines were used intensively throughout the world. In view of the different conditions in developing countries, the literal application of the WHO *Air Quality Guidelines for Europe* could be misleading. Factors such as high and low temperature, humidity, altitude, background concentrations and nutritional status could influence the health outcomes after the population has been exposed to air pollution. To make the WHO *Air Quality Guidelines for Europe* globally applicable, a task force group meeting was convened at WHO Headquarters from 2-5 December 1997. The outcome of that meeting is this publication of globally applicable air quality guidelines.

The objective of WHO's *Guidelines For Air Quality* is to help countries derive their own national air quality standards. The guidelines are technologically feasible and consider socio-economic and cultural constraints. They provide a basis for protecting public health from the adverse effects of air pollution and for eliminating, or reducing to a minimum, those air pollutants that are likely hazardous to human health. Consequently, the instruments of air quality management are also addressed in this publication.

## **2. *Factors affecting the concentrations of air pollutants***

Local concentrations of air pollutants depend upon the strength of their sources and the efficiency of their dispersion. Day to day variations in concentrations are more affected by meteorological conditions than by changes in source strengths. Wind is of key importance in dispersing air pollutants and for ground level sources pollutant concentrations are inversely related to wind speed. Turbulence is also important: a "rough" terrain, as produced for example by buildings, tends to lead to increased turbulence and better dispersion of pollutants.

## **3. *Exposure to air pollutants***

The total daily exposure of an individual to air pollution is the sum of the separate contacts to air pollution experienced by that individual as he/she passes through a series of environments (also called micro-environments) during the course of the day (e.g. at home, while commuting, in the streets, etc.). Exposures in each of these environments can be estimated as the product of the concentration of the pollutant in question and the time spent in the environment..

There are many factors that can account for the substantial differences between the concentrations of pollutants measured at central sites and those in the breathing zone of residents

of the community. Many of these factors can be modelled and such models have been used for estimating dose distributions associated with ambient air concentrations.

#### **4. Health significance of air pollution**

A new database of epidemiological studies emerged in the late 1980s and 1990s. This database of time-series studies was developed first in the United States and later in Europe and other areas. In essence the time series approach takes the day as the unit of analysis and relates the daily occurrence of events such as deaths or admissions to hospital to daily average concentrations of pollutants whilst taking careful account of confounding factors such as season, temperature and day of the week. Powerful statistical techniques have been applied and coefficients have been produced that relate the daily average concentrations of pollutants to their effects. Associations have been demonstrated between daily average concentrations of particles, ozone, sulphur dioxide, airborne acidity, nitrogen dioxide, and carbon monoxide. Although the associations for each of these pollutants were not significant in all studies, taking the body of evidence as a whole the consistency is striking. For particles and ozone it has been accepted by many that the studies provide no indication of any threshold of effect.

#### **5. Air pollutant concentrations and factors affecting susceptibility**

The concentrations of classical pollutants in ambient air of European countries and of the United States have been extensively discussed in the *Air Quality Guidelines for Europe* (WHO 1999a). In developing countries, by contrast, the concentrations of pollution levels in ambient air are higher by an order of magnitude, according to the main source of information on air pollution in developing countries, the Air Management Information System (AMIS).

Indoor air pollutants usually differ in type and concentration from outdoor air pollutants. Indoor pollutants include environmental tobacco smoke, biological particles, non-biological particles, volatile organic compounds, nitrogen oxides, lead, radon, carbon monoxide, asbestos, various synthetic chemicals and others. Degradation of indoor air quality has been associated with a range of health effects, from discomfort and irritation to chronic pathologies and cancers.

On a global scale, biomass fuels are used daily in about half the world's households as energy for cooking and/or heating. Biomass smoke contains significant amounts of several important pollutants: carbon monoxide, particulate matter, hydrocarbons and to a lesser extent, nitrogen oxides. However, biomass smoke also contains many organic compounds, including PAH that are thought to be toxic, carcinogenic, mutagenic or otherwise of concern. In China, coal burning is a major source of indoor air pollution and coal smoke contains all of these pollutants as well as additional ones, e.g. sulphur oxides and heavy metals such as lead.

An unknown, but significant, proportion of biomass fuel burning takes place in conditions where much of the air-borne effluent is released into poorly ventilated living areas. Therefore, some of the highest concentrations of particulate matter and other pollutants occur in rural, indoor environments in developing countries. Due to the high pollutant concentrations and the large populations involved, the total human exposure to many important air pollutants can be much higher in homes of the poor in developing countries than in the outdoor air of cities in the developed world.

Altitude, temperature and humidity vary significantly across the globe. At increased altitude the partial pressure of oxygen falls and inhalation increases in compensation. For particles, this

increased inhalation will lead to an increased intake of airborne particles. On the other hand, for gaseous pollutants no increase in effects over those experienced at sea level would be expected. Temperature has a very significant effect on health, whereas humidity is unlikely to have a significant effect on the toxicity of gaseous pollutants.

The age structure of populations differs markedly from country to country. Old people tend to show increased susceptibility to air pollution. Very young children may also be at increased risk. People with a poor standard of living suffer from nutritional deficiencies, infectious disease due to poor sanitation and overcrowding, and tend to be provided with a poor standard of medical care. Each of these factors may render individuals more susceptible to the effects of air pollution. Diseases which produce narrowing of the airways, a reduction in the area of the gas-exchange surface of the lung and an increased alteration of inhalation-perfusion ratios are likely to make the subject more susceptible to the effects of a range of air pollutants.

## **6.      *Role of guidelines and standards***

The purpose of the *Guidelines for Air Quality* is to provide a basis for protecting public health from adverse effects of air pollution and for eliminating, or reducing to a minimum, those contaminants that are known to be, or likely to be, hazardous to human health and well being. The *Guidelines* should provide background information for nations engaged in setting air quality standards, although their use is not restricted to this. These *Guidelines* are not intended as standards. In moving from guidelines to standards, prevailing exposure levels and environmental, social, economic and cultural conditions in a nation or region should be taken into account. In certain circumstances there may be valid reasons to pursue policies which will result in pollutant concentrations above or below the guideline values.

In the updated version of the Air Quality Guidelines for Europe, a similar approach to that in the 1987 air quality guidelines was used. However, total tolerable intakes were calculated for multimedia pollutants first, and then adequately partitioned among the different exposure routes. The term "protection factor" used in the 1987 guidelines was abandoned. Instead, uncertainty factors were used to account for the extrapolation from animal to man (alternatively, human equivalent concentrations were calculated), and to account for individual variability. Wherever information on inter- and intraspecies differences in pharmacokinetics was available, data-derived uncertainty factors were employed. Additional uncertainty factors were applied whenever necessary to account for the nature and severity of the observed effects and for the adequacy of the database. For most of the compounds considered, information on the dose/exposure response relationship was provided, to give policymakers clear guidelines on the possible impact of the pollutant at different exposure levels and to permit an informed decision making process to take place. For some compounds, e.g. platinum, a guideline value was considered unnecessary as exposure through ambient air levels was considerably below the lowest level at which effects were seen. For other compounds, for example particulate matter (PM<sub>10</sub>), no threshold of effect(s) could be found and therefore no guideline value could be derived. Instead, exposure-effect information highlighting the public health impact of different pollutant levels was provided.

In the updating process for carcinogens, a more flexible approach than in the 1987 air quality guidelines was applied. As a default approach, low-dose risk extrapolation was conducted for the IARC groups 1 (proven human carcinogen) and 2A (probable human carcinogen, limited evidence), and an uncertainty factor was applied for agents in IARC groups 2B (probable human carcinogen, inadequate evidence) and 3 (unclassified chemicals). However, the mechanism of action of the

carcinogen was the determining factor for the method of assessment. Hence, it was decided that compounds classified under 1 or 2A could be assessed using uncertainty factors, if evidence for a non-threshold mechanism of carcinogenicity existed. By way of contrast, compounds classified under 2B could be assessed by low-dose extrapolation methods, if a non-threshold mechanism of carcinogenicity in animals was proven. Flexibility was also given in the choice of the extrapolation model, depending on the available data (including data for PBPK modelling). The linearized multistage model was used as a default approach. Besides providing unit risk estimates in cases where low dose risk extrapolation was conducted, levels associated with excess cancer risk of 1 : 10000, 1 : 100 000 and 1 : 1000 000 were calculated.

## **7. *Exposure-response relationships***

These guidelines place some emphasis on epidemiological data. Epidemiological studies are sometimes preferable to controlled exposure studies in that they provide information on responses in populations and on the effects of real exposures to pollutants and pollutant mixtures. However, the results of epidemiological studies are less easy to use than the results of controlled exposure studies in defining guidelines.

For both particles and ozone an assumption of linearity was made when defining the exposure-response relationships included in the revised guidelines. Extrapolation beyond the available data is dangerous; however, as there is evidence to suggest that the exposure-response relationship may become less steep as ambient levels of particles rise. For ozone, the relationship at low concentrations may be concave upwards. These are important points to be considered if the guidelines are to be used in countries with levels of pollution different from the range covered by the guidelines.

## **8. *Moving from guidelines to standards***

An air quality standard is a description of a level of air quality, adopted by a regulatory authority as enforceable. At its simplest, an air quality standard should be defined in terms of one or more concentrations and averaging times. Further information on the form of exposure (e.g. outdoor), on monitoring to assess compliance with the standard, and on methods of data analysis and Quality Assurance and Quality Control requirements should be added. Other factors to be considered in setting an air quality standard include the nature of the pollution effects and whether they represent adverse health effects; and whether special populations are at risk.

The development of air quality standards is only a part of an adequate air quality management strategy. Legislation, identification of authorities responsible for enforcement of emission standards and penalties for exceeding standards are also necessary. Emission standards may play an important role in the management strategy especially if exceeding air quality standards is used as a trigger for abatement measures. These may be needed at both the national and the local level. Air quality standards are also important in informing the public about air quality. Used in this way they are a double edged weapon as the public commonly assumes that once a standard is exceeded adverse effects on health will occur. This may not be the case.

The transfer of the dose-response relationships to other parts of the world, especially for particulate matter, should be conducted with caution for several reasons. These include:

1. The chemical composition of the particles.
2. The concentration range.
3. The responsiveness of the population.

4. The limitations of the established relationships.

## **9. *Cost-benefit analysis and other factors***

Cost-benefit analysis is one way of formally weighing the costs of reducing air pollution against the benefits produced. The concept is that emissions are reduced until the marginal costs and benefits are equal. While the cost of abatement measures may be relatively easy to quantify, this may not be the case when non-technical measures are employed. In any case, it is likely to be more difficult to assign monetary values to the benefits obtained. Some aspects of reduced morbidity, such as the reduced use of hospital facilities and drugs are comparatively easy to cost; others, such as reductions in premature deaths and symptoms, are not. Applying monetary values based on a "willingness to pay" basis has been suggested, and has been accepted as appropriate by many health economists. This approach has been seen as preferable to one based only on such indices as loss of production, earnings or hospital expenses.

Factors other than monetary factors also need to be considered when considering the setting of national air quality standards. These include the technical capacity of a country to achieve and maintain an air quality within the desired standards, the social implications of adopting certain standards to ensure equity of costs and benefits among the population, and environmental costs and benefits.

## **10. *Health-based guidelines***

For the purpose of presenting the health-based air quality guidelines, the key air pollutants, also termed "classical" air pollutants - SO<sub>2</sub>, NO<sub>2</sub>, CO, O<sub>3</sub>, SPM, and lead are briefly described with respect to health risk evaluations and recommended guideline values. Particular emphasis is given to suspended particulate matter <10 µm diameter (PM<sub>10</sub>) and < 2.5 µm diameter (PM<sub>2.5</sub>). The guidelines are presented in Chapter 3 in tables 3.1 to 3.5 and in figures 3.1 to 3.9. The information available for a number of other air pollutants (including carcinogens and non-carcinogens) is also summarized and presented in synoptic tables.

## **11. *Classical air pollutants: applicability of WHO Air Quality Guidelines for Europe on a world wide scale***

In the derivation of the WHO *Air Quality Guidelines for Europe*, assumptions were made for some compounds, which may not be applicable in some parts of the world. For example, the importance of different routes of exposure for some pollutants may vary from country to country. It should be understood that if such factors were to be taken into account then different guidelines could be derived. For a number of pollutants a Unit Risk (UR) assessment has been provided. These assessments are also dependent upon considerations of the comparative importance of different routes of exposure.

It is important that regulatory authorities should evaluate whether local circumstances give cause to doubt the validity of the guideline set out in the WHO *Guidelines for Air Quality* as a basis for setting local guidelines or standards.



## **12. Indoor air quality**

Indoor spaces are important microenvironments when assessing risks from air pollution. For many air pollutants most of the daily exposure by inhalation occurs indoors because of the amount of time spent indoors or because of the pollutant concentration levels encountered. The air quality inside buildings is affected by many factors. In an effort to conserve energy, modern building design has favoured tighter structures with lower rates of ventilation. By contrast, in some areas of the world, natural ventilation only is used; in others, mechanical ventilation is common. In modern buildings most of the pollution problems arise from low ventilation rates and the presence of products and materials that emit a large variety of compounds, whereas the inhabitants of many less developed countries face problems related to pollutants generated by human activities, in particular by combustion processes.

If only the health effects of air pollution are being considered, it does not matter if a pollutant is inhaled by breathing outdoor or indoor air. However, there are important differences in the composition of pollutant mixtures in outdoor and indoor air. For example, in outdoor air there are traffic-generated emissions, whereas indoor air pollution is generated from tobacco smoke or from cooking with biomass-fuelled stoves. Not all of these compositions have been taken into account in developing the *Guidelines for Air Quality*, and they may not be applicable under all circumstances, so care should be taken to avoid misinterpretation.

## **13. Ambient air quality monitoring and assessment**

The three main air quality assessment tools are: i) ambient monitoring; ii) models and iii) emission inventories/measurement.

The ultimate purpose of monitoring is not merely to collect data, but to provide the necessary information required by scientists, policy makers and planners to enable them to make informed decisions on managing and improving the environment. Monitoring fulfils a central role in this process, providing the necessary scientific basis for policy and strategy development, objective setting, compliance measurement against targets and enforcement action. However, the limitations of monitoring should be recognised. No monitoring programme, however well funded and designed, can hope to comprehensively quantify patterns of air pollution in both space and time. In many circumstances, measurements alone may be insufficient or impractical for the purpose of fully defining population exposure in a city or country. Monitoring therefore often needs to be used in conjunction with other objective assessment techniques, including modelling, emission measurement and inventories, interpolation and mapping. At best, monitoring provides an incomplete, but useful, picture of current environmental quality.

Reliance on modelling alone also is not recommended. Although models can provide a powerful tool for interpolation, prediction, and optimisation of control strategies, they are effectively useless unless properly validated by real-world monitoring data. It is important, also, that the models utilised are appropriate to local conditions, sources and topography, as well as being selected for compatibility with available emission and meteorological datasets. Many models depend on the availability of reliable emission data.

A complete emissions inventory for a city or country may need to include emissions from point, area and mobile sources. In some circumstances, assessment of pollutants transported into the area under study may also need to be considered. Inventories will, for the most part, be

estimated using emission factors appropriate to the various source sectors (verified by measurement), and used in conjunction with surrogate statistics such as population density, fuel use, vehicle kilometres or industrial throughput. Emission measurements will usually only be available for large industrial point sources or from representative vehicle types under standardised driving conditions.

All three assessment tools are interdependent in scope and application. Accordingly, monitoring, modelling and emission assessments should be regarded as complementary components in any integrated approach to exposure assessment or in determining compliance with air quality criteria.

## **14. Ambient air quality management**

Some basic principles guide international and national policies for the management of all forms of air pollution. An important global initiative occurred in 1983 when the UN General Assembly established the World Commission on Environment and Development, headed by Gro Harlem Brundtland. The report produced by the Commission was entitled *Our Common Future* and it was presented by the UN General Assembly in 1987 and endorsed by it. It has been influential in bringing environmental issues into the global arena, and in expressing some concepts that have been influential in air quality management.

The Brundtland Commission suggested that to meet the legitimate aspirations of the world's population without destroying the environment, sustainable development would be required. It defined **sustainable development** as: *development that meets the needs of the present without compromising the ability of future generations to meet their own needs*. This concept has been embraced as an apparent means of integrating environmental policy and economic development.

Following from the Brundtland Commission, the UN Conference on the Environment and Development was held in Rio in 1992. The aim was to ensure that practical foundations for sustainable development were put into place. The Agenda 21 document and the Rio declaration were the most obvious results of this conference. Agenda 21 is a document covering sustainable development which is not binding on countries, but national implementation is reviewed by the Sustainable Development Commission and the UN General Assembly. Agenda 21 supports a number of environmental management principles on which some government policies including air quality management are based. These include:

**precautionary principle** - where there is a clear possibility of damaging environmental consequences, action should be taken to protect the environment without awaiting the full scientific proof that the environment will be damaged by the proposal.

**polluter pays principle**- the full costs associated with pollution (including monitoring, management, clean-up and supervision) should be met by the organisation responsible for the source of the pollution.

In addition, many countries have adopted the principle of **pollution prevention**, which aims to reduce pollution at sources.

The responsibility of national governments for international reporting on the environment of their country has enabled greater exchange of air quality information around the world.

The foundation for air quality management is the government policy framework. Without a suitable policy framework and adequate legislation it is difficult to maintain an active or successful air quality management programme. A policy framework refers to transport, energy, planning, development, and policy in other areas, as well as environmental policy. Air quality objectives are more readily achieved if these interconnected government policies are compatible, and if mechanisms exist for co-ordinating responses to issues, which cross different areas of government policy. Measures to achieve some integration of air quality policy with health, energy, transport and other policy areas have been adopted in many developed countries.

The goal of air quality management is commonly stated to be to maintain a quality of air that protects human health and welfare. This goal recognises that air quality must be maintained at levels, which protect human health, but it also must provide protection of animals, plants (crops, forests and natural vegetation), ecosystems, materials and aesthetics, such as natural levels of visibility. To achieve an air quality goal requires the development of policies and strategies.

## **15. *Management of indoor air quality***

Most human beings spend most of their time in indoor environments, where they can be exposed to poor air quality. Pollution and degradation of indoor air cause illness, increased mortality, loss of productivity and have major economic and social implications. Health effects can include increased rates of cancer, lung disease, allergy and asthma as well as fatal conditions such as carbon monoxide poisoning and legionnaires' disease, as discussed in Section 4.1. The medical and social cost associated with these illnesses, and the related reduction in human productivity, result in staggering economic losses.

Indoor air quality problems affect all types of buildings including homes, schools, offices, health care facilities and other public and commercial buildings. Indoor air problems can be reduced by better urban planning, design and operation, as well as maintenance of buildings, materials and equipment in buildings.

This document considers the management of indoor air quality in developed countries, and in some situations in developing countries, and then focuses on the important and widespread problem of how to manage indoor air quality associated with biomass fuel combustion in developing countries.

## **16. *Priority setting in air quality management***

It is important to give guidance to countries on how to set priorities in rational air quality management. Actual priorities will differ for each country; therefore, each country sets priorities in air quality management according to its policy objectives, needs and capabilities. Priority setting in air quality management refers to prioritising health risks to be avoided, with corresponding prioritisation of air pollutant compounds, and concentrating on the most important sources of the pollutants. Conceptually, prioritising health risks is straightforward. High priority of health risks will be given to those compounds for which “high” toxicity and “high” exposure of the population are entailed. Conversely, low priority health risks involve agents of “low” toxicity and “low” exposure. “Medium” priority risks include compounds in which either toxicity or exposure is “low” while the other is “high”.

A framework for a political, regulatory and administrative approach is required to guarantee a

consistent and transparent derivation of air quality standards and to ensure a basis for making decisions on risk-reducing measures and abatement strategies. In such a framework the following considerations need to be included:

- The legal aspects.
- The potential of air pollution to cause adverse effects on health, taking into account the populations at risk.
- The exposure-response relationships of pollutants and pollutant mixtures and the actual exposure responsible for related health and/or environmental risks.
- The acceptability of risk.
- The cost-benefit analysis.
- The stakeholder contribution in standard setting.

## **17. *Enforcement of air quality standards: clean air implementation plans***

The enforcement of air quality standards aims to evaluate the need for control action on emission sources to attain compliance with the standards. The instruments used to achieve this goal are the Clean Air Implementation Plans (CAIPs). The outline of such a plan should be defined in regulatory policies and strategies. Clean air implementation plans were developed in several developed countries during the 1970s and 1980s. Air pollution was characterized by a multitude of sources of many different types of air pollutants. Consequently it was extremely difficult to assess the public health risks associated with a single source, or even a group of sources. As a consequence, on the basis of the polluters pay principle (Chapter 6), sophisticated tools were developed which assessed the sources, air pollutant concentrations, health and environmental effects and control measures, and which made a causal link between emission, air pollution and the necessary control measures. A typical clean air implementation plan (CAIP) includes:

A description of the area.

An emissions inventory.

An air pollutant concentrations inventory - monitored and simulated.

A comparison with emissions and air quality standards or guidelines.

An inventory of the effects on public health and the environment.

A causal analysis of the effects and their attribution to individual sources.

Control measures and their costs.

Transportation and land-use planning.

Enforcement procedures.

Resource commitment.

Projections for the future.

In developing countries, the air pollution situation is often characterized by a multitude of sources of few types, or sometimes few sources. Using the experience obtained in developed countries, the control action to be taken is very often obvious. As a consequence, in cases where little useful monitoring data are available, less monitoring could be sufficient, and dispersion models could help to simulate spatial distributions of pollutant concentrations. Much simplified CAIPs would have to be developed for cities of developing countries or countries in transition. At present, the main sources of emissions in many cities of the developing world are old vehicles and some industrial sources such as power plants, brick kilns, cement factories and a few others. Their relative contribution to air pollution could be determined by use of rapid emission inventories. The emission factors used in such inventories are published and a PC programme

is available, which enables an estimation of emissions and ambient air concentrations, and evaluates the impact of possible control measures. Projections for the future can also be evaluated by the programme.

# 1. Introduction

Air pollution is a major environmental health problem affecting developed and developing countries around the world. Increasing amounts of potentially harmful gases and particles are being emitted into the atmosphere on a global scale, resulting in damage to human health and the environment. It is damaging the resources needed for the long-term sustainable development of the planet.

The sources of air pollution resulting from human activities are of three broad types.

Stationary sources. These can be subdivided into:

Rural area sources such as agricultural production, mining and quarrying.

Industrial point and area sources such as manufacturing of chemicals, non-metallic mineral products, basic metal industries, power generation.

Community sources, e.g. heating of homes and buildings, municipal waste and sewage sludge incinerators, fireplaces, cooking facilities, laundry services and cleaning plants.

Mobile sources. These comprise of any form of combustion-engine vehicles, e.g. light duty gasoline-powered cars, light and heavy-duty diesel-powered vehicles, motorcycles, aircraft, and including line sources such as fugitive dusts from vehicle traffic.

Indoor sources. These include: tobacco smoking, biological sources (such as pollen, mites, moulds, insects, micro-organisms, pet allergens etc.), combustion emissions, emissions from indoor materials or substances such as volatile organic compounds, lead, radon, asbestos, various synthetic chemicals and others.

In addition, there are also natural sources of pollution, e.g. eroded areas, volcanoes, certain plants that release great amounts of pollen, sources of bacteria, spores and viruses, etc. These natural physical and biological sources of pollution are not discussed in this publication.

In recent years it has become clear that indoor air pollution from the use of open fires for cooking and heating may be a serious problem in developing countries. It has been estimated that about 2 800 000 people die annually from exposure to high concentrations of suspended particulate matter in the indoor air environment; and the excess mortality due to suspended particulate matter and sulphur dioxide in the ambient air amounts to about 500 000 people annually (Murray and Lopez 1996; Schwela 1996a; WHO 1997a). Although the indoor air database is weak due to the scarcity of monitoring results, these estimates indicate that a serious indoor air problem may exist in developing countries.

Air pollutants are usually classified into suspended particulate matter (dusts, fumes, mists, smokes), gaseous pollutants (gases and vapours) and odours.

*Suspended particulate matter (SPM)* Particulate matter suspended in air includes total suspended particles (TSP), PM<sub>10</sub>, (SPM with median aerodynamic diameter less than 10 µm), PM<sub>2.5</sub> (SPM with median aerodynamic diameter less than 2.5 µm), fine and ultrafine particles, diesel exhaust, coal fly-ash, mineral dusts (e.g. coal, asbestos, limestone, cement), metal dusts and fumes (e.g. zinc, copper, iron, lead), acid mists (e.g. sulphuric acid), fluoride particles, paint pigments, pesticide mists, carbon black, oil smoke and many others. Suspended particulate pollutants provoke respiratory diseases, and can cause cancers, corrosion, destruction to plant life, etc. They can also constitute a nuisance (e.g. accumulation of dirt), interfere with sunlight (e.g. light

scattering from smog and haze) and also act as catalytic surfaces for reaction of adsorbed chemicals.

*Gaseous pollutants:* Gaseous pollutants include sulphur compounds (e.g. sulphur dioxide (SO<sub>2</sub>) and sulphur trioxide (SO<sub>3</sub>)), carbon monoxide (CO), nitrogen compounds [e.g. nitric oxide (NO), nitrogen dioxide (NO<sub>2</sub>), ammonia (NH<sub>3</sub>)], organic compounds [e.g. hydrocarbons (HC), volatile organic compounds (VOC), polycyclic aromatic hydrocarbons (PAH) and halogen derivatives, aldehydes, etc.], halogen compounds (HF and HCl) and odourous substances.

Secondary pollutants may be formed by thermal, chemical or photochemical reactions. For example, by thermal action SO<sub>2</sub> can be oxidised to SO<sub>3</sub> which, dissolved in water, gives rise to the formation of sulphuric acid mist (catalysed by manganese and iron oxides). Photochemical reactions between NO<sub>x</sub> and reactive hydrocarbons can produce ozone (O<sub>3</sub>), formaldehyde (HCHO) and peroxyacetyl nitrate (PAN); reactions between HCl and HCHO can form bis-chloromethyl ether.

*Odours:* While some odours are known to be caused by specific chemical agents such as hydrogen sulphide (H<sub>2</sub>S), carbon disulphide (CS<sub>2</sub>) and mercaptans (R-SH, R<sub>1</sub> S R<sub>2</sub>), others are difficult to define chemically.

An air pollutant concentrations inventory summarizes the results of monitoring ambient air pollutants. The data are expressed in terms of annual means, percentiles and trends of the parameters measured. In most developed countries compounds measured for such an inventory include SO<sub>2</sub>, nitrogen oxides (NO<sub>x</sub>), SPM, CO, O<sub>3</sub>, heavy metals, PAH, and VOC. In developing countries the “classical” compounds SO<sub>2</sub>, NO<sub>x</sub>, SPM, CO, O<sub>3</sub> and lead are commonly monitored.

Trends in air pollution exposure are usually shown as annual arithmetic or geometric means and as statistical measures of short-term exposure such as high percentiles, or maximal or second highest values of a sample. The general picture for the “classical” compounds considered in this publication is that SO<sub>2</sub> and SPM concentrations are decreasing in developed countries while NO<sub>x</sub> and O<sub>3</sub> concentrations are either constant or increasing (UNEP/WHO 1992). In many countries in transition and in developing countries, SO<sub>2</sub> and SPM concentrations are increasing as a consequence of increasing combustion, as are NO<sub>x</sub> and O<sub>3</sub> due to increasing traffic exhaust and emissions of VOC by industrial sources as precursors of O<sub>3</sub>.

WHO's air quality guidelines were first published as *Air Quality Guidelines for Europe* in 1987 (WHO 1987). Since 1993 the *Air Quality Guidelines for Europe* has been revised and updated after a review of the literature published since 1987 (WHO 1992a; WHO 1994a; WHO 1995a; WHO 1995b; WHO 1995c; WHO 1996a; WHO 1998a; WHO 1999a). Also, the following additional compounds were considered in the review procedure: 1,3 butadiene, environmental tobacco smoke (ETS), fluoride, man-made-vitreous fibres (MMVF) and platinum. Parallel to the review of the air quality guidelines for Europe, the Environmental Health Criteria series of the International Programme on Chemical Safety has continued and the health risks of more than 120 chemical compound and mixtures were assessed between 1987 and 1998.

Trends of ambient air pollution were assessed in the WHO/UNEP Global Environmental Monitoring System/Air Pollution (GEMS/Air) which operated from 1973 to 1995 (UNEP/WHO 1993). The GEMS/Air programme has been replaced by a new programme under the umbrella of WHO's Healthy Cities Programme: Air Management Information System (AMIS). AMIS is intended as an information turntable which collects information on all issues of air quality

management from its participants and distributes this information among them via the information centre at WHO. Several databases have already been developed (WHO 1997b; WHO 1998b). The AMIS core database of ambient air pollutant concentrations contains summary data, including annual means, percentiles and the number of days on which WHO Air Quality Guidelines are exceeded, from more than 100 cities in the world. A database on air quality guidelines and air quality standards contains data from about 60 countries. A database on air pollution management capabilities contains data from 70 cities. A database of the AMIS focal points helps AMIS participants in different countries to communicate with each other. A database on indoor air pollutant concentrations and a noise database have been developed and will be available in the near future.

The WHO Air Quality Guidelines for Europe (WHO 1987) were based on evidence from the epidemiological and toxicological literature published in Europe and North America. They did not consider exposure to ambient air concentrations in developing countries and the different conditions in these countries. However, these guidelines were used intensively throughout the world. In view of the different conditions in developing countries, the literal application of the WHO Air Quality Guidelines for Europe could be misleading. Factors such as high and low temperature, humidity, altitude, background concentrations and nutritional status could influence the health outcome after exposure of the population to air pollution. To make the WHO *Air Quality Guidelines for Europe* globally applicable, a task force group meeting was convened at WHO Headquarters from 2-5 December 1997. The outcome of this meeting is this publication of the globally applicable *Guidelines for Air Quality*.

The objective of WHO's *Guidelines for Air Quality* is to help countries derive their own national air quality standards, to help protect human health from air pollution. The guidelines are technologically feasible and consider socio-economic and cultural constraints. They provide a basis for protecting public health from the adverse effects of air pollution, and for eliminating or reducing to minimum, air pollutants likely to be hazardous to human health. Consequently, the instruments of air quality management are also addressed in this publication.

## **2. Air quality and health**

### **2.1 Basic facts**

Pure air comprises oxygen (21%) and nitrogen (78%) and a number of rarer gases, of which argon is the most plentiful. Carbon dioxide (CO<sub>2</sub>) is present at a lower percentage concentration (0.03%) than argon (0.93%). Water vapour, up to 4% by volume, is also present. Oxygen is produced by plants as a by-product of photosynthesis and the earth's atmosphere is now described as oxidant, or oxidising, in comparison with the hydrogen-rich reducing atmosphere that was present before life began. The increase in oxygen has led to the development of anti-oxidant defenses in many living organisms.

The atmosphere contains a number of gases which, at higher than usual concentrations, are poisonous to humans and animals and damaging to plants. These include O<sub>3</sub>, SO<sub>2</sub>, NO<sub>2</sub>, CO and a wide range of VOC. Some of the latter are carcinogenic, for example benzene and butadiene. All these potentially toxic gases are referred to as air pollutants.

As well as gases, the atmosphere contains a wide variety of particulate matter, both solid and liquid,



ranging in size from a few nanometres to about 0.5 mm. Small particles ( $<2.5\ \mu\text{m}$ ) persist in the air for long periods, forming a more or less stable aerosol. Larger particles are more quickly lost as their mass leads to rapid sedimentation.

This publication is focused on gases and particulate materials that have been accepted as posing a threat to health. The relative importance of the different pollutant gases and particles varies with their concentrations over both time and distance. This implies that the extent of the effects of air pollutants on health may vary from country-to-country. Careful monitoring of the concentrations of polluting gases and the particle size distribution, concentration, and composition is thus needed before an acceptable estimate of effects can be produced. The picture is further complicated as some combinations of pollutants act in an additive manner and some perhaps synergistically.

### **2.1.1 Physico-chemical aspects of air pollution and units used to describe concentrations of air pollutants**

A consistent system of units is necessary if concentrations of air pollutants in different countries are to be compared. For both gases and particles WHO has adopted a mass per unit volume system, with concentrations generally expressed as  $\mu\text{g}/\text{m}^3$ . The volume of a mass of air varies with ambient temperature and atmospheric pressure and thus these conditions should be specified. In considering pollutants on a global scale this is clearly important.

The alternative system, the volume mixing ratio, is applicable only to gases. In this system the concentration of gas is expressed as parts per billion, for example, and assuming ideal gas behaviour, does not depend upon the conditions of sampling because these will affect the air containing the pollutant and the pollutant itself to the same extent. A gas present at one part per million thus occupies  $1\ \text{cm}^3$  per  $\text{m}^3$  of polluted air; is present as 1 molecule per  $1 \times 10^6$  molecules and exerts a partial pressure of  $1 \times 10^{-6}$  atmospheres.

The two systems are interconvertible as under ideal conditions, 1 Mole of gas occupies 22.4 litres at 273K and 13mb pressure, dry air Standard Temperature and Pressure Dry (STPD). The interconversion formula is:

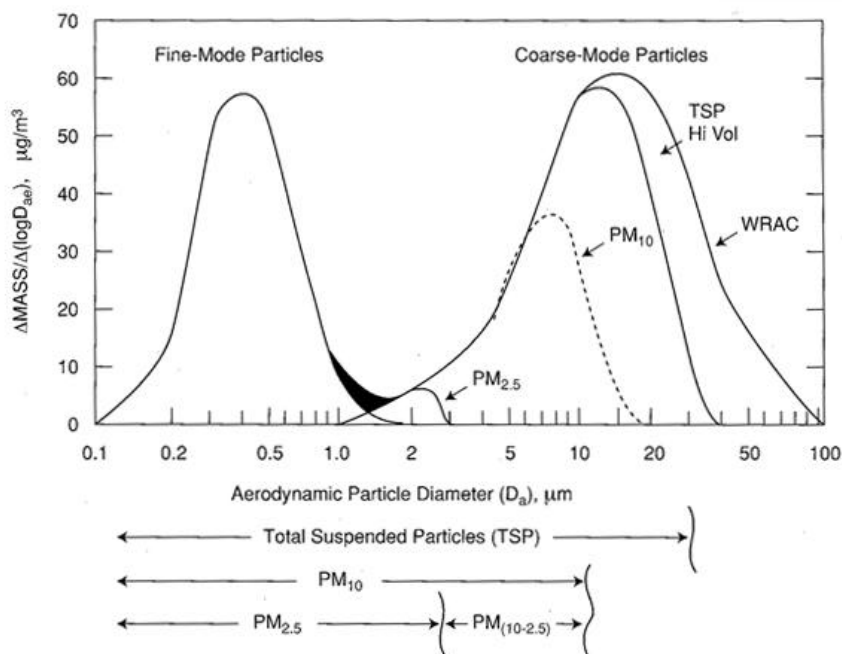
$$\text{mg}/\text{m}^3 = \text{ppm} \times (\text{molecular weight}/\text{molar volume})$$

$$\text{molar volume} = 22.4 \times T \times 1013/273 \times P$$

T = absolute temperature (K)

P = atmospheric pressure (mb)

For particles, the mass per unit volume approach is used. Particle deposition in the respiratory tract depends upon the dimensions of the particles (WHO 1979a). Thus, in describing the particle loading of the air, information on the distribution of particle size should be given in addition to the mass concentration. A representative size distribution of urban particulate matter is provided in figure 2.1. It may also be important to specify the number of particles present in each of several specific size ranges per unit volume of air.



Source: USEPA, 1996

**Figure 2. 1. Representative example of a mass distribution of ambient PM as function of aerodynamic particle diameter. A wide-ranging aerosol collector (WRAC) provides an estimate of the full coarse mode distribution. Inlet restrictions of the high volume sampler for TSP, the PM<sub>10</sub> sampler, and the PM<sub>2.5</sub> sampler reduce the total mass reaching the sampling filter.**

The distribution of sizes of particles arising from each source of aerosols has been shown to follow a log-normal distribution: thus the geometric mean (or median) diameter and the geometric standard deviation are often quoted and specify the distribution. In defining the median diameter of the particles of an aerosol it should be specified whether this value reflects the mid-point of the distribution of the mass or number of particles present. Thus the Mass or Count Median Diameter (MMD or CMD) should be specified. An additional refinement involves adjusting for the aerodynamic properties of the particles and so the Mass or Count Aerodynamic Median Diameters should be quoted. In naturally occurring aerosols the geometric standard deviation tends to vary from about 2 to 4  $\mu\text{m}$  with 84% of the distribution being of size less than that specified by the median diameter multiplied by the geometric standard deviation. It is incorrect to refer to the median diameter of a single particle: the term refers to the distribution of sizes present in an aerosol cloud.

Current techniques used to measure the mass concentration of particles in air make use of size-specific sampling devices and thus the mass of particles of less than 10  $\mu\text{m}$  diameter may be determined (PM<sub>10</sub>) as an index of the mass concentration of particles that can penetrate into the human thorax. Sampling devices allow a fairly definite separation of particles of greater or less than the specified size. To be precise, the percentage of particle mass in the specified size range accepted by the sampling system, should be specified (e.g. 10  $\mu\text{m}$  in the above example). A PM<sub>10</sub> sampling head accepts 50% of particles of aerodynamic diameter exactly 10  $\mu\text{m}$ , the acceptance fraction rising rapidly for particles of smaller diameter and declining rapidly for particles of greater diameter.

The mass concentration of particles of less than 2.5  $\mu\text{m}$  diameter (PM<sub>2.5</sub>) is a means of measuring the total gravimetric concentration of several chemically distinctive classes of particles that are emitted into or formed within the ambient air as very small particles. In the former category

(emitted) are carbonaceous particles in wood smoke and diesel engine exhaust. In the latter category (formed) are carbonaceous particles formed during the photochemical reaction sequence that also leads to O<sub>3</sub> formation, as well as sulphate and nitrate particles resulting from the oxidation of SO<sub>2</sub> and nitrogen oxide released during fuel combustion and their reaction products.

The coarse particle fraction, i.e., those particles with aerodynamic diameters larger than about 2.5 µm, are largely composed of soil and mineral ash that are mechanically dispersed into the air.

Both the fine and coarse fractions are complex mixtures in a chemical sense. To the extent that they are in equilibrium in the ambient air, it is a dynamic equilibrium in which they enter the air at about the same rate as they are removed. In dry weather, the concentrations of coarse particles are balanced between dispersion into the air, mixing with air masses, and gravitational fallout, while the concentrations of fine particles are determined by rates of formation, rates of chemical transformation, and meteorological factors. Concentrations of both fine and coarse particulate matter are effectively depleted through in-cloud and below-cloud scavenging by precipitation. Further elaboration of these distinctions is provided in Table 2.1.

**Table 2.1. Comparisons of ambient fine and coarse mode particles**

	<b>Fine Mode</b>	<b>Coarse Mode</b>
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts.
Composed of:	Sulphate, $\text{SO}_4^{2-}$ ; nitrate $\text{NO}_3^-$ ; ammonium, $\text{NH}_4^+$ ; hydrogen ion, $\text{H}^+$ ; elemental carbon; organic compounds (e.g., PAHs); metals (e.g. Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); particle-bound water.	Resuspended dusts (e.g., soil dusts, street dust); coal and oil fly ash, metal oxides of crustal elements (Si, Al, Ti, Fe); $\text{CaCO}_3$ , NaCl, sea salt; pollen, mould spores; plant/animal fragments; tire wear debris
Solubility	Largely soluble, hygroscopic and deliquescent	Largely insoluble and non-hygroscopic
Sources	Combustion of coal, oil, gasoline, diesel, wood; atmospheric transformation products of $\text{NO}_x$ , $\text{SO}_2$ and organic compounds including biogenic species (e.g. terpenes) high temperature processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads; suspension from disturbed soil (e.g. farming, mining, unpaved roads); biological sources; construction and demolition; coal and oil combustion; ocean spray
Lifetimes	Days to weeks	Minutes to hours
Travel Distance	100s to 1000s of kilometres	< 1 to 10s of kilometres

Source: USEPA (1995a, b)

As indicated in Table 2.1, fine and coarse particles generally have distinct sources and formation mechanisms, although there may be some overlap. Primary fine particles are formed from condensation of high temperature vapours during combustion. Secondary fine particles are usually formed from gases in three ways:

Nucleation (i.e., gas molecules coming together to form a new particle).

Condensation of gases onto existing particles.

By reaction of absorbed gases in liquid droplets.

Particles formed from nucleation also coagulate to form relatively larger aggregate particles or droplets with diameters between 0.1 - 1.0  $\mu\text{m}$ , and such particles normally do not grow into the coarse mode. Particles form as a result of chemical reaction of gases in the atmosphere that lead

to products that either have a low enough vapour pressure to form a particle, or react further to form a low vapour pressure substance. Some examples include:

The conversion of  $\text{SO}_2$  to sulphuric acid droplets ( $\text{H}_2\text{SO}_4$ ).

Reactions of  $\text{H}_2\text{SO}_4$  with  $\text{NH}_3$  to form ammonium bisulphate ( $\text{NH}_4\text{HSO}_4$ ) and ammonium sulphate ( $(\text{NH}_4)_2\text{SO}_4$ ).

The conversion of  $\text{NO}_2$  to nitric acid vapour ( $\text{HNO}_3$ ), which reacts further with  $\text{NH}_3$  to form particulate ammonium nitrate ( $\text{NH}_4\text{NO}_3$ ).

Although some directly emitted particles are found in the fine fraction, secondary particles formed from gases dominate the fine fraction mass. By contrast, most of the coarse fraction particles are formed directly as particles, and result from mechanical disruption such as crushing, grinding, evaporation of sprays, or suspension of dust from construction and agricultural operations. Basically, most coarse particles are formed by breaking up bigger masses into smaller ones. Energy considerations normally limit coarse particle sizes to greater than  $1.0\text{ }\mu\text{m}$  in diameter. Some combustion-generated mineral particles, such as fly ash, are also found in the coarse fraction. Biological material such as bacteria, pollen, and spores may also be found in the coarse mode.

In general, fine and coarse particles exhibit different degrees of solubility and acidity. With the exception of carbon and some organic compounds, fine particles are largely soluble in water and hygroscopic (i.e., fine particles readily take up and retain water). Except under fog conditions, the fine particle mode also contains almost all of the strong acid. By contrast, coarse mineral particles are mostly insoluble, non-hygroscopic, and generally basic.

Fine and coarse particles typically exhibit different behaviour in the atmosphere. These differences affect several exposure considerations including the representativity of central-site monitored values and the behaviour of particles that were formed outdoors after they penetrate into homes and buildings where people spend most of their time.

Fine accumulation mode particles typically have longer atmospheric life times (i.e. days to weeks) than coarse particles, and tend to be more uniformly dispersed across an urban area or large geographic region. Atmospheric transformations can take place locally, during atmospheric stagnation, or during transport over long distances. For example, the formation of sulphates from  $\text{SO}_2$  emitted by power plants with tall stacks can occur over distances exceeding 300 kilometres and 12 hours of transport time; therefore, the resulting particles are well mixed in the air shed. Once formed, the very low dry deposition velocities of fine particles contribute to their persistence and uniformity throughout an air mass.

Larger particles generally deposit more rapidly than small particles; as a result, total coarse particle mass is less uniform in concentration across a region than that of fine particles. The larger coarse particles ( $> 10\text{ }\mu\text{m}$ ) tend to fall out of the air rapidly and have atmospheric lifetimes of only minutes to hours depending on their size, wind velocity, and other factors. Their spatial impact is typically limited by a tendency to fall out in the nearby downwind area. The atmospheric behaviour of the small particles within the “coarse fraction” ( $\text{PM}_{10-2.5}$ ) is intermediate between that of the larger coarse particles and fine particles. Thus, some of the smaller coarse fraction particles may have lifetimes on the order of days and travel distances of up to 100 km or more. In some locations, source distribution and meteorology affects the relative homogeneity of fine and coarse particles, and in some cases, the greater measurement error in estimating coarse fraction mass precludes clear conclusions about relative homogeneity.

The composition of airborne particles is seldom routinely determined though this can vary significantly from site to site. This is important in interpreting the results of epidemiological studies of the effects of particles on health. Extrapolation from data collected in one country to conditions in another may be unwise unless some comparability of particle composition has been established.

### **2.1.2 Sources of air pollutants**

The sources of air pollutants may be divided into anthropogenic and natural. However, as human activity disturbs natural systems, the distinction may become blurred. Natural sources include dust storms, volcanic action, forest fires and the formation of radioactive particles from gases such as radon. Incursions from the stratosphere increase ground level (tropospheric) concentrations of  $O_3$ . For some pollutants, e.g.  $SO_2$ , natural sources exceed anthropogenic sources on a global scale. However, when considering the effects of air pollutants on health, especially in urban areas where population densities are high, anthropogenic sources are very important and are those to which attention is usually directed with a view to control.

Most anthropogenic sources of fine particles, i.e. those less than  $2.5\ \mu m$  in aerodynamic diameter, involve combustion of some sort. Materials of biological origin (e.g. wood, coal and oil) burn in air by virtue of their carbon content. If a substance containing only hydrocarbon compounds burns with complete efficiency, only water and  $CO_2$  are produced. Such combustion demands a stoichiometric ratio of oxygen to fuel and, in practice, is never attained. Unburned fragments of combustible material, semi-volatile organic compounds, which vaporise and subsequently recondense as tarry droplets and incombustible matter are usually emitted as components of smoke during and following the combustion process. Improving the mix of air and fuel and pre-removal of volatile compounds may reduce smoke production. Smokeless fuel is prepared and burnt in this way. If the supply of oxygen is inadequate, large increases in CO production occur.

In most countries, motor vehicles, industrial activity and the generation of electricity account for a large percentage of the anthropogenic production of the oxides of nitrogen and sulphur. These, in addition to CO, particles and VOC are described as primary pollutants in that they are produced directly by the combustion process. Reactions taking place in the troposphere generate secondary pollutants:  $O_3$  is a classic example.  $NO_2$  breaks down photochemically under the action of ultra-violet light to generate NO and atomic oxygen. The latter combines with molecular oxygen to produce  $O_3$ . The presence of peroxy radicals derived from atmospheric reactions of HC and other organic compounds ensures that NO is oxidized back to  $NO_2$  without loss of  $O_3$ . Thus an  $O_3$ -generating series of reactions is established. The formation of  $O_3$  typically occurs as polluted air drifts away from sites of production including urban areas;  $O_3$  may thus occur at large distances from sources of  $NO_2$  and HC.

$NO_2$  is both a primary and a secondary pollutant. Motor vehicles emit both NO and  $NO_2$ . In the atmosphere, NO is oxidized to the dioxide, slowly by oxygen but rapidly by  $O_3$ . This explains the low concentrations of  $O_3$  generally found close to sources of oxides of nitrogen.

In addition to the above, sulphur in fuel also gives rise to both primary and secondary pollutants.  $SO_2$  is formed by oxidation during combustion. Further oxidation of  $SO_2$  leads to  $SO_3$ , which rapidly undergoes hydration to form sulphuric acid and this, in turn, is neutralized by  $NH_3$  to

ammonium bisulphate and ammonium sulphate. These compounds make an important contribution to the ambient fine particle aerosol.

The combustion of oil and petrol in internal combustion engines leads to the release of organic compounds, which condense in the air to produce small particles of the order of 1  $\mu\text{m}$  in diameter. These and the freshly formed sulphuric acid droplets of similar size are described as nucleation mode particles. Such particles have a short lifetime ( $< 1$  hour) and aggregate or agglomerate to produce particles in the 0.2-2.0  $\mu\text{m}$  diameter range which are defined as accumulation mode particles. These particles are stable and long-lived, and may be transported many hundreds of kilometres before being eventually lost from the air, generally as a result of below-cloud scavenging by precipitation.

### **2.1.3 Factors affecting the concentrations of air pollutants**

Local concentrations of air pollutants depend upon the strength of their sources and the efficiency of their dispersion. Day to day variations in concentrations are more affected by meteorological conditions than by changes in source strengths. Under some conditions both factors may play a part: in cold, still weather, dispersion is reduced whilst production is increased by the increased use of domestic space heating.

Wind is of key importance in dispersing air pollutants: concentrations being inversely related to wind speed for ground level sources. Turbulence is also important: a "rough" terrain, as produced for example by buildings, tends to lead to increased turbulence and better dispersion of pollutants.

Temperature inversions are of great importance in controlling the depth of the layer of air adjacent to the ground in which pollutants are well mixed (the mixing depth). As a mass of air rises it is exposed to decreasing atmospheric pressure and expands accordingly. This causes the temperature of the air mass to fall. The rate at which temperature falls with height is described as the adiabatic lapse rate: for dry air the rate of decline of temperature is about  $1^{\circ}\text{C}$  for each 100 m of height. Air saturated with water vapour loses heat more slowly than dry air, since the heat capacity of water vapour is twice that of dry air. As temperature falls and the saturated vapour pressure also falls, water condenses out as droplets and latent heat is released. As air containing water vapour, but not saturated, cools on rising it will reach saturation and thereafter the adiabatic lapse rate will be reduced.

As a mass of air rises it cools but as long as its temperature remains greater than that of the surrounding air it will retain buoyancy and continue to rise. Conversely if the actual temperature falls more slowly than that of the mass of air, or even increases, the cooling air will rapidly become heavier than the surrounding air and it will fail to rise. Consequently, a temperature inversion occurs when the air temperature rises with height above the ground.

At night, with low wind speeds and clear skies, rapid cooling of the ground and the adjacent air causes air to be coldest close to the ground and thus air cannot rise. Polluted air will not rise in the layer in which the usual temperature gradient is reversed and thus the concentration of pollutants in this layer will increase, sometimes leading to a thick layer of polluted air close to the ground.

Temperature inversions occur in summer as well as in winter. With strong sunlight and high traffic density, temperature inversions contributed to the high incidence of photochemical smog first described in the early 1950s in Los Angeles and now seen commonly in other large cities surrounded by mountains, such as Mexico City, Sao Paulo, and Caracas.

#### **2.1.4 Exposure to air pollutants**

The total daily exposure of an individual to air pollution is the sum of the separate contacts to air pollution experienced by that individual as he/she passes through a series of environments during the course of the day (also called micro-environments, e.g. at home, while commuting, in the streets, etc.). Exposures in each of these environments can be estimated as the product of the concentration of the pollutant in question and the time spent in the environment. In this model, the concentration of pollutants is assumed to be approximately constant during the time a person spends time in it. Exposure should not be confused with dose: i.e., the amount of pollutant absorbed. As the number of micro-environments studied is increased, a better estimate of total daily exposure is produced. The daily average concentration of a pollutant recorded at a single, fixed-site outdoor monitoring point provides only a very approximate guide to actual exposure.

One obvious and important micro-environment is the indoor environment where the types and concentrations of pollutants may be very different from those outdoors. For example, O<sub>3</sub> concentrations are generally much lower indoors in the absence of indoor sources, and O<sub>3</sub> penetrating from outdoors is destroyed by reaction with interior surfaces. By contrast, indoor concentrations of a chemically non-reactive fine particle such as sulphate may reach 90% of those outdoors. For some pollutants, indoor concentrations usually exceed outdoor concentrations.

In some cool climate countries people living in urban areas spend as much as 90% of their time indoors; this should be considered in interpreting the results of epidemiological studies relating outdoor concentrations of pollutants to effects on health. In other countries where climates are warm and many occupational activities are conducted outdoors, the percentage of the day spent indoors may be very much less. In some developing countries, indoor air pollution may be much higher than outdoor air pollution due to use of biomass fuels in open stoves (Section 4.2).

Besides varying temporally, outdoor concentrations of air pollutants vary from place-to-place. For example, concentrations of primary pollutants generated by motor vehicles decline rapidly as one moves away from busy roads. However, concentrations of pollutants generated by motor vehicles may be significantly higher inside motor vehicles than indicated by single site monitors and thus the motor car may itself be a significant micro-environment. Some pollutants are comparatively evenly distributed across large areas: O<sub>3</sub> and fine particles are examples. For such pollutants, monitoring at a limited number of sites may provide an adequate indication of concentrations over wide regions.

Personal monitoring devices have been developed for some pollutants. At their simplest these provide an integrated assessment of personal exposure over a given period. An overview of some aspects of the technology of monitoring devices is provided in Chapter 5.



### 2.1.5 Health significance of air pollution

Exposure to air pollution is probably as old as human exposure to fire. There is a large amount of archaeological evidence that indoor air pollution must have been troublesome to early humans, who used fire in confined spaces (Brimblecombe 1987). The classical writers record the oppressive fumes of Rome. Attention to effects of air pollution on health was focused during the early and mid 20th Century by a series of air pollution episodes, which produced dramatic effects on health. The Meuse Valley in Belgium (1930), Donora in the USA (1948) and London, England (1952) all experienced air pollution episodes which were investigated in some detail. In the 1952 London air pollution episode it was estimated that 4000 extra deaths occurred as a result of a smog largely consisting of high concentrations of SO<sub>2</sub> and particulate matter (Brimblecombe 1987), and in Donora some 43% of the population were affected by symptoms including headache, eye irritation, dyspnoea and vomiting. Analysis of the London episode showed that the elderly, especially those suffering from pre-existing cardio-respiratory disorders and the very young were at greatest risk. Later studies demonstrated a decline in urban levels of chronic bronchitis as concentrations of air pollutants fell (Chin et al 1981).

Emphasis on severe episodes of pollution may have distracted attention from the effects of long term exposure to pollution. Studies in London in the 1950s and 60s showed that the self-reported state of health of a panel of patients suffering from chronic bronchitis varied with day-to-day levels of air pollution (Waller 1971). It was noted, using simple methods of analysis, that symptoms did not increase unless the concentrations of smoke (measured as Black, or British Smoke) and SO<sub>2</sub> exceeded 250 and 500 µg/m<sup>3</sup>, respectively. It is likely that, had more searching methods of analysis been applied, effects would have been seen at lower concentrations.

Since the 1950s a great body of evidence has accumulated showing that air pollutants have a damaging effect on health. Some of the key studies are reviewed in Chapter 3 of this publication. Two especially important groups of studies will be dealt with briefly here as they have played an important role in the formulation of these guidelines.

When the WHO Air Quality Guidelines for Europe were developed in 1987 (WHO 1987) emphasis was placed on the results of studies of volunteers exposed to air pollutants under controlled conditions. Where such studies demonstrated a Lowest Observed Effect, or Adverse Effect Level this was used as a starting point for deriving the relevant air quality guideline. Epidemiological studies that demonstrated a threshold of effect were used in the same way.

A new database of epidemiological studies emerged in the late 1980s and 1990s. This database of time-series studies was developed first in the United States and later in Europe and other areas (Schwartz et al 1996). In essence the time series approach takes the day as the unit of analysis and relates the daily occurrence of events, such as deaths or admissions to hospital, to daily average concentrations of pollutants whilst taking careful account of confounding factors such as season, temperature and day of the week. Powerful statistical techniques have been applied and coefficients relating daily average concentrations of pollutants to effects have been produced. The results of these studies have been remarkably consistent and have withstood critical examination well (Samet et al. 1995). Such methods cannot, of course, be expected to prove the possible or probable causal nature of the associations demonstrated, but detailed examination of the data and application of the usual tests for likelihood of causality have convinced many that it would be unwise to disregard the findings.

Associations have been demonstrated between daily average concentrations of particles, O<sub>3</sub>, SO<sub>2</sub>, airborne acidity, NO<sub>2</sub>, and CO. The associations for each of these pollutants were not significant in all studies though, taking the body of evidence as a whole, the consistency is striking. More remarkable than the consistency of the results was the demonstration of associations at levels of pollution hitherto expected to be quite safe: indeed, below the levels recommended in the 1987 WHO Air Quality Guidelines for Europe.

For particles and O<sub>3</sub> it has been accepted by many that the studies provide no indication of any threshold of effect. This was reflected in the tables relating small differences in daily concentrations of particles and O<sub>3</sub> to effects on health (Chapter 3).

In time - series studies, daily counts of events are related to the daily average concentration of pollutants measured, usually at a single, fixed, monitoring site or predicted from such measurements. In any city it is likely that there will be a distribution of personal exposure across the population. Thus, on days when the measured or predicted level of pollution is low, some individuals may be exposed to greater than the reported concentration. If such exposure exceeded some threshold then effects would be recorded and attributed to occurring as a result of exposure to the recorded or predicted concentration. It might then be asked whether time series studies are capable of discerning a threshold of effect, especially if the threshold is low. This problem is by no means limited to particulate matter and O<sub>3</sub>: similar difficulties in identifying a threshold of effect at a population level apply to lead. This is an important point with regard to defining an air quality guideline based on such data: it is unlikely that a single guideline value can be derived from such a database and thus the "guideline" should be accepted to be a relationship relating events to airborne concentrations. This is a significant departure from the concept of a guideline value as a level of exposure at which the great majority of people, even in sensitive groups, would be unlikely to experience any adverse effects. Translation of this new form of guideline into an air quality standard is likely to be difficult. Junker and Schwela further discussed this issue in some detail (Schwela and Junker 1978; Junker and Schwela 1998).

Time-series studies relate the concentrations of air pollutants to their effects on health: in fact they provide the slope of a regression line relating concentrations to health events. There are no grounds for simple extrapolation of the concentration-exposure relationship to high levels of pollution. Several studies have shown that the slope of regression line is reduced when the annual average concentration of pollution is high (Schwartz and Marcus 1990).

Elevations in daily rates for various adverse health outcomes are sometimes referred to as the acute effects of air pollutants. For example, an increase in pollutant concentration might cause an increase in asthma attacks. It is assumed that without an increase in pollution, neither would asthma attacks increase. It is also likely that long-term exposure to air pollution produces chronic effects on health. For example, lifelong exposure to air pollution in England amongst those born in the late 19th Century is likely to have increased their chances of developing chronic bronchitis and dying earlier than expected as a result of the illness. (Chinn et al. 1981). In the United States, cohort studies in a range of towns have demonstrated associations between long term average concentrations of fine particles (PM<sub>2.5</sub> and sulphates) and the Standardized Mortality Ratios of communities (Dockery *et al* 1993; Pope *et al* 1995). Attempts to estimate the public health impact of air pollution have been made on the basis of both the cohort studies and the time-series studies. On the basis of one of the cohort studies (Pope et al. 1995), Brunekreef (1997) has reported that exposure to current levels of air pollution in the Netherlands may lead to a average reduction in longevity of 1 year. Work

reported from the United States reports slightly larger effects: perhaps 2 years are lost in polluted communities compared with unpolluted areas of the United States. Loss of life expectancy may be distributed statistically across the affected population. This is the case amongst cigarette smokers where the average loss of life expectancy is of the order of 3-5 years, though some smoking-related deaths occur among people in their forties.

## **2.2     *Air pollutant concentrations and factors affecting susceptibility***

The concentrations of emitted pollutants and population exposures to air pollution vary substantially from country to country. In addition, human responses to air pollutant exposure also vary. Outdoor and indoor concentrations of air pollutants, and a number of examples of factors affecting responses to pollutants, are considered in this section.

### **2.2.1     Concentrations of classical pollutants in ambient air**

There are far more data available on the ambient outdoor concentrations of certain classic air pollutants in many countries around the world than for any of the other pollutants, as monitoring records on black smoke (BS) and SO<sub>2</sub> in particular go back for five decades or more. There are, however, relatively few locations where all of the classical air pollutants have been measured simultaneously, or over extended periods. Additionally, historical data are often of limited value for retrospective or cross-sectoral analyses of air quality and health. Only recently, and only for a limited number of sites, have the specificity of analyses, the validity of calibrations, the identification of site representativity for the specific sampling purpose, the consistency of averaging times and/or sampling intervals, and the frequency and data management procedures been standardized to appropriate quality assurance procedures (see Chapter 5).

Available air pollutant concentration data were reviewed by the Task Group and selected data summaries are presented here to give the reader some general perspectives on recent pollutant levels and trends in the various WHO regions. The presentation is organized into three categories for each of the classical air pollutants.

The first category contains data on air quality in the European region on the basis that these summary data provided key input to the *Air Quality Guidelines for Europe* (WHO 1999a), which in turn provided the basis for the WHO *Guidelines for Air Quality* summarized in this publication. These data underwent a limited peer review by the WHO/EURO Working Group that judged them to be sufficiently representative and reliable for inclusion in the *Air Quality Guidelines for Europe*.

The second category contains data on ambient outdoor air quality in other WHO regions that were collected from countries by representatives from those regions on the WHO Global Air Quality Guidelines Task Group. In most cases, the Task Group was not able to assess data quality. Thus, it was not possible to endorse these data in terms of their accuracy and representativity. Although some data may be of high quality, some of the data were based on intermittent sampling programmes and cannot be reliably used for determining longer-term average concentrations.

Despite these limitations, the Task Group considered that presentation of some of the available summary data would provide a valuable frame of reference for the readers of this report. Accordingly, for each WHO Region other than Europe, a restricted set of data was selected for this report. Wherever possible, they represent: (a) at the one extreme, data for point source monitoring in regions as being representative of high-end human exposures; (b) non-typical levels, selected from data for urban sites not greatly affected by industrial point sources; and (c) at the other extreme, non-urban site data selected as being representative of the low end of concentrations for the country.

Each region was represented by data from a limited number of countries in that region, that differed in size and extent of industrial development, to demonstrate the extent of potential exposure of people in that region. Wherever possible, data that are summarized include available information on the source of the data, averaging times, and the quality assurance procedures followed in producing the data.

The third category of data consists of summaries from the WHO Air Management Information System (AMIS) programme (WHO 1997b, WHO 1998b). Since AMIS collects data from collaborating centres in all WHO regions, there is some overlap in coverage with the data summarized in the regional reports within the second category. The primary justification for including the AMIS data as a separate category is that the procedures used to generate and report these data are more uniform and were subjected to more validation, providing an independent source of data of assured quality.

An examination of the data summaries that follow clearly shows that air quality in large cities in many developing countries is remarkably poor, and that very large numbers of people in those countries are exposed to ambient concentrations of air pollutants well above the WHO *Guidelines for Air Quality*.

## **Air quality data in developing countries**

The main source of information on air pollution in developing countries is the Air Management Information System AMIS (WHO 1997b) set up by the WHO as a continuation of GEMS/Air (UNEP/WHO 1993). AMIS is based on voluntary reporting of data by municipalities of the WHO member states. The AMIS core data base collects information on annual (arithmetic) mean and high (95-, 98-) percentiles of daily mean concentrations of SO<sub>2</sub>, NO<sub>2</sub>, O<sub>3</sub>, CO, SPM, lead and other potentially monitored compounds. In principle, data from three types of monitoring stations are stored: “industrial,” reflecting levels in areas affected by emissions from industry; “city center / commercial,” which will be mostly affected by traffic; and “residential,” which should reflect the best basic level of population exposure. Until now the coverage of the system has been limited to 100 cities, but the intention is to acquire current information from some 300 cities by the end of 2000. The analysis of the data and its limitation is discussed by Krzyzanowski and Schwela (1999).

## Air pollution levels and trends

### Sulphur dioxide

In most analysed cities, the annual mean concentrations of SO<sub>2</sub> in residential areas have not exceeded 50 µg/m<sup>3</sup>. Notable exceptions are several cities in China, with the SO<sub>2</sub> concentration of 330 µg/m<sup>3</sup> in Chongqing and 100 µg/m<sup>3</sup> in Beijing in 1994. In some Chinese cities, the levels reported from “residential” locations exceed those from “commercial” regions of the city and are comparable with the levels in industrial zones. This may reflect the impact of combustion of sulphur-containing coal for domestic heating and cooking.

High levels of SO<sub>2</sub> may also be seen in other developing countries, especially in those with cold winters, as illustrated by the report from Nepal (Sharma 1997). Daily mean concentration of SO<sub>2</sub> was in the range 273 - 350 µg/m<sup>3</sup> in residential areas of Kathmandu during September - December 1993. In monitoring sites close to main roads, the reported range is 310-875 µg/m<sup>3</sup>, reflecting the influence of emissions from traffic. More than half of the vehicles registered in the city are equipped with two-stroke engines and many are old and ill maintained.

In most of the cities with data allowing trend assessment a decline in mean annual SO<sub>2</sub> concentration was seen over the 1990s. The most dramatic reduction of air pollution with SO<sub>2</sub> was reported from Mexico City, where the concentration in various residential areas dropped from 100-140 µg/m<sup>3</sup> in 1990-1991 to 32-37 µg/m<sup>3</sup> in 1995-1996. In the most polluted Chinese cities an annual means declined between 1% and 10%.

### Suspended particulate matter

The most commonly monitored and reported indicator of this type of air pollution is the mass concentration of TSP. In most of the cities, the TSP annual mean concentration exceeds 100 µg/m<sup>3</sup>, with the levels exceeding 300 µg/m<sup>3</sup> in several cities of China and India. There is no evidence of any overall systematic and significant change in TSP levels: the data from the 1990s show increasing as well as decreasing trends in a similar number of cities. The most visible relative decrease of TSP concentrations is shown by the data from Bangkok, but the progress is not steady there either. More consistent, though with a smaller relative rate, is the decrease in TSP concentration in Mexico City. The opposite tendency can be seen in some Chinese cities, with the most rapid increase of TSP concentration in Guangzhou (from less than 150 µg/m<sup>3</sup> in 1990-1992 to more than 300 µg/m<sup>3</sup> in more recent years).

In a limited number of cities reporting data to AMIS levels of PM<sub>10</sub> are also measured. The most commonly registered annual average PM<sub>10</sub> levels ranged from 50 - 100 µg/m<sup>3</sup> in the years 1995-1996. The highest concentrations, exceeding 250 µg/m<sup>3</sup>, were observed in Calcutta and New Delhi. In most towns with high PM<sub>10</sub> average in the last year, an increase in the pollutant concentration was seen over the 1990s. In most cases, this increase has occurred even when the decrease in TSP was reported. An opposite trend and a decrease in PM<sub>10</sub> level were seen in the Central and Southern America cities. In Mexico City, the relative decrease in PM<sub>10</sub> was faster than that of TSP.

This limited information on the size-specific particulate pollution allows a comparison of the mass concentration of TSP and PM<sub>10</sub>. For most sites and years with data on both indicators, the PM<sub>10</sub> to TSP ratio was in the range between 0.4-0.8. However, in a few cases, the ratio exceeded

1.0. This fact indicates that the measurements reported to AMIS might not have been done at the same locations and/or periods. In a southeastern part of Mexico City, the ratio remained between 0.25 and 0.32 in all years 1991-1996, while in the southwestern part of the city it was consistently between 0.44 and 0.55. More specific studies of the size distribution of airborne particles, conducted in the northern cities of China in the mid-1980s, indicate that some 70% of the mass concentration of TSP are due to the PM<sub>10</sub> (Ning et al 1996). During the heating season, particles with diameter less than 2 µm were found to make some 30-50% of TSP. The elemental analysis of the particles confirmed that human activities are the main source of the fine fraction of particulate matter. Similar results were reported from Jakarta, where particles with diameter less than 7.2 µm contributed more than 80% of TSP (Zou et al 1997). Traffic-related compounds contributed significantly to the overall pollution mass, and especially to the fine particle fraction.

## **Nitrogen dioxide**

In most of the cities reporting to AMIS the annual mean concentrations of NO<sub>2</sub> remain moderate or low, not exceeding 40 µg/m<sup>3</sup>. However, in Mexico City and in Cape Town, the annual average of 70 µg/m<sup>3</sup> has been exceeded regularly in the 1990s. A paper based on data from centrally located monitors in Sao Paulo indicates annual mean of 240 µg/m<sup>3</sup> in 1990/91 (Saldiva et al. 1995). The trends vary between the cities, but a 5-10% annual increase in concentration of this pollutant was more common than a decrease.

The observed pattern is consistent with the volume of car traffic in each city. The highest pollution levels, and the increasing trends, are observed in the cities with high and increasing car traffic. In Southern Asia or in Latin America, this high NO<sub>2</sub> concentration combined with the intense UV radiation results in photochemical smog with high oxidant concentrations. It is illustrated by the analysis of temporal and spatial patterns of tropospheric O<sub>3</sub> in New Delhi (Singh et al 1997). The build-up of O<sub>3</sub> over the day is faster than scavenging of O<sub>3</sub> by the NO<sub>2</sub>. In Mexico City, the mixture of high NO<sub>2</sub> emissions from gasoline combustion and intense UV radiation is the cause of the notorious photochemical smog in that city. According to the data reported to AMIS, the O<sub>3</sub> concentration exceeded a concentration of 120 µg/m<sup>3</sup> in over 300 days a year in 1994-96, and the 95<sup>th</sup> percentile of maximum daily 1-hour average O<sub>3</sub> concentration was around 500 µg/m<sup>3</sup>. Some decrease was seen, however, in the annual mean O<sub>3</sub> concentration, indicating slow improvement of air quality in non-extreme days.

### **2.2.2 Factors affecting susceptibility to indoor air pollution**

Indoor air pollutants usually differ in type and concentration from outdoor air pollutants. Indoor pollutants include environmental tobacco smoke, biological particles (such as pollen, mites, moulds, insects, microorganisms, pet allergens etc.), non-biological particles (such as smoke), VOC, NO<sub>x</sub>, lead, radon, CO, asbestos, various synthetic chemicals and others. Degradation of indoor air quality has been associated with a range of health effects, including discomfort, irritation, chronic pathologies and various cancers.

With growing public concern about indoor air quality, action has been taken in many developed countries to characterize levels of indoor air pollutants, to improve ventilation and fuel emissions, and to reduce exposure to environmental tobacco smoke, biological contamination and radon among other actions. Even though there is considerable evidence that indoor air quality is a serious and widespread problem in many developing countries, the information and

resources to control indoor air quality are often lacking (Ferrari et al. 1995). Management of indoor quality is discussed in section 6.2.

Perhaps the most important factor that causes qualitatively and quantitatively different exposures to air pollutants across different countries is that of indoor heating and cooking by solid fuel burning. This topic deserves special attention. The emissions, concentrations, exposures and health effects are discussed in detail in Chapter 4. On a global scale, biomass fuels (wood, crop residues, dung and grass) are used daily in about half the world's households as energy for cooking and/or heating. In China, for example, it has been estimated that coal burning results in particle concentrations up to  $5000 \mu\text{g}/\text{m}^3$  in indoor living areas, whereas smoky houses in Nepal and Papua New Guinea have peak levels of  $10\,000 \mu\text{g}/\text{m}^3$  or more (Smith 1996). An unknown, but significant, proportion of this activity takes place in conditions where much of the airborne effluent is released into the living area. Therefore, some of the highest concentrations of particulate matter other pollutants occur in rural, indoor environments in developing countries.

Biomass smoke contains significant amounts of several important pollutants: CO, particles, HC and to a lesser extent,  $\text{NO}_x$ . However, biomass smoke also contains many organic compounds, including PAH, that are thought to be toxic, carcinogenic, mutagenic or otherwise of concern. Coal smoke contains all of these as well as additional pollutants, e.g. sulphur oxides and heavy metals such as lead. In many parts of the world these pollutants are released from stoves in poorly ventilated homes or in enclosed courtyards. Due to the high concentrations and the large populations involved, the total human exposure to many important air pollutants can be much higher in the homes of the poor in developing countries than in the outdoor air of cities in the developed world.

### **2.2.3 Meteorological factors**

At increased altitude the partial pressure of oxygen falls and inhalation increases in compensation. For gaseous pollutants no increase in effects over those experienced at sea level would be expected as a result of the increased inhalation, as the partial pressures of the pollutant gases will fall in line with that of oxygen. For particles, on the other hand, increased inhalation volumes will lead to increased intake of airborne particles and perhaps changes in patterns of deposition. Differences in effects between those who have always lived at high altitude and those who have recently relocated there might be expected.

Temperature has a very significant effect on health and has been shown to be an important confounding factor when examining the effects of air pollutants. The relationship between ambient temperature and ill health is "U"- or "V"-shaped with excess daily deaths increasing in both cold and hot conditions. Local populations tend to be acclimatised to local conditions and cope better with changes in temperature than do immigrants from other countries. The effect of low temperatures in winter is more marked in countries with temperate climates, than in much colder countries. Inhaled volumes increase under hot conditions, and thus the intake of pollutants also increases. In addition, warm days encourage people to spend more time out-of-doors and so personal exposure patterns to pollutants may change. Of course, living in well-ventilated houses in warm weather, when doors and windows may be open, may decrease exposure to pollutants from indoor sources and increase exposure to outdoor sources.

Humidity is unlikely to have a significant effect on the toxicity of gaseous pollutants, and it may reduce the effects of some particles by permitting hygroscopic growth in particle size prior to

inhalation, thus changing the patterns of deposition from smaller to larger airways in the lung.

#### **2.2.4 Demographic factors**

The age structure of populations differs markedly from country to country. Old people tend to show increased susceptibility to air pollution as a result of reduced functioning of physiological defence mechanisms, reduced physiological reserves and the increased prevalence of disease.

Very young children may also be at increased risk due to incompletely developed defence mechanisms, higher ventilation rates per unit body mass and a tendency to spend more hours out of doors than adults.

#### **2.2.5 Socio-economic factors**

People with a poor standard of living suffer from nutritional deficiencies, from infectious diseases due to poor sanitation and overcrowding, and tend to be provided with a poor standard of medical care. Each of these factors may render individuals more susceptible to the effects of air pollution. A dietary lack of anti-oxidant factors may decrease defence mechanisms against oxidant pollutants such as O<sub>3</sub> and NO<sub>2</sub>. Delayed clearance of particles in airways already damaged by infection is likely. In developing countries, poor air quality may be closely associated with the incidence of infectious diseases.

#### **2.2.6 Effects of differing levels of disease in the population**

Diseases which produce narrowing of the airways, a reduction in the area of the gas-exchange surface of the lung and an increased alteration of inhalation-perfusion ratios are likely to make the subject more susceptible to the effects of a range of air pollutants. Epidemiological studies have shown that patients suffering from asthma or chronic obstructive pulmonary disease suffer an increase in symptoms when levels of pollutants are raised (see Chapter 3). It should be noted that asthma is less common in developing than in developed countries. However, the prevalence of infectious disease in developing countries, including tuberculosis, may militate against the development of the IgE antibody response, which is characteristic of asthma.

#### **2.2.7 Specific differences in prevalence levels of air pollutants**

Concentrations of air pollutants vary greatly from country to country. In countries where indoor air pollution is common, due to cooking over open fires with poor ventilation, indoor exposure may be an important cause of damage to health especially among women. In other countries, including those of the Middle East, particle concentrations in outdoor air are high due to wind-blown dust. In desert areas this dust contains a high proportion of silica, and silicotic nodules have been described in the lungs of residents. However, high concentrations of volcanic ash do not seem to be associated with acute effects on health. Specific examples of the levels of ambient urban air quality, and indoor air quality in various countries around the world are provided by the AMIS (WHO 1997b; WHO 1998b).

Countries burning brown coal (or lignite) for domestic heating are likely to experience high concentrations of smoke and SO<sub>2</sub>. To these may be added the pollutants produced by motor



vehicles. Leaded motor vehicle fuel is in use in many parts of the world and in these areas airborne lead particles make an important contribution to total lead intake both by inhalation and by ingestion.

## **2.3    *Exposure to air pollutants***

An ideal characterization of the distribution of human exposures would be based on direct measurements of each pollutant concentration in the breathing zone of each member of a representative cross section of the population of interest. At present, however, such a programme is technically impossible and probably impractical as well. Instead, ambient air quality measurements at central, fixed, air monitoring sites are widely used surrogates for population exposures, and are generally the only widely available quantitative resource that can be related to exposures. Personal monitors for exposure estimates could overcome some of the shortcomings of ambient air monitors, but they can be applied only in a small sample of the population.

There are many factors that can account for the substantial differences between the concentrations of pollutants measured at central sites and those in the breathing zone of residents of the community. Air pollutants emitted into outdoor air can be attenuated during infiltration into indoor air. This attenuation can be expected to be minimal for all pollutants of outdoor origin when barriers such as windows and doors are open or absent. In contrast, attenuation can be very large for tightly sealed buildings during times of maximal heating or cooling needs.

The attenuation of indoor air pollutant concentrations by removal to indoor surfaces is highly dependent on the physico-chemical characteristics of the pollutant. At one extreme is a chemically stable fine particle component such as sulphate ion, where indoor concentrations are typically 90% or more of outdoor concentrations. At the other extreme, indoor concentrations can be low for larger particles deposited by sedimentation in the relatively still air.

For a relatively non-reactive gas, such as CO, the indoor-outdoor concentration ratio is usually near unity in a home without indoor CO sources. However, indoor concentrations can be much higher than outdoor concentrations when there are sources such as burning cigarettes and open flames used for cooking or space heating. By contrast, chemically reactive gases, such as O<sub>3</sub> and SO<sub>2</sub> fairly rapidly diffuse to, and react with, interior surfaces. As a result, indoor-outdoor concentration ratios are typically much lower than unity.

Lead is the only classic air pollutant that can gain access to humans through indirect transport routes. Where leaded motor vehicle fuels are used, fine particle emission from vehicle exhausts can be inhaled. In addition, the particles that deposit on terrestrial surfaces can be ingested, either directly from soil in play yards, or after being carried indoors as a component of house dust. Furthermore, particulate lead deposited on plants or agricultural fields can be retained in food products and add to body burdens. Similar pathway considerations also apply to toxic air pollutants other than lead.

Humans engage in a variety of daily activities, and the concentrations of air contaminants in their breathing zone can vary substantially as they move through various microenvironments, each of which may be affected by different attenuation factors or increments from indoor sources. Furthermore, even a complete knowledge of the concentrations of all relevant pollutants in each

microenvironment would not provide an adequate basis for predicting physiological and pathological responses to their exposures. Pollutant uptake could also be greatly affected by ventilation rate and pattern, entry of air via the nose or mouth, airway sizes (which exhibit great individual variability), past and current history of exposure to other toxicants (such as cigarette smoke), and prior disease histories and genetic predispositions. Many of these factors can be modelled and such models have been used for estimating dose distributions associated with ambient air concentrations.

## **Sulphur dioxide**

SO<sub>2</sub> is a colourless pungent, irritating, water-soluble and reactive gas. Concentrations in ambient air in cities of developed countries have mostly decreased in the last two or three decades due to tighter emissions control, increased use of low sulphur fuels and industrial restructuring. Consequently, high ambient concentrations in earlier decades have been replaced by annual mean concentrations of about 20-40 µg/m<sup>3</sup> in most cities in developed countries and daily means rarely exceed 125 µg/m<sup>3</sup>.

However the situation is more complex in developing countries. In cities, the annual mean concentrations of SO<sub>2</sub> in ambient air may range from very low levels up to 300 µg/m<sup>3</sup> (WHO 1998b). Peak concentrations measured as ten-minute averages may exceed 2000 µg/m<sup>3</sup> under conditions of poor atmospheric dispersion such as inversions (Section 2.2.2), or when emissions from a major source are brought to ground levels by certain atmospheric conditions. SO<sub>2</sub> can also reach high concentrations in air in some indoor environments through the use of sulphur containing fuels such as coal for heating and cooking (Section 4.2).

As it is highly reactive, SO<sub>2</sub> has a highly non-uniform dose distribution along the conductive airways of the respiratory tract. For low to moderate tidal volumes and nasal breathing, the penetration into the lungs is negligible. For larger tidal volumes and oral inhalation, doses of interest may extend into segmental bronchi. SO<sub>2</sub> can only reach the gas-exchange region of the lungs after sorption onto fine particles; and the available particle surface is limited except when very large mass concentrations of fine particles are present (WHO 1987; WHO 1994a).

Another special consideration for SO<sub>2</sub> is that there is a great variation in susceptibility to a bronchoconstrictive response. Persons having asthma or atopy can be about 10 times more responsive than healthy subjects.

## **Nitrogen dioxide**

Ambient concentrations of NO<sub>2</sub> in air are variable. Natural background concentrations in ambient air can be less than 1 µg/m<sup>3</sup> to more than 9 µg/m<sup>3</sup>. In ambient air in cities annual mean concentrations can range from 20-90 µg/m<sup>3</sup> with hourly maximum concentrations from 75-1000 µg/m<sup>3</sup> (WHO, 1994a). Concentrations of NO<sub>2</sub> in indoor air can reach average concentrations of 200 µg/m<sup>3</sup> over several days, with hourly maximum concentrations of 2000 µg/m<sup>3</sup> where there are unvented gas heating appliances and poor ventilation (WHO, 1994a).

NO<sub>2</sub> is a relatively water-insoluble gas and appreciable amounts of inhaled NO<sub>2</sub> can penetrate to, and elicit biological responses in, small lung airways. As with SO<sub>2</sub>, there is much greater susceptibility to a bronchoconstrictive response in individuals with asthma.

## **Carbon monoxide**

Natural ambient concentrations of CO range between 0.01-0.23 mg/m<sup>3</sup> (WHO 1994a). In urban environments, mean concentrations over eight hours are usually less than 20 mg/m<sup>3</sup>, and one-hour peak levels are usually less than 60 mg/m<sup>3</sup>. Highest concentrations are usually measured near major roads, as vehicles are the major source of CO. Concentrations of CO can be high in vehicles, underground car parks, road tunnels and in other indoor environments where combustion engines operate with inadequate ventilation. In these circumstances, mean concentrations of CO can reach up to 115 mg/m<sup>3</sup> for several hours. In houses with unflued combustion heaters, peak CO concentrations can reach up to 60 mg/m<sup>3</sup> (WHO 1994a).

CO exerts its toxic effects after binding with hemoglobin in the capillaries of the lungs. It is not removed in larger airways.

## **Ozone**

Background concentrations of O<sub>3</sub> in remote and relatively unpolluted parts of the world are often in the range of 40 to 70 µg/m<sup>3</sup> as a one-hour average. In cities and areas downwind of cities, maximum mean hourly concentrations can be as high as 300 to 400 µg/m<sup>3</sup>. High O<sub>3</sub> concentrations can persist for 8 to 12 hours per day for several days, when atmospheric conditions favour O<sub>3</sub> formation and poor dispersion conditions exist (Section 2.2.2). O<sub>3</sub> is normally at higher concentrations in ambient air outdoors than in indoor air.

O<sub>3</sub> is a relatively water-insoluble gas. It reacts and produces toxic effects on small airway surfaces. The dose-delivery is greatest in terminal and respiratory bronchioles. Unlike NO<sub>2</sub> and SO<sub>2</sub>, there is very little difference in lung function responsiveness between asthmatics and healthy subjects. There is, however, a great variability in individual responsiveness that is not yet understood.

## **Particulate matter (PM)**

Concentrations of particulate matter in air are highly variable. In some areas very high levels occur naturally due to wind-blown dust from arid soils. Human activities, such as fires, overgrazing, agricultural practices and mining, can increase particle concentrations in air in remote areas. In Western Europe and North America efforts to control emissions of particulate matter have generally resulted in lower levels of particles in ambient air. In many cities the annual average concentrations of PM<sub>10</sub> are in the range 20 to 50 µg/m<sup>3</sup> for ambient air (WHO 1999a). However, annual average concentrations in some cities in Eastern Europe and in some developing countries can be above 100 µg/m<sup>3</sup>. Concentrations of PM<sub>2.5</sub> are usually about 45 to 65% of the concentrations of PM<sub>10</sub>.

Concentrations of particulate matter in indoor air can be extremely high when biomass fuels such as wood, crop residues and dung, or coal are used for cooking or heating. Indoor concentrations of up to 2000 to 5000 µg/m<sup>3</sup> of total suspended particulate matter have been measured in some circumstances during cooking with biomass fuels in developing countries (Section 4.2).

Particle size is a critical factor in internal dose distribution. The location of initial deposition in the airways depends on particles size, with coarse particles being deposited in the upper respiratory tract and fine particles being transported to the lower respiratory tract. The rate of deposition in conductive airways also depends on particle size (see Section 2.1).

## Lead

Levels of lead found in air, food, water and soil/dust vary widely throughout the world and depend on the degree of industrial development, urbanization and lifestyle factors. Ambient air levels over  $10\text{ }\mu\text{g}/\text{m}^3$  have been reported in urban areas near smelters, whereas lead levels below  $0.1\text{ }\mu\text{g}/\text{m}^3$  have been found in cities where leaded petrol is no longer used. In cities of developing countries traffic-related lead levels range between  $0.3$  and  $1\text{ }\mu\text{g}/\text{m}^3$  with extreme annual mean values between  $1.5$ - $2\text{ }\mu\text{g}/\text{m}^3$ .

Lead is inhaled as fine particles and deposited in the lungs. Since lead uptake by blood is dependent on deposition pattern and solubility (which is influenced by chemical form and particle size), total lead content is only a surrogate for the biologically effective dose. Furthermore, as noted in earlier sections, airborne lead can also reach humans indirectly via deposition on soil and vegetation, and through food chains.

## Other air pollutants

In nearly all countries routine air quality monitoring programmes are concentrated almost exclusively on selected classic pollutants. Relatively few of the other air pollutants (considered in detail in Section 3.2) are routinely monitored, except in a few occupational environments. Data are sometimes collected on personal exposures to classic and other air pollutants, but seldom are there standardised protocols for sample collection and analysis, and data processing and storage. As a result, estimates for personal exposures are generally based on highly uncertain models and the assumptions built into them. In general, the situation with respect to ambient concentrations of other air pollutants considered in section 3.2 is characterized as described in the second columns of Tables 3.2 and 3.3 in that section.

## 2.4 Role of guidelines and standards

The purpose of these guidelines is to provide a basis for protecting public health from the adverse effects of air pollution and for eliminating, or reducing to a minimum, those air contaminants that are known to be, or are likely to be, hazardous to human health and well being (WHO 1987).

These *Guidelines* should provide background information for nations engaged in setting air quality standards, although their use is not restricted to this. The *Guidelines* are not intended to be standards. In moving from guidelines to standards, prevailing exposure levels and environmental, social, economic and cultural conditions in a nation or region should be taken into account (see Section 2.4.4). In certain circumstances there may be valid reasons to pursue policies, which will result in pollutant concentrations above or below the guideline values (WHO 1987).

### 2.4.1 The 1987 WHO Air Quality Guidelines for Europe

Already in 1958, WHO recognized that air pollution was a threat to the health and well-being of peoples throughout the world. As a consequence, WHO has taken its first steps to marshal the facts and to suggest procedures by which preventive and remedial action may be taken by its member countries, before serious harm is done to the health of their people (WHO 1958). In a

forthcoming Technical Report, criteria for guidelines for air quality are described as tests, which permit the nature and magnitude of air pollution on man and the environment to be determined. Guidelines were defined as sets of concentrations and exposure times that are associated with specific effects of varying degrees of air pollution on man, animals, vegetation and on the environment in general (WHO 1964). In 1972, guidance as to the levels of ambient air pollutants that constitute hazards to health were first formulated for the “classic” compounds SO<sub>2</sub>, SPM, CO and photochemical oxidants (WHO 1972). These attempts culminated in 1987 in the publication of the Air Quality Guidelines for Europe for a much extended set of air pollutants (WHO 1987).

In the *Air Quality Guidelines for Europe* (WHO 1987), relevant information on the pollutants was carefully considered during the process of establishing guideline values. It was noted that ideally, guideline values should represent concentrations of chemical compounds in air that would not pose any hazard to the human population. However, the realistic assessment of human health hazards necessitated a distinction between absolute safety and acceptable risk. To aim at achieving absolute safety, one would need to know the complete dose-response relationships in individuals in relation to all sources of exposure. Moreover, the type of toxic effect elicited by specific pollutants or their mixtures; the existence (or not) of “thresholds” for specified toxic effects; the significance of interactions; and the variation in sensitivity and exposure levels within the human population would all have to be known. However, such comprehensive and conclusive data on environmental contaminants are not always available. Scientific judgement and consensus, therefore, play an important role in establishing acceptable levels of population exposure.

### **Criteria for endpoints other than carcinogenicity**

For compounds reportedly without carcinogenic effects, or for which data on carcinogenicity were lacking or insufficient, the starting-point for the derivation of guideline values was to define the lowest concentration at which effects are observed in humans, animals and plants. The difference between the lowest level at which an effect is observed, and the level, at which no effect is observed, is among the factors included in judgements concerning the appropriate margin of protection. In the case of irritant and sensory effects on human, it was considered desirable where possible to determine the no-effect level.

### **Criteria for selection of a lowest-observed adverse-effect level (LOAEL)**

The distinction between adverse and non-adverse effects was stated to pose considerable difficulty. The definition of an adverse effect was given as “any effect resulting in functional impairment and/or pathological lesions that may affect the performance of the whole organism, or which contributes to a reduced ability to respond to an additional challenge”. Even with such a definition, a significant degree of subjectivity and uncertainty was found to be present. To resolve this difficulty, data were ranked in three categories: (i) Single observations, even of potential health concern, were not readily used as a basis for guideline values; (ii) A lowest-observed-effect level might result in pathological change, and therefore was considered a higher degree of health concern; (iii) A substantial change in the direction of pathological effects has had a major influence on guideline considerations.

### **Criteria for selection of uncertainty factors**

The toxicology of pollutants, including the type of metabolites formed, variability in metabolism, or response in humans suggesting hypersusceptible groups, and the likelihood that the compound or its metabolites will accumulate in the body, was taken into account by uncertainty factors. Uncertainty factors were essentially determined through scientific judgement in consensus.

### **Criteria for selection of averaging times**

As a chemical may cause acute, minor, reversible effects after brief exposure, and irreversible or incapacitating effects after prolonged exposure, expert judgement had to be applied, based on the weight of the evidence available. Generally, when short-term exposures lead to adverse effects, short-term averaging times were recommended. In other cases, exposure-response knowledge was sufficient to recommend a long-term average.

### **Criteria for consideration of sensory effects**

Some of the substances selected for evaluation have malodorous properties at concentrations far below those at which toxic effects occur. Although odour annoyance cannot be regarded as an adverse health effect in a strict sense, it affects the quality of life. Therefore, odour threshold levels (detection threshold, recognition threshold, and nuisance threshold) for such chemicals have been indicated where relevant and used as a basis for separate guideline values.

### **Criteria for Carcinogenic Endpoint**

Cancer risk assessment involves a qualitative assessment of how likely it is that an agent is a human carcinogen, and a quantitative assessment of the cancer rate the agent is likely to cause at given level and duration of exposure.

### **Quantitative assessment of carcinogenicity**

The decision to consider a substance as a carcinogen is based on the classification criteria of the International Agency for Research on Cancer:

Group 1	Proven human carcinogens.
Group 2	Probable human carcinogens. This category is divided into two subgroups according to higher (Group 2A) and lower (Group 2B) degrees of evidence.
Group 3	Unclassified chemicals

It was decided that for all chemicals not categorized in Groups 1 and 2A guideline values based on non-carcinogenic health endpoints were to be given.

### **Quantitative assessment of carcinogenic potency**

Quantitative risk assessment was found to include the extrapolation of risk from relatively high dose levels to relatively low dose levels. High dose levels are characteristic of animal experiments or occupational exposures, where cancer responses can be measured. Low dose levels are of concern in environmental protection, where such risks are too small to be measured directly, either in animal or epidemiological studies.

In the 1987 guidelines, the risk associated with lifetime exposure to a certain concentration of a carcinogen in the air has generally been estimated by linear extrapolation. The carcinogenic potency has been expressed as the incremental unit risk estimate. The incremental unit risk estimate of an air pollutant was defined as “the additional lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1  $\mu\text{g}/\text{m}^3$  of the agent in the air they breathe”.

Necessary assumptions for the average relative risk method were: (i) the response (measured as relative risk) is some function of cumulative dose or exposure; (ii) there is no threshold dose for carcinogens; (iii) the linear extrapolation of the dose-response curve towards zero gives an upper-bound conservative estimate of the true risk function, if the unknown (true) dose-response curve has a sigmoidal shape; (iv) there is constancy of the relative risk in the specific study situation.

Advantages and limitations of the method used in the 1987 guidelines were extensively discussed.

#### **2.4.2 The development of the guideline setting process**

During the development of the 1987 WHO *Guidelines*, emphasis was placed on specifying the guidelines in terms of a concentration and averaging time, which would define an exposure unlikely to produce adverse effects, even in the majority of those members of groups with increased sensitivity to the pollutant in question. Small changes, or so called physiological changes, for example in indices of lung function, were agreed to fall outside the definition of “adverse effects”.

For many of the classic air pollutants the guidelines were based on controlled exposure studies, or on epidemiological studies which demonstrated a threshold of effect. Uncertainty factors, or protection factors, were applied to the published data to allow for more sensitive individuals who might not have been adequately represented in the studies. The guidelines were statements of levels of exposure at which, or below which, no adverse effects can be expected. This does not imply that as soon as a guideline is exceeded adverse effects occur, but rather that the likelihood of such effects occurring would be increased. The guidelines have sometimes been misinterpreted as Lowest-Observed-Adverse-Effect Levels (LOAEL), which they are not.

Genotoxic carcinogens were treated differently: a Unit Risk was estimated from calculating the additional risk from a lifetime exposure to a unit concentration of the carcinogen. For a few pollutants, including  $\text{O}_3$ , the guideline was specified as a range of concentrations.

During the period between the publication of the 1987 *Guidelines* and their revision, a number of meetings were held to consider how the guidelines might be updated (WHO 1992a; WHO 1994a; WHO 1995a; WHO 1995b; WHO 1995c; WHO 1996a). A number of important decisions were made and these are detailed in the reports of the meetings. Among these, the desirability of providing guidance on the exposure-response relationship for as many pollutants as possible was stressed. This has been an important feature of the revised guidelines.

In the updated version of the *Air Quality Guidelines for Europe*, a similar approach was applied as in the 1987 air quality guidelines. However, total tolerable intakes were calculated for multimedia pollutants first, and then adequately partitioned among the different exposure routes. The term “protection” factor used in the 1987 guidelines was abandoned. Instead, uncertainty

factors to account for the extrapolation from animal to man (alternatively, human equivalent concentrations were calculated), and to account for individual variability. Wherever information on inter- and intraspecies differences in pharmacokinetics was available, data-derived uncertainty factors were employed. Additional uncertainty factors were applied whenever necessary to account for the nature and severity of the observed effects and for the adequacy of the database. For most of the compounds considered, information on the dose/exposure-response relationship was provided, both to give policy makers clear guidelines on the possible impact of the pollutant at different exposure levels, and to permit an informed decision making process to take place. For some compounds, e.g. platinum, a guideline value was considered unnecessary as exposure through ambient air levels was considerably below the lowest level at which effects were seen. For other compounds, for example PM<sub>10</sub>, no threshold of effect could be found and therefore no guideline value could be derived. Instead, exposure-effect information highlighting the public health impact of different pollutant levels was provided.

In the updating process for carcinogens, a more flexible approach than in the 1987 Air Quality Guidelines was applied. Although, as a default approach, low-dose risk extrapolation was conducted for groups 1 and 2A, and an uncertainty factor approach applied in the case of agents in groups 2B and 3, the mechanism of action was the determining factor for the method of assessment. Hence, it was decided that compounds classified under 1 or 2A could be assessed using uncertainty factors, if evidence for a non-threshold mechanism of carcinogenicity existed. By way of contrast, compounds classified under 2B could be assessed by low-dose extrapolation methods, if a non-threshold mechanism of carcinogenicity in animals was proven. Flexibility was also given in terms of the choice of the extrapolation model, depending on the available data (including data for PBPK modelling). The linearized multistage model was used as a default approach. Besides providing unit-risk estimates in cases where low-dose risk extrapolation was conducted, levels associated with excess cancer risk of 1:10 000, 1:100 000 and 1:1 000 000 were calculated.

In evaluating ecotoxic effects of major air pollutants, the effects of O<sub>3</sub>, nitrogen-containing compounds and SO<sub>2</sub> on vegetation (crops, forests) were evaluated. Besides the deposition effects of nitrogen compounds, those of sulphates and total acidity were also evaluated. The principles applied were those developed by the Working Group on Effects under the Convention on Transboundary Air Pollution of the UNECE, and the evaluations were carried out jointly with that group. Critical levels and critical loads were derived. Critical levels are concentrations of pollutants in the atmosphere above which direct adverse effects on receptors such as plants, ecosystems or materials may occur. Critical loads represent quantitative estimates of an exposure, in the form of deposition, to one or more pollutants, below which significant harmful effects on specified sensitive elements of the environment will not occur.

### **2.4.3 Exposure-response relationships**

These guidelines place some emphasis on epidemiological data. Epidemiological studies are sometimes preferable to controlled exposure studies in that they provide information on responses in populations and on the effects of real exposures to pollutants and pollutant mixtures. However, the results of epidemiological studies are less easy to use than the results of controlled exposure studies in defining guidelines.

Most epidemiological studies relate responses to concentrations of pollutants, often measured



at single fixed site monitors. These data tell us little about the exposure-response relationships of individuals but, rather, tell us about the concentration-response relationship of the population studied. This relationship depends upon the pattern of exposure of the population considered and thus the relationship may vary from country-to-country. When the results of time-series studies on the effects of particles in the USA and Europe were compared, only small differences were seen (Wilson and Spengler 1996). But whether the differences were, in fact, due to differences in exposure patterns, or to differences in the toxicity of the ambient particle aerosol, or differences in the particle indices that were measured, remains unknown. Differences in response to air pollution may occur between developed and developing countries.

For both particles and O<sub>3</sub> an assumption of linearity was made when defining the exposure-response relationships included in the revised guidelines. Extrapolation beyond the available data is unwise, since there is evidence to suggest the exposure-response relationship may become less steep as ambient levels of particles rise (Schwartz and Marcus 1990; Lippmann and Ito 1995). For O<sub>3</sub>, the relationship at low concentrations may be concave upwards. These are important points to be considered if the guidelines are to be used in countries with levels of pollution different from the range covered by the guidelines.

#### **2.4.4 Moving from guidelines to standards**

An air quality standard is a description of a level of air quality that is adopted by a regulatory authority as enforceable. At its simplest, an air quality standard should be defined in terms of one or more concentrations and averaging times. In addition, other data should be added, including information on the form of exposure (e.g. outdoor), on monitoring which is relevant in assessing compliance with the standard, and on methods of data analysis, quality assurance and quality control.

In some countries the standard is further qualified by defining an acceptable level of attainment or compliance. Levels of attainment may be defined in terms of the fundamental units that define the standard. For example, if the unit defined by the standard is the day, then a requirement for 99% compliance allows the standard to be exceeded by three days a year. The cost of meeting any standard is likely to depend on the degree of compliance required. Consequently, it may be sensible to consider carefully the costs and benefits of different levels of compliance when deciding on the standard.

It is important to remember that the development of air quality standards is only a part of an adequate air quality management strategy (see Chapter 6). Legislation, identification of authorities responsible for enforcement of emission standards and penalties for exceedances are all also necessary. Emission standards may play an important role in the management strategy, especially if exceedance of air quality standards is used as a trigger for abatement measures. These may be needed at both the national and the local level.

Air quality standards are also important in informing the public about air quality. Used in this way they are a double-edged weapon as the public commonly assumes that once a standard is exceeded adverse effects on health will occur. This may not be the case, as discussed in Section 2.4.2.

#### 2.4.5 Factors to be considered in setting an air quality standard

The process of setting standards is simplified when the WHO *Guidelines* provide a guideline value. In general, local review of the health effects database may be unnecessary. However, when published studies on associations between air pollutants and health effects in the local region are available, it is prudent for the authorities responsible for setting national standards to give them due consideration in their evaluation of the applicability of the WHO *Guidelines for Air Quality*. If no single value is offered but rather a Unit Risk estimate, or a concentration-response relationship is defined, then the following should be considered in setting standards:

The nature of the effects indicated should be examined and decisions made as to whether they represent adverse health effects.

Special populations at risk should be considered.

Sensitive populations or groups are defined here as those impaired by concurrent disease or other physiological limitations and those with specific characteristics that make the health consequences of exposure more significant (e.g. developmental phase in children). In addition, other groups may be judged to be at special risk because of their exposure patterns and because the effective dose for a given exposure may be increased, as in the case of children for example. The sensitive populations may vary between countries due to differences in the number of people with inadequate access to medical care, in the prevalence of certain endemic diseases, in the prevailing genetic factors, or in the prevalence of debilitating diseases or nutritional deficiencies. The regulator needs to decide which specific groups at risk should be protected by the standards.

These factors have been considered in the development of these guidelines and have been included when a guideline value has been offered.

The WHO Guideline for SPM was developed to address the health effects associated with exposures to particulate matter released into the ambient outdoor environment, as well as the secondary ambient particulate matter found in the atmosphere from gaseous precursors (e.g. sulphate, nitrate, and the organic products of photochemical reaction sequences). The exposures take place in the outdoor air and in indoor microenvironments following infiltration of the particles into occupied indoor spaces. The numerical effects relationships described in the *Guideline* were based on size-selective mass concentration data that were obtained from numerous, and generally consistent, study results for urban population in North and South America and Europe. However, the transfer of these relationships to other parts of the world should be conducted with caution for several reasons. These include:

1. The chemical composition of the particles may be substantially different in the nation developing the air quality standard, when compared with the regions in which the community studies were conducted and which contributed to the development of the guideline. Mass concentration in selected particle size ranges (i.e. PM<sub>10</sub> and/or PM<sub>2.5</sub>) is, at best, a surrogate index for the biologically active components in the mixture. The mixture in the communities studied in the development of the guideline was dominated by primary and secondary effluents from motor vehicles, central station power generation, and space heating by natural gas and light oil combustion. The mixtures in communities in less developed countries may be different. They may be dominated by the effluents of inefficient combustion units and wind-blown soil, with quite different toxic properties from those in the studies

used by WHO.

2. The particle concentration range may be substantially different.  
The WHO response-concentration relationships for particulate matter are based on a linear model of response, which is a suitable approximation within the range of particle concentrations typically found in the studies used by WHO. However, it is well established that the coefficient tends to decrease toward the upper end of the concentration range. In addition, the slope established for the lower concentrations cannot reliably be used to predict responses at the higher mass concentration levels that may be observed in urban areas in less developed countries.
3. The responsiveness of the population may be substantially different.  
The WHO response-concentration relationships were based on responses of populations that were mostly well nourished and who had access to modern health services. By contrast, the populations exposed to higher concentrations of particles in less developed countries are likely to have lower quality nutrition and health care. Alternatively, they may well be a hardy survivor population with fewer people in a fragile condition of health. It is currently unclear whether the responsiveness of the populations in other parts of the world differ from those studies in North and South America and Europe.

For these reasons, the WHO response-concentration relationships should be used with caution as predictors of health impacts in less developed countries. In particular, the relationships should not be extrapolated to concentrations beyond the ranges given in Figures 3.6 to 3.8.

#### **2.4.6 Uncertainty factors**

In development of these guidelines, the size of uncertainty factors applied to published data in deriving a guideline was considered to be a matter for expert judgement, rather than prescription (WHO 1987). Where the database was strong, smaller uncertainty factors were used than where the database was weak. Database strength depends upon the availability of published studies relevant to the circumstances of a country for which the guidelines are intended. In moving from guidelines to country-specific standards, the size of the uncertainty factors may require revision.

Impact assessment or risk assessment plays an important part in setting standards. This will depend on exposure and an assessment of population exposure will be required. In considering the appropriate form of exposure assessment needed attention should be paid to the database from which the guideline was derived.

Acceptability of risk varies from country to country and is in part dependent on social conditions, priorities and on the other risks to which a population is exposed. In some countries a risk that would be unacceptable elsewhere might be considered small.

#### **2.4.7 Cost-benefit analysis and other factors**

The costs of reducing levels of air pollution should be weighed against the benefits produced. Cost-benefit analysis is one way of formally setting out this process, and it uses money as a common currency for costs and benefits.

The concept is that pollutant concentrations are reduced at least until the associated costs and

benefits are balanced: more strictly, emissions are reduced until the marginal costs and benefits are equal. While the cost of abatement measures may be relatively easy to quantify, this may not be the case when non-technical measures are employed. In any case, it is likely to be more difficult to assign monetary values to the benefits obtained. Some aspects of reduced morbidity, such as a reduction in the use of hospital facilities and drugs, are comparatively easy to cost; others such as reductions in premature deaths and symptoms are not. Applying monetary values based on a "willingness to pay" basis has been suggested, and has been accepted as appropriate by many health economists. This approach has been seen as preferable to one based only on such indices as loss of production, earnings or hospital expenses. Cost-benefit analysis is discussed in detail in Section 7.9.

In practice the strict theoretical precepts of cost-benefit analysis should be supplemented by broader social and economic considerations. This process is sometimes described as "Stakeholder Input". Stakeholders are defined as those who have an interest in the outcome of a decision making process. The aim is to ensure as far as possible social equity and fairness to all involved parties. An adequate and early involvement of all concerned stakeholders will increase the transparency of the process and is likely to increase the acceptability of the outcome.

Factors other than monetary concerns also need to be evaluated when considering the setting of national air quality standards. These include the technical capacity of a country to achieve and maintain an air quality within the desired standards; the social implications of adopting certain standards to ensure an equity of costs and benefits among the population; and environmental costs and benefits.

### 3. Health-based Guidelines

In this chapter the key air pollutants, also termed “classic” air pollutants - SO<sub>2</sub>, NO<sub>2</sub>, CO, O<sub>3</sub>, SPM and lead - are briefly described with respect to health risk evaluations and recommended guideline values. Particular emphasis is given to PM<sub>10</sub> and PM<sub>2.5</sub>. The information available for a number of other air pollutants (including inorganic compounds, organic volatile components and certain indoor air pollutants such as radon) is also summarized and presented in a synoptic table. These sections are based upon papers prepared for the updating of the *Air Quality Guidelines for Europe* (WHO 1999a) and exposure information obtained from various regions. A third section considers factors, such as altitude, humidity, temperature, nutritional status, health status, vulnerability etc., that affect the actual health impact of air pollutants on the individual and vulnerable groups.

#### 3.1 Key air pollutants

##### Sulphur dioxide

*Short-period exposures (less than 24 hours)*

Most information on the acute effects of SO<sub>2</sub> comes from controlled chamber experiments on volunteers exposed to SO<sub>2</sub> for periods ranging from a few minutes up to one hour (WHO 1999a). Acute responses occur within the first few minutes after commencement of inhalation. Further exposure does not increase effects. Effects include reductions in the mean forced expiratory volume over one second (FEV<sub>1</sub>), increases in specific airway resistance (sRAW), and symptoms such as wheezing or shortness of breath. These effects are enhanced by exercise that increases the volume of air inspired, as it allows SO<sub>2</sub> to penetrate further into the respiratory tract.

A wide range of sensitivity has been demonstrated, both among normal subjects and among those with asthma. People with asthma are the most sensitive group in the community. Continuous exposure-response relationships, without any clearly defined threshold, are evident. To develop a guideline value, the minimum concentrations associated with adverse effects in asthmatic patients exercising in chambers have been considered. An example of an exposure-response relationship for asthmatic patients is shown in Figure 3.1, expressed in terms of change in FEV<sub>1</sub> after a 15-minute exposure.

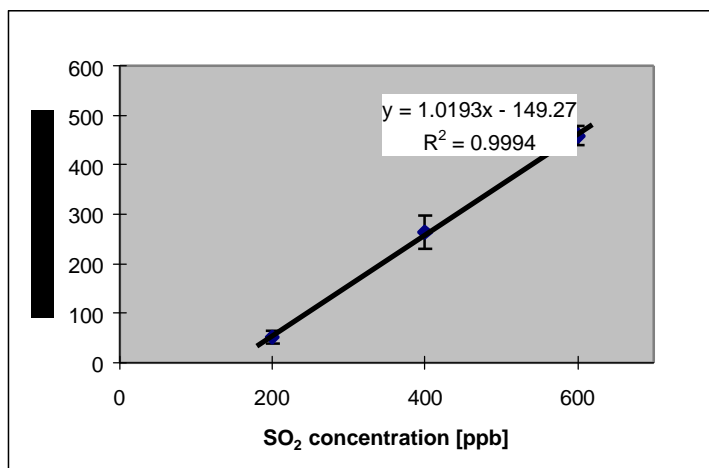
*Exposure over a 24-hour period*

Information on the effects of exposure averaged over a 24-hour period is derived mainly from epidemiological studies in which the effects of SO<sub>2</sub>, SPM and other associated pollutants are considered. Exacerbation of symptoms among panels of selected sensitive patients seems to arise in a consistent manner when the concentration of SO<sub>2</sub> exceeds 250 µg/m<sup>3</sup> in the presence of SPM. Several more recent studies in Europe have involved mixed industrial and vehicular emissions now common in ambient air. At low levels of exposure (mean annual levels below 50 µg/m<sup>3</sup>; daily levels usually not exceeding 125 µg/m<sup>3</sup>) effects on mortality (total, cardiovascular and respiratory) and on hospital emergency admissions for total respiratory causes and chronic obstructive pulmonary disease (COPD), have been consistently demonstrated. These results have been shown, in some instances, to persist when black smoke and SPM levels were

controlled for, while in others no attempts have been made to separate the pollutant effects. In these studies no obvious threshold levels for SO<sub>2</sub> has been identified.

### *Long-term exposure*

Earlier assessments examined findings on the prevalence of respiratory symptoms, respiratory illness frequencies, or differences in lung function values in localities with contrasting concentrations of SO<sub>2</sub> and SPM, using data from the coal-burning era in Europe. The lowest-observed-adverse-effect level of SO<sub>2</sub> was judged to be at an annual average of 100 µg/m<sup>3</sup>, when present with SPM. More recent studies related to industrial sources of SO<sub>2</sub>, or to the changed urban mixture of air pollutants, have shown adverse effects below this level. But a major difficulty in interpretation is that long-term effects are liable to be affected not only by current conditions, but also by the qualitatively and quantitatively different pollution of earlier years. However, cohort studies on differences in mortality between areas with contrasting pollution levels indicate that mortality is more closely associated with SPM, than with SO<sub>2</sub>.



**Figure 3.1** Mean change of FEV<sub>1</sub> in asthmatics with changing SO<sub>2</sub> concentrations

### *Guidelines*

Based upon controlled studies with asthmatics exposed to SO<sub>2</sub> for short periods, it is recommended that a value of 500 µg/m<sup>3</sup> (0.175 ppm) should not be exceeded over averaging periods of 10 minutes. Because exposure to sharp peaks depends on the nature of local sources, no single factor can be applied to estimate corresponding guideline values over longer periods, such as an hour. Day-to-day changes in mortality, morbidity, or lung function related to 24-hour average concentrations of SO<sub>2</sub> are necessarily based on epidemiological studies, in which people are in general exposed to a mixture of pollutants; and guideline values for SO<sub>2</sub> have previously been linked with corresponding values for SPM. This approach led to a previous guideline 24-hour average value of 125 µg/m<sup>3</sup> (0.04 ppm) for SO<sub>2</sub>, after applying an uncertainty factor of two to the lowest-observed-adverse-effect level. In more recent studies, adverse effects with significant public health importance have been observed at much lower levels of exposure. However, there is still uncertainty as to whether SO<sub>2</sub> is the pollutant responsible for the observed adverse effects, or whether it is a surrogate for SPM with diameters below 10 µm or 2.5µm, or even for some other correlated substance. There is no basis for numerical changes of the 1987 guideline values for SO<sub>2</sub> and thus 125 µg/m<sup>3</sup> for an averaging time of 24 hours and 50 µg/m<sup>3</sup> as an annual mean are recommended. However, the current guideline values are no longer linked

with SPM.

## **Nitrogen dioxide**

### ***Short-term exposure effects***

Available data from animal toxicology experiments indicate that acute exposure to NO<sub>2</sub> concentrations of less than 1880 µg/m<sup>3</sup> (1 ppm) rarely produce observable effects. Normal healthy humans, exposed at rest or with light exercise for less than two hours to concentrations above 4700 µg/m<sup>3</sup> (2.5 ppm), experience pronounced decreases in pulmonary function; generally, normal subjects are not affected by concentrations less than 1880 µg/m<sup>3</sup> (1.0 ppm). One study showed that the lung function of subjects with chronic obstructive pulmonary disease is slightly affected by a 3.75-hour exposure to 560 µg/m<sup>3</sup> (0.3 ppm).

A wide range of findings in asthmatics has been reported. Asthmatics are likely to be the most sensitive subjects, although uncertainties exist in the health database. The lowest concentration causing effects on pulmonary function was reported from two laboratories that exposed mild asthmatics for 30-110 minutes to 565 µg/m<sup>3</sup> (0.3ppm) NO<sub>2</sub> during intermittent exercise. However, neither of these laboratories was able to replicate these responses with a larger group of asthmatic subjects. One of these studies indicated that NO<sub>2</sub> can increase airway reactivity to cold air in asthmatic subjects. At lower concentrations, the pulmonary function of asthmatics was not changed significantly.

NO<sub>2</sub> increases bronchial reactivity, as measured by the response of normal and asthmatic subjects following exposure to pharmacological bronchoconstrictor agents, even at levels that do not affect pulmonary function directly in the absence of a bronchoconstrictor. Some, but not all, studies show increased responsiveness to bronchoconstrictors at NO<sub>2</sub> levels as low as 376-565 µg/m<sup>3</sup> (0.2 to 0.3 ppm); in other studies, higher levels had no such effect. Because the actual mechanisms of effect are not fully defined and NO<sub>2</sub> studies with allergen challenges showed no effects at the lowest concentration tested (188 µg/m<sup>3</sup>; 0.1 ppm), full evaluation of the health consequences of the increased responsiveness to bronchoconstrictors is not yet possible. Recent studies have shown an increased reactivity to natural allergens in the same concentration range. The results of repetitive exposures of such individuals, or the impact of single exposures on more severe asthmatics, are not known.

### ***Long-term exposure effects***

Studies with animals have clearly shown that several weeks to months of exposure to NO<sub>2</sub> concentrations of less than 1880 µg/m<sup>3</sup> (1ppm) causes a range of effects, primarily in the lung, but also in other organs such as the spleen and liver, and in blood. Both reversible and irreversible lung effects have been observed. Structural changes range from a change in cell type in the tracheobronchial and pulmonary regions (at a lowest reported level of 640 µg/m<sup>3</sup>), to emphysema-like effects. Biochemical changes often reflect cellular alterations, with the lowest effective NO<sub>2</sub> concentrations in several studies ranging from 380-750µg/m<sup>3</sup>.

NO<sub>2</sub> levels of about 940 µg/m<sup>3</sup> (0.5ppm) also increase susceptibility to bacterial and viral infection of the lung. There are no epidemiological studies that can be confidently used to quantify a long-term NO<sub>2</sub> exposure or concentration likely to be associated with the induction of unacceptable health risks in children or adults. Homes with gas cooking appliances have peak levels of NO<sub>2</sub> in the same range as levels causing effects in some animal and human clinical

studies. Epidemiological studies evaluating the effects of NO<sub>2</sub> exposures in such homes have been conducted. In general, epidemiological studies of adults and infants (less than 2 years old) show no significant effect of the use of gas cooking appliances on respiratory illness; nor do the few available studies of infants and adults show any associations between pulmonary function changes and gas stove use. However, children 5-12 years old are estimated to have a 20% increased risk for respiratory symptoms and disease for each increase of 28 µg/m<sup>3</sup> NO<sub>2</sub> (2-week average), where the weekly average concentrations are in the range of 15-128 µg/m<sup>3</sup> or possibly higher. However, the observed effects cannot clearly be attributed to either the repeated short-term high level peak, or to long-term exposures in the range of the stated weekly averages (or possibly both).

The results of outdoor studies consistently indicate that children with long-term ambient NO<sub>2</sub> exposures exhibit increased respiratory symptoms that are of longer duration, and show a decrease in lung function. However, outdoor NO<sub>2</sub> epidemiological studies, as with indoor studies, provide little evidence that long-term ambient NO<sub>2</sub> exposures are associated with health effects in adults. None of the available studies yields confident estimates of long-term exposure-effect levels, but available results most clearly suggest respiratory effects in children at annual average NO<sub>2</sub> concentrations in the range of 50-75 µg/m<sup>3</sup> or higher.

### ***Guidelines***

Despite the large number of acute controlled exposure studies in humans, several which used multiple concentrations, there is no evidence for a clearly defined concentration-response relationship for NO<sub>2</sub> exposure. For acute exposures, only very high concentrations (>1,000 ppb; 1,990 µg/m<sup>3</sup>) affect healthy people. Based on small changes in lung function, often less than a 5% drop in FEV<sub>1</sub> with NO<sub>2</sub> exposure, and changes in airway responsiveness in studies on asthmatics and patients with chronic obstructive pulmonary disease, a range of 365-565 µg/m<sup>3</sup> (0.20 to 0.30 ppm) 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 with exposure to 188 µg/m<sup>3</sup>, and because of a meta-analysis suggesting changes in airway responsiveness below 365 µg/m<sup>3</sup>. However, the significance of the response at 188 µg/m<sup>3</sup> has been questioned on the basis of an inappropriate statistical analysis and a failure to replicate the findings. Based on these human clinical data, a one-hour guideline of 200 µg/m<sup>3</sup> is proposed. At double this recommended guideline (400 µg/m<sup>3</sup>), there is evidence to suggest possible small effects in pulmonary function of asthmatics. Should the asthmatic be exposed either simultaneously or sequentially to NO<sub>2</sub> and an aero-allergen, the risk of an exaggerated response to the allergen is increased.

Although there is no particular study or set of studies that clearly supports selection of a specific numerical value for an annual average guideline, there is need to protect the public from chronic NO<sub>2</sub> exposures. Based on the studies reviewed, it is not currently possible to select a well-supported value; but a previous review on NO<sub>2</sub> recommended an annual value of 40 µg/m<sup>3</sup> (WHO 1997c). In the absence of support for an alternative value, this figure is recognized as an air quality guideline.

### **Carbon monoxide**

CO diffuses rapidly across alveolar, capillary and placental membranes. Approximately 80-90 % of the absorbed CO binds with hemoglobin to form carboxyhemoglobin (COHb), which is a



specific biomarker of exposure in blood. The affinity of hemoglobin for CO is 200-250 times that for oxygen. During exposure to a fixed concentration of CO, the COHb concentration increases rapidly at the onset of exposure, starts to level off after 3 hours, and reaches a steady-state after 6-8 hours of exposure. It is noted that the elimination half-life in the fetus is much longer than in the pregnant mother.

The binding of CO with hemoglobin to form COHb reduces the oxygen-carrying capacity of the blood and impairs the release of oxygen from hemoglobin. These are the main causes of tissue hypoxia produced by CO at low exposure levels. At higher concentrations, the rest of the absorbed CO binds with other heme proteins such as myoglobin and with cytochrome oxidase and cytochrome P-450. The toxic effects of CO first become evident in organs and tissues with high oxygen consumption, such as the brain, heart, exercising skeletal muscle and the developing fetus.

Severe hypoxia due to acute CO poisoning may cause both reversible, short-lasting, neurological deficits and severe, often delayed, neurological damage. The neurobehavioural effects include impaired coordination, tracking, driving ability, vigilance and cognitive performance at COHb levels as low as 5.1-8.2%.

In apparently healthy subjects, the maximal exercise performance decreases at COHb levels as low as 5%. The regression between the percentage decrease in maximal oxygen consumption and the percentage increase in COHb concentration appears to be linear, with a fall in oxygen consumption of approximately 1% for each 1% rise in COHb level above 4%.

In controlled studies involving patients with documented coronary artery disease, mean pre-exposure COHb levels of 2.9-5.9% (corresponding to post-exercise COHb levels of 2.0-5.2%) have been associated with a significant shortening in the time to onset of angina, with increased electrocardiographic changes and with impaired left ventricular function during exercise. In addition, ventricular arrhythmias may be increased significantly at the higher range of mean post-exercise COHb levels. Epidemiological and clinical data indicate that CO from smoking and environmental or occupational exposures may contribute to cardiovascular mortality and to the early course of myocardial infarction. Current data from epidemiological studies and experimental animal studies indicate that common environmental exposures to CO in the developed world would not have atherogenic effects on humans (WHO 1999a).

During pregnancy, endogenous production of CO is increased so that maternal COHb levels are usually about 20% higher than the non-pregnant values. At steady-state, the fetal COHb levels are as much as 10-15% higher than the maternal COHb levels. There is a well-established and probably causal relationship between maternal smoking and low birth weight at fetal COHb levels of 2-10%. In addition, maternal smoking seems to be associated with a dose-dependent increase in perinatal deaths and with behavioural effects in infants and young children.

### ***Guidelines***

Endogenous production of CO results in COHb levels of 0.4-0.7% in healthy subjects. During pregnancy, elevated maternal COHb levels of 0.7-2.5% have been reported, mainly due to increased endogenous production. The COHb levels in non-smoking general populations are usually 0.5-1.5% due to endogenous production and environmental exposures. Non-smoking people in certain occupations (car drivers, policemen, traffic wardens, garage and tunnel workers, firemen etc.) can have long-term COHb levels up to 5%, and heavy cigarette smokers have

COHb levels up to 10% (WHO 1999a). Well-trained subjects engaging in heavy exercise in polluted indoor environments can increase their COHb levels quickly up to 10-20%. Epidemic CO poisonings in indoor ice arenas have been reported.

To protect non-smoking, middle-aged and elderly population groups with documented or latent coronary artery disease from acute ischemic heart attacks, and to protect fetuses of non-smoking pregnant mothers from untoward hypoxic effects, a COHb level of 2.5% should not be exceeded.

The guideline values (ppm values rounded), and periods of time-weighted average exposures, have been determined in such a way that the COHb level of 2.5% is not exceeded, even when a normal subject engages in light or moderate exercise. The guideline values for CO are 100 mg/m<sup>3</sup> (90 ppm) for 15 minutes, 60 mg/m<sup>3</sup> (50 ppm) for 30 minutes, 30 mg/m<sup>3</sup> (25 ppm) for 1 hour, and 10 mg/m<sup>3</sup> (10 ppm) for 8 hours.

### **Ozone and other photochemical oxidants**

O<sub>3</sub> toxicity occurs in a continuum in which higher concentrations, longer exposure duration, and greater activity levels during exposure cause greater effects. Short-term acute effects include pulmonary function changes, increased airway responsiveness and airway inflammation, and other symptoms. These health effects are statistically significant at 160 µg/m<sup>3</sup> (0.08 ppm) for 6.6 hour exposures in a group of healthy exercising adults, with the most sensitive subjects experiencing a more than 10% functional decrease within 4-5 hours. Controlled exposure of heavily exercising adults, or children to an O<sub>3</sub> concentration of 240 µg/m<sup>3</sup> (0.12 ppm) for 2 hours, also produced decreases in pulmonary function. There is no question that substantial acute adverse effects occur during exercise with one hour exposure to concentrations of 500 µg/m<sup>3</sup> or higher, particularly in susceptible individuals or subgroups.

Field studies in children, adolescents, and young adults have indicated that pulmonary function decrease can occur as a result of short term exposure to O<sub>3</sub> concentrations in the range 120-240 µg/m<sup>3</sup> and higher. Mobile laboratory studies have observed changes in pulmonary function in children or asthmatics exposed to O<sub>3</sub> concentrations of 280-340 µg/m<sup>3</sup> (0.14-0.17 ppm) for several hours. Respiratory symptoms, especially coughing, have been associated with O<sub>3</sub> concentrations as low as 300 µg/m<sup>3</sup> (0.15 ppm). O<sub>3</sub> exposure has also been reported to be associated with increased respiratory hospital admissions and exacerbation of asthma. The effects are observed with exposures to ambient O<sub>3</sub> (and co-pollutants) and with controlled exposures to O<sub>3</sub> alone. This demonstrates that the functional and symptomatic responses can be attributed primarily to O<sub>3</sub>.

A number of studies evaluating animals (rats and monkeys) exposed to O<sub>3</sub> for a few hours or days have shown alterations in the respiratory tract, in which the lowest-observed-effect levels were in the range of 160-400 µg/m<sup>3</sup> (0.08-0.2 ppm). These included the potentiation of bacterial lung infections, inflammation, morphological alterations in the lung, increases in the function of lung enzymes active in oxidant defenses, and increases in collagen content. Long-term exposure to O<sub>3</sub> in the range of 240-500 µg/m<sup>3</sup> (0.12 to 0.25 ppm) causes morphological changes in the epithelium and interstitium of the centri-acinar region of the lung, including fibrotic changes.

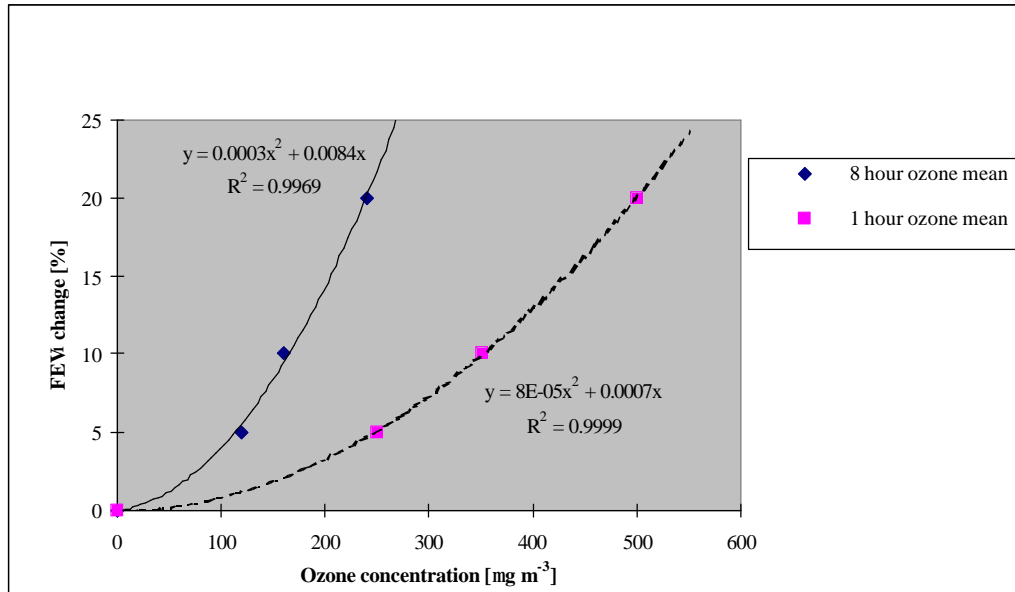
## ***Guidelines***

Establishing guidelines for ambient O<sub>3</sub> concentrations is complicated by the fact that detectable responses occur at, or close to, the upper bounds of background concentrations. Thus it is not possible to base the guidelines on a no-observed-adverse-effect level (NOAEL) or LOAEL. At O<sub>3</sub> levels of 200 µg/m<sup>3</sup> and lower (for 1-8 hour exposure periods), there are statistically significant decreases in lung function, airway inflammatory changes, exacerbation of respiratory symptoms, and symptomatic and functional exacerbation of asthma in susceptible people during exercise. Functional changes and symptoms, as well as increased hospital admissions for respiratory causes, are also observed in population studies.

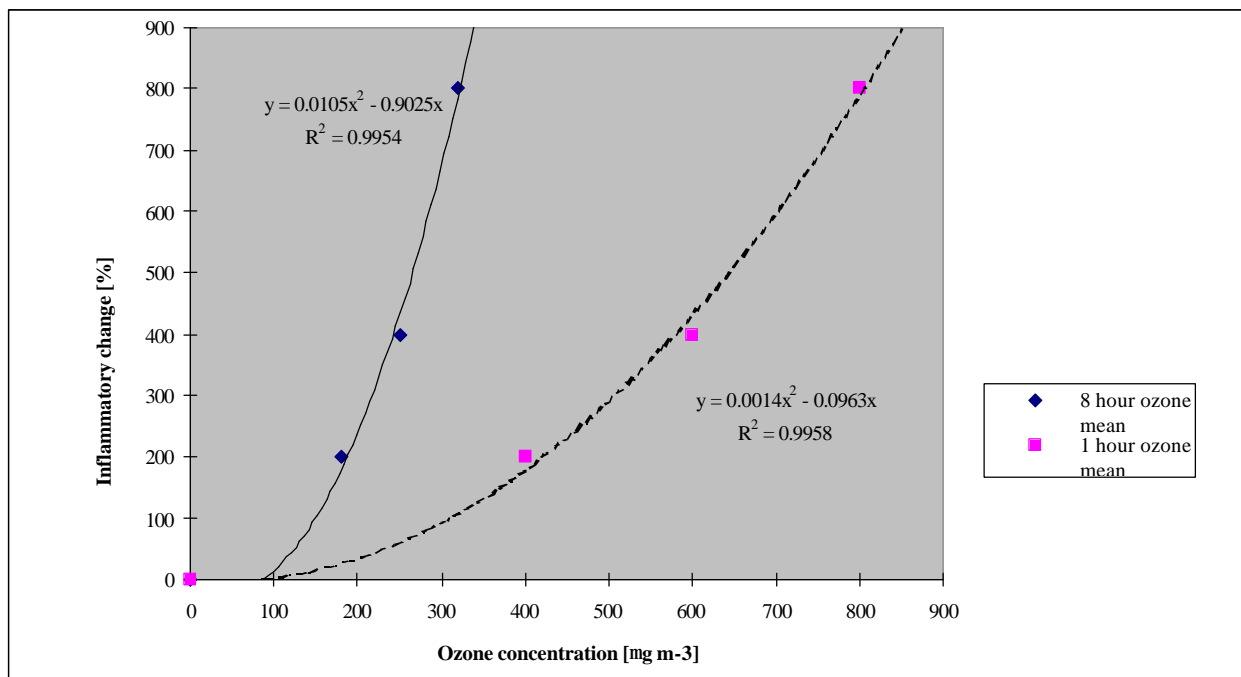
To select a guideline, one must accept the premise that some detectable functional responses are of little or no health concern, and that too few people may respond to the effects of O<sub>3</sub> exposure to warrant designation as a group needing protection from exposure to ambient O<sub>3</sub>. In the case of respiratory function responses, a judgement could be made that O<sub>3</sub>-related reductions of FEV<sub>1</sub> at, for example, less than 10% were of no clinical concern. The balance of evidence indicates that reductions of FEV<sub>1</sub> of more than 10% occurred at O<sub>3</sub> levels of 160 µg/m<sup>3</sup> and higher. It is generally accepted that the exposure duration to O<sub>3</sub> is important in controlling the response and that exposures to raised concentrations for periods of eight hours are not unlikely. On this basis, a guideline value for ambient air of 120 µg/m<sup>3</sup> for a maximum period of eight hours per day has been established as a level at which acute effects on public health are likely to be small.

For those public health authorities that cannot accept such levels of health risk, an alternative is to explicitly select some other level of acceptable exposure and associated risk using the dose response relationships given in Figures 3.2-3.5. These figures, which are based on corresponding tables in the *Air Quality Guidelines for Europe* (WHO 1999a), summarize the ambient O<sub>3</sub> concentrations that are associated with levels of responses among population subgroups. Although chronic exposure to O<sub>3</sub> may cause effects, quantitative information from humans is inadequate for estimating the degree of protection from chronic effects offered by these *Guidelines*. In any case, the O<sub>3</sub> concentration at which any adverse health outcome is expected will vary with the duration of the exposure and with the volume of air inhaled during the exposure. As there is a strong correlation in field studies between the one-hour and eight-hour O<sub>3</sub> concentration and hospital admissions (Figure 3.5), the reduction in health risk associated with decreasing one-hour or eight-hour O<sub>3</sub> levels should be very similar.

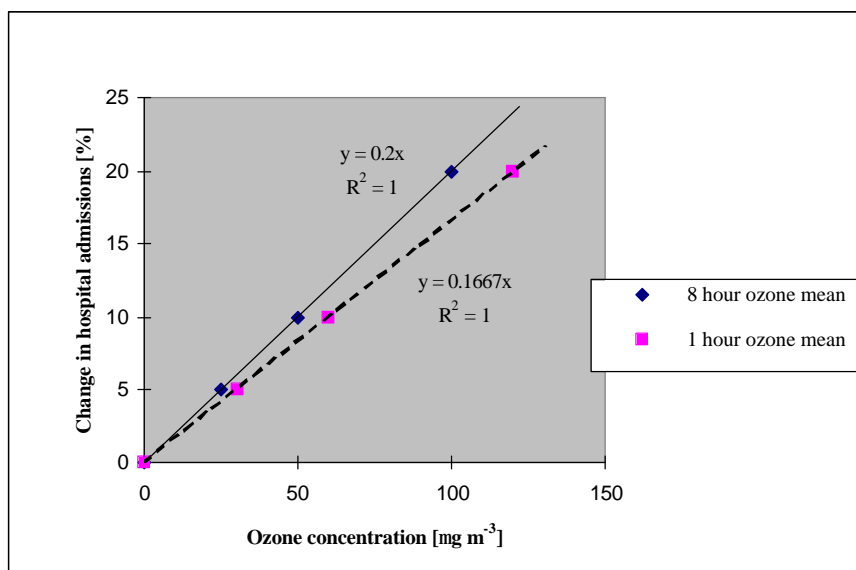
Thus, the amount of time spent outdoors and the typical level of activity are factors which should be considered in risk evaluation. Figures 3.2 and 3.5 summarize the O<sub>3</sub> levels at which two representative adverse health outcomes, based on controlled exposure experiments, may be expected. The dose-response relationships in these figures represent expert judgment based on the collective evidence from numerous studies and linear extrapolation in a few cases where data were limited. Interestingly, these dose-response relationships appear to be non linear.



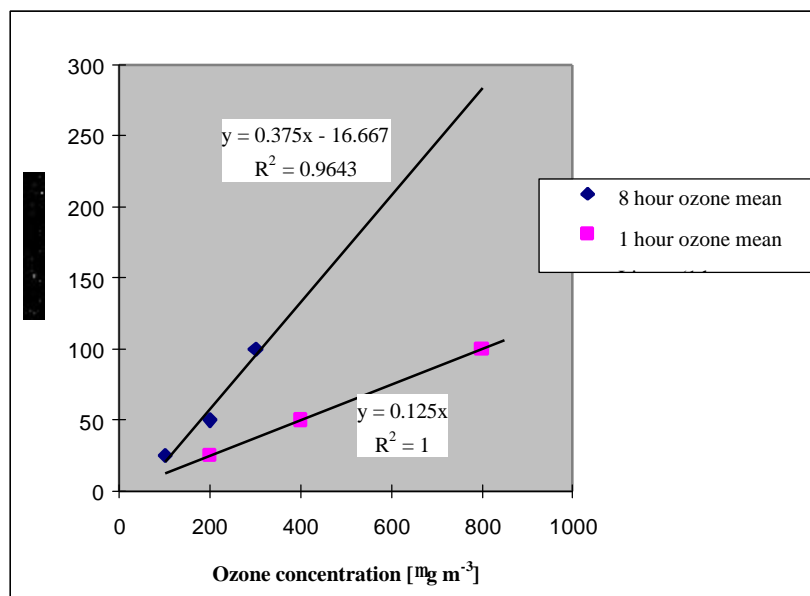
**Figure 3.2. Change in FEV<sub>1</sub> as a function of O<sub>3</sub> concentration in the most sensitive 10% of active young adults and children.**



**Figure 3.3. Inflammatory change (neutrophil influx in lungs of healthy young adults exercising outdoors at more than 40l/min expiratory volume in the lung) as a function of O<sub>3</sub> concentration.**



**Figure 3.4.** Increase in hospital admissions for respiratory conditions as a function of  $\text{O}_3$  concentration.



**Figure 3.5.** Change in symptom exacerbation among adults and asthmatics as a function of  $\text{O}_3$  concentration.

Epidemiological data show relationships between changes in various health outcomes and changes in the peak daily ambient  $\text{O}_3$  concentration. Two examples of such relationships are shown in Figures 3.4 and 3.5. Short-term increases in levels of ambient  $\text{O}_3$  are associated both with increased hospital admissions with a respiratory diagnosis, and with respiratory symptom exacerbation in healthy people and asthmatics. These observations may be used to quantify the expected improvements in health outcomes that may be associated with a lower ambient  $\text{O}_3$  concentration. The dose-response relationships presented in Figures 3.4 and 3.5 assume a linear relationship between  $\text{O}_3$  concentration and health outcome. However, uncertainties exist concerning the forms of these relationships and it is unclear whether similar response slopes can be expected at widely different ambient  $\text{O}_3$  levels. In the event that such relationships are curvilinear (i.e., concave upwards), the benefits of lowering the  $\text{O}_3$  concentration are likely to

be greater when the average ambient level is higher. Conversely, if the ambient O<sub>3</sub> concentration is already low, the benefits of lowering the concentration may be less than would be suggested by these figures. Another important area of uncertainty is the degree to which other pollutants influence these relationships.

The previous WHO guidelines (WHO 1987) included a one-hour guideline value of 150-200 µg/m<sup>3</sup> for O<sub>3</sub>. Although recent research does not indicate that this guideline would necessarily be erroneous, the 8-hour guideline would protect against acute one-hour exposures in this range and thus it is concluded that a one-hour guideline value would not be necessary. The health problems of greatest concern are increased hospital admissions, exacerbation of asthma, inflammatory changes in the lung and structural alterations in the lung. These are more appropriately addressed by a guideline value which limits average daily exposure, and consequently inhaled dose and dose rate, rather than addressing the rare short duration deterioration of air quality that may be associated with unusual meteorological conditions.

A guideline for PAN is not warranted at present, as it does not seem to pose a significant health problem at levels that are observed in the environment.

### *Suspended particulate matter*

Health effects of SPM in humans depend on particle size and concentration, and can fluctuate with daily fluctuations in PM<sub>10</sub> or PM<sub>2.5</sub> levels. They include acute effects such as increased daily mortality, increased rates of hospital admissions for exacerbation of respiratory disease, fluctuations in the prevalence of bronchodilator use and cough and peak flow reductions. Long-term effects of SPM refer also to mortality and respiratory morbidity, but only few studies on the long-term effects of SPM exist. Air pollution by particulate matter has been considered to be primarily an urban phenomenon, but it is now clear that in many areas of developed countries, urban-rural differences in PM<sub>10</sub> are small or even absent, indicating that PM exposure is widespread. This is not to imply that exposure to primary, combustion-related PM may not be higher in urban areas.

A variety of methods exist to measure different fractions of particulate matter in air, with different health significance (see Section 2.1.1). This evaluation has tended to focus on studies in which PM exposure was expressed as PM<sub>10</sub> and PM<sub>2.5</sub>. Health effect studies conducted with various TSP and BS as exposure indicators have provided valuable additional information. However, they are less suitable for deriving exposure-response relationships for PM because TSP includes particles that are too large to be inhaled, or because the health significance of particle opacity as measured by the Black Smoke method is uncertain. Methods for measuring particle concentrations are discussed in section 5.7.

The current time-series epidemiological studies are unable to define a threshold below which no effects occur. Recent studies suggest that even at low levels of PM (less than 100 µg/m<sup>3</sup>), short-term exposure is associated with health effects. At low levels of PM<sub>10</sub> (0 - 100 µg/m<sup>3</sup>), the short-term exposure-response curve fits a straight line reasonably well (Figures 3.6 to 3.8). However, there are indications from several studies that at higher levels of exposure (several hundreds of µg/m<sup>3</sup> of PM<sub>10</sub>), at least for effects on mortality, the curve is flatter than at low levels of exposure. This is discussed later in this section.

Although many studies have obtained acute effect estimates for PM<sub>10</sub> that are reasonably

consistent, this does not imply that particle composition or size distribution within the  $PM_{10}$  fraction is unimportant. Limited evidence from studies on dust storms indicates that such  $PM_{10}$  particles are much less toxic than those associated with combustion sources. Recent studies in which  $PM_{10}$  size fractions and/or constituents have been measured suggest that the observed effects of  $PM_{10}$  are largely associated with fine particles and not with the coarse fraction ( $PM_{10}$  minus  $PM_{2.5}$ ). In some areas strong aerosol acidity or sulphate may be the cause of the effects associated with  $PM_{2.5}$ .

Evidence is also emerging that long-term exposure to low concentrations of PM in air is associated with mortality and other chronic effects, such as increased rates of bronchitis and reduced lung function. Two cohort studies conducted in the U.S.A. suggest that life expectancy may be 2-3 years shorter in communities with high PM than in communities with low PM. This is consistent with earlier cross-sectional studies, which compared age-adjusted mortality rates across a range of long-term average PM concentrations. The results showed that long-term average exposures to low PM levels, starting at about  $10 \mu g/m^3$  of fine particulate matter, were associated with a reduction in life expectancy. Whilst such observations require further corroboration, preferably also from other areas in the world, these new studies suggest that the public health implications of PM exposure may be large.

Figures 3.6-3.8 show summary estimates of the relative increase in various health parameters as a function of PM concentration. These figures are based on data reported in studies in which  $PM_{10}$  and/or  $PM_{2.5}$  have been measured. They were not inferred from other measures such as Coefficient of Haze, Black Smoke or SPM. The database for parameters other than  $PM_{10}$  is still limited, so the evaluation of health effects, especially the short-term effects, is largely expressed in terms of  $PM_{10}$ . However, future regulations and monitoring activities should give emphasis to the ultrafine and fine fractions in addition to, or even instead of,  $PM_{10}$ .

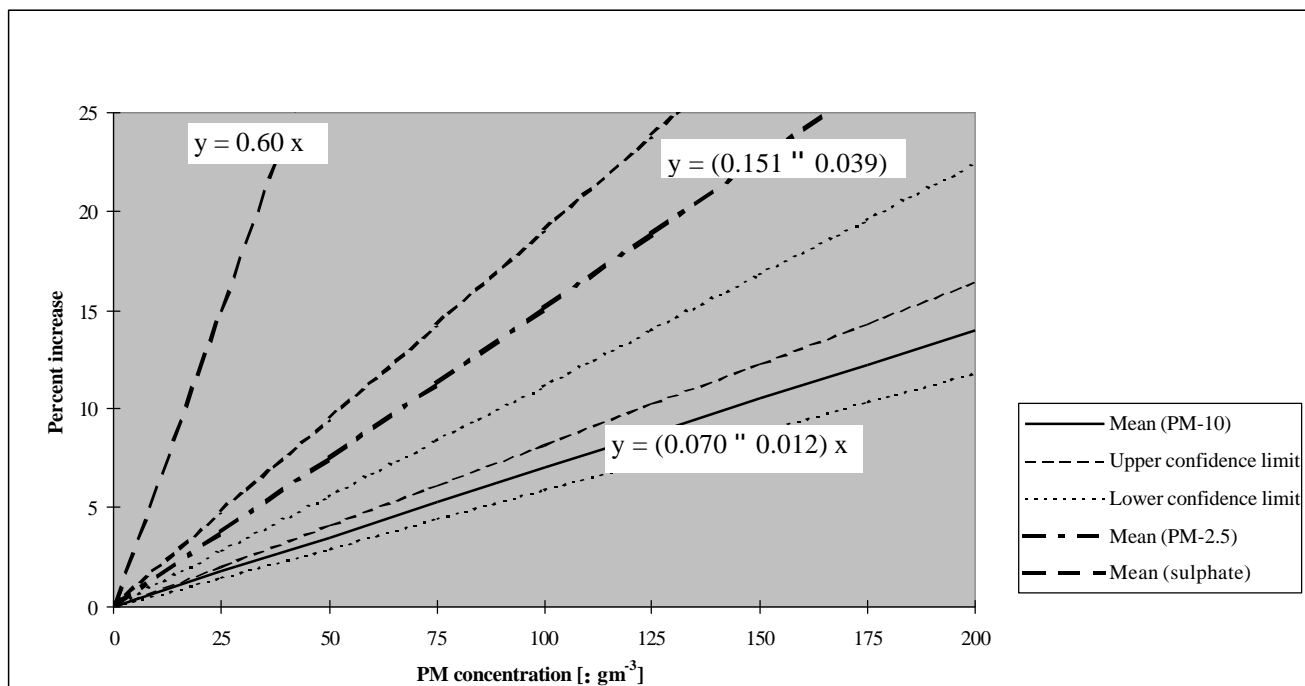


Figure 3.6. Increase in daily mortality as a function of PM concentration.

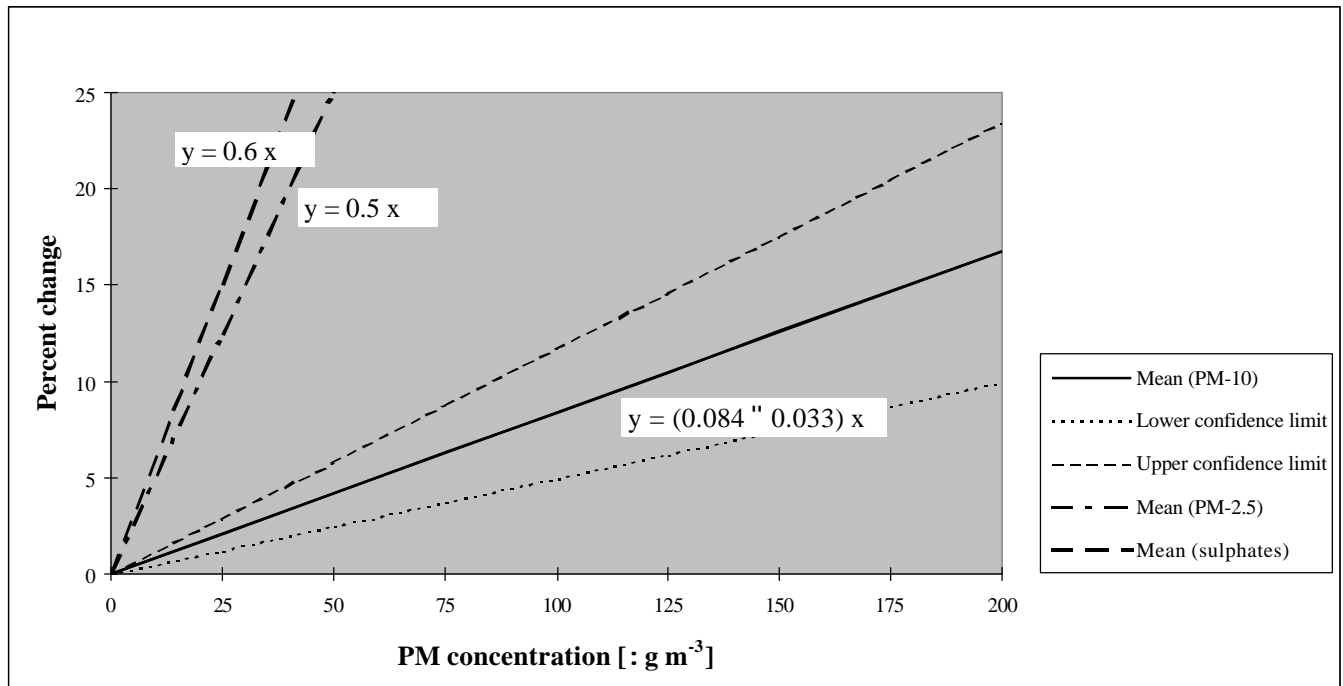


Figure 3.7. Percent change in hospital admissions assigned to  $PM_{10}$ ,  $PM_{2.5}$  and sulphates.

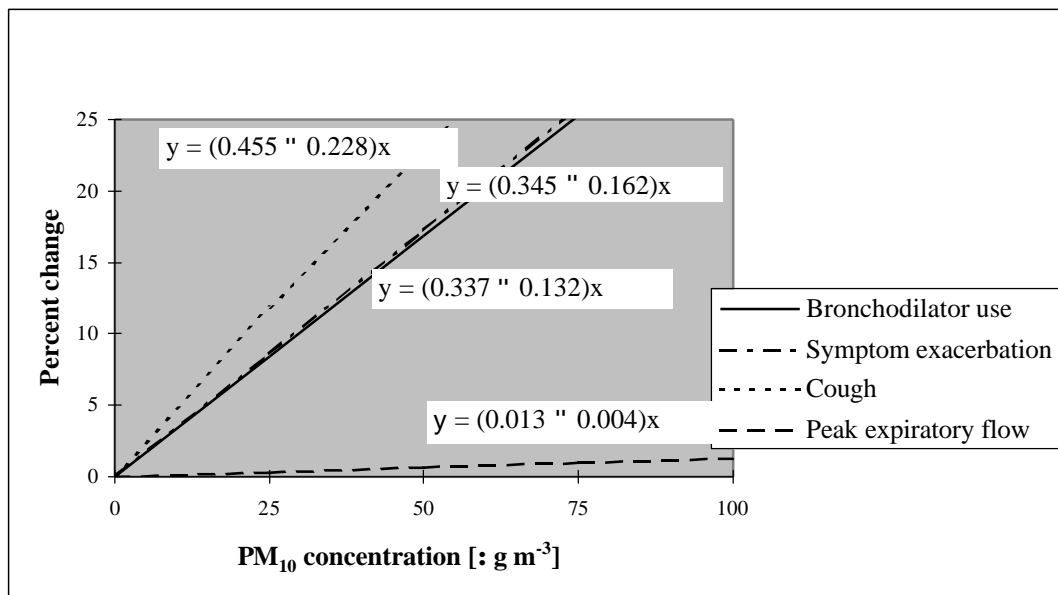


Figure 3.8. Change in health endpoints as a function of  $PM_{10}$  concentration.

The following issues should be considered when using these graphs:

- (1) The graphs should not be used for  $PM_{10}$  concentrations below  $20 \mu\text{g}/\text{m}^3$ , or above  $200 \mu\text{g}/\text{m}^3$ ; or for  $PM_{2.5}$  concentrations below  $10 \mu\text{g}/\text{m}^3$ , or above  $100 \mu\text{g}/\text{m}^3$ . This caution is required as mean 24-hour concentrations outside of the quoted ranges were not used for the risk assessment, and extrapolations beyond them would be invalid.
- (2) The areas close to the straight lines in Figures 3.6–3.8 should be considered as ‘shaded’ areas representing uncertainty, indicated by the 95% confidence intervals (CI).



- (3) There is a fundamental difference between the guidelines for PM<sub>10</sub> or PM<sub>2.5</sub> and the guideline values for respirable particulate matter derived in the WHO *Air Quality Guidelines for Europe* (WHO 1987). The guidelines for PM<sub>10</sub> and PM<sub>2.5</sub> are relationships between a health endpoint and the PM concentration. The percent change is related to the risk of health effects occurring. In consequence, when deriving an air quality standard for PM<sub>10</sub> or PM<sub>2.5</sub> using these relationships, it has to be decided which curve should be used and the risk has to be fixed. This is a new situation with respect to the derivation of air quality standard from an air quality guideline value, in which a risk is assumed without it being explicitly stated.
- (4) Figures 3.6-3.8 can be used with caution to estimate how many subjects would be affected over a short period of time with increased PM levels, for a population of a given size, mortality and morbidity characteristics. There is need for caution because of variation in results between studies for some effects.
- (5) With information on the average number of deaths and the average number of hospital admissions due to respiratory illness in a particular population, the trendlines in Figures 3.6 and 3.7 allow an estimation of the number of subjects that would be affected by an episode of PM<sub>10</sub>, or PM<sub>2.5</sub>. Similarly, with information on the number of asthmatics using bronchodilators, or experiencing asthma symptoms on a particular day, the trendlines in Figure 3.8 allow an estimate of the expected number of affected subjects. An instructive example is explained in the *Air Quality Guidelines for Europe* (WHO 1999a).
- (6) There is little current information to quantify the reduction in life expectancy associated with daily mortality increases related to PM exposure. If effects are restricted to subjects in poor health, effects on age at death may be relatively small.

### **Guidelines**

Evidence from epidemiological studies consistently points to associations between short-term exposure to PM and adverse effects on human health, even at low levels of PM commonly encountered in developed countries. The database does not, however, enable the derivation of specific guideline values at present. Most of the information currently available comes from studies in which particles in air have been measured as PM<sub>10</sub>. There is now also an increasing body of information on PM<sub>2.5</sub>, and the most recent studies show that, in general, PM<sub>2.5</sub> is a better predictor of health effects than PM<sub>10</sub>. Evidence is also emerging that constituents of PM<sub>2.5</sub>, such as sulphates and strongly acidic particles, are sometimes better predictors of health effects than PM<sub>2.5</sub>.

Many studies relate day-to-day variations in PM to day-to-day variations in health parameters. They provide quantitative estimates of effects of PM that are generally consistent. The available information does not allow a judgement to be made of concentrations below which no effects would be expected. For this reason, no guideline value for short-term average concentrations is recommended. Risk managers are referred to the risk estimates provided in the Figures 3.6-3.8 for guidance in setting standards for PM.

There is less information on the long-term effects of PM on health. Some studies have suggested that long-term exposure to PM is associated with reduced survival, and a reduction of life expectancy in the order of 2-3 years. Other recent studies have shown that the prevalence of bronchitis symptoms in children, and of reduced lung function in both children and adults, are associated with PM exposure. For this reason, no guideline value for long-term average concentrations is recommended. Risk managers are referred to the risk estimates provided in Figures 3.6-3.8 for guidance regarding standards for PM.

## **Lead**

The level of lead in blood is the best available indicator of current and recent past environmental exposure and, with stable exposures, may also be a reasonably good indicator of lead body-burden. The biological effects of lead can therefore be related to blood lead levels as an indicator of internal exposure. The relationship between blood lead concentrations and exposure to lead in air exhibits downward curvilinearity where the range of exposures is sufficiently large. At low levels of exposure the deviation from linearity is negligible and linear models of the relationship between intake and blood lead levels are satisfactory approximations.

The LOAEL for hematological and neurological effects of lead in adults and children can be summarized as follows. Frank anemia is exhibited in adults at blood lead levels above 800 µg/l, and in children above about 700 µg/l. Hemoglobin production is reduced in adults at blood lead levels above 500 µg/l and in children above 250-300 µg/l. The presence of lead in the blood also inhibits delta-aminolaevulinic acid dehydrase (ALAD), an enzyme involved in heme biosynthesis, resulting in an accumulation of its substrate, ALA, in blood, plasma and urine (WHO 1987). Urinary ALA and coproporphyrin are elevated in both adults and children above blood lead levels of about 400 µg/l. Erythrocyte protoporphyrin is found to increase in male adults at blood lead levels above 200-300 µg/l, and in female adults and children above 150-200 µg/l. A reduction in vitamin D<sub>3</sub> occurs in children at blood lead levels above 100-150 µg/l. Consequently, inhibition of ALAD in adults and children is likely to occur at blood lead levels of about 100 µg/l. However, because of its uncertain biological significance for the functional reserve capacity of the heme biosynthetic system, ALAD inhibition is not treated as an adverse effect here. Encephalopathic signs and symptoms appear not to occur in adults at lead concentrations in blood below 1000-1200 µg/l, and in children below 800-1000 µg/l.

Cognitive effects in lead workers have not been observed at blood lead levels below 500 µg/l, although reductions in nerve conduction velocity were found at concentrations as low as 300 µg/l. Elevation of free erythrocyte protoporphyrin has been observed at blood lead levels of 200-300 µg/l. Central nervous system effects, as assessed by neurobehavioural endpoints, appear to occur in children at levels below 200 µg/l. Consistent effects have been reported for global measures of cognitive functioning, such as the psychometric intelligence quotient, at blood lead levels between 100-150 µg/l. Some epidemiological studies have indicated effects such as hearing impairment at blood lead levels below 100 µg/l. Animal studies provide qualitative support for the claim that lead is a causative agent for hearing impairment.

## ***Guidelines***

The guidelines for lead in air are based on the effects of lead in blood. Critical effects to be considered in the adult organism include elevation of free erythrocyte protoporphyrin, whereas for children cognitive deficits, hearing impairment and disturbed vitamin D metabolism are taken

as the decisive effects. All of these effects are considered adverse. A critical level of lead in blood is 100 µg/l. It should be stressed that all of these values are based on population studies yielding group averages, and apply to the individual child only in a probabilistic manner.

For the derivation of a guideline value the following arguments have been considered:

Currently measured "baseline" blood lead levels of minimal anthropogenic origin are probably between 10-30 µg/l.

Various international expert groups have determined that the earliest adverse effects of lead in populations of young children begin at 100-150 µg/l. Although it cannot be excluded that population effects may occur below this range, it is prudent to derive a guideline value based on the lowest value of this range (100 µg/l).

It can be assumed that inhalation of airborne lead is a significant route of exposure for adults (including pregnant women), but it is of less significance for young children, for whom other pathways of exposure such as ingestion are generally more important than inhalation.

It appears that 1 µg Pb/m<sup>3</sup> of air directly contributes approximately 19 µg Pb/l of blood in children and about 16 µg Pb/l of blood in adults, although it is accepted that the relative contribution of lead in air is less significant in children than in adults. These values are approximations, recognizing that the relationships are curvilinear in nature and will apply principally at lower blood lead levels.

It must be taken into account that in typical situations an increase of lead in air also contributes to increased lead uptake by indirect environmental pathways. To correct for uptake by other routes, it is assumed that 1 µg Pb/m<sup>3</sup> in air would contribute to 50 µg Pb/l in blood.

It is recommended that efforts should be undertaken to ensure that at least 98% of an exposed population, including pre-school children, should have blood lead levels that do not exceed 100 µg/l. In this case, the median level of lead in blood would not exceed 54 µg/l. On this basis, the annual average concentration of lead in air should not exceed 0.5 µg/m<sup>3</sup> in blood. These estimates are assumed to also protect adults.

To prevent further increases of lead in soils, and the consequent increases in exposure of future generations, the levels of lead in air should be kept as low as possible.

As both direct and indirect exposure of young children to lead in air occurs, the guidelines for lead in air should be accompanied by other preventive measures. These should specifically take the form of monitoring the lead content of dust and soils arising from the fallout of lead in air. The normal hand-to-mouth behaviour of children necessitates that dust and soil be defined as potentially serious sources of exposure. A specific monitoring value is not recommended. Some data indicate that lead fallout in excess of 250 µg m<sup>-2</sup>/day will increase blood lead levels.

In summary, the WHO guideline values for the "classic" air pollutants are provided in Table 3.1.

**Table 3.1. WHO guideline values for the "classical" air pollutants (WHO 1999a)**

Compound	Annual ambient air concentration [mg/m <sup>3</sup> ]	Health endpoint	Observed effect level [mg/m <sup>3</sup> ]	Uncertainty factor	Guideline Value [mg/m <sup>3</sup> ]	Averaging time
Carbon monoxide	500-7000	Critical level of COHb < 2.5%	n.a.	n.a.	100 000	15 minutes
					60 000	30 minutes
					30 000	1 hour
					10 000	8 hours
Lead	0.01-2	Critical level of Pb in blood < 100-150 mg Pb/l	n.a.	n.a.	0.5	1 year
Nitrogen dioxide	10-150	Slight changes in lung function in asthmatics	365-565	0.5	200	1 hour
					40	1 year
Ozone	10-100	Respiratory function responses	n.a.	n.a.	120	8 hours
Sulphur dioxide	5-400	Changes in lung function in asthmatics	1000	2	500	10 minutes
		Exacerbations of respiratory symptoms	250	2	125	24 hours
		in sensitive individuals	100	2	50	1 year

n.a. not applicable

### 3.2 Other air pollutants

This section briefly describes the health-based guidelines for airborne inorganic and organic compounds for non-carcinogenic and carcinogenic health endpoints. Also some compounds relevant for indoor air pollution will be covered. In the process of revising and updating the WHO *Air Quality Guidelines for Europe* and the *Environmental Health Criteria* series, no guideline value and no risk-concentration relationship could be derived for several compounds. The compounds are fluorides and platinum for non-carcinogenic endpoints and 1,3 butadiene and cadmium<sup>VI</sup> for carcinogenic health endpoints.

#### *Guidelines based on noncarcinogenic health endpoints*

In the updated and revised document of the WHO *Air Quality Guidelines for Europe* (WHO 1999a) the following compounds with noncarcinogenic endpoints were considered: cadmium, dichloromethane, fluorides, HCHO, manganese, mercury, styrene, tetrachloroethylene, and toluene.

Data for CS<sub>2</sub> and H<sub>2</sub>S were not revised, and the original guidelines (WHO 1987) are still applicable.

In addition, some compounds were not considered in the process of updating and revising the *Air Quality Guidelines for Europe*. The guidelines for these compounds were taken from the published documents of the *Environmental Health Criteria* series (EHC) of the International Programme for Chemical Safety and the Concise International Chemical Assessment Documents (CICAD) of the Inter-Organization programme for the sound Management of Chemicals. For non-carcinogenic health endpoints these include the compounds: acetaldehyde (EHC 167, WHO 1995d); acetone (EHC 207, WHO 1998c); acrolein (EHC 127, WHO 1992b); acrylic acid (EHC 191, WHO 1997d); 2-butoxyethanol (CICAD 10, WHO 1998d); carbon tetrachloride (EHC 208, WHO 1999b); chloroform (EHC 163, WHO 1994b); cresol (EHC 128, WHO 1995e); 1,4-dichlorobenzene, monochlorobenzene, and trichlorobenzene (EHC 128, WHO 1991a); di-n-butyl phthalate (EHC 189, WHO 1997e); diesel exhaust (EHC 171, WHO 1996b); 2-ethoxyethanol, 2-ethoxyethanolacetate, and methoxyethanol (EHC 115, WHO 1990a); ethylbenzene (EHC 186, WHO 1996c); hexachlorocyclopentadiene (EHC 120, WHO 1991b); isophorone (EHC 174, WHO 1995f); methanol (EHC 196; WHO 1997f); methyl bromide (EHC 166, WHO 1995g); methylmethacrylate (CICAD 4, WHO 1998e); propanols (EHC 102, WHO 1990b; EHC 103, WHO 1990c); 1,1,1,2-tetrafluoroethane (CICAD 11, WHO 1998f); and xylenes (EHC 190, WHO 1997g).

The starting point for the air quality guidelines for non-carcinogenic air pollutants from the Environmental Health Criteria documents were the concepts of NOEL, NOAEL, LOEL and LOAEL (WHO 1987; WHO 1994c). Uncertainty factors were applied to these values to derive the guidelines. These uncertainty factors take into account intraspecies variation, interspecies variation, quality of data, and extrapolations from LOAEL to NOAEL and from subchronic to chronic effects. Examples for such factors and their application in deriving the guidelines are given in EHC 170 (WHO 1994c). For interspecies (extrapolation from animal to human) variation, usually a factor of 10 was applied. For intraspecies variation a factor of 5–10 was used. For use of an effect level rather than a no-effect level a factor of 2–10 was also applied, depending on the quality of the data. It was usually assumed that an uncertainty factor of 1000, based on interspecies variation (factor of 10), intraspecies variation (factor of 10) and LOAEL to NOAEL extrapolation (factor of 10), also accounted for variations in exposure time and the limitations of the database. If occupational data were the basis of a guideline derivation, a factor accounting for the number of hours per week divided by the number of working hours was applied. The choice of uncertainty factors was subject to individual expertise and judgement.

Some general considerations have to be considered in deriving guideline values in the Environmental Health Criteria (EHC) documents and in their interpretation and use:

A consistent methodology has been used in the derivation of quantitative guideline values for human exposures to chemical substances present in food, drinking-water, air and other media by *ad hoc* IPCS Task Groups (of varying membership) reviewing and evaluating data and finalizing EHC monographs on various chemicals. This approach embodies the concept that, to the extent possible, guidance values for the protection of human health should reflect consideration of total exposure to the substance whether present in air, water, soil, food or other media. Guideline values should be derived for a clearly defined exposure scenario, based on the data for the reference man (as defined in Appendix 4 of WHO 1994c), and therefore might not represent national or local circumstances.

The precision of the guidance values is dependent upon the validity and reliability of the available data. Frequently, there are sources of uncertainty in the derivation of TIs and in their allocation as a basis for guideline values, so that the resulting values represent a best estimate

based on the available data at the time. The description of the derivation of guideline values clearly indicates the nature and sources of uncertainty and the manner in which they have been taken into account in the derivation. The numerical values of guideline values should reflect the precision present in their derivation; usually guideline values are given to only one significant figure.

Establishing tolerable intakes (TIs comprising tolerable daily intakes (TDIs) or acceptable daily intakes (ADIs), in units mg/(kg bw d) or µg/(kg bw d), bw bodyweight) is central to the determination of guidance values. A TDI or ADI is defined as an estimate of the intake of a substance over a lifetime that is considered to be without appreciable health risk. It may have different units depending upon the route of administration upon which it is based and is generally expressed on a daily or weekly basis. Though not strictly an “intake”, TIs for inhalation are generally expressed as airborne concentrations (i.e. µg or mg per m<sup>3</sup>).

Two areas are critical in the methodology for the derivation of guidance values for human exposures to chemical substances in the environment:

***Development of a tolerable intake on the basis of interpretation of the available data on toxicity.*** For practical purposes, toxic effects are considered to be of two types, threshold and non-threshold. For substances where the critical effect is considered to have a threshold (including non-genotoxic carcinogenesis for which there is adequate mechanistic data), a TI is developed usually on the basis of a NOAEL.

***Allocation of the proportions of the tolerable intake to various media.*** Development on available information, the development of guidance values for compounds present in more than one environmental medium will require the allocation of proportions of the TI to various media (for example, air, food and water). For the derivation of guidance values, the allocation will be based on information on relative exposure via different routes.

Media exposure allocations of TIs for the derivation of guidance values in EHC monographs are based on relative exposure by different routes for a given scenario. Though this is suggested as a practical approach, the use of allocations based on exposure in different media does not preclude the development of more stringent limits. It is also important to recognize that the proportions of total intake from media may vary, based on circumstances. Site- or context-specific guideline values better suited to local circumstances and conditions could be developed from TIs presented in the EHC in situations where relevant data on exposure are available, and particularly where there are other significant sources of exposure to a chemical substance (e.g., in the vicinity of a waste site). Regulatory authorities may also take control to develop risk management strategies appropriate for local circumstances, although the ultimate objective of control should be reduction of exposure from all sources to less than the TIs. In addition, where data on organoleptic thresholds are included in EHC monographs, these can also be considered by relevant authorities in the development of limits.

Polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs) constitute a group of persistent environmental chemicals. A number of dioxin or furan congeners, as well as some co-planar PCBs have been shown to exert a number of toxic responses similar to those of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), the most toxic dioxin. These effects include dermal toxicity, immunotoxicity, reproductive effects and teratogenicity, endocrine disruption and carcinogenicity. For dioxin-like compounds a TDI

was derived in units of toxicity equivalent (TEQ) uptakes (WHO 1998k), which is supposed to represent a tolerable daily intake for life-time exposure. Occasional short-term excursions above the TDI would have no health consequences provided that the averaged intake over long periods is not exceeded. It was stressed that the upper range of the TDI of 4 pg TEQ/kg bw should be considered a maximal tolerable intake on a provisional basis and that the ultimate goal is to reduce human intake levels below 1 pg TEQ/kg bw/day.

The air quality guidelines for non-carcinogenic pollutants can only be applied if the averaging times are specified. The averaging time associated with a guideline value depends on the type of effects that are caused by short-term exposure producing acute effects, or long-term exposure producing chronic effects. Typical averaging times are 30 minutes for odorous pollutants, 24 hours to 1 week for acute exposures and 1 year for chronic health effects. The decision on the averaging time for a guideline needs careful screening of the toxicological and epidemiological findings and expertise in judging the results. As a consequence, the choice of an averaging time can be subjective, as is the choice of an uncertainty factor.

The air quality guidelines for compounds with non-carcinogenic health endpoints are summarized in Table 3.2.

**Table 3.2. Guidelines for air quality: compounds with non-carcinogenic health endpoints**

Compound	Average ambient air concentration [: g/m <sup>3</sup> ]	Health endpoint	Observed effect level [mg/m <sup>3</sup> ]	Uncertainty factor	Guideline Value (GV) or Tolerable Concentration (TC) [: g/m <sup>3</sup> ]	Averaging time	Source	
Acetaldehyde	5	Irritancy in humans Carcinogenicity related irritation in rats	45 (NOEL) 275 (NOEL)	20 1000	2 000 (TC) 50 (TC)	24 hours 1 year	WHO 1995d	EHC 167
Acetone	0.5-125	Odour annoyance	240 (OT)	n.a.	n.p.	-	WHO 1998c	EHC 207
Acrolein	15	Eye irritation in humans Odour annoyance	0.13 0.07	n.p. n.a.	50 (GV) -	30 min 30 min	WHO 1992b WHO 1992b	EHC 127 EHC 127
Acrylic acid	No data	Nasal lesions in mice	15 (LOAEL)	50	54 (GV)	1 year	WHO 1997d	EHC 191
2-Butoxyethanol	0.1-15	Haematotoxicity in rats	242 (NOAEL)	10	13100 (TC)	1 week	WHO 1998d	CICAD 10
Cadmium	(0.1-20) . 10 <sup>-3</sup>	Renal effects in the population	n.a.	n.a.	5 x 10 <sup>-3</sup> (GV)	1 year	WHO 1999a	
Carbon disulphide	10-1500	Functional CNS changes in workers Odour annoyance	10 (LOAEL) 0.2 (OT)	100 n.a.	100 (GV) 20 (GV)	24 hours 30 min	WHO 1987 WHO 1987	
Carbon Tetrachloride	0.5-1	Hepatotoxicity in rats	6.1(NOAEL)	1000	6.1 (TC)	1 year	WHO 1999b	EHC 208
1,4 Dichlorobenzene	0.2-3.5	Increase in organ weight and urinary proteins	450 (NOEL)	500	1000 (TC)	1 year	WHO 1991a	EHC 128
Dichloromethane	< 5	COHb formation in normal subjects		n.a.	3000 (GV)	24 hours	WHO 1999a	
Diesel exhaust	1.0 - 10.0	Chronic alveolar inflammation in humans Chronic alveolar inflammation in rats	0.139 (NOAEL)* 0.23 (NOAEL)*	25 100	5.6 (GV) 2.3 (GV)	1 year 1 year	WHO 1996b	EHC 171

\* For diesel exhaust two approaches were applied, which based on a NOAEL of 0.41 mg/m<sup>3</sup> in rats. The corresponding levels were converted to a continuous exposure scenario. n.a. not applicable; n.p. not provided.



**Table 3.2 Guidelines for air quality: compounds with non-carcinogenic health endpoints (cont.)**

Compound	Average Concentration [: g/m <sup>3</sup> ]	Health endpoint	Observed effect level [mg/m <sup>3</sup> ]	Uncertainty factor	Guideline Value (GV) or Tolerance Concentration (TC) [: g/m <sup>3</sup> ]	Averaging time	Source	
2-Ethoxyethanol	No data	Developmental effects in rats	37 (NOEL)	n.p.	n.p.	1 year	WHO 1990a	EHC 115
2-Ethoxyethylacetate	No data	Developmental effects in rats	170 (NOEL)	n.p.	n.p.		WHO 1990a	EHC 115
Ethylbenzene	1-100	Increase of organ weight	2150 (NOEL)	100	22 000 (GV)	1 year	WHO 1996c	EHC 186
Fluorides	0.5 - 3	Effects on livestock	n.a.	n.a.	1 (GV)	1 year	WHO 1999a	
Formaldehyde	(1-20) . 10 <sup>-3</sup>	Nose, throat irritation in humans	0.1 (NOAEL)	n.a.	100 (GV)	30 min	WHO 1999a	
Hexachlorocyclopentadiene	No data	Inhalation effects in rats	0.45 (NOEL)	n.p.	n.p.	1 year	WHO 1991b	EHC 120
Hydrogen sulphide	0.15	Eye irritation in humans Odour annoyance	15 (LOAEL) (0.2-2.0) x 10 <sup>-3</sup> (OT)	100 n.a.	150 (GV) 7 (GV)	24 hrs 30 min	WHO 1987 WHO 1987	
Isophorone	No data	Odour annoyance	1.14 (OT)	n.a.	-	30 min	WHO 1995f	EHC 174
Manganese	0.01 - 0.07	Neurotoxic effects in workers	0.03 (NOAEL)	200	0.15 (GV)	1 year	WHO 1999a	
Mercury, inorganic	(2-10) . 10 <sup>-3</sup>	Renal tubular effects in humans	0.020 (LOAEL)	20	1 (GV)	1 year	WHO 1999a	
2-Methoxyethanol	No data	Developmental toxicity in rats	31 (NOEL)	n.p.	n.p.		WHO 1990a	EHC 115
Methyl bromide	0.05-0.8	Reduction in fertility index in rats	12 (NOEL)	n.p.	n.p.		WHO 1995g	EHC 166
Methyl Methacrylate	2.4 x 10 <sup>-4</sup>	Degenerate changes in olfactory epithelium in rodents	102.5 (NOEL)	100	200 (TC)	1 year	WHO 1998e	CICAD 4
Monochlorobenzene	0.2-3.5	Decreased food intake, increased organ weight, lesions and changes in blood parameters	341 (LOAEL)	1000	500 (TC)	1 year	WHO 1991a	EHC 128

n.a. not applicable; n.p. not provided.

**Table 3.2 Guidelines for air quality: compounds with non-carcinogenic health endpoints (cont.)**

Compound	Average ambient air concentration [: g/m <sup>3</sup> ]	Health endpoint	Observed effect level [mg/m <sup>3</sup> ]	Uncertainty factor	Guideline Value (GV) or Tolerance concentration (TC) [: g/m <sup>3</sup> ]	Averaging time	Source	
1-Propanol	0.05	Reproduction in pregnant rats	9001 (NOEL)	n.p.	n.p.		WHO 1990b	EHC 102
2-Propanol	1500-35000	Developmental toxicity in rats	9001 (LOEL)	n.p.	n.p.		WHO 1990c	EHC 103
Styrene	1.0 -20.0	Neurological effects in workers Odour annoyance	107 (LOAEL) 0.07 (OT)	40 n.a.	260 (GV) 7 GV)	1 week 30 minutes	WHO 1999a WHO 1987	
Tetrachloroethylene	1 - 5	Kidney effects in workers Odour annoyance	102 (LOAEL) 8	400 n.a.	250 (GV) 8000 (GV)	24 hours 30 minutes	WHO 1999a WHO 1987	
1,1,1,2-Tetrafluoroethane	No data	Development toxicity in animals	41700 (NOAEL)	n.p.	n.p.		WHO 1998f	CICAD 11
Toluene	5 - 150	Effects on CNS in workers Odour annoyance	332 (LOAEL) 1 (OT)	1260 n.a.	260 (GV) 1000 (GV)	1 week 30 minutes	WHO 1999a WHO 1987	
1,3,5 Trichlorobenzene	0.5-0.8	Metaplasia and hyperplasia of respiratory epithelium in rats	100 (NOEL)	500	200 (TC)	1 year	WHO 1991a	EHC 128
1,2,4 Trichlorobenzene	0.02-0.05	Increase in urinary porphyrins in rats	22.3 (NOAEL)	500	50 (TC)	1 year	WHO 1991a	EHC 128
Vanadium	0.05 - 0.2	Respiratory effects in workers	0.02 (LOAEL)	20	1 (GV)	24 hours	WHO 1987	
Xylenes	1 - 100	CNS effects in human volunteers Neurotoxicity in rats Odour annoyance	304 (NOAEL) 870 (LOAEL) 4.35 (OT)	60 1000 n.a.	4800 (GV) 870 (GV) -	24 hours 1 year 30 minutes	WHO 1997g WHO 1997g WHO 1997g	EHC 190 EHC 190 EHC 190

n.a. not applicable; n.p. not provided.

**Table 3.2 Guidelines for air quality: compounds with non-carcinogenic health endpoints (cont.)**

Compound	Average ambient air concentration [ $\mu\text{g}/\text{m}^3$ ]	Health endpoint	Observed effect level [mg/kg bw d]	Uncertainty factor	Tolerable Daily intake (TDI or ADI) [ $\mu\text{g}/\text{kg bw d}$ ]	Averaging Time (over lifetime)	Source	
Chloroform	0.3-10	Hepatotoxicity in beagles	15 (LOEL)	1000	15 (TDI)	24 hours	WHO 1994b	EHC 163
Cresol	1-10	Reduced body weight and tremors in mice	50 (LOAEL)	300	170 (ADI)	24 hours	WHO 1995e	EHC 168
Di-n-butyl Phthalate	(3-80) $\cdot 10^{-3}$	Developmental/Reproductive toxicity	66 (LOAEL)	1000	66 (ADI)	24 hours	WHO 1997e	EHC 189
			Estimated human daily intake [pg/kg bw d]					
Dioxin-like compounds	n.p.	Neurobehavioural effects/ Endometriosis in monkey offspring Decreased sperm count/immune suppression/increase genital malformations in rat offspring	14-37 (LOAEL)*	10	[TEQ/kg bw d] 1-4 (TDI)	24 hours	WHO 1998k	

\* Estimated from the maternal body burden of exposed rats and monkeys by applying a factor of 2..  
 kg bw d = kilogramme bodyweight per day

Additional air pollutants were considered for which it was not possible to derive guideline values. For non-carcinogenic health endpoints these compounds include dioxins, fluorides, platinum and other compounds, for which the existing information can be extracted from the EHC series compiled in Appendix 4.

***Guidelines based on carcinogenic health endpoints***

In the revision of the WHO *Air Quality Guidelines for Europe* (WHO 1999a) the following compounds with carcinogenic endpoints were considered: arsenic, benzene, chromium (VI), man-made vitreous fibres, nickel, PAH, radon, trichloroethylene, and toluene. The data for acrylonitrile and vinylchloride were not revised and updated and the original guidelines are still applicable (WHO 1987). Additional carcinogenic compounds, for which unit risks could be derived from the EHC series publications, are included in the guidelines. These include acetaldehyde (EHC 167, WHO 1995d); bis(chloromethyl)ether (EHC 201, WHO 1998h); 1,2-dichloroethane (CICAD 1, WHO 1998g); diesel exhaust (EHC 171, WHO 1996b); selected non-heterocyclic PAH (EHC 202, WHO 1998i); and 1,1,2,2-tetrachloroethane (CICAD 3, WHO 1998j).

In addition, for some carcinogenic compounds, such as 1,3 butadiene and cadmium, guidelines could not be derived. Existing information on these compounds can be taken from WHO 1999a and, for other compounds, from the published documents of the *Environmental Health Criteria* series compiled in Appendix 4.

**Table 3.3 Guidelines for air pollutants with carcinogenic health endpoints**

<b>Compound</b>	<b>Average ambient air concentration [mg/m<sup>3</sup>]</b>	<b>Health endpoint</b>	<b>Unit risk [mg/m<sup>3</sup>]<sup>-1</sup></b>	<b>IARC classification</b>	<b>Source</b>	
Acetaldehyde	5	Nasal tumours in rats	$(1.5-9) \times 10^{-7}$	2B	WHO 1995d	EHC 167
Acrylonitrile	0.01 - 10	Lung cancer in workers	$2 \times 10^{-5}$	2A	WHO 1987	
Arsenic	$(1 - 30) \cdot 10^{-3}$	Lung cancer in exposed humans	$1.5 \times 10^{-3}$	1	WHO 1999a	
Benzene	5.0 - 20.0	Leukemia in exposed workers	$(4.4-7.5) \times 10^{-6}$	1	WHO 1999a	
Benzo[a]pyrene		Lung cancer in humans	$8.7 \times 10^{-2}$	1	WHO 1999a	
Bis(chloromethyl)ether	No data	Epitheliomas in rats	$8.3 \times 10^{-3}$	1	WHO 1998h	EHC 201
Chloroform	0.3-10	Kidney tumours in rats	$4.2 \times 10^{-7}$	2B	WHO 1994b	EHC 163
Chromium <sup>VI</sup>	$(5-200) \cdot 10^{-3}$	Lung cancer in exposed workers	$(1.1-13) \times 10^{-2}$	1	WHO 1999a	
1,2-Dichloroethane	0.07 - 4	Tumour formation in rodents	$(0.5-2.8) \times 10^{-6}$	2B	WHO 1998g	CICAD 1
Diesel exhaust	1.0 - 10.0	Lung cancer in rats	$(1.6-7.1) \times 10^{-5}$	2A	WHO 1996b	EHC 171

**Table 3.3** Guidelines for air pollutants with carcinogenic health endpoints (cont.)

Compound	Average ambient air concentration [mg/m <sup>3</sup> ]	Health endpoint	Unit risk [mg/m <sup>3</sup> ] <sup>-1</sup>	IARC classification	Source	
ETS	1-10	Lung cancer in exposed humans	10 <sup>-3</sup>		WHO 1999a	
Nickel	1-180	Lung cancer in exposed humans	3.8 x 10 <sup>-4</sup>	1	WHO 1999a	
PAH (BaP)	(1-10) . 10 <sup>-3</sup>	Lung cancer in exposed humans	8.7 x 10 <sup>-2</sup>	1	WHO 1999a	
1,1,2,2-Tetrachloroethane	0.1 - 0.7	Hepatocellular carcinomas in mice	(0.6-3.0) x 10 <sup>-6</sup>	3	WHO 1998j	CICAD 3
Trichloroethylene	1 -10	Cell tumours in testes of rats	4.3 x 10 <sup>-7</sup>	2A	WHO 1999a	
Vinylchloride	0.1 - 10	Hemangiosarkoma in exposed workers Liver cancer in exposed workers	1 x 10 <sup>-6</sup>	1	WHO 1987	

For the compounds noted in Table 3.3 estimation of the unit risks is described in the references quoted. Unit risks for mixtures such as petrol exhaust, roofing tar, smokeless and smoky coal, and wood smoke can, in principle, be estimated from the potencies of these mixtures and the unit risk of benzo[a]pyrene (BaP) by use of the formula:

$$UR_{\text{mixture}} = (\text{potency of mixture})/(\text{potency of "coke oven top"}) \times UR_{\text{BaP}} \times (\text{content of BaP in mixture}).$$

In this relationship the potencies of the mixture and the potency of "coke oven top" are taken from Table A.I.17 of EHC 202 (WHO 1998i);  $UR_{\text{mixture}}$  denotes the unit risk of the mixture and  $UR_{\text{BaP}}$  that of BaP; the unit of the content of BaP in the mixture is microgram per gram of mixture. Table 3.4 reflects the relative potencies of the mixtures, which defined as the potencies of the mixtures divided by the potency of "coke oven top" (see EHC 202, WHO 1998i).

**Table 3.4. Relative potencies of certain mixtures**

Mixture	Relative potency of mixture
<i>Petrol exhaust</i>	0.736
Roofing tar	0.145
<i>Smokeless coal</i>	0.368
Smoky coal	1.026
Wood smoke	0.759

For example, the BaP content of wood smoke has been estimated to range between 1 and 29 [mg BaP/g of mixture] (Ward 1999). Inserting all quantities into the above equation leads to a unit risk for wood smoke in the range of  $(0.07-1.9) \times 10^{-7} [\mu\text{g}/\text{m}^3]^{-1}$ . If the BaP content of other mixtures are known the unit risk can be estimated in a similar way.

Using the potencies of other non-heterocyclic polycyclic hydrocarbons relative to BaP (see Table A.I.9 of EHC 202, WHO 1998i), unit risks can also be given as a rough estimate for these compounds by using of the formula (results are given in Table 3.5)

$$UR_{\text{compound}} = (\text{potency of compound})/(\text{potency of BaP}) \times UR_{\text{BaP}}$$

**Table 3.5. Estimate of unit risks for several polycyclic aromatic hydrocarbons**

Compound	Relative potency range compared to BaP	Unit risk [mg/m <sup>3</sup> ] <sup>-1</sup>
Anthanthrene	0.28 - 0.32	(2.4 - 2.8) x 10 <sup>-2</sup>
Benz[a]anthracene	0.014 - 0.145	(1.2 - 13) x 10 <sup>-4</sup>
Benzo[a]pyrene	1	8.7 x 10 <sup>-2</sup>
Benzo[b]fluoranthene	0.1 - 0.141	(0.87 - 1.2) x 10 <sup>-2</sup>
Benzo[j]fluoranthene	0.045 - 0.1	(0.4 - 0.87) x 10 <sup>-2</sup>
Benzo[k]fluoranthene	0.01 - 0.1	(8.7 - 87) x 10 <sup>-4</sup>
Chrysene	0.001 - 0.1	(8.7 - 870) x 10 <sup>-5</sup>
Cyclopenta[cd]pyrene	0.012 - 0.1	(1 - 8.7) x 10 <sup>-3</sup>
Dibenzo[a,e]pyrene	1	8.7 x 10 <sup>-2</sup>
Dibenz[a,c]anthracene	0.1	8.7 x 10 <sup>-3</sup>
Dibenz[a,h]anthracene	0.89 - 5	(7.7 - 43.5) x 10 <sup>-2</sup>
Dibenzo[a,l]pyrene	100	8.7 x 10 <sup>-0</sup>
Dibenzo[a,e]fluoranthene	1	8.7 x 10 <sup>-2</sup>
Dibenzo[a,h]pyrene	1 - 1.2	(8.7 - 10.4) x 10 <sup>-2</sup>
Dibenzo[a,i]pyrene	0.1	8.7 x 10 <sup>-3</sup>
Fluoranthene	0.001 - 0.01	(8.7 - 87) x 10 <sup>-5</sup>
Indeno[1,2,3,-cd]pyrene	0.067 - 0.232	(5.8 - 20.2) x 10 <sup>-3</sup>

Air quality guidelines for man-made vitreous fibres and radon were also revised. Man-made vitreous fibre (MMVF) concentrations have been measured in only a few studies and have been found to average about 340 fibres per cubic metre (F/m<sup>3</sup>) in ambient air and 570 F/m<sup>3</sup> in indoor air. Maximum values were 2400 F/m<sup>3</sup> in ambient air and 5600 F/m<sup>3</sup> in indoor air. Several types of refractory ceramic fibres were found to be carcinogenic in inhalation studies in animals. The IARC classified ceramic fibres as possibly carcinogenic to humans (Group 2B). From inhalation studies in animals, the unit risk for lung tumours for a lifetime exposure to 1000 F/m<sup>3</sup> was estimated to be 10<sup>-6</sup> per fibre/m<sup>3</sup> for fibres of length below 5 µm.

Radon is another indoor air pollutant known to cause lung cancer in humans. Average indoor concentrations range between 20 and 200 Bq/m<sup>3</sup>. A study of lung cancer in workers showed a linear increase in lung cancer in response to increases in estimated radon exposure (Pershagen et al. 1994). Figure 3.9 shows the estimated proportion of lung cancers that can be attributed to residential radon. This figure can be used to assess the risk of radon exposure.



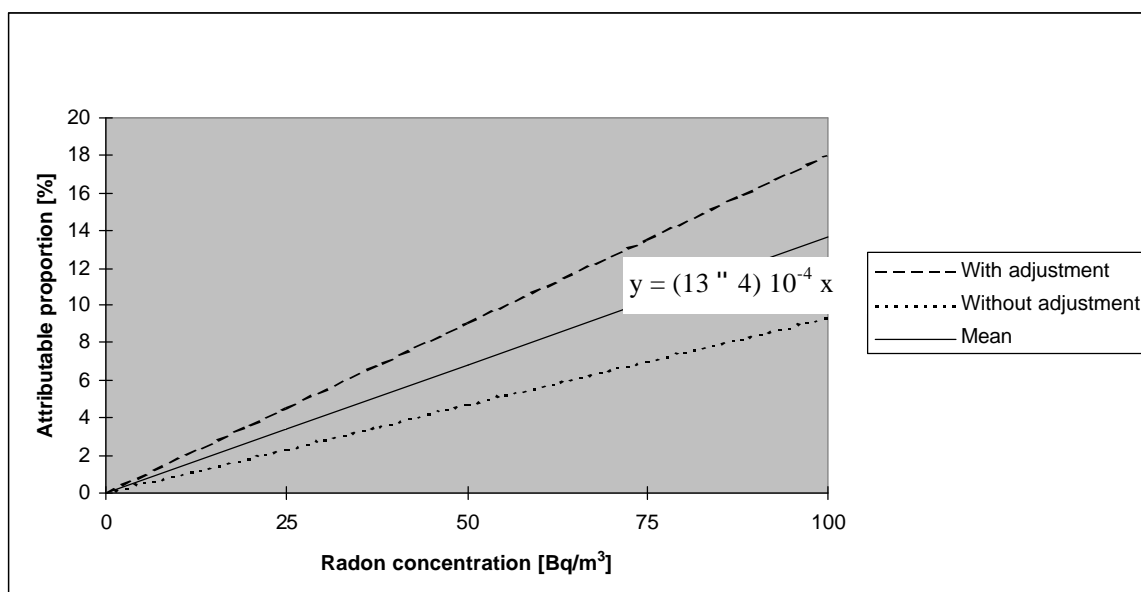


Figure 3.9. The proportion of lung cancers attributable to radon exposure.

### 3.3 Classical air pollutants: applicability of WHO Air Quality Guidelines for Europe on a world-wide scale

In the derivation of the WHO *Air Quality Guidelines for Europe*, assumptions were made for some compounds, which may not be applicable in some parts of the world. For some, but not all, pollutants the importance of different routes of exposure may vary from country to country. It should be understood that if such factors were to be taken into account then different guidelines could be derived. It is important that regulatory authorities should answer the following question before adapting for local use a guideline from the *Air Quality Guidelines for Europe*: Do local circumstances give cause to doubt the likely validity of the guideline set out in the WHO *Air Quality Guidelines* as a basis for setting local guidelines or standards? For a number of pollutants a unit risk assessment has been provided. These assessments are also dependent upon considerations of the comparative importance of different routes of exposure.

### 3.4 Studies of effects of air pollutants on health in WHO regions

As discussed above, the effects of air pollutants on health vary depending on several factors. These include the level of exposure and the susceptibility of the exposed population. The susceptibility of the population is affected by factors such as the numbers of young children and older people, as well as the proportion of people suffering from asthma and other chronic respiratory conditions. Epidemiological studies reflect this variation in sensitivity by showing different associations between levels of exposure and health effects for different subpopulations. In addition, sources and patterns of exposure, e.g. indoor and outdoor exposures, are likely to differ substantially from region to region. In part this is dependent upon weather conditions.

These factors and the variation in response-concentration relationships are powerful arguments for health studies being undertaken in the different WHO Regions on the effects of air pollutants.

It could be a mistake to simply adopt response-concentration relationships derived from Western European or North American studies for general use.

No general review drawing together the results of epidemiological studies on air pollution across the WHO Regions has been published. Regions differ significantly in terms of the number of studies undertaken and in the quality of those studies. Many, perhaps most, studies are done with the intention of characterising the local problem and quantifying the effects of air pollution on health. Preliminary studies to assess whether there is a problem are common.

Recent developments in our understanding of the effects of air pollutants on health suggest that, at least for particulate matter and O<sub>3</sub>, all levels of exposure above zero are associated with effects on health. That pollutants such as sulphur and NO<sub>2</sub> should be regarded as no-threshold compounds seems toxicologically implausible, although such a conclusion is difficult to avoid given the current time-series data.

## **Sulphur dioxide**

### **Latin America**

Few epidemiological studies conducted in Latin America have investigated the effect of SO<sub>2</sub> on health. In a study conducted in Chile close to an industrial area where SO<sub>2</sub> annual means ranged from 101-145 µg/m<sup>3</sup>, and maximum daily averages from 405-1230 µg/m<sup>3</sup>, an increase of 50 µg/m<sup>3</sup> in the SO<sub>2</sub> daily mean value was related to a 4% increase in cough frequency (95% CI: 1-7%), a 3% increase in phlegm production (95% CI: 0-6%) and a 4% increase in wheeze occurrence (95% CI: 0-11%), with a one-day lag among children with chronic respiratory symptoms (Sanchez-Cortez 1997). A significant change in evening peak flow measurements was also observed. No effects were observed in children without chronic respiratory symptoms. In this study, health effects were observed at levels lower than 125 µg/m<sup>3</sup> (the WHO guideline) among susceptible children. However, SO<sub>2</sub> may have interacted with PM<sub>10</sub> levels, which ranged from 5 to 125 µg/m<sup>3</sup> in this study.

In the same study, when areas with different long-term ambient levels of SO<sub>2</sub> were compared (70 µg/m<sup>3</sup> vs. 130 µg/m<sup>3</sup> annual mean over 3 years), the prevalence of chronic respiratory symptoms was higher in the area with the higher SO<sub>2</sub> annual means (30% vs. 14% for chronic cough and 14.3% vs. 6.1% for wheezing). The differences were statistically significant (Sanchez-Cortez 1997). PM<sub>10</sub> annual means were low in both areas.

### **Mediterranean Region**

Few studies have investigated the effects of air pollution on health in the Eastern Mediterranean region. In one study of residents of the Shoubra El-Kheima industrial area of Egypt results showed that 37.4% of the examined sample (4730 subjects) suffered from chronic obstructive pulmonary diseases (COPD) and the prevalence increased with age (El-Samara et al. 1984). This study found that 1478 students (out of the studied group of 6380 students) were suffering from COPD. A strong positive correlation was recorded between PM<sub>10</sub> level and incidence of asthma.

## **Western Pacific Region**

### **Japan**

An epidemiological survey in Japan from 1981 to 1983 involved schoolchildren aged 6-12 years (Nitta et al. 1993; Ono et al. 1990; Nakai et al. 1995). Annual mean concentrations in urban areas ranged from 26.8-30.9  $\mu\text{g}/\text{m}^3$   $\text{SO}_2$ . Suburban area levels ranged from 20.5-23.9  $\mu\text{g}/\text{m}^3$  and background levels from 13.3-22.9  $\mu\text{g}/\text{m}^3$ . Comparison of the effect of  $\text{SO}_2$  on human health in the different areas showed that the prevalence of asthmatic symptoms, of chest congestion and of phlegm significantly correlated with annual mean levels of  $\text{SO}_2$ .

### **China**

Epidemiological investigations in China show short-term exposure to 280  $\mu\text{g}/\text{m}^3$   $\text{SO}_2$  was correlated with apparent effects on the health of traffic police, whose respiratory function was reduced by 29-64%, and whose incidence of chronic rhinitis and pharyngitis was raised by 30-90%, compared with the control group (BMEPB 1980). Where the annual average air concentration of  $\text{SO}_2$  was 260  $\mu\text{g}/\text{m}^3$ , secondary and elementary school students had a much higher incidence of chronic respiratory diseases than in less polluted areas. For example, the incidence of tonsil suppuration was increased 5.1-fold, simple rhinitis by 1.1-fold and nose engorgement by 0.9-fold (BMEPB 1980). Under long exposure to an annual average of 175  $\mu\text{g}/\text{m}^3$   $\text{SO}_2$  (with 550  $\mu\text{g}/\text{m}^3$  particulate matter also present), the three-year average mortality from pulmonary heart disease and respiratory diseases in the community was twice that of the control group (GMEPB 1980).

A study was conducted on the influence of  $\text{SO}_2$  pollution on lung function of children and women (Chen et al. 1993). It found that at annual average concentration of 140  $\mu\text{g}/\text{m}^3$  (with 150  $\mu\text{g}/\text{m}^3$  particulate matter),  $\text{SO}_2$  is associated with lower levels of lung function in children at the ages of 10-12, with major decreases in FVC and  $\text{FEV}_1$ . For each 60  $\mu\text{g}/\text{m}^3$  increment in annual average concentration of  $\text{SO}_2$ , there was an average 99 ml drop in the children's FVC and a 70 ml drop in  $\text{FEV}_1$ . The FVC of women was decreased by 57 ml under the same conditions. In addition, it was found that  $\text{SO}_2$  can affect women's non-specific immunity in parts of their respiratory passages, lowering their average concentration of saliva lysozyme by 5.6  $\mu\text{g}/\text{ml}$  and specific immunoglobuline by 32  $\mu\text{g}/\text{ml}$  (Chen et al. 1995).

### **South East Asia**

The results of epidemiological studies in India indicate that adverse health effects can be associated with ambient air  $\text{SO}_2$  at an annual average concentration of 40  $\mu\text{g}/\text{m}^3$ . Interpretation of these findings is complicated by the high co-existing particle levels, as well as by a number of additional local factors. These include high indoor and occupational exposure to air pollutants, below average health conditions and poor nutritional status, unsafe water supply, poor general hygiene etc.

A study of 4129 community residents of three areas of Bombay, representing three grades of air pollution conditions (based on secondary data), and a fourth area 40 km away towards the south-east as a control, found:

- i. Higher morbidity in the two more polluted areas for breathing problems, cough and common colds. The city's residents in the polluted zone were the healthiest, even in comparison to rural populations. Other symptoms related to pollution were headache, eye irritation, chest

pain, skin lesions and intermittent cough.

ii. In the urban low pollution area there was a larger prevalence of cardiac complaints.

**Table 3.6. Standardised prevalence of selected diseases in Bombay (after Kamat and Doshi 1987)**

Disease	Urban SO <sub>2</sub> levels			
	Low (<50 µg/m <sup>3</sup> )	Intermediate (51-100 µg/m <sup>3</sup> )	High (>100 µg/m <sup>3</sup> )	Rural (control)
Dyspnea	3.2	6.0	7.3	5.5
Chronic cough	1.7	2.7	5.1	3.3
Intermittent cough	0.4	5.8	15.6	3.7
Frequent colds	12.1	20.8	18.0	11.0
Chronic bronchitis	2.3	4.5	4.5	5.0
Cardiac disorders	8.2	4.3	6.8	2.7

A study (Kamat et al. 1992) of 4 comparable communities in central and north-eastern Bombay (2 each) among randomly matched 349 subjects in 1988-1989, along with ambient SO<sub>2</sub>, NO<sub>2</sub> and SPM air monitoring was carried out in Parel, Maravali, Deonar and Dadar. Air pollutant levels in winter were higher particularly for SO<sub>2</sub> in Parel (up to 584 µg/m<sup>3</sup>) and Maravali; Deonar showed lower pollution. Clinical respiratory symptoms were higher in Parel and Maravali (cough 12% and 11.2%, dyspnea 17% and 13.3% respectively). Cardiac problems were commoner in Parel (11.0%). Maravali had a high prevalence for headache and eye irritation (9.5%). Those using kerosene suffered more than those using gas (22.2% as compared to 9.2%) Lung functions (FVC, FEV<sub>1</sub>) were lowest in Parel for males and in Maravali for females. Expiratory flow rates were lower at Dadar, followed by Maravali. Despite lower SO<sub>2</sub> pollution, symptoms in Maravali residents were comparable to those in Parel. It was conjectured that this may be due to added effect of diesel exhausts (NO<sub>2</sub>, SPM) or other unmeasured chemicals.

## Nitrogen dioxide

### Latin America

There are few data from Latin America on the impact of outdoor sources of NO<sub>2</sub> exposure on health. As in many Latin American cities, NO<sub>2</sub> levels are usually low (WHO 1998b). However, in a preliminary study conducted in Sao Paulo, Brazil (Saldiva et al. 1995), a 75 µg/m<sup>3</sup> increase in NO<sub>2</sub> was related to a 30% increase in mortality for respiratory illness among children less than five years old.

In Mexico City, a time-series study of hospital emergency visits among children less than 15 years of age found NO<sub>2</sub> daily levels correlated with upper respiratory illnesses (Tellez-Rojo et al. 1999). Stronger associations were observed during the winter months, when NO<sub>2</sub> levels ranged from 40-160 µg/m<sup>3</sup> (mean 90 µg/m<sup>3</sup>), and O<sub>3</sub> levels from 82-740 µg/m<sup>3</sup> (mean 368 µg/m<sup>3</sup>). The correlation coefficient between pollutants and illness was 0.44. The highest indicated effect of NO<sub>2</sub> was observed with a two-day lag. A 56 µg/m<sup>3</sup> increase in daily NO<sub>2</sub> ambient concentration was associated with a 39% increase in upper respiratory illnesses (95% CI: 28-51%). However, given the mixture of contaminants, and the general low NO<sub>2</sub> levels observed in this study, it is not possible to ascertain that NO<sub>2</sub> is the contaminant responsible for the observed effects.

## **Western Pacific Region**

### **Japan**

From 1992 to 1995, the Japanese Environment Agency surveyed the health effects of air pollutants in about 15 000 schoolchildren (EA 1997). The results showed that the prevalence of asthmatic symptoms was higher at NO<sub>2</sub> levels above 37.6 µg/m<sup>3</sup> than below this level. In general, however, the levels of NO<sub>2</sub> in Japan are not high enough to demonstrate a clear cause-effect relationship between the prevalence of asthmatic symptoms and NO<sub>2</sub> concentration. But neither are they low enough to rule out a causal relationship.

A survey of respiratory symptoms as a function of distance from roads with heavy traffic showed that the prevalence rate of respiratory symptoms, such as chronic cough and wheezing, was higher in residents nearer roads (Nitta et al. 1993; Ono et al. 1990). When there were no indoor NO<sub>2</sub> sources except for gas cooking stoves, both indoor and individual levels of NO<sub>2</sub> were attributable primarily to automobile exhaust (Nakai et al. 1995).

It has been reported that an interaction between air pollution, especially NO<sub>2</sub>, and high temperature, may synergistically increase lung cancer mortality rates, since regional differences in age-adjusted lung cancer rates were explained by an interaction between NO<sub>2</sub> and temperature (Choi et al. 1997).

### **China**

In recent years, epidemiological studies examined NO<sub>2</sub> concentrations in kitchens of 160 city dwellers, as well as urine hydroxyproline (HOP) levels of individuals after 24-hour exposures. The results showed that in liquid petroleum gas (LPG) -fuelled kitchens, NO<sub>2</sub> peak concentrations can reach 990-1,809 µg/m<sup>3</sup> at the moment of ignition, 17-37.5 -fold higher than the daily average concentration of 50 µg/m<sup>3</sup> (background concentration). Also, the urine HOP levels of individuals cooking in LPG-fuelled kitchens were higher than those cooking in coal fuelled kitchens (Zhang Jinhiang et al. 1996). In contrast, NO<sub>2</sub> exposures produced by burning coal was significantly higher than those resulting from the burning of LPG.

A survey in four cities showed that the daily average value of indoor NO<sub>2</sub> concentration was 53 µg/m<sup>3</sup>, and elevated levels of SO<sub>2</sub>, CO and TSP were recorded. Studies of primary school students aged 10-15 years residing in this environment showed 30-70% suffer from coughing, and 7-40% suffer from phlegm; and the incidence of tonsillitis and hyperplasia of retropharyngeal lymph folliculi are 7-17% and 15-16%, respectively. In addition, effects on immunity indices (such as PHA skin test and saliva lysozyme) were also observed (Wang Jin et al. 1989; Qin Yuhui et al. 1990).

Studies on 60 healthy Beijing children aged 9-11 years, and exposed to NO<sub>2</sub> at a daily average level of 70-110 µg/m<sup>3</sup>, with the peak values of 150-260 µg/m<sup>3</sup> for two months, reported a negative correlation between NO<sub>2</sub> concentration and peak expiratory flow rates (PEFR). The results indicate that increased NO<sub>2</sub> level could affect children's respiratory function, aggravate air duct blocking and subsequently reduce PEFR (Wang Lihua et al. 1994). Long-term exposure to 50-100 µg/m<sup>3</sup> NO<sub>2</sub> may significantly affect children's respiratory and immunity systems; and it may have similar effects on sensitive adults.

### **Australia**

Morgan et al. (1998) examined the effects of outdoor air pollutants on daily hospital admissions in Sydney, Australia. A time-series analysis of counts of daily hospital admissions and outdoor air pollutants (1990-1994) showed that an increase in the daily maximum 1-hour concentration of NO<sub>2</sub> from the 10th to the 90th percentile was associated with an increase of 5.29% (95% CI: 1.07% to 9.68%) in childhood asthma admissions and 4.60% (95% CI: -0.17% to 9.61%) in COPD admissions. A similar increase in daily maximum 1-hour particle concentration was associated with an increase of 3.01% (95% CI: -0.38% to 6.52%) in COPD admissions. An increase from the 10th to the 90th percentile in daily maximum 1-hour NO<sub>2</sub> was associated with an increase of 6.71% (95% CI: 4.25% to 9.23%) in heart disease admissions among those 65 years and older. Increases in heart disease, COPD and childhood asthma were associated with increased NO<sub>2</sub> levels.

### **Carbon monoxide**

### **Mediterranean Region**

In Cairo, CO concentrations greater than the WHO *Guidelines for Air Quality* values were recorded in streets having moderate-to-heavy traffic densities in residential areas and in the city centre (Nasralla 1997). These concentrations resulted in high levels of COHb in the blood of traffic policemen, sometimes reaching more than 10%. This study also found a significant direct relationship between ischemic heart disease and COHb level in Cairo traffic policemen (Salem 1990).

### **Western Pacific Region**

### **China**

Chinese middle-school students residing in a relatively low-pollution district of Shenyang, and undergraduate students studying at a relatively low-pollution district of Beijing, had average blood COHb concentrations of 0.8 % and 0.5 % respectively. Research on the effect of indoor CO on children aged 8-13 years showed that for rooms with individual heating the average CO content was 12.4 mg/m<sup>3</sup> and the COHb blood levels in these children was 4.17%. In rooms with central heating, the CO concentration was 6.4 mg/m<sup>3</sup> and the COHb levels in was 1.79% (Liu Jifang et al. 1992). This study also showed that in individually heated rooms the children's saliva lysozyme exhibited lower activity than that in centrally heated rooms; and immunoglobulin G content of the former is less than that of the latter. This phenomenon suggests that CO pollution could result in hyp immunity for children (Liu Jifang et al. 1992).

### **Ozone and other photochemical oxidants**

### **Latin America**

Several studies conducted in Mexico City have illustrated the association of acute peak daily O<sub>3</sub> concentration with respiratory health. A study conducted among children reported both acute and subacute effects of O<sub>3</sub> on lung functions (Castillejos et al. 1992). A 106 µg/m<sup>3</sup> rise in the mean 48-hour O<sub>3</sub> levels was associated with a decrease of 2% in FEV<sub>1</sub>, and a 7.4% decrease in the forced expiratory flow FEF<sub>25-75</sub>. A greater decrease in these parameters was observed in children with chronic cough, chronic phlegm or wheeze. In another study, conducted among

school children from Mexico City, that compared quintiles of O<sub>3</sub> concentration, a decrease of 1.43% in FVC and 2.85% in FEV<sub>1</sub> was reported in the highest quintile of O<sub>3</sub> concentration (364-730 µg/m<sup>3</sup>) (Castillejos et al. 1995). This change in FEV<sub>1</sub> is less than that predicted by Figure 3.2.

In a study conducted among pre-school children, an increase in school absenteeism for respiratory illnesses was observed among children exposed to higher O<sub>3</sub> concentrations (Romieu et al. 1992). Children exposed for two consecutive days to peak daily O<sub>3</sub> levels above 260 µg/m<sup>3</sup> had a 20% increase in risk of respiratory illness. For children exposed for 2 consecutive days to high O<sub>3</sub> levels (above 260 µg/m<sup>3</sup>) and the previous day were exposed to low temperature, the risk of respiratory illness reached 40%. It is important to note that in Mexico City, and in some areas of Sao Paulo, levels of 260 µg/m<sup>3</sup> are frequently reached on several consecutive days.

O<sub>3</sub> exposure has also been related to emergency department visits for acute upper respiratory illness among children in Mexico City. An increase of 100 µg/m<sup>3</sup> in the 1-hour daily maximum was related to a 10% increase (95% CI: 7-13%) in upper respiratory illnesses during winter time. An increase of 100 µg/m<sup>3</sup> in the 1-hour daily maximum during 5 consecutive days was related to a 30% increase in upper respiratory illnesses (95% CI: 23-37%) (Tellez-Rojo et al. 1997). In this study a non-linear effect was observed in relation to O<sub>3</sub> levels. The upper respiratory illnesses increased linearly from 160-300 µg/m<sup>3</sup> and then tended to level off. A further increase in risk was observed at levels close to 440 µg/m<sup>3</sup>. Effects at low concentrations of O<sub>3</sub> could not be studied.

Asthmatic children may be more susceptible than others to the effects of O<sub>3</sub> exposure. Studies conducted in Mexico City have shown that asthma-related emergency department visits increased 43% (95% CI: 24-66%) for an increase of 50 ppb in the daily 1-hour maximum O<sub>3</sub> level, with a 1-day lag (Romieu et al. 1995). In this study, peak O<sub>3</sub> concentrations ranged from 20-500 µg/m<sup>3</sup>, with a mean of 180 µg/m<sup>3</sup>.

In panels of asthmatic children, O<sub>3</sub> exposure has been related to a decrease in peak expiratory flow rate and an increase in respiratory symptoms (Romieu et al. 1996; Romieu et al. 1997). In general an increase of 100 µg/m<sup>3</sup> of daily peak O<sub>3</sub> concentrations led to an 11% increase (95% CI: 5-19%) of lower respiratory symptoms and a significant decrease in peak expiratory flow rate.

The decreased respiratory function observed among children exposed to O<sub>3</sub> in Mexico City seems to be smaller than that observed in children who are not chronically exposed to high levels of O<sub>3</sub>, suggesting the existence of a phenomenon of "tolerance". This finding supports studies showing that repetitive exposures tend to produce smaller responses (Hackney et al. 1997; Folinsbee 1991). The potential adverse effect of such "tolerance", or functional adaptation, is not known, but the absence of a protective response to O<sub>3</sub> exposure (bronchoconstriction) could lead to a higher exposure of children and therefore a more severe long-term effect. Experimental studies in animals and humans have shown that O<sub>3</sub> increases airway permeability and particle clearance, causes airway inflammation and a decrease in bacterial capacity, causes structural alteration in the lung and accelerate ageing of the lung (Lippmann 1989; and Section 3.1).

**Western Pacific Region****China**

An investigation has been conducted in China on the effect of short-term O<sub>3</sub> exposure on lung function for male non-smokers. During the test, volunteers undertook a moderate amount of exercise at intervals; and parameters of vital capacity were monitored. The study data showed that under the condition of short-term exposure  $180 \pm 40 \mu\text{g}/\text{m}^3$  is the threshold concentration for acute lung dysfunction; and  $100\mu\text{g}/\text{m}^3$  is the threshold concentration for general malaise (Fang Qisheng et al. 1991).

**Australia**

A time-series analysis of counts of daily hospital admissions and outdoor air pollutants in Sydney (Morgan et al. 1998) found that an increase in the daily maximum 1-hour O<sub>3</sub> concentration was associated with a 2.45% (95% CI: -0.37, 5.35) increase in heart disease admissions among those 65 years and older.

A study of daily mortality in the Brisbane region (Simpson et al. 1997) indicated that O<sub>3</sub> levels (maximum daily O<sub>3</sub> levels were about  $240 \mu\text{g}/\text{m}^3$ ) were significantly associated with total daily mortality. There was little evidence of interaction between the O<sub>3</sub> effects (mainly in summer) and particles or with SO<sub>2</sub> and NO<sub>2</sub>. The associations between O<sub>3</sub> and daily mortality were significant only for individuals who were older than 65 years of age. Positive associations were also found with cardiovascular disease categories and the regression coefficients, when significant, were higher than those for total mortality. The results indicated a possible threshold for O<sub>3</sub> levels.

**Suspended Particulate Matter****Latin America*****Evaluation of the effects of short-term exposure on morbidity and mortality***

Various studies in Latin America have assessed the effect of particulate matter pollution on health. These included mortality studies, and studies of the health effects of particulate matter on respiratory symptoms and functions among children and adults. Studies related to the effects of particulate matter pollution on mortality have been conducted in Brazil, Chile and Mexico. An increase of  $10 \mu\text{g}/\text{m}^3$  PM<sub>10</sub> in Sao Paulo was related to an increase in daily mortality of 3% among adults older than 65 years of age (Saldiva et al. 1995). In Chile, a 0.8% increase (95% CI: 0.6-1.2%) in daily mortality was reported for an increase of  $10 \mu\text{g}/\text{m}^3$  PM<sub>10</sub> (Ostro et al. 1999). In Mexico, a 0.5% increase (95% CI: 0.3-0.7%) in daily mortality was found for a similar increase in daily TSP (Borja-Aburto et al. 1997). These results are concordant with similar studies conducted in other parts of the world (Pope et al. 1995).

Studies conducted to determine the impact of particulate matter pollution on respiratory emergencies and medical visits have also suggested a positive association (Molina Esquivel et al. 1989; Ara-Seebla 1990; Arranda et al. 1994). In a study conducted in Santiago, Chile, respiratory-related emergency visits were related to ambient levels of PM<sub>10</sub> and PM<sub>2.5</sub> during the winter months. In this study, PM<sub>10</sub> levels ranged from 16-270  $\mu\text{g}/\text{m}^3$  and PM<sub>2.5</sub> levels from 10-156  $\mu\text{g}/\text{m}^3$ . It was observed that an increase of 63.5  $\mu\text{g}/\text{m}^3$  in PM<sub>10</sub> (1 quartile of the



distribution) was related to a 2% increase (95% CI: 0.5-3.4%) in respiratory-related emergency department visits, with a 2-day lag during the winter months. A  $36.5 \mu\text{g}/\text{m}^3$  in  $\text{PM}_{2.5}$  was related to a 2.2% increase in the number of emergency department visits for acute respiratory illnesses (95% CI: 0.9-3.6%) with a 2-day lag. A similar increase in  $\text{PM}_{2.5}$  was related to a 5.4% increase in the risk of acute pneumonia (95% CI: 1.9-5.6%) with a 3-day lag, and to a 3.7% increase in the risk of upper respiratory illnesses (95% CI: 1.9-5.6%) with a 2-day lag during winter (Ilabaca Marileo 1996). In this study, the  $\text{PM}_{2.5}$  daily mean ranged from 10-156  $\mu\text{g}/\text{m}^3$ , and the relation appeared to be linear over the range of concentration studied.

The dose-response curves of this study, for emergency department visits of patients with severe and not-so-severe respiratory diseases related to  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ , had smaller slopes than those provided in the WHO Guidelines (Figure 3.7). In fact, for  $\text{PM}_{10}$  the slope fell below the lower confidence limit provided. For  $\text{PM}_{2.5}$ , the slope was considerably smaller than that shown in the WHO Guidelines. However, when the relationship of  $\text{PM}_{2.5}$  with pneumonia-related emergency department visits, a severe respiratory illness, was considered, the slope was larger and above the upper limit of the  $\text{PM}_{10}$  effect predicted by the WHO Guidelines.

Results from a panel study conducted in Puchucavi, Chile, indicated an increase of 5% in cough (95% CI: 1-10%) among children with chronic respiratory symptoms was associated with an increase of 30  $\mu\text{g}/\text{m}^3$  in the 24-hour average levels of  $\text{PM}_{10}$  (Sanchez-Cortez 1997).

Studies conducted in Mexico among asthmatic children have documented an increase in respiratory symptoms and a decrease in lung function related to exposure to  $\text{PM}_{10}$ . During the study, daily  $\text{PM}_{10}$  ambient levels ranged from 29-363  $\mu\text{g}/\text{m}^3$ , with a mean of 167  $\mu\text{g}/\text{m}^3$ , and daily  $\text{PM}_{2.5}$  levels ranged from 23-177  $\mu\text{g}/\text{m}^3$ , with a mean of 86  $\mu\text{g}/\text{m}^3$ . The results suggested that an increase of 10  $\mu\text{g}/\text{m}^3$  in  $\text{PM}_{10}$  levels was associated with a 4% increase in minor respiratory symptoms, and a 0.35% decrease in peak expiratory flow rate (Romieu et al. 1996). In the same study, an increase of 10  $\mu\text{g}/\text{m}^3$  in the  $\text{PM}_{2.5}$  daily mean level was associated with an 8% increase (95% CI: 3-14%) in the incidence of symptoms in the lower respiratory tract. It is important to note that results of this study suggest a synergistic effect of  $\text{PM}_{10}$  and  $\text{O}_3$  exposure on the incidence of symptoms in the lower respiratory tract among these children.

### ***Evaluation of the effects of long-term exposure on mortality and morbidity.***

Few studies have investigated the long-term health effects of particulate matter in Latin America. In a study conducted in Rio de Janeiro, an association was found between the annual average TSP levels in different districts of the city and mortality for pneumonia among infants (Penna and Duchiade 1991). For each 10  $\mu\text{g}/\text{m}^3$  increase in TSP, the infant mortality from pneumonia was estimated to increase by 2.2 per 10,000 population.

Studies conducted in Cubatao, Brazil, have documented the decrease in pulmonary functions among children chronically exposed to high particle levels (Hofmeister 1987; Spektor et al. 1991). Children residing in the most polluted areas had lower pulmonary functions. Studies conducted in Chile (SERPLAC 1989; Arranda et al. 1993) reported a higher incidence of respiratory symptoms and lower pulmonary functions in children resident in Santiago than in a control city. The results suggested an association between cough, nocturnal respiratory symptoms and hoarseness, and  $\text{PM}_{10}$  levels. However, these studies do not provide sufficient data to quantitatively evaluate the risk.

### **Mediterranean Region**

A study showed a significant increase of chest diseases occurred in schoolchildren living in Kafr El-Elwe (a residential settlement close to a cement company) and Helwan City, as compared with those living in Shebin El-Kom, a more rural area (Hussein 1988; Nasralla 1992). It was found that 29.2% of schoolchildren in the first two settlements have obstructive lung diseases compared to only 9% in Shebin El-Kom. Furthermore, the high rate of mortality due to chest and cardiovascular diseases among the population of Helwan and Maadi was related to the prevalence of high concentrations of suspended particles and SO<sub>2</sub> in the atmosphere (Hussein 1988; Nasralla 1992).

### **Western Pacific Region**

#### **Japan**

An epidemiological survey of schoolchildren in Japan showed that the prevalence of asthmatic symptoms, and congestion in chest and phlegm, was significantly correlated with levels of SPM (Nitta et al. 1993; Ono et al. 1990; Nakai et al. 1995). The annual mean concentrations of SPM in urban, suburban and background areas were 45.1-52.7 µg/m<sup>3</sup>, 36.5-43.3 µg/m<sup>3</sup> and 27.8-32.4 µg/m<sup>3</sup>, respectively. The Japanese Environment Agency surveyed the health effects of air pollutants in about 15,000 schoolchildren (EA 1997). The results revealed a correlation between the prevalence of asthmatic symptoms and SPM at annual mean levels of 25-57 µg/m<sup>3</sup> SPM. An epidemiological study in 185 schoolchildren (Shima and Adachi 1996) has shown that children with high IgE levels appear to be particularly susceptible to the effects of automobile exhaust at annual average concentrations of SPM of about 34 µg/m<sup>3</sup>.

A study of the morbidity of allergic rhinitis based on Japan National Health Insurance records showed a three-fold increase in the rate of allergic rhinitis (AR) over 10 years (Miyao et al. 1993). Additionally, results suggested possible correlations between the morbidity of AR and the mean yearly levels of the pollutant components SPM and NO<sub>2</sub>.

#### **China**

Epidemiological studies in China show that under long-term exposure, there is a correlation between particle concentrations and mortality from lung cancer. An investigation based on data for 50 million people in 26 cities showed that the average PM<sub>10</sub> pollution in urban districts and in control districts were 460 µg/m<sup>3</sup> and 220 µg/m<sup>3</sup>, respectively, and the corresponding average mortality from lung cancer was 14.0% and 7.0% (He Xingzhou et al. 1984; Fang Qisheng et al. 1991). The incidence of respiratory diseases, mainly chronic broncho-pneumonia and emphysema, with symptoms of coughing and dyspnea, increased with increasing particle level. Every 100 µg/m<sup>3</sup> increase in TSP concentration led to a 6.75% increase in the incidence of chronic broncho-pneumonia in this coal-burning area. The results showed that exposure to 200 µg/m<sup>3</sup> of TSP can cause upper-respiratory diseases in children; and that 290-470 µg/m<sup>3</sup> of TSP significantly depressed immune functions in children. TSP concentrations less than 160 µg/m<sup>3</sup> had no obvious effect on the incidence of respiratory tract diseases. Another study found that organic extracts from TSP of different sizes had different strengths of mutagenic effects. The smaller the particle, the stronger its mutagenic effects (Li Xiuyun et al. 1992).

Exposure to TSP (with the daily-average concentration below 150 µg/m<sup>3</sup>) produced an increased

frequency of attacks of asthma in some asthma patients. The lung function of children was reduced after short-term exposure to TSP concentrations over  $250 \mu\text{g}/\text{m}^3$ . When TSP concentration were higher than  $750 \mu\text{g}/\text{m}^3$ , middle-aged and old people, people with respiratory disease, and cardiovascular patients exhibited higher mortality (Li Xiuyun et al. 1992).

### Australia

In Sydney, a time-series analysis of counts of daily hospital admissions and outdoor air pollutants (Morgan et al. 1998) showed that an increase in daily maximum 1-hour particle concentration was associated with an increase of 3.01% (95% CI: -0.38% to 6.52%) in hospital admissions for chronic obstructive pulmonary disease. An increase from the 10th to the 90th percentile in daily mean particle concentrations was associated with an increase in heart disease admissions among those 65 years and older of 2.82% (95% CI: 0.90 to 4.77), respectively.

A study of daily mortality in the Brisbane region (Simpson et al. 1997) indicated that the associations between total daily mortality and particle levels that were found in the United States and other countries might also be applicable in Brisbane. The associations between particulate matter and daily mortality were significant only for individuals who were older than 65 years of age; positive associations were also found with cardiovascular disease categories. And the regression coefficients, when significant, were higher than those for total mortality. The results did not indicate a threshold for particle levels.

### Africa

A paucity of data exists in Africa about health effects associated with exposure to specific air pollutants. However, numerous studies in South Africa have indicated associations between a variety of respiratory symptoms and air pollution in urban, industrial and informal settlement areas. For example, high prevalence rates for respiratory illness were found in a residential suburb within an industrial area, relative to a suburb further away. Similarly, when compared with areas using cleaner fuel, raised levels of respiratory effects have been identified in informal settlements, where coal and wood were commonly used for domestic purposes (Opperman et al. 1993; Terblanche et al. 1992; Terblanche et al. 1993).

## **Lead**

### Latin America

Lead is transported to the fetus across the placenta since there is no metabolic barrier to fetal lead uptake. Parental exposure to lead produces toxic effects on the human fetus including reductions in gestational age, birthweight and mental development. A study conducted in Mexico has shown that the concentration of lead in the bone of a mother was significantly related to low birthweight (Gonzalez-Cossio et al. 1997).

The central nervous system is the primary target organ for lead toxicity in children (Needleman and Galsonis 1990), as discussed in Section 3.3. In agreement with these findings a study, conducted in Mexico City among schoolchildren from low-to-medium social status and aged 9-12 years, showed a strong negative correlation between blood lead level, and intellectual coefficients and teacher grading. There was no evidence of a threshold level (Muñoz et al. 1993).

The intensity of vehicular traffic, as a surrogate for exposure to ambient air lead, has been related to blood lead levels. In a study conducted in Mexico, children residing near a road with high traffic volumes had significantly higher levels of lead in blood than did children residing in a residential neighbourhood with smaller traffic volumes (Romieu et al. 1992). In another study conducted in Mexico among two hundred children younger than five years of age, the concentration of lead in ambient air was a significant predictor of blood lead levels (Romieu et al. 1995). The concentration of lead in ambient air (24-hour average) ranged from 0.20-0.52  $\mu\text{g}/\text{m}^3$ . The correlation coefficient between lead in the blood and lead in ambient air was 0.30. It was estimated that for each increase of 1.5  $\mu\text{g}/\text{m}^3$  of lead in ambient air, the concentration of lead in blood would increase by 1  $\mu\text{g}/\text{dl}$ .

### *Africa*

Studies conducted in Johannesburg indicated that approximately 60% of children have blood lead levels exceeding 10  $\mu\text{g}/\text{dl}$ . Children from an informal settlement group, where coal was largely used for cooking purposes, had significantly higher blood lead levels than their inner city and suburban counterparts. In Cape Town, about 13% of coloured pre-school and first-grade children had blood lead levels above 25  $\mu\text{g}/\text{dl}$  (Deveaux et al. 1986; von Schirnding 1989; von Schirnding et al 1991).

## **4 . Indoor Air Quality**

Most people spend a large of their time indoors, which makes indoor spaces important microenvironments when addressing risks from air pollution. Most of a person's daily exposure to many air pollutants comes through inhalation of indoor air, both because of the amount of time spent indoors and because of the higher pollution levels indoors. The air quality inside buildings is affected by many factors. In an effort to conserve energy, modern building design has favoured tighter structures with lower rates of ventilation. By contrast, in some areas of the world only natural ventilation is used; in other areas mechanical ventilation is common. Factors that can have a negative effect on health and comfort in buildings range from chemical and biological pollutants, to occupant perceptions of specific stresses such as temperature, humidity, artificial light, noise and vibration.

Although there is a tendency to use similar types of construction all over the world, especially for office buildings, indoor problems are often different in developed countries when compared with less developed countries. While in the former most of the problems arise from low ventilation rates and the presence of products and materials that emit a large variety of compounds, the inhabitants of many less developed countries face problems related to pollutants generated by human activities, in particular by combustion processes.

If health effects of air pollution are being considered, it does not matter if a pollutant is inhaled by breathing outdoor or indoor air. However, outdoor air has a different pollutant composition than that found in indoor air. Traffic-generated emissions are an example of outdoor air pollution; indoors, pollution sources include tobacco smoke and combustion products generated with biomass-fuelled stoves. Not all of these compositions have been taken into account in developing the air quality guidelines, and they may not be applicable under **all** circumstances, so care should be taken to avoid misinterpretation.

### **4.1 *Indoor air pollution in developed countries***

#### **4.1.1 Important indoor air pollutants and their sources**

Important sources of chemical indoor pollutants include outdoor air, the human body and human activities, emissions from building materials, furnishings and appliances and use of consumer products. Microbial contamination is mostly related to the presence of humidity. The heating, ventilating and air conditioning system can also act as a pollutant source, especially when it is not properly maintained. For example, improper care of filters can lead to re-emission of particulate contaminants. Biological contamination can proliferate in moist components of the system and be distributed throughout the building.

Indoor air pollutants can be classified in different ways. One approach is to divide them into chemical, physical and biological agents. Another approach is to classify them according to their origin. The origin of a particle has an important impact on its composition, which may include chemical and biological agents besides the physical nature of the particle itself. For example, combustion-generated tobacco smoke contains a complex mixture of pollutants.

The sources of indoor air pollution and the principal pollutants, grouped by outdoor and indoor

origin, are summarized in Table 4.1. This is not a complete listing of all sources of indoor air pollutants, as there is continuous air exchange between indoors and outdoors, and most pollutants present in the outdoor air are also found indoors. Moreover, indoor sources may lead to an accumulation of some compounds that are rarely present in the ambient air. The most important compounds in indoor air environments include SPM, SO<sub>2</sub>, NO<sub>x</sub>, CO, photochemical oxidants and lead. In developed countries, pollutant concentrations indoors are similar to those outdoors, with the ratio of indoor to outdoor concentration falling in the range 0.7-1.3. Concentrations of combustion products in indoor air can be substantially higher than those outdoors when heating and cooking appliances are used. This is particularly true in developing countries where ovens and braziers are used with imperfect kitchen and stove designs.

**Table 4.1. - Principal pollutants and sources of indoor air pollution, grouped by origin**

<b>Principal pollutants</b>	<b>Sources, predominantly outdoor</b>
SO <sub>2</sub> , SPM/RSP	Fuel combustion, smelters
O <sub>3</sub>	Photochemical reactions
Pollens	Trees, grass, weeds, plants
Pb, Mn	Automobiles
Pb, Cd	Industrial emissions
VOC, PAH	Petrochemical solvents, vaporization of unburned fuels
<b>Principal pollutants</b>	<b>Sources both indoor and outdoor</b>
NO <sub>x</sub> , CO	Fuel burning
CO <sub>2</sub>	Fuel burning, metabolic activity
SPM & RSP	Environmental tobacco smoke, resuspension, condensation of vapours and combustion products
Water vapour	Biological activity, combustion, evaporation
VOC	Volatilization, fuel burning, paint, metabolic action, pesticides, insecticides, fungicides
Spore	Fungi, moulds
<b>Principal pollutants</b>	<b>Sources, predominantly indoor</b>
Radon	Soil, building construction materials, water
HCHO	Insulation, furnishing, environmental tobacco smoke
Asbestos	Fire-retardant, insulation
NH <sub>3</sub>	Cleaning products, metabolic activity
PAH, Arsenic, Nicotine, Acrolein	Environmental tobacco smoke
VOC	Adhesives, solvents, cooking, cosmetics
Mercury	Fungicides, paints, spills or breakage of mercury-containing products
Aerosols	Consumer products, house dust
Allergens	House dust, animal dander
Viable organisms	Infections

Adapted from Suess 1992; WHO 1995i.

#### **4.1.2 Concentrations of indoor air pollutants**

Indoor concentrations of air pollutants are influenced by outdoor levels, indoor sources, the rate of exchange between indoor and outdoor air, and the characteristics and furnishings of buildings. Indoor concentrations of air pollutants are subject to geographical, seasonal and diurnal variations.

In developed countries indoor levels of NO<sub>2</sub>, for example, are affected by gas heaters and cooking ranges (used in 20-80% of houses in some countries). In five European countries, the average NO<sub>2</sub> concentrations (over 2-7 days) were in the range of 20-40 µg/m<sup>3</sup> in living rooms and 40-70 µg/m<sup>3</sup> in kitchens, for dwellings with gas equipment and 10-20 µg/m<sup>3</sup> in dwellings without gas equipment. These values may be doubled in rooms facing streets with heavy motor traffic. These exposure levels may have an effect on respiratory function, as discussed in Chapter 3. People may be exposed to higher NO<sub>2</sub> levels under certain circumstances, such as in dwellings equipped with unvented cooking ranges. In addition, short-term measurements reveal NO<sub>2</sub> concentrations that may be five-fold higher than those averaged over several days. Peak values of up to 3800 µg/m<sup>3</sup> for 1 minute have been measured in the Netherlands in kitchens with unvented gas cooking ranges (ECA 1989; Seifert 1993).

In general, average short-term CO concentrations at kerbside locations in developed countries are about 60 mg/m<sup>3</sup> for 30 minutes or 30 mg/m<sup>3</sup> for 1 hour. In kitchens with gas stoves, short-term values of up to 15 mg/m<sup>3</sup> have been measured. High values were also measured in bars and pubs, where smoking is common, with average concentrations of 10-20 mg/m<sup>3</sup> and peak levels up to 30 mg/m<sup>3</sup> (Seifert 1993).

In five developed European countries HCHO concentrations in indoor air were reported to range from 9-70 µg/m<sup>3</sup>. Higher values are occasionally encountered, especially in dwellings with urea-formaldehyde foam insulation (ECA 1990).

In general, average indoor levels of radon are 20-70 Bq/m<sup>3</sup> (ECA 1995), although they may be ten times higher in certain areas.

Exposure to environmental tobacco smoke is an important factor in indoor air quality assessment. The particle and vapour phases of environmental tobacco smoke are complex mixtures of several thousand chemicals, including known carcinogens such as nitrosamines and benzene. One of the most commonly used indicators of environmental pollution by tobacco smoke is the concentration of PM<sub>10</sub>. This is 2-3 times higher in houses with smokers than in other houses (Schwartz and Zeger 1990). Nicotine is present in the vapour phase, with concentrations of up to 10 µg/m<sup>3</sup> in houses with smokers. Data from nine European countries revealed that 33-66% of households had at least one smoker. The proportion of children with mothers smoking at home varied from 20-50%, and the proportion of children with fathers smoking at home ranged from 41-57%. Tobacco smoke, and particularly the exposure of children, is therefore a major problem for indoor air quality and environmental health.

#### **4.1.3 Health effects and symptoms**

Most indoor air pollutants directly affect the respiratory and cardiovascular systems, and have been discussed in detail in Chapter 3. In this section, health effects of indoor air pollutants not discussed in Chapter 3 will be summarized.

The direct human health effects of indoor air pollution on the respiratory system vary according to both the intensity and the duration of exposure, and also with the health status of the population exposed. Certain parts of the population may be greater risk, for example, the very young and elderly, those already suffering from respiratory disease, hyper-responders and people exercising.

The active and passive inhalation of tobacco smoke can lead to reduced pulmonary function, to an increased incidence of respiratory symptoms and infections, and to an increased incidence of lung cancer.

Inhalation of infectious microorganisms discharged by people and animals is a primary mechanism of contagion for most acute respiratory infections. In indoor environments characterized by reduced ventilation and increased use of untreated recirculated air concentrations of microorganisms may increase.

Outdoor allergens, house dust mites, and moulds in indoor environments of high humidity can cause allergic asthma (reversible narrowing of lower airways), allergic rhinoconjunctivitis in children and young adults, and recurrent bouts of pneumonitis or milder attacks of breathlessness.

The main acute effects of HCHO include odour perception and irritation of eyes, nose and throat. Discomfort, lacrimation, sneezing, coughing, nausea and dyspnea have also been observed, depending on the HCHO concentration.

Health effects reported for VOC range from sensory irritation to behavioural, neurotoxic, hepatotoxic and genotoxic effects. Concentrations at which identified health effects occur are usually much greater than those measured in indoor air. Exposure to mixtures of VOC may be an important cause of Sick Building Syndrome (SBS).

Asbestos and other mineral fibres may be a cause of an increased incidence of lung cancer. Acute exposure to asbestos and glass fibres can cause severe skin irritation.

More complex health effects are SBS and Building Related Illnesses (BRI). SBS is the occurrence of specific symptoms with unspecified aetiology, and are experienced by people while working or living in a particular building, but which disappear after they leave it. Symptoms include mucous membrane, skin and eye irritation, chest tightness, fatigue, headache, malaise, lethargy, lack of concentration, odour annoyance and influenza symptoms. SBS usually cannot be attributed to excessive exposure to known contaminant or to a defective ventilation system. A number of factors may be involved:

- Physical factors, including temperature, relative humidity, ventilation rate, artificial light, noise and vibration,
- Chemical factors, including environmental tobacco smoke, HCHO, VOC, pesticides, odorous compounds, CO, CO<sub>2</sub>, NO<sub>2</sub> and O<sub>3</sub>.
- Biological and psychological factors.

It is assumed that the interaction of several factors, involving different reaction mechanisms, cause the syndrome, but there is yet no clear evidence of any exposure-effect relationship. BRI is an illness related to indoor exposures to biological and chemical substances (e.g. fungi, bacteria, endotoxins, mycotoxins, radon, CO, HCHO). It is experienced by some people working or living in a particular building and it does not disappear after leaving it. Illnesses include respiratory tract infections and diseases, legionnaires' disease, cardiovascular diseases and lung cancer.



## **4.2    *Indoor air quality in less-developed countries***

Air quality in buildings in developing countries can have similar problems to those found in developed countries, particularly in the large modern urban areas in developing countries. As smoking rates in developing countries increase, exposure to environmental tobacco smoke can also be expected to increase. In addition, some hazardous materials, particularly pesticides, are becoming so widely used in developing countries that there may be higher indoor exposures than in developed countries.

There can be significant and widespread indoor exposures to many of the classical air pollutants, specifically sulphur dioxide, particulate matter, carbon monoxide, and nitrogen dioxide, in developing countries. A particular issue for developing countries is exposure to emissions from cooking and heating which may produce the highest air pollution exposures to many pollutants. Today about half the population of the world continues to rely for cooking and associated space heating on simple household stoves using unprocessed solid fuels that have high emission factors for a range of health-damaging air pollutants. This section briefly summarizes what is known about the emissions, exposures, and health effects. Possible ways of managing the problems are discussed in Chapter 6.

### **4.2.1    Emissions**

Although part of human experience since the first controlled use of fire, air pollution from simple open combustion of biomass has been scientifically characterized only in the last two decades, largely due to rising concerns about woodsmoke pollution in developed countries. Studies have shown high emission factors for many important pollutants, including respirable particulate matter, carbon monoxide, polycyclic aromatic hydrocarbons, such as benzo-a-pyrene, and volatile organic compounds, such as formaldehyde and benzene. Biomass fuels emit hundreds of chemicals during small-scale combustion, such as in household cooking or heating stoves (Smith 1987).

By comparison to modern cooking fuels, such as kerosene and gas, unprocessed solid fuels produce 10-100 times more respirable particulate matter per meal as the result of low (combustion and heat-transfer) efficiencies. Although biomass makes up only 10-15% of total human fuel use, compared to modern fuels a much larger fraction is burned indoors, since nearly one-half of humanity cooks and/or heats with simple stoves burning traditional biomass fuels (WHO 1997a).

Household use of coal is common in China and Eastern Europe. In Eastern Europe, coal is used mainly for heating in devices and emissions are vented outdoors, a process usually resulting in less human exposure than from using coal for cooking.

### **4.2.2    Concentrations**

It is not known what fraction of biomass-burning households cook indoors on unvented stoves, although it is clear that many hundreds of millions do so during some or all seasons of the year. There is also little information about the ventilation rates in the many thousands of housing types in developing countries or countries in transition.

Unfortunately, relatively little monitoring has been done in these indoor environments and none of it has been done in a way to provide statistically valid samples of large populations. The results that have been obtained, nevertheless, are striking. Table 4.2 for example, lists results for particulate matter in indoor air obtained in a number of indoor air quality studies. Other classical pollutants also reach significant levels in these circumstances.

Important non-classical pollutants, such as formaldehyde, polycyclic aromatic hydrocarbons, benzene, and 1,3-butadiene also have been found to reach levels much higher than any but occupational settings in developing countries. In some areas of China and India, household coal use leads to high indoor concentrations of fluorine and arsenic with consequent health effects.

#### **4.2.3 Exposures**

Population exposure to an air pollutant is defined here as the simple combination of the concentration of the pollutant in air being inhaled, the duration of time over which it is inhaled, and the number of people exposed. As half the households in the world use solid fuels on a daily basis and it is activities such as cooking that generate most indoor emissions, there is a confluence of emissions, people, and time in places which may have relatively little ventilation. Consequently, globally there are high levels of indoor exposure to emissions from solid fuels (Smith 1993).

These high exposures are suggested by the data on personal exposure concentrations experienced by women during cooking over solid fuel stoves listed in Table 4.3 Global particulate concentrations and exposures in urban and rural outdoor and indoor environments are shown in Table 4.4.

#### **4.2.4 Health Effects**

Relatively few studies have been conducted to determine the health effects of indoor exposures to air pollutants in developing countries. Enough data has become available in recent years, however, to obtain some preliminary information on the type and very approximate magnitude of effects (Chen et al. 1990).

The following categorizes some major categories of effects where there is reasonable evidence from smoking studies, urban air studies, and multiple studies of solid-fuel use in developing countries. Also listed, where known, are the apparent odds ratios comparing the risk of these diseases between people living in houses using unvented biomass fuel and similar households not using such fuels. All the odds ratios reported here are statistically significant results, mostly of multivariate analyses in which a number of potentially confounding variables were included:

Acute respiratory infections in children: This is the chief cause of ill-health in the world and strongly associated with indoor use of solid fuels for cooking in a number of studies in Asia and Africa (OR = 2 - 6) (e.g., Pandey et al. 1989; Collings et al. 1990; Mtango et al. 1992; O'Dempsey et al. 1996).

Chronic obstructive pulmonary disease: This has been shown to be strongly associated with use of solid fuels in non-smoking women often along with *cor pulmonale* in studies from Latin America, South Asia and Saudi Arabia (OR = 3.4-15) (e.g., Dennis 1996; Dossing et al. 1994;

Pandey 1984; Sandoval et al. 1993; Albalak et al. 1999)

Lung cancer: Lung cancer has been shown in many Chinese studies to be statistically associated with use of coal for cooking and heating, but not biomass fuels (OR = 3-9) (Smith and Liu 1994; Shields et al. 1995).

There is some evidence from studies of solid-fuel use in developing countries indicating a relationship between adverse pregnancy outcomes, the third most important category of ill-health in the world, and smoke exposure. After multivariate analyses, stillbirth has been associated with biomass fuel use by pregnant women in one Indian study (OR = 1.5) (Mavalankar et al. 1991) and with low birth-weight in Guatemala (Boy et al. 1999). After multivariate analyses, TB and blindness (cataracts) have been shown to be related to use of biomass fuels in two national and two local studies in India (Mishra et al. 1999a; Gupta et al. 1997; Mishra et al. 1999b; Mohan et al. 1989). Unfortunately all these studies relied on the type of stove or fuel as the indicator of pollution. More studies are needed that measure concentrations and exposures to indoor air pollutants so that exposure-response relationships can be more firmly determined.

#### **4.2.5 Application of air quality guidelines to indoor air pollutant exposure**

The magnitude and population distribution of indoor air pollution exposure from unvented solid fuel use tends to differ from the outdoor urban air pollution exposures that have been the basis of most of the health effects research cited in Chapter 3. In many situations, for example, exposure levels may be high during cooking periods, with relatively low exposures between cooking periods.

##### **Classical gaseous pollutants**

All of the classical gaseous pollutants except ozone can be found in indoor solid fuel smoke and these can be a health concern in households with poor ventilation. Although there have been relatively few measurements of gaseous pollutants in developing countries, emissions estimates from solid fuel burning suggest that levels exceeding the air quality guidelines may be widespread in developing countries (WHO 1992c; WHO 1997a).

##### **Particulate matter**

The WHO air quality guidelines and most other particulate matter standards do not specify the chemical composition of particles. However, the health effects may vary with differences in particle compositions (see Section 2.4). Most of the epidemiological studies used to derive the air quality guidelines for particulate matter were conducted in cities where fossil-fuel particulate matter dominated and some even had significant contributions from coal burning, sometimes at household scale. Thus, it is important to consider the chemical composition of indoor air particulate matter when considering health effects of emissions from solid fuel combustion.

Very high concentrations of particles in indoor air can occur, sometimes for short duration, such as during cooking over solid fuel fires in rooms with poor ventilation. As discussed in Section 2.4, extrapolations of the air quality guidelines health impacts slope for particulate matter beyond 150 µg/m<sup>3</sup> PM<sub>10</sub> must be done with extreme care because there may be a flattening of the exposure/response slope at higher exposure concentrations.

Although some epidemiological studies of particle air pollution were conducted in cities with significant emissions from woodsmoke during some seasons, there is insufficient information to consider the applicability of the new air quality guideline for particulate matter to biomass smoke. Many researchers believe that the chemical composition of fresh biomass smoke from open fires is too different from the aged fossil-fuel particulate matter upon which most of the epidemiological studies have been based to make such an extrapolation with current knowledge. At this stage, no judgment can be made about whether biomass particulate matter is less or more unhealthy than the same exposure concentration of urban outdoor particulate matter, but only that they may induce a different response because of their different composition. Thus, even though it is clear from the existing epidemiological literature that significant ill-effects do occur, it is not possible at this point to be confident about the precise exposure-response relationships.

Tobacco smoke is a fresh biomass smoke, which has been studied far more than any other pollutant mixture. In the form of ETS, it is associated with adverse health impacts in adults and children at particle concentrations similar to those at which the epidemiological studies of health effects of outdoor particulate matter have been conducted (Section 3.4). Even though it is not clear whether particulate matter is the best single measure by which to characterize ETS, the large health impact at concentrations commonly found leads to the conclusion that no level above zero could be considered acceptable (see Section 3.4). It should also be kept in mind that exposure to ETS and other air pollutants can act synergistically to produce adverse health effects (WHO 1999c).

There are similarities between ETS and biomass smoke from stoves, as hundreds of the organic compounds they both contain are similar. This supports evidence that exposure to biomass smoke from open stoves causes considerable human ill-health world wide. Nevertheless, until more evidence becomes available from studies done in biomass-using households, it is considered prudent not to extrapolate the guidelines described for particulate matter in section 3.1 to higher PM concentrations but rather use a conservative approach or alternatively apply the 1987 Air Quality Guidelines for particulate matter (WHO 1987).

**Table 4.2. Indoor particle air pollution from biomass combustion in developing countries: partial list of studies measuring area concentrations (Smith 1996).**

Country	Year of publication	Description of sample	Concentration [mg/m <sup>3</sup> ]
Papua New Guinea	1968	n=9, overnight, floor level	5200
	1974	n=6, overnight, sitting level	1300
Kenya	1971/2	n=8, overnight, highlands/lowlands	4000/800
	1988	n=64, 24 h, thatched/iron roof	1300/1500 (R)
India	1982	n=64, 30 min, wood/dung/charcoal	15,800/18,300/5500
	1988	n=390, cooking, 0.7m/ceiling	4000/21,000
	1992	n=145, cooking/non-cooking/living	5600/820/630
	1994	n=61, 24 h, ag-resid/wood	2800/2000 (I)
	1995	n=50, breakfast/lunch/dinner	850/1250/1460 (I)
	1996	n=136, urban, cooking/sleeping	2860/880 (I)
Nepal	1986	n=17, 2 h	4400 (I)
China	1986	n=64	2570
	1987	n=4, 8 h	10,900 (I)
	1988	n=9, 2 houses, 12 h	2900
	1988	n=12, 4 houses, dung	3000 (I)
	1990	15 houses, dung, winter/summer	1670/830 (I)
	1991	straw, avg summer-winter, kitchen/living room/dung	1650/610/1570 (I)
	1991	1-story/2-story houses	80/170
	1993	4 kitchens	1060 (I)
Gambia	1988	n=36, 24 h, dry/wet season	2000/2100 (I)
Zimbabwe	1990	n=40, 2 h	1300 (I)
Brazil	1992	n=11, 2-3 h, trad/impr	1100/90 (I)
Guatemala	1993	n=44, 24 h, trad/impr	1200/530 (I)
	1996	n=18, 24 h, trad/impr	720/190 (I), 520/90 (R)
	1996	n=43, 24 h, trad/impr	870/150 (R)
South Africa	1993	n=20, 12 h, kitchen/bedroom	1720/1020
Mexico	1995	n=31, 9 h	335 (R)/439 (I)

(Woodfuel, rural, and TSP unless otherwise stated; I=inhalable=cutoff at approx. 10mm; R=respirable=cutoff at 5mm or smaller; Trad/impr=traditional open stove compared to improved stove with flue)

**Table 4.3. Indoor particle air pollution from biomass combustion in developing countries: partial list of studies of individual breathing area concentrations (women during cooking, unless otherwise stated) (Smith 1996).**

Country	Year of publication	Description of sample	Concentration [mg/m <sup>3</sup> ]
India	1983	n=65, 4 villages	6800
	1987	n=165, 8 villages	3700
	1987	n=44, 2 villages	3600
	1988	n=129, 5 villages	4700
	1991	n=95, winter/summer/monsoon	6800/5400/4800
	1996	n=40, two urban slums, infants, 24 h	400/520 (I)
Nepal	1986	n=49, 2 villages	2000
	1990	n=40, trad/impr	8200/3000
Zambia	1992	n=184, 4 h, urban, wood/charcoal	470/210 (R)
Ghana	1993	n=143, 3 h, urban, wood/charcoal	590/340 (R)
South Africa	1993	n=15, 12 h, children, winter/summer	2370/290

(Woodfuel, rural, and TSP unless otherwise stated; I=inhalable=cutoff at approx. 10mm; R=respirable=cutoff at 5mm or smaller; Trad/impr=traditional open stove compared to improved stove with flue)

**Table 4.4. Particle concentrations and exposures in the eight major global microenvironments (Smith 1996).**

Region	Concentrations		Exposures		
	Indoor (µg/m <sup>3</sup> )	Outdoor (µg/m <sup>3</sup> )	Indoor (%)	Outdoor (%)	TOTAL (%)
<u>Developed</u>					
Urban	100	70	7	1	7
Rural	80	40	2	0	2
<u>Developing</u>					
Urban	250	280	25	9	34
Rural	400	70	52	5	57
		TOTAL (%)	==	86	14
					100

Note: Population exposures expressed as a percentage of the world total. Here exposure is defined to equal to the number of people exposed multiplied by the duration of exposure and the concentration breathed during that time.

## 5. Ambient air quality monitoring and assessment

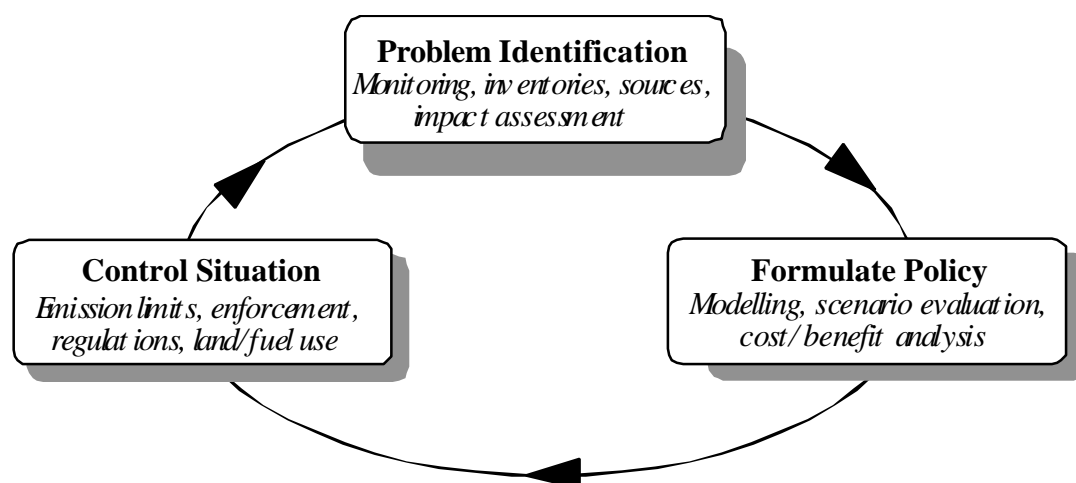
### 5.1 Assessment tools and functions

This chapter reviews some of the methodologies and systems used for the assessment of ambient air quality, with particular reference to the requirement for population exposure assessment and for determining compliance with standards or guidelines. The pollutants considered in detail are, SO<sub>2</sub>, NO<sub>2</sub>, CO, O<sub>3</sub>, SPM and lead. These have a variety of potentially acute and chronic population health impacts, discussed in Chapter 3. Accordingly, the evaluation of air quality against guidelines may need to consider a range of time scales for effects, ranging from 10 minutes (SO<sub>2</sub>) to one year (NO<sub>2</sub>, SO<sub>2</sub>, lead).

The three main air quality assessment tools are:

ambient monitoring  
models  
emission inventories/measurement

The ultimate purpose of monitoring is not merely to collect data, but to provide the information necessary for scientists, policy makers and planners to make informed decisions on managing and improving the environment. Monitoring fulfils a central role in this process, providing the necessary sound scientific basis for policy and strategy development, objective setting, compliance measurement against targets and enforcement action (Figure 5.1).



**Figure 5.1. The Role of Monitoring in Air Quality Management**

However, the limitations of monitoring should be recognized. In many circumstances, measurements alone may be insufficient -or impractical- for the purpose of fully defining population exposure in a city or country. No monitoring programme, however well funded and designed, can hope to comprehensively quantify patterns of air pollution in both space and time. At best, monitoring provides an incomplete - but useful - picture of current environmental quality. Monitoring therefore often needs to be used in conjunction with other objective assessment techniques, including modelling, emission measurement and inventories,

interpolation and mapping. These are discussed in greater detail in Chapter 6.

Conversely, reliance on modelling alone is not recommended. Although models can provide a powerful tool for interpolation, prediction, and optimization of control strategies, they depend on the availability of reliable emission data. A complete inventory for a city or country may need to include emissions from point, area and mobile sources; in some circumstances, assessment of pollutants transported into the area under study may also need to be considered. It is important, also, that the models utilized are appropriate to local conditions, sources and topography, as well as being selected for compatibility with available emission and meteorological datasets.

Inventories will, for the most part, be estimated using emission factors appropriate to the various source sectors (verified by measurement), and used in conjunction with surrogate statistics such as population density, fuel use, vehicle kilometres or industrial throughput. Emission measurements will usually only be available for large industrial point sources, or from representative vehicle types under standardized driving conditions.

All three assessment tools are interdependent in scope and application. Accordingly, monitoring, modelling and emission assessments should be regarded as complementary components in any integrated approach to exposure assessment or determining compliance against air quality criteria. Thus, for a reasonably complete picture of population exposure, ambient monitoring data will need to be supplemented by corresponding information from microenvironment and individual exposure surveys. This chapter focuses on ambient monitoring techniques and systems. Historically, these have provided most of the data used for exposure assessment. Recent publications have dealt in some detail with microenvironment and individual exposure monitoring (WHO 1999a). These issues are discussed in Chapter 4.

## **5.2 *Monitoring objectives***

The first step in designing or implementing any monitoring system is to define its overall objectives. Setting diffuse, overly restrictive or ambitious monitoring objectives will result in cost-ineffective programmes with poor data utility. In such circumstances, it will not be possible to make optimal use of the available manpower and resources. Thus it is vital that clear, realistic and achievable monitoring objectives be set. This enables appropriate Data Quality Objectives (DQOs) to be defined (Box 5.2). In turn, this makes it possible for a targeted and cost-effective Quality Assurance Programme (QAP) to be developed. Overall requirements for such a programme are addressed in outline in section 5.3. A clear definition of overall monitoring objectives and DQOs is therefore essential to enable networks to be optimally designed, priority pollutants and measurement methods to be selected, and data management/reporting requirements to be identified (Figure 5. 2).

The relationships between the data collected and the information to be derived from it must be taken into account when a monitoring programme is planned. This emphasizes the need for users and potential users of the data to be involved in the planning of surveys, not only to ensure that they are appropriate to their needs, but also to justify the resource commitment. It should be recognized that monitoring networks are invariably designed for a variety of functions. These may include policy and strategy development, local or national planning, measurement against international standards, identification/quantification of risk and public awareness. Typical



monitoring functions are summarized in Box 5.1. Every monitoring survey or network is therefore different, being influenced by a unique mix of local/national issues and objectives.

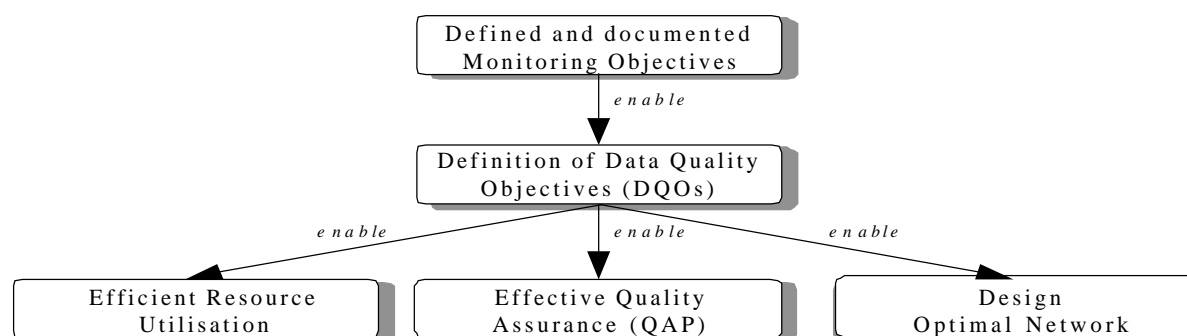
#### **Box 5.1 - Key Monitoring Objectives**

- Determining population exposure and health impact assessment.
- Informing the public about air quality and raising awareness.
- Identifying threats to natural ecosystems.
- Determining compliance with national or international standards.
- Providing objective inputs to Air Quality Management, traffic and land-use planning.
- Source apportionment and identification.
- Policy development and prioritisation of management actions.
- Development/validation of management tools (models, Geographical Information Systems etc.).
- Assessing point or area source impacts.
- Trend qualification, to identify future problems or progress against management/control targets.

#### **Box 5.2 - Data Quality Objectives**

*The essential requirements to be met by measurements, if overall monitoring objectives are to be achieved.*

- Measurement accuracy and precision.
- Traceability to metrology standards.
- Temporal completeness (data capture).
- Spatial representativeness and coverage.
- Consistency - from site to site and over time.
- International comparability/harmonization.



**Figure 5.2 The Importance of Objective Setting**

### 5.3 *Quality assurance and quality control (QA/QC)*

Quality assurance and control (QA/QC) is an essential part of any air monitoring system. It is a programme of activities that ensures that measurements meet defined and appropriate standards of quality, with a stated level of confidence. It should be emphasized that the function of QA/QC is not to achieve the highest possible data quality. This is an unrealistic objective, which cannot be achieved under practical resource constraints. Rather, it is a set of activities, which ensures that measurements comply with the specific DQOs for the monitoring programme. In other words, QA/QC ensures that data are fit for the purpose. Major QA/QC objectives are summarized in Box 5.3, whilst the functional components of a QA/QC programme are identified in Box 5.4.

Quality assurance activities cover all pre-measurement phases of monitoring, including determining monitoring and data quality objectives, system design, site selection, equipment evaluation and operator training. Quality control functions affect directly measurement-related activities such as site operation, calibration, data management, field audits and training. The successful implementation of each component of a QA/QC scheme is necessary to ensure the success of the complete programme. QA/QC may be regarded as a chain of activities designed to deliver credible and accurate data, but a chain is only as strong as its weakest link!

#### ***Box 5.3 - QA/QC for Air Monitoring: overall objectives***

- Measurements accurate, precise and credible.
- Data representative of ambient or exposure conditions.
- Results comparable and traceable.
- Measurements consistent over time.
- High data capture, evenly distributed.
- Optimal use of resources.

#### ***Box 5.4 - QA/QC for Air Monitoring: the major components***

- |                          |   |
|--------------------------|---|
| <i>Quality Assurance</i> | <ul style="list-style-type: none"> <li>• Definition of monitoring and data quality objectives.</li> <li>• Network design, management and training systems.</li> <li>• Site selection and establishment.</li> <li>• Equipment evaluation and selection.</li> <li>• Routine site operations.</li> </ul> |
| <i>Quality Control</i>   | <ul style="list-style-type: none"> <li>• Establishment of calibration/traceability chain.</li> <li>• Network audits and inter-calibrations.</li> <li>• System maintenance and support.</li> <li>• Data review and management.</li> </ul>  |

Although the main principles of QA/QC system design apply to most network or instrumentation types, there are often characteristic differences in their emphasis and practical implementation. It is a common oversight to place too much emphasis on laboratory-based quality assurance activities, as these are often easier to control and monitor.

Although such QA/QC tasks are vital, particularly for sampler-based measurement programmes involving substantial laboratory analysis, considerable emphasis in any network quality system needs to be focused on the point of measurement. Mistakes or problems at the start of the measurement chain cannot be readily corrected afterwards. Sample system design and maintenance (see Section 5.4.3), regular site visits, audits and inter-calibrations therefore play an important role in network quality assurance.

Another unifying feature of network quality systems is the need for effective data screening and validation. In any measurement programme -however well designed or operated- equipment malfunction, human error, power failures, interference and a variety of other disturbances may result in the collection of spurious data. To maximize data integrity and utility, therefore, these must be identified and removed before a final, definitive dataset can be generated or used.

The design of an effective and targeted QA/QC programme is only the first step in the process of quality management. The programme needs to be fully documented, and compliance with its procedures and requirements actively monitored. Monitoring programmes often evolve over time as objectives, legislation, resources or air pollution problems change. Quality assurance programmes therefore also need to be regularly reviewed, to ensure that they remain properly targeted and fit for purpose.

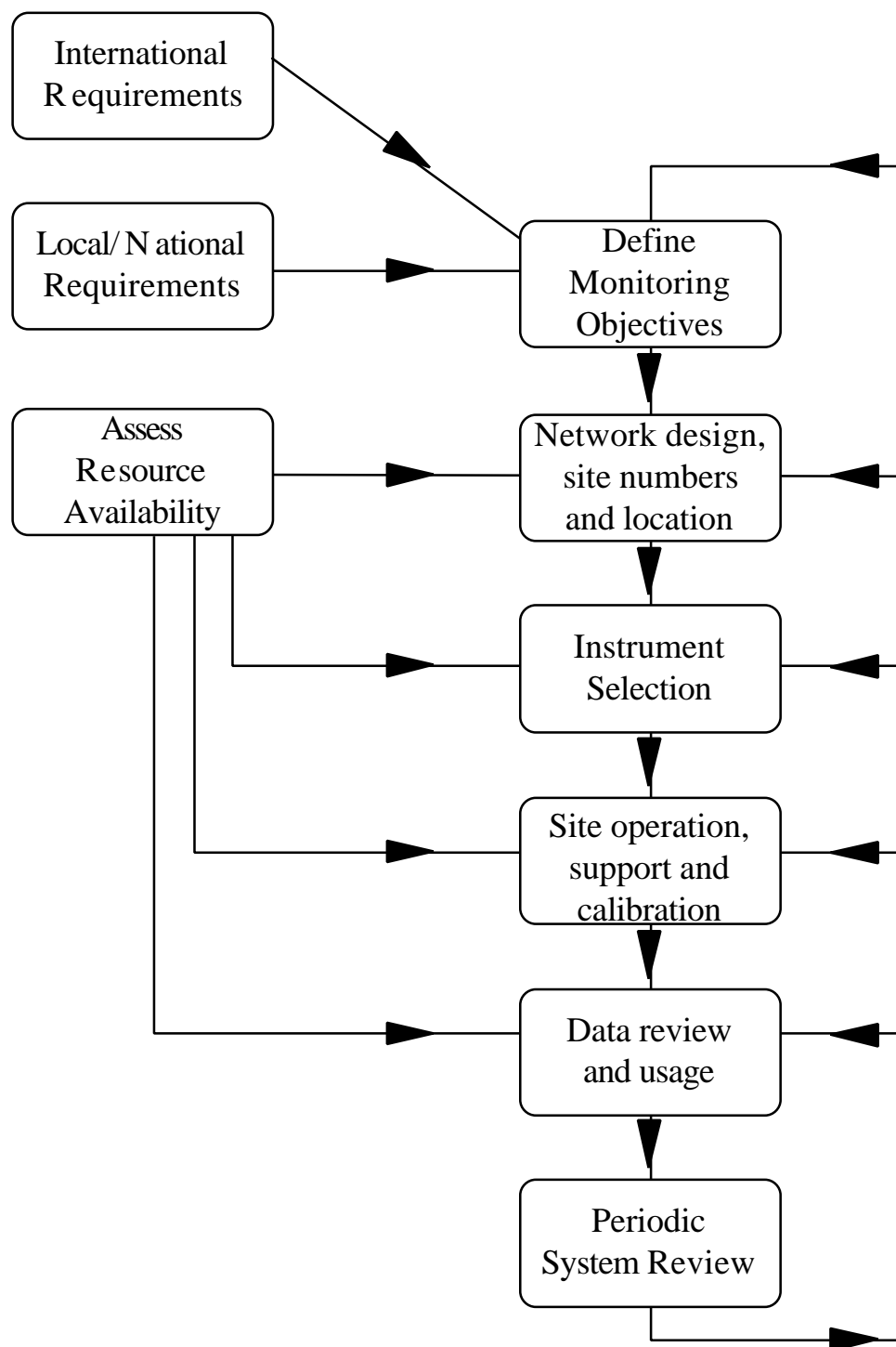
A step-by-step model for the development and implementation of QA/QC programmes for air monitoring is depicted in Figure 5.3. QA/QC systems are considered in greater detail elsewhere (UNEP/WHO 1994a; Bower 1997).

## **5.4 Network design**

There are no universal rules for network design, since any decisions will be determined ultimately by the overall monitoring objectives and resource availability. Although monitoring systems can have just a single, specific objective, it is more common for them to have a broad range of targeted programme functions. No survey design can hope to completely address all the possible monitoring objectives listed in Box 5.1. However, the design of surveys to meet these individual requirements often has common features, and can use common data (to avoid duplication of effort) and overlapping data to verify the credibility of results and conclusions. The overall design goal is to ensure that the maximum information can be derived from the minimum effort. In some countries, networks may be operated by a variety of organisations, including different Government Departments. In such a circumstance, harmonization of the programmes and sharing of data is vital to avoid unnecessary effort and to maximize overall cost-effectiveness.

### **5.4.1 Resource constraints and issues**

A key issue, which needs to be addressed at a very early stage of the network design process, is that of resource availability (Box 5.5). In practice, this is usually the major determinant in network design, which will exert a particularly strong influence on the choice of site numbers, pollutants to be monitored and instrumentation selected.



**Figure 5.3. QA/QC for Air Monitoring: a Step-by-step Approach**

A wide range of commitments and costs is likely to be incurred in any air monitoring programme. Some of these are listed in Box 5.6. Before any firm capital or resource commitment, it is therefore essential to plan the survey, assess resource availability, select the most appropriate equipment and choose monitoring sites.

**Box 5.5 - Network Design: important resource constraints**

- \$ money (capital and ongoing).
- ☺ skilled manpower.
- 🕒 time.

When any equipment purchase must be made, consideration is needed of its long-term operational or financial sustainability. Local sustainability requires the continuing availability of agents (or an in-house capability) for repair and maintenance, together with the necessary skill-base for routine equipment operation and calibration. Financial sustainability requires an ongoing budget for equipment operation, typically amounting to about 10% per annum of the initial capital expenditure.

**Box 5.6 - Costs of Air Monitoring**

- Capital purchase of analysers, samplers, site and laboratory infrastructure.
- Equipment service, maintenance and repair.
- Staff and subcontractor costs - operational and management.
- QA/QC audits, intercalibrations, training, data management.
- Running costs - site rental, electricity, consumables, spare parts, calibration gases, telephone, lab analysis, transport etc.

An ongoing resource commitment to QA/QC is also required in any monitoring survey or network, to ensure that its measurement quality and availability are fully consistent with overall programme objectives. Typically, a budget of between 20-40% of the total annual operating costs may be appropriate for this purpose, depending on the complexity of the programme and the stringency of its DQOs.

#### **5.4.2 Site numbers and selection**

For the purposes of designing a network to assess population exposure and compliance with health guidelines, a number of basic issues need to be addressed (Box 5.7).

**Box 5.7 - Compliance Monitoring- basic issues**

- Where is the population?
- What pollutant concentrations are they exposed to?
- ... and for how long?
- In what areas and micro-environments is exposure important?

In practice, the number and distribution of air quality monitoring stations required in any network, or the number of samplers used in a survey, also depend on the area to be covered, the spatial variability of the pollutants being measured and the required data usage (Box 5.8).

**Box 5.8 - Network Design: Site Numbers**

*Will depend on:*

- required data use/objectives.
- area to be covered.
- spatial variability of pollutants.
- resource availability.
- instruments deployed.

There are a number of approaches to network design and site selection. Exposure assessment, in particular, will often need to target both source-oriented monitoring sites (often synonymous with worst-case or 'hot-spot' environments) and background locations optimized for quantifying general population exposure. Depending on the pollutants under assessment, data from a wide variety of location types may therefore be necessary to build up a reasonably complete picture of exposure patterns (Box 5.9).

Although the overall requirement of any network or survey is to maximize spatial coverage and representativeness, in practice this goal is only approached by grid-based monitoring strategies: these can be optimized to provide detailed information on spatial variability and exposure patterns for priority pollutants. However, this approach is highly resource-intensive and not, in consequence, widely used. To reduce resource requirements, a grid approach can be utilized in conjunction with intermittent or mobile sampling, although use of this technique is not consistent with the need to maximize temporal representativeness as well as spatial coverage (see section 5.4.3).

A more flexible approach to network design, appropriate over city-wide or national scale, involves siting monitoring stations or sampling points at carefully selected representative locations, chosen on the basis of required data and known emission/dispersion patterns of the pollutants under study. This approach to network design requires considerably fewer sites than grid strategies and is, in consequence, cheaper to implement. However, sites must be carefully selected if measured data are to be useful. Moreover, modelling and other objective assessment

techniques may need to be utilized to ‘fill in the gaps’ in any such monitoring strategy.

<b>Box 5.9 – Possible Monitoring Locations Relevant to Exposure Assessment</b>	
<b>Site Classification</b>	<b>Description</b>
• city/urban centre	An urban location representative of general population exposure in towns or city centres, e.g. pedestrian precincts and shopping areas
• urban background	An urban location distanced from sources and therefore broadly representative of city-wide background conditions
• suburban/residential	A location type situated in a residential area on the outskirts of a town or city
• kerbside/near road	A site sampling within 1-5 metres of a busy road
• industrial	An area where industrial sources make an important contribution to long-term or peak concentrations
• rural	An open countryside location distanced as far as possible from roads, populated and industrial areas.
• source/target-oriented	Any special source-orientated or micro-environment site. For example, garages, car parks or tunnels, or a site located at a targeted receptor point such as schools or hospitals
• indoor	Will include domestic and office environments (excluding occupational), together with in-car and commuting environments- see Chapter 6.

Some general points to consider when selecting a site location are:

**Overall monitoring objectives.** These usually determine the target areas for study, the priority pollutants and the number of sites required.

**Sources and emissions.** Compilations of emission data can assist substantially in site selection. These will help to identify the most polluted areas, as well as other location types where population exposure may be significant. If a full emission inventory is not available, then surrogate statistics such as population density, traffic flows and fuel consumption may be of use in estimating likely pollution ‘hot spots’, where target receptor exposure may be maximized.

**Meteorology and topography.** The prevailing weather conditions and local topography will strongly influence the dispersion of air pollutants or, in the case of secondary pollutants, affect their production in the atmosphere.

**Model simulations.** The results of dispersion modelling, if available, can be used to predict pollutant dispersion and deposition patterns, thereby helping to identify areas where exposure may be maximized. To be of real use, reliable emissions and meteorological data are needed, together with an appropriate and validated model.

**Existing air quality data.** If monitoring has already been carried out in the area of interest, the data from previous studies may prove useful in targeting problem areas. If no such studies have been carried out, special screening surveys may be designed to provide area-wide or local information on pollution problems. These often involve passive samplers and/or mobile monitoring laboratories.

**Other information** such as demographic, health, population and land-use information are invaluable in targeting locations representative of both baseline and worst-case exposure. The use of Geographical Information Systems (GIS), in particular, allow both ambient measurements and other geo-co-ordinated datasets to be used for exposure assessment, epidemiological studies and a range of air quality management activities.

The site-selection process must also take into account the spatial distribution and variability of criteria pollutants within urban environments. For example, concentrations of primary traffic pollutants such as CO are highest at roadside locations, whereas O<sub>3</sub> levels have higher spatial uniformity but will be lowest in near-road locations due to scavenging by vehicle NO<sub>x</sub> emissions. For this reason, it is usually not possible to optimize measurements for all pollutants at any one site location. In such circumstances, some degree of compromise will often be required. In general, the spatial variability of secondary pollutants, such as NO<sub>2</sub> and O<sub>3</sub>, tends to be more homogeneous than for primary pollutants such as CO and SO<sub>2</sub>. This greater variability of primary pollutants, in particular in proximity to sources, will have obvious implications for monitoring site density and numbers required in any survey.

Micro-scale siting considerations are also important in ensuring that meaningful and representative measurements are made. If baseline concentrations are to be assessed, then monitoring sites should be adequately separated from local pollutant sources (for example, roads or small boilers) or sinks (such as dense vegetation). Probe aerodynamics and site sheltering will also often be important. Free airflow around the sampling inlet will be necessary to ensure representative sampling; for this reason, sampling in a stagnant or sheltered micro-environment should be avoided.

A variety of practical considerations also apply when selecting monitoring sites. They must be accessible for site visits, but the potential for public interference or vandalism must also be recognized. Electricity for pollutant analysers and station infrastructure must be available, together with a telephone linkage for data telemetry, if utilized (Box 5.10).

#### **Box 5.10 - Network Design: Micro-Scale**

*Need to consider -*

- public safety.
- visual intrusiveness/aesthetics of site.
- security/vandalism.
- access to utilities and maintenance.
- planning permission.
- local sources/sinks.
- aerodynamic clearance/sheltering.

### **5.4.3 Sampling strategies and systems**

Monitoring involves assessing pollutant behaviour in both space and time. A good network design should therefore seek to optimize both spatial and temporal coverage, within available resource constraints (UNEP/WHO 1994a; Bower 1997). The previous section dealt with maximizing spatial coverage and obtaining representative measurements. Achieving good time-



domain performances is not a problem for most commonly-used air monitoring methodologies (see Section 5.5). However, once priority pollutants selected must be capable of a time resolution consistent with the pollutant averaging times specified in guidelines.

Continuously operating automatic analysers may be used to assess compliance with short- or long-term guidelines. Well-recognized semi-automatic methods such as acidimetric SO<sub>2</sub> samplers (see Section 5.7.1) will be perfectly adequate for measurement against daily standards or criteria. For automatic analysers or samplers to reliably measure ambient pollutant concentrations, it is essential that these pollutants are transferred unchanged to the instrument reaction cell. The sampling manifold is a crucial and often overlooked component of any monitoring system, which strongly influences the overall accuracy and credibility of all the measurements made.

Integrating measurement methods such as passive samplers, although fundamentally limited in their time resolution, are useful for the assessment of long-term exposure, as well as being invaluable for a variety of area-screening, mapping and network design functions (UNEP/WHO 1994b). Problems can arise, however, when using manual sampling methods in an intermittent, mobile or random deployment strategy. Such an approach is usually adopted for operational or instrumentation reasons, or simply because it would not be possible to analyse the sample numbers or data produced by continuous operation. Intermittent sampling is still widely used world-wide. However, this sampling strategy may be of limited utility in assessing diurnal, seasonal or annual pollutant patterns or, indeed, for a reliable assessment of population exposure patterns.

When auditing monitoring sites world-wide, sampling system deficiencies are by far the most commonly encountered problem. Usually, these result from inappropriate designs or inadequate cleaning of the sampling system. Some design requirements, common to all gas sampling systems for analysers or samplers, are summarized in Box 5.11. Corresponding requirements for SPM are complex, and these are discussed in detail elsewhere (UNEP/WHO 1994c).

***Box 5.11 - Key Air Sampling System Requirements***

- Inertness to pollutants being sampled.
- Minimized air-residence time.
- Low airstream/sample line interaction.
- Excess flow above total analyser demand.
- Minimized pressure drop.
- Removal of interferences such as water vapour/pollutants.
- Avoidance of thermal "shock" (when hot, humid, ambient air is sampled into an air-conditioned enclosure).
- ease of cleaning and maintenance...
- ...which must be done regularly!

## **5.5 Instrument issues**

The capabilities of air monitoring methodologies, as well as their inevitable resource implications, exert a strong influence on network design. This section reviews some of these issues. Specific monitoring methods applicable to individual criteria pollutants are reviewed in

## Section 5.7.

Air monitoring methodologies can be divided into four main generic types, covering a wide range of costs and performance levels. These are passive samplers, active samplers, automatic analysers and remote sensors. The main advantages and characteristics of these monitoring technologies are summarized in Box 5.12.

<b>Box 5.12 - Air Monitoring Techniques</b>			
<b>Method</b>	<b>Advantages</b>	<b>Disadvantages</b>	<b>Capital Cost</b>
<b>Passive Samplers</b>	<ul style="list-style-type: none"> <li>• Very low cost.</li> <li>• Very simple.</li> <li>• No dependence on mains electricity.</li> <li>• Can be deployed in very large numbers</li> <li>• Useful for screening, mapping and baseline studies.</li> </ul>	<ul style="list-style-type: none"> <li>• Unproven for some pollutants.</li> <li>• In general only provide monthly and weekly averages.</li> <li>• Labour-intensive deployment/analysis.</li> <li>• Slow data throughput.</li> </ul>	US\$10-70 per sample.
<b>Active Samplers</b>	<ul style="list-style-type: none"> <li>• Low cost.</li> <li>• Easy to operate.</li> <li>• Reliable operation/performance.</li> <li>• Historical dataset.</li> </ul>	<ul style="list-style-type: none"> <li>• Provide daily averages.</li> <li>• Labour-intensive sample collection and analysis.</li> <li>• Laboratory analysis required.</li> </ul>	US\$1000-3000 per unit.
<b>Automatic Analysers</b>	<ul style="list-style-type: none"> <li>• Proven.</li> <li>• High performance.</li> <li>• Hourly data.</li> <li>• On-line information.</li> </ul>	<ul style="list-style-type: none"> <li>• Complex.</li> <li>• Expensive.</li> <li>• High skill requirement.</li> <li>• High recurrent costs.</li> </ul>	US\$10 000-15 000 per analyser.
<b>Remote sensors</b>	<ul style="list-style-type: none"> <li>• Provide path or range-resolved data.</li> <li>• Useful near sources.</li> <li>• Multi-component measurements.</li> </ul>	<ul style="list-style-type: none"> <li>• Very complex and expensive.</li> <li>• Difficult to support, operate, calibrate and validate.</li> <li>• Not readily comparable with point data.</li> <li>• Atmospheric visibility and interferences.</li> </ul>	US\$70 000 - 150 000 per sensor, or more.

**Passive samplers**

These offer a simple and cost-effective method of screening air quality in an area. A sample integrated over a defined exposure time (typically a week to a month) is collected by molecular diffusion to a pollutant-specific absorbent material. The low unit costs permit sampling at a number of points in the area of interest. This is useful in highlighting “hot-spots” of high pollutant concentrations, such as major roads or emission sources, where more detailed studies may be needed. Careful survey design and attention to laboratory-based QA/QC of the sample analysis process is necessary to make best use of this technique.

### ***Active samplers***

Pollutants samples are collected either by physical or chemical means for subsequent analysis in a laboratory. Typically, a known volume of air is pumped through a collector such as a filter or chemical solution for a known period of time, which is then removed for analysis. There is a long history of active sampler measurements in many parts of the world, providing valuable baseline data for trend analyses and comparison. Sampling systems (for gases), sample conditioning, weighing systems (for SPM) and laboratory procedures are key factors influencing the quality of the final data.

### ***Automatic analysers***

These can provide high-resolution measurements (typically hourly averages or better) at a single point for most of the criteria pollutants (SO<sub>2</sub>, NO<sub>2</sub>, CO and SPM), as well as for other important species such as VOC. The sample is analysed on-line and in real-time, usually by electro-optic methods: UV or IR absorption, fluorescence or chemiluminescence are common detection principles. To ensure the data from automatic analysers are accurate and reliable, a high standard of maintenance, operational and quality assurance/control procedures is invariably required.

### ***Remote sensors***

These are recently developed instruments which use long-path spectroscopic techniques to make real-time concentration measurements of a range of pollutants. The data are obtained by integrating along a path between a light source and a detector. Long-path monitoring systems can have an important role to play in a number of monitoring situations, particularly in proximity to sources. A high standard of operational, calibration and data screening/management practice is essential if meaningful data are to be produced by such systems.

### ***General advice on instrument selection***

It is advisable to always choose the simplest technique that will do the job. Inappropriate, too complex or failure-prone equipment can result in poor network performance, limited data utility and - worst of all - a waste of money. Although monitoring objectives are the major factor to consider, resource constraints and the availability of skilled manpower must also be considered. There is a clear trade-off between equipment cost, complexity, reliability and performance. More advanced systems can provide increasingly refined data, but are usually more complex and difficult to handle.

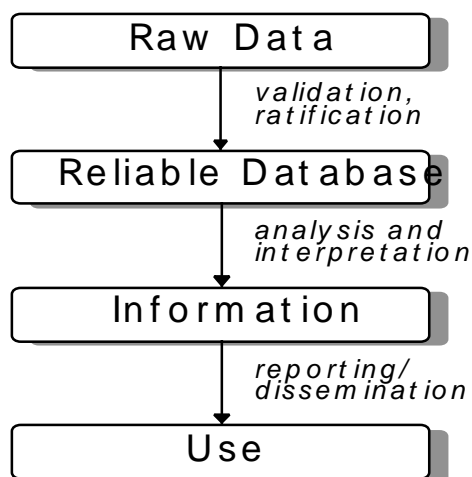
Sampler methods are not necessarily less accurate than automatic analysers. For instance, data from co-located chemiluminescence NO<sub>x</sub> analysers and diffusion tubes can show excellent agreement, to within plus or minus 10%, providing both techniques are subject to high standards of quality assurance and operational practice (Smith et al. 1997). In practice, the combined use of samplers and automatic analysers in a 'hybrid' monitoring programme can offer a versatile and cost-effective approach to network design over a municipal or national scale. Such a network design will use passive or active samplers to provide good spatial coverage and area-resolution of measurements. Automatic analysers, deployed at carefully selected locations, can provide more detailed time-resolved data for assessing peak concentrations or comparison with short-term standards.

In some circumstances, additional use may be made of passive or active samplers. Reasonably robust statistical relationships can often be derived between peak, upper percentile and long-term average pollutant concentrations (Carless et al. 1994). Although these semi-empirical relationships may differ from pollutant to pollutant, as well as with generic site type, they may enable long-term datasets from sampler surveys to be used to assess broad compliance with short-term guidelines; or at least to identify areas where exceedances are likely. This indirect assessment technique should, however, always be used with caution.

Deducing the levels of one pollutant from measurements of another may be possible when the local air pollution climate is dominated by emissions from one source sector, and where robust and well-established emission ratios exist for the species in question. For example, traffic-related benzene and lead levels may in some circumstances be estimated from corresponding CO concentrations. However, surrogate measurements of this kind must always be used with caution.

## 5.6 Turning data into information

As emphasised in the introduction to this chapter, the purpose of monitoring is not merely to collect data, but to produce useful information for planning, health professional, regulatory and public end-users (Figure 5.4). Raw data by themselves are of very limited utility. These first need to be screened (by validation) and collated to produce a reliable and credible dataset (UNEP/WHO 1994a; Bower 1997). In effective Air Quality Management Information Systems, the validated measurements will be archived together with corresponding emission datasets, model predictions and other input relevant to decision-making.



**Figure 5.4** Data throughput from a monitoring programme

The next stage in data management is analysis and interpretation, designed to provide useful information in an appropriate format for end-users. A variety of proven analytical methodologies are available for air quality datasets. However, the appropriate level and method of data treatment will be determined by the ultimate end-use. A minimum level of data management could be the production of daily, monthly and annual summaries, involving simple statistical and graphical analyses that show both time and frequency distributions of the monitoring data. The use of Geographical Information Systems (GIS) should be considered, particularly when the intention is to combine pollution data with those from epidemiological and other geo-co-

ordinated social, economic or demographic sources.

The information derived from measured data must be reported or otherwise disseminated in a timely manner to end-users. This can be in the form of complete datasets, processed summaries, peak or average statistics, exceedances of standards or targets, analytical results, graphs or maps. Formats for information transfer should be designed which are both appropriate to the capabilities of the network and to the requirements of the users. Communicating data or information may involve a number of transmission methods, including paper reports, CD-ROMs, electronic, broadcast and INTERNET media. Public information systems, often exploiting innovative broadcast and world wide web media, play an increasingly important role in many countries in raising awareness, warning of pollution episodes and advising susceptible population subgroups.

## **5.7 Key pollutants and measurement methods**

This section summarizes the measurement techniques available for determining ambient concentrations of the main "classic" pollutants, SO<sub>2</sub>, NO<sub>2</sub>, CO, O<sub>3</sub>, SPM and lead. There is some overlap between these techniques and corresponding methodologies used for individual exposure and micro-environment surveys. At extreme ambient concentrations, moreover, some occupational exposure measurement systems, such as detector tubes, may be used in a semi-quantitative manner (Saltzman and Caplan 1995).

### **Sulphur dioxide**

As the main source of this pollutant is the combustion of fossil fuels containing sulphur, either in power stations or domestic/commercial space heating, the major local source types strongly influences monitoring and assessment strategies. Automatic analyzers need to be used if compliance against a short-term guideline is to be determined; a variety of active samplers are suitable for comparison with daily or annual guidelines. Passive samplers may be used to provide data for comparison with the long-term annual guideline.

#### ***Passive samplers***

There are currently no national or international standards governing the application of SO<sub>2</sub> diffusion tubes to ambient air monitoring, nor for their laboratory preparation and analysis. Protocols for sample preparation and analysis by spectrophotometry and ion exchange chromatography have, however, been published in scientific literature (Bennett et al. 1992; Downing et al. 1994; Hargreaves and Atkins 1988).

A variety of passive sampling techniques are available (UNEP/WHO 1994b). The most widely used include:

The triethanolamine (TEA)/glycol/spectrophotometry method (Hangartner et al. 1989).

The potassium hydroxyde (KOH)/glycerol/spectrophotometry method (Hargreaves and Atkins 1988).

The sodium carbonate (Na<sub>2</sub>CO<sub>3</sub>)/glycerine/ion-exchange chromatography method (Ferm 1991).

Hybridization of these techniques is widespread. In the UK, for instance, KOH or NaOH is used

as absorbent, but with the tube membrane proposed by Ferm (1991) and using ion-exchange chromatography as the analysis method. In practice, the ion-exchange chromatographic technique has been informally accepted as the standard method for SO<sub>2</sub> diffusion tube analysis. The typical sensitivity of this hybrid technique is  $\pm 8.5 \mu\text{g}/\text{m}^3$ : some under-reading against automatic analysers has been observed (about 30%), although agreement with active samplers is better (Downing et al. 1994).

### *Active samplers*

The equipment required for sampling gaseous sulphur compounds in ambient air is described in full in International Standard ISO 4219 (ISO 1979). This standard gives details of the equipment necessary to sample gaseous pollutants by absorption in a liquid bubbler. The standard also includes guidance for siting and installation of the apparatus. The principle of active-sampling methodologies is to draw ambient air through a collecting medium (typically a liquid bubbler), for a specified time, typically 24 hours. The volume of air is metered. The collecting medium is subsequently analysed and the concentration of pollutant in the sampled air determined. This proven method is well established, and has been used in many monitoring networks worldwide for a number of years. In consequence, there is a long history of active sampler SO<sub>2</sub> measurements available for trend assessment.

There are several methods of SO<sub>2</sub> monitoring based on this principle, which can be carried out using the apparatus specified in ISO 4219. They differ with respect to the solutions used in the bubblers for SO<sub>2</sub> absorption and the method of analysis. The four most widely used methods are described below.

*Acidimetric (total acidity) method.* This method, given in ISO 4220 (ISO 1983), is used to determine a gaseous acid air pollution index. Although this method measures total acidity, and is not specific for SO<sub>2</sub>, it is adequate for general use. The simplicity of the method, and the fact that the reagents are relatively safe, makes it a popular choice for routine monitoring (AEA 1997). An accuracy of  $\pm 10\%$  has been estimated for SO<sub>2</sub> measurements using the total acidity method, taking account of all contributory factors. A precision of  $\pm 4 \mu\text{g}/\text{m}^3$  is achievable for this widely-used method (AEA 1997).

*Ion-exchange chromatography.* A variation on the above technique. The exposed peroxide solutions are analysed for sulphate ions by means of ion-exchange chromatography, rather than titration. This has the advantage of being sulphate-specific, but requires the use of an expensive ion-exchange chromatograph.

*Tetrachloromercurate (TCM) method.* This is also known as the Pararosaniline method ISO 6767 (ISO 1990). This is the reference method specified in the EC Directive on SO<sub>2</sub> and suspended particulate matter (EC 1980). However, the reagents used are very toxic, and for this reason the method is not widely used.

*Thorin method.* This method is given in ISO 4221 (ISO 1980). The reagents used include perchloric acid, barium perchlorate, dioxane and thorin. These are hazardous and must be handled and disposed of with care. Accordingly, this method is not commonly used world-wide.

### ***Automatic analysers***

The measurement of SO<sub>2</sub> in ambient air using automatic analysers is covered by ISO/DIS 10498 (ISO/DIS 1999). Well-established automatic monitoring techniques are available. The most widely used method for automatic SO<sub>2</sub> measurement is ultraviolet fluorescence (UVF). SO<sub>2</sub> molecules in the sample airstream are excited to higher, unstable energy states by UV radiation at 212 nm. These energy states decay, causing an emission of secondary fluorescent radiation with an intensity proportional to the concentration of SO<sub>2</sub> in the sample.

The accuracy of data from automatic SO<sub>2</sub> analysers depends on a range of factors encompassing the entire measurement chain. These include accuracy of calibration standards, analyser stability and sample losses in the measurement system. An accuracy of  $\pm 10\%$  has been estimated for SO<sub>2</sub> measurements in UK national automatic networks, taking account of all contributory factors. The precision of SO<sub>2</sub> measurements, determined from long-term variations in baseline response of in-service analysers, is estimated to be  $\pm 3 \mu\text{g}/\text{m}^3$  (AEA 1996).

### ***Remote sensors***

Remote optical sensor systems, such as the Differential Optical Absorption System (DOAS), use a long-path spectroscopic technique to make real-time measurements of the pollutant concentration by integrating readings along a path between a light source and a detector. Long-path monitoring systems can be used to measure SO<sub>2</sub>, but the methodology is less well established than that for automatic point monitors. The accuracy and precision of the data from these instruments are, therefore, much more difficult to determine. The method does not conform to ISO 7996 (ISO 1985b). Particularly careful attention needs to be paid to instrument calibration and quality assurance to obtain meaningful data from remote sensing instruments.

### **Nitrogen dioxide**

Automatic analysers must be used for the direct determination of compliance against the hourly guideline, although much useful information can be inferred using passive samplers (see section 4.5). Either technique is applicable for comparing ambient levels against the annual guideline.

### ***Passive samplers***

Monitoring ambient NO<sub>2</sub> concentrations using passive diffusion tube samplers is now well established. This method provides an integrated, average concentration for the pollutant over the exposure period (typically 2-4 weeks) and is particularly well suited to baseline and screening studies for assessing the spatial distribution of NO<sub>2</sub> concentrations in an urban environment. The most widely used techniques are variants on the Palmes-type sampler, originally developed for the assessment of occupational exposure. This uses a tube sampler, employing TEA as absorbent. Sample analysis, after thermal desorption, is by spectrophotometry or ion-exchange chromatography (Palmes et al. 1976). Very large scale mapping surveys are possible using diffusion tubes, but careful attention both to the harmonization of analytical procedures and to the outputs from different analytical laboratories is essential for the success of large-scale passive sampler surveys.

Although extensively used throughout the UK and Europe there are, at present, no national or international standards governing the application of diffusion tubes for ambient air quality

monitoring, nor for the laboratory preparation and analysis of diffusion tubes. Protocols for sampler preparation and analysis by spectrophotometry have, however, been published in the scientific literature (Palmes et al. 1976; Atkins et al. 1986); these have been informally accepted as standard procedures for NO<sub>2</sub> diffusion tube preparation and analysis.

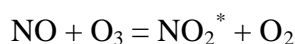
Recent comparisons of NO<sub>2</sub> diffusion tube measurements with co-located chemiluminescent NO<sub>x</sub> analysers show good agreement (Smith et al. 1997; Gerboles and Amantini 1993). Over the range of concentrations generally encountered in urban areas (20-80 µg/m<sup>3</sup>), it was found that on average NO<sub>2</sub> diffusion tubes, exposed for one month, tended to overestimate ambient NO<sub>2</sub> by approximately 10% compared with a chemiluminescent NO<sub>x</sub> analyser. Precision estimates of the diffusion tube technique have been quoted as 5-8% in similar studies.

### ***Active samplers***

A variety of active sampler technologies are available (UNEP/WHO 1994b). The best known of these is the Griess-Saltzman method, covered by ISO 6768 (ISO 1985a). Although this method is sensitive and requires a relatively simple, inexpensive sampling apparatus, there are a number of disadvantages. It is a relatively skilled and labour-intensive technique, uses corrosive chemicals and is not readily applicable to sampling periods longer than 1-2 hours. There also remain doubts about calibration methods, collection efficiency and possible side-reactions. In consequence, this method cannot be recommended for general baseline monitoring applications.

### ***Automatic analysers***

The reference method for automatic measurement of nitrogen oxide concentrations, as defined for compliance with EC Directive 85/203/EEC (EC 1985), is the automatic chemiluminescence method described in ISO standard 7996 (ISO 1985b). This method is widely used world wide. The method is based on the chemiluminescence energy emitted when NO in the sample airstream reacts with O<sub>3</sub> in an evacuated chamber to form an excited energy state of NO<sub>2</sub>. The chemiluminescent reaction is:



Emitted light from the excited NO<sub>2</sub><sup>\*</sup> is converted to an output voltage by a photomultiplier tube and amplifier.

Automatic NO<sub>2</sub> analysers based on liquid-phase chemiluminescence, produced by reacting NO<sub>2</sub> with a chemical solution, are also available. These highly sensitive but relatively fragile instruments are mostly employed for research applications and are not generally regarded as being suitable for routine baseline monitoring purposes.

The accuracy of data from automatic NO<sub>2</sub> analysers depends on a range of factors encompassing the entire measurement chain. These include the accuracy of calibration standards, analyser stability and sample losses in the measurement system. Final accuracy can therefore vary from network to network. An accuracy of ± 8% has been estimated for NO<sub>2</sub> measurements in well-run automatic networks, taking account of all contributory factors (AEA 1996). The precision of NO<sub>2</sub> measurements is estimated to be ±6.5 µg/m<sup>3</sup>, determined from long-term variations in the baseline responses of in-service analysers.



### ***Remote sensors***

Long-path monitoring systems are available for the measurement of NO<sub>2</sub>, but the methodology is less well established than that for automatic point monitors. The accuracy and precision of the data from these instruments are, therefore, much more difficult to determine. The method does not conform to ISO 7996 (ISO 1995b) and, as noted previously, careful attention needs to be given to instrument calibration and quality assurance to obtain meaningful data.

### **Carbon monoxide**

CO in urban areas results almost entirely (typically ~90%) from road traffic emissions. Since CO is a primary pollutant, its ambient concentrations closely follow emissions. In urban areas, concentrations are therefore highest at the kerbside and decrease rapidly with increasing distance from the road. Mostly automatic analysers are being used for the direct assessment of ambient levels against guidelines.

### ***Passive samplers***

A passive sampler has been developed for CO, utilizing a zeolite absorber and a narrow filamental diffusion passage to optimize uptake, and involving GC/FID analysis after thermal desorption (Lee et al. 1992). This technique may be useful for screening, mapping and 'hot-spot' identification. Its use does not, however, appear to be widespread at the present time.

### ***Active samplers***

Grab samples may be collected for subsequent laboratory analysis. However, this technique is not known to be widely used.

### ***Automatic analysers***

The measurement of CO in ambient air is covered by international standards ISO/FDIS 4224 (ISO/FDIS 1999a) and ISO 8186. (ISO 1989)

Baseline ambient CO monitoring is normally carried out using IR analysers. A number of electrochemical CO analysers are available, but these are generally of low sensitivity and not suitable for routine ambient monitoring. However, they may have application in areas of high concentrations. A version of this sensor is incorporated in a commercially available roadside pollution monitoring system.

CO analysis is based on the absorption of IR radiation at wavelengths of 4.5-4.9 micrometres. Since other gases and particles can also absorb IR, the analyser must distinguish between absorption by CO and absorption by interferences. In the most common analyser type, this is done using a gas filter correlation wheel containing a cell of pure nitrogen and a cell of nitrogen plus CO. The cell containing CO removes the CO-sensitive wavelengths before the IR signal enters the absorption chamber, whilst all wavelengths are transmitted by the other cell. The difference in the intensity of the two absorption signals, divided by the intensity of the IR source, provides a measure of the ambient CO concentration.

The accuracy of data from automatic CO analysers depends on a range of factors encompassing the entire measurement chain. These include accuracy of calibration standards, analyser stability

and sample losses in the measurement system. An accuracy of  $\pm 8\%$  and a precision of  $\pm 0.5 \text{ mg/m}^3$  may be achieved using this technique in well-managed and quality-assured programmes.

## **Ozone**

O<sub>3</sub> is not emitted directly from man-made sources in any significant quantities, but is formed in the atmosphere by sunlight-driven chemical reactions involving NO<sub>x</sub> and VOC (see Section 2.1.2). These reactions are not immediate, but may take from hours to days to complete. O<sub>3</sub> is chemically scavenged by primary NO<sub>x</sub> emissions from traffic, and is also removed from the atmosphere by deposition to the ground.

Both spatial and temporal distributions of O<sub>3</sub> differ markedly from those of other pollutants. In particular, significant impacts may occur in areas up to hundreds of kilometres downwind of the original precursor emissions, as a result of long-range as a result of long-range transport. Ambient concentrations and population exposure may often be maximized in suburban and rural areas. This has important implications for monitoring system design.

### ***Passive samplers***

A variety of techniques are available (UNEP/WHO 1994b). These include:

1,2-di-(4-pyridyl) ethylene absorbent- spectrophotometry (Monn and Hangartner 1990).  
 KI –spectrophotometry (Grosjean and Hisham 1992).  
 NaNO<sub>2</sub>/Na<sub>2</sub>CO<sub>3</sub>/glycerine -ion chromatography (Koutrakis et al. 1990).  
 Indigo carmine-reflectance (Alexander et al. 1991).

These methods are not as widely used or validated as corresponding samplers for NO<sub>2</sub> and no clear consensus as to a standard technique has yet emerged.

### ***Active samplers***

The most widely used active sampler technique was the Neutral Buffered Potassium Iodide (NKBI) method. Although relatively simple and inexpensive, there are practical problems with deterioration of the iodine complex and interference (most notably from NO<sub>2</sub> and SO<sub>2</sub>). These issues have reduced its use to the extent that the technique may now be regarded as obsolete.

### ***Automatic analysers***

ISO 10313 (ISO 1993a) is not of real relevance, as the chemiluminescence detection technique it describes is no longer widely used. The most commonly used technology is now that of UV absorption; this is specified as the reference method for the purposes of EC Directive 92/72/EEC (EC 1992). An ISO standard is being developed for the UV method.

UV absorption is a robust, well-developed technique. Ambient O<sub>3</sub> concentrations are calculated from the absorption of UV light at 254 nm wavelength. The sample passes through a detection cell of known length (l). An O<sub>3</sub>-removing scrubber is used to provide a zero reference light intensity, I<sub>0</sub>. The analyser alternately measures the absorption of air in the cell with no O<sub>3</sub> present and the absorption in the experimental sample cell, I<sub>s</sub>. The ambient O<sub>3</sub> concentration, c, may be simply calculated using the Beer-Lambert equation:

$$I_s = I_0 e^{-alc}$$

where  $a$  is the relevant absorption coefficient at 254 nm.

Given appropriate attention to system design, calibration and equipment support a typical measurement accuracy of  $\pm 11\%$  and a precision of  $\pm 4 \mu\text{g}/\text{m}^3$  should be readily achievable in well-run automatic networks.

### ***Remote sensors***

Open-path optical remote sensing techniques such as DOAS are available for  $\text{O}_3$ , although the associated practical issues noted in previous sections are applicable.

### ***Suspended particulate matter***

SPM is a generic term embracing all airborne particulate matter. This therefore encompasses a wide range of size fractions, morphologies and chemical compositions, as discussed in Chapter 2. Although coarse particle size ranges may cause significant local nuisance or soiling, it is the finer fractions, such as  $\text{PM}_{2.5}$ , that are capable of deep lung/airway penetration. Concern about the potential health impacts of fine particulate matter has increased rapidly over recent years.

SPM monitoring is fundamentally different from the measurement of gaseous pollutants, and the methods are generally less precise. A wide variety of different sampling and detection methodologies is available, including the Tapered Element Oscillating Microbalance (TEOM),  $\beta$ -ray analysis, gravimetric sampling (low or high-volume) and a number of indirect optical, particle counting and light-scattering methods. The sampling system strongly affects the measurement process and appropriate aerodynamically designed inlets are essential for proper sample-fractionated determinations (UNEP/WHO 1994c).

### ***Active samplers***

Gravimetric samplers collect particulate matter onto a filter using high-volume (about  $100 \text{ m}^3/\text{hour}$ ) or low-volume (about  $1 \text{ m}^3/\text{hour}$ ) pumped sample flows. The weight of particulate matter deposited on the filter is used to calculate a 24-hour average mass concentration. No ISO or CEN standards have yet been promulgated for ambient measurement of  $\text{PM}_{10}$  particulate matter using gravimetric samplers, although these are under development at the present time. An ISO standard for evaluating  $\text{PM}_{10}$  inlet heads is, however, available (EN 1999). A United States Environmental Protection Agency procedure for  $\text{PM}_{10}$  using the high-volume sampler is given in Federal Register 40 CFR Part 50 (CFR 1993). However, compliance with this procedure does not ensure consistency with the anticipated CEN standards.

The various SPM monitoring techniques may not necessarily produce comparable measurements. Different sampling systems, operating temperature, filter media and filter history may also potentially affect measurement equivalence. The accuracy and precision of any measured mass concentration is, therefore, liable to a wide margin of error. A target accuracy of  $<10 \mu\text{g}/\text{m}^3$  and a precision of  $<5 \mu\text{g}/\text{m}^3$  (for daily average concentrations  $<100 \mu\text{g}/\text{m}^3$ ) are given for  $\text{PM}_{10}$  measurements by EN 12341 (EN 1999).

Medium- or low-volume gravimetric samplers are more portable and less noisy than high-volume samplers, making them more suitable for use in urban areas. However, the mass of particles

collected is far less than with high-volume samplers, giving a greater potential for errors due to filter weighing. According to a recent large-scale instrument comparison, a number of commercially available high- and medium-volume samplers are equivalent to a reference Wide Ranging Aerosol Collector (WRAC) (EN 1999).

Correct filter handling, documentation and analysis is fundamental for obtaining valid data. The filters must be conditioned in a temperature- and humidity-controlled environment, typically 20 °C and 50% relative humidity, for at least 24 hours before and after exposure. The filters must be accurately weighed using a suitable balance, that has been calibrated using an accredited method.

### ***Automatic analysers***

Instruments are commercially available using the following techniques:

Tapered Element Oscillating Microbalance (TEOM).

Beta-ray absorption analysers (ISO/FDIS 1999b).

Light scattering systems.

Of the automatic instrument types available, the TEOM and  $\beta$ -ray systems have been operated widely for many years and are well tested in the field. The light scattering type of instrument has been developed more recently, and is therefore less well proven in service. Operating experience and co-located measurement campaigns indicate that measurements from the different instruments are not always equivalent or comparable

For traceable and robust measurements, samplers must be fitted with a tested PM<sub>10</sub> inlet head and an accurate flow control system. The PM<sub>10</sub> sampling inlet should be tested to ISO Standard 7708 (ISO 1995) to ensure accurate size fractionation at the point of sampling. A target accuracy figure of  $<10\mu\text{g}/\text{m}^3$  and precision of  $<5\mu\text{g}/\text{m}^3$  (for daily average concentrations  $<100\mu\text{g}/\text{m}^3$ ) are given in EN 12341 (EN 1999). Tests on in-service TEOM analysers deployed in UK networks demonstrate these figures to be realistic and achievable.

## **Lead**

The main sources of lead in air are the combustion of petrol containing lead-based additives and industrial emissions.

### ***Active samplers***

These are based on pumped sampling of large quantities of ambient air, capturing fine ambient particulate matter on a filter for subsequent analysis. Analysis of filters for lead is covered by ISO 9855 (E), which specifies atomic absorption spectroscopy as the standard analytical method (ISO 1993b). There is no standard sampling method, although the EC Directive does specify some relevant sampling and filter criteria (EC 1982).

A variety of sampling methods are used, including high-, medium-, and low-volume samplers. There is no standard or reference sampling method. The UK method is broadly typical: this utilises an “M Type” sampler designed specifically for this purpose. Its flow rate is controlled to 5.4-7.1 m<sup>3</sup>/day, and Millipore Aerosol Field Monitor filters are exposed and changed weekly. Passive sampling methods are not applicable.

## 6. Air Quality Management

### 6.1 Introduction

Basic principles guide international and national policies for the management of all forms of air pollution. An important global initiative occurred in 1983 when the UN General Assembly established the World Commission on Environment and Development, headed by Gro Harlem Brundtland. The report produced by the Commission, *Our Common Future*, was presented to the UN General Assembly in 1987 and endorsed by it. It has been influential in bringing environmental issues into the global arena, and in expressing some concepts that have been influential in air quality management (WCEDC 1987).

The Brundtland Commission suggested that sustainable development would be required to meet the legitimate aspirations of the world population without destroying the environment. It defined **sustainable development** as: "development that meets the needs of the present without compromising the ability of future generations to meet their own needs." This concept has been embraced as an apparent means of integrating environmental policy and economic development.

Following the Brundtland Commission, the UN Conference on the Environment and Development was held in Rio, in 1992 (UNCED 1992). The aim was to ensure that practical foundations for sustainable development were put in place. The Agenda 21 document and the Rio declaration were the most obvious results of this conference. Agenda 21 is a document covering sustainable development, which is not binding on countries. However, national implementation is reviewed by the Sustainable Development Commission and the UN General Assembly. Agenda 21 supports a number of environmental management principles on which some government policies are based, including air quality management. These include:

**precautionary principle** - where it is clear that a proposal will damage the environment, action should be taken to protect the environment without awaiting scientific proof of damage.

**polluter pays** - the full costs associated with pollution (including monitoring, management, clean-up and supervision) should be met by the organization responsible for the source of the pollution.

In addition, many countries have adopted the principle of **pollution prevention**, which aims to reduce pollution at sources.

The responsibility of national governments for international reporting on the environment of their country has enabled greater exchange of air quality information around the world.

#### *Strategy for air quality management*

The goal of air quality management is to maintain a quality of air that protects human health and welfare. This goal recognizes that air quality must be maintained at levels that protect human health, but must also provide protection of animals, plants (crops, forests and natural vegetation), ecosystems, materials and aesthetics, such as natural levels of visibility (Murray 1997). And to achieve this air quality goal, it is necessary to develop policies and strategies.

Government policy is the foundation for air quality management. Without a suitable policy framework and adequate legislation it is difficult to maintain an active or successful air quality management programme. A policy framework refers to policies in several areas, including transport, energy, planning, development and the environment. Air quality objectives are more readily achieved if these interconnected government policies are compatible, and if mechanisms exist for co-ordinating responses to issues which cross different areas of government policy. Measures that have been adopted in many developed countries for integrating air quality policy with health, energy, transport and other areas are summarized in a report of the United Nations Economic Commission for Europe (UNECE 1995; UNECE 1999).

This following section (6.1) will discuss management of ambient air, and section 6.2 the management of indoor air.

### **6.1.1 Strategies for ambient air quality management**

This section reviews the development of an air quality strategy, including some basic principles of ambient air quality management, ambient air quality standards, source emission inventories, emissions control for point, mobile and area sources, management of “non-classic” pollutants, communication and international air quality management.

Ambient air quality management has a long history. Complaints of air pollution led to studies of air pollutants and their effects. However, a substantial excess mortality rate during the economic expansion after the Second World War resulted in the initiation of pollution control. High air pollution levels in cities in the US and Europe resulted in excess deaths, including more than 4000 excess deaths in London from a stagnant atmosphere of fog, smoke and SO<sub>2</sub> during five days in December 1952 (Brimblecombe 1987). As a consequence of this disaster, there was increased public pressure for better air quality in cities in developed countries around the world. With the availability of relatively cheap and clean fuels, and in an environment of strong economic growth and increasing incomes, governments in developed countries slowly introduced measures to improve air quality in cities. Particular emphasis in the early stages was on reducing particle and SO<sub>2</sub> concentrations in cities.

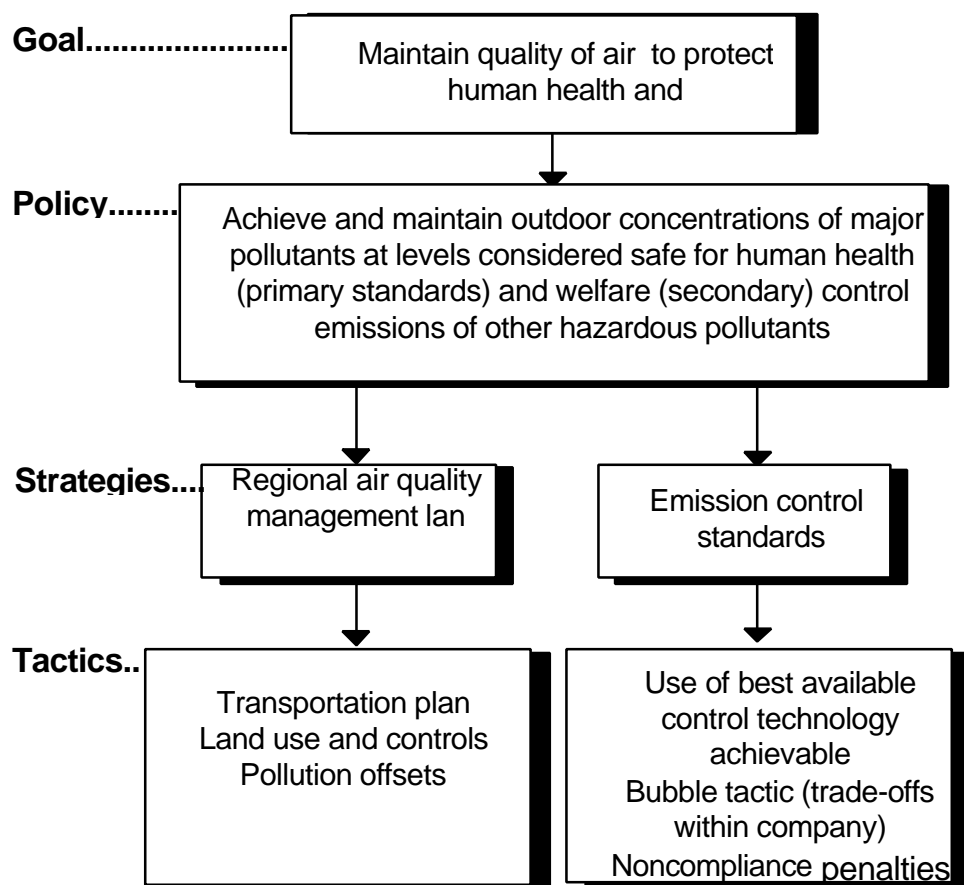
In the last two or three decades, air quality management in the cities of developed countries has broadened in scope. However, the emphasis and success of management activities has been varied. Although considerable progress to improve air quality has been achieved in some large cities of developing countries, many large cities face very significant challenges to implementing effective action. Also, it is now recognized that urban air pollution can travel long distances, affecting areas outside the local and national boundaries in which the polluting event occurs. Polluted air crosses regional and national boundaries, affecting health and environments in rural areas and in other countries.

In response, more effective international action has been implemented. International guidelines on ambient air quality have been produced by organisations such as WHO (WHO 1987), and international policies are being co-ordinated under conventions such as the Convention on Long-Range Transboundary Air Pollution (UNECE 1995; UNECE 1999).

### **6.1.2 Stages in the development of ambient air quality management**

A legal framework is needed to provide a context for ambient air quality management. While there are many possible models, one example is illustrated by Figure 6.1.

**Figure 6.1. The Structure of the U.S. ambient air quality legislation as established by the Clean Air Act 1970, and amendments (after Westman 1985).**



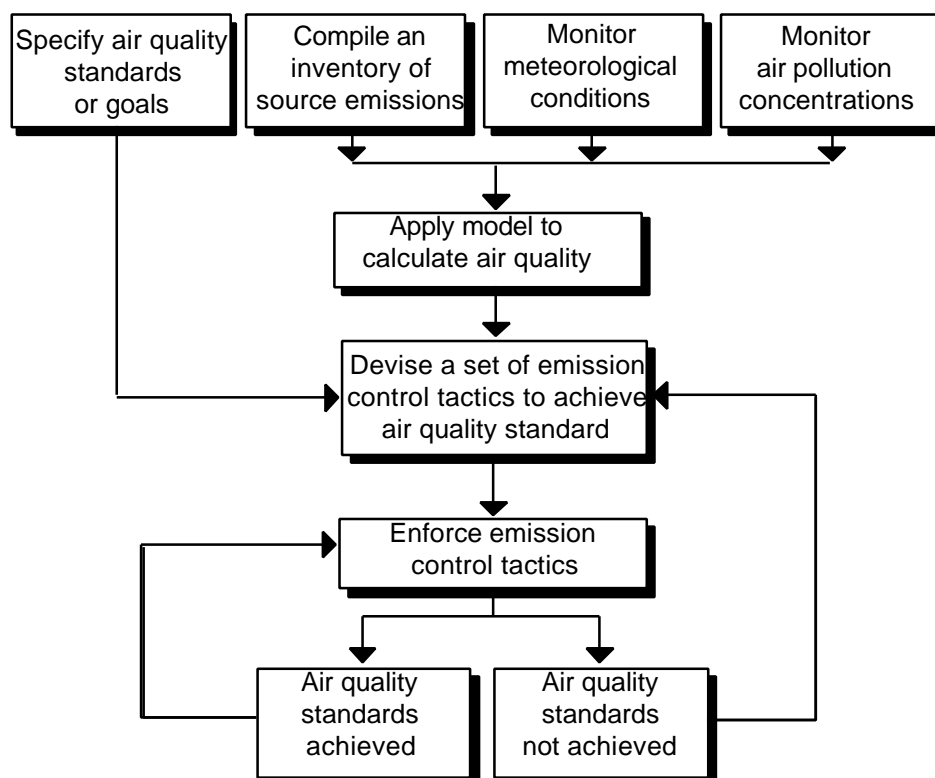
When goals and policies have been developed, the next stage is the development of a strategy or plan. Figure 6.2 summarizes the stages involved in the development of an air quality management strategy and can start with the development of ambient air quality standards or guidelines. It may also involve the development of an emissions inventory. The monitoring of both meteorological conditions and air pollutant concentrations would also normally occur, as these data are required by models used to estimate air quality, and to validate the model output. Air quality standards and model outputs or measurements may be considered in devising emission control tactics aimed at achieving the air quality standards. The tactics need to be enforced, and if the standards are achieved, they need continued enforcement. If the standards are not achieved after a reasonable period of time, the emission control tactics may need to be revised.

National air quality standards are usually based on a consideration of international guidelines, such as the WHO *Guidelines for Air Quality*, as well as national criteria documents that consider dose-response relations for the effects of each pollutant on human health, livestock, wildlife, crops, forests, natural ecosystems, materials, aesthetics etc. In some cases, studies of combinations of pollutants may be required. National standards take into account the technical,



social, economic and political factors within the nation. National approaches to the establishment of air quality standards in some developed countries have been summarized by the Economic Commission for Europe (UNECE 1995; UNECE 1999).

**Figure 6.2. Stages involved in the development of an air quality management strategy (Elsom 1992).**



In some cases, monitoring may show that ambient air pollutant concentrations are considerably higher than some options for standards. An issue for those developing standards is whether national standards should reflect the need to protect human health and the environment, when this objective is unlikely to be achieved in the short- to medium-term with the available resources. In some countries, the standards are set at realistically attainable levels, given the prevailing national technical, social, economic and political conditions, even though they may not be fully consistent with the levels needed to fully protect human health and the environment. Over time, air quality standards may also change, after review, as conditions within a nation change, and as the scientific relationship between air quality, the health of the population and the quality of the environment becomes better understood.

As discussed in Chapter 3, there are considerable differences between the “classic” air pollutants such as SO<sub>2</sub>, particles, NO<sub>2</sub>, CO, O<sub>3</sub>, and the “non-classic” air pollutants (Table 6.1). Different approaches may be needed to develop standards for the two types of air pollutants. Air quality monitoring (Chapter 5) is used to assess whether air quality at particular locations is in compliance with the standards selected.

**Table 6.1. A comparison of the “classic” and “non-classic” air pollutants**

“CLASSIC” POLLUTANTS	“NON-CLASSIC” POLLUTANTS
Few	Numerous
Not bioaccumulated (except Pb)	Some bioaccumulate
Lung is primary target (except CO and Pb)	There may be many target organs
Human health effects are generally well known (except NO <sub>x</sub> )	Human dose-response data rarely available
Effects occur from minutes (acute) to years later (chronic)	Effects occur from minutes (acute) to years later (chronic)

### 6.1.3 Source emission inventories

A crucial component of an air quality management plan is a reasonable quantitative knowledge of the sources of the various emissions (Figure 6.2). An emissions inventory is essential. In some cases, emissions are described in source groups. These may be:

Point sources such as major industrial sites.

Mobile sources such as motor vehicles.

Area sources such as domestic emissions and emissions from light industry and commercial areas.

Biogenic or natural sources.

For some components of an emissions inventory accurate data may be available. For example, accurate emissions data may be available for some industrial sites from measurements of stack emissions. In other cases, emissions can be calculated from estimates of process inputs. For example, the emissions of SO<sub>2</sub> from coal-fired electricity generation plants can often be calculated with reasonable accuracy from the knowledge of the throughput and sulphur content of the fuels and other information.

While estimates of emissions are needed to develop emission inventories, measurements to confirm the veracity of the estimates are highly desirable. Surveys may be used for point sources such as large industrial facilities to provide data on their emissions. However, reporting by companies is not always complete, particularly for fugitive emissions (such as leaks of volatile substances, equipment leaks and loss of fine particles from stockpiles), and for combustion products such as PAH for which sufficient data may not be available.

When source data are missing, it is common to use general emission factors for both point and diffuse sources. Emissions from diffuse sources include emissions from motor vehicles and off-road mobile sources, and area sources such as light industry, domestic and wood burning, as well as biogenic emissions from natural sources such as vegetation. Emission factors for diffuse sources are usually calculated using data specific for each source type. For example motor vehicle emissions may be estimated by calculations involving the distance traveled by vehicles, the number of vehicles, temperature, fuel consumption and the composition and properties of the fuels used. General emission factors for various industrial processes are available from published sources (such as EEA (undated); USEPA 1985; USEPA 1987; USEPA 1995 and more

recent supplements and updates). However, these emission factors need to be used with care, as adjustments in emission factors may be needed to take into account differences in operating conditions, fuels and feed materials.

It should be noted that emissions inventories must also manage those pollutants, which form in the air from reactions among other pollutants. O<sub>3</sub> and some other components of the photochemical smog complex are not directly emitted into the atmosphere, but form from reactions among NO<sub>x</sub> and reactive organic compounds. The control of photochemical smog requires, among other things, an understanding of the sources and emissions of NO<sub>x</sub> and reactive organic compounds.

In some developing countries, reliable statistical information for producing accurate emissions estimates is lacking. However, where action is needed to improve air quality, the absence of this information should not prevent the development of preliminary emissions estimates. Basic information about the population, transportation, industry, fuels and other information can be used to calculate preliminary emissions estimates (Kato and Akimoto 1992), which can then be used to develop and implement air quality management plans. The preliminary emissions estimates can be revised as more accurate information becomes available. Sources of information on how to prepare rapid emissions inventories include WHO 1993a; WHO 1993b; WHO 1995h; WHO 1997b.

#### **6.1.4 Meteorology and mathematical models**

A knowledge of meteorological conditions in an area is useful when applying mathematical models to calculate air quality. As indicated in Chapter 4, modelling is a powerful tool for the interpolation, prediction and optimization of control strategies. Models allow the consequences of various options for improving air quality to be compared. However, models need to be validated by monitoring data. Their accuracy depends on many factors, including the accuracy of the source emissions data, the quality of knowledge of meteorological conditions in the area, and the assumptions about physical and chemical processes in the atmosphere involving the transport and transformation of pollutants.

#### **6.1.5 Emissions control approaches**

##### ***Command and control***

Laws and regulations are at the heart of air quality management strategies. The traditional approach for developing and implementing air quality management strategies has been the "command and control" approach. This approach has several major features centred around the regulation of emissions. The command and control approach usually involves the development and regulation in law of emissions standards, the licensing of emissions sources, the monitoring and reporting of emissions, and penalties for exceeding license conditions. Under this system, the techniques to be used in areas such as pollution control are prescribed by government, and compliance with conditions is checked by government inspectors. Licences are issued, standards are set, compliance with standards are checked, non-compliance cases commonly go to court, mitigating circumstances are considered by the court, and penalties are imposed. New developments or major changes to sources are usually subject to environmental impact assessment, and new sources may be subject to tighter performance standards than existing

operations.

The "command and control" approach is the most widely used technique around the world as it has many strengths. This system has some public confidence, and provides a degree of certainty to industry and the public. However, it is also time consuming, expensive and legalistic. As the penalties imposed by the courts may be light, the outcomes may be unsatisfactory for all involved. The command and control approach is also rigid, with the potential for arbitrary decisions and a focus on end-of-pipe solutions, instead of more comprehensive pollution prevention approaches. While it may establish a minimum condition, it provides no incentive to minimize emissions. It usually ignores equity, often requiring highly expensive best-available technology for new sources, while existing sources with a lower level of technology and performance continue to pollute. However, in some situations the command and control approach has worked extremely well, and many countries have reduced emissions of SO<sub>2</sub>, coarse particles and reduced or eliminated lead emissions from petrol.

In many countries the reform of regulations in the last 10-15 years has reduced dependence on the traditional command and control approach. In recent years, the trend in most developed countries has been towards increased use of other forms of regulatory control. One such approach is self-regulation (Table 6.2). It is argued that some industry groups, for example the chemical industry or the petroleum industry, are familiar with current best practice within their own industry, and can set codes of practice, industry standards and targets. Individual companies conduct self-monitoring of compliance and are subject to audit. However, self-regulation measures can provide less certainty to industry and may inspire less public confidence than regulatory control by government.

**Table 6.2. Types of environmental regulation (after Bradfield et al. 1996)**

Type	Description	Example
Command and control	Issue of licences, setting of standards, checking for compliance with standards, sanctions for non-compliance	Air pollution control Government audits Emission standards
Economic instruments	Use of pricing, subsidies, taxes, and charges to alter production and consumption patterns of organisations and the public	Load-based emission charges Tradeable emission permits  <i>Differential taxes</i>  True cost pricing of resources
Co-regulation	Formulation and adoption of rules, regulations and guidelines in consultation with stakeholders, negotiated within prescribed boundaries	National registers of pollution emission inventories
Self-regulation	Self-imposition of regulations and guidelines and environmental audits by industry groups. Voluntary adoption of environmental management measures.	Voluntary codes of practice Self-audit Emission reduction targets Environmental management systems

***Economic instruments***

Another approach adopted in many countries involves the use of economic instruments (UNECE 1995; UNECE 1999). This approach decreases the operating costs for pollution prevention. Examples include load-based emission charges, which increase operating costs for industry if pollution discharge increases; higher tax charges for leaded petrol compared with unleaded petrol; product charges and environmental taxes on fertilisers, batteries, pesticides, etc.; reducing subsidies for energy use; and subsidising zero emissions products. Pricing policies are a powerful economic instrument for air quality improvements. It has been estimated that direct energy subsidies in developing countries total nearly US\$230 billion each year (El-Ashry 1993). Reducing subsidies for energy use encourages energy conservation, reduces emissions from power stations, and frees investment to be used for other purposes, such as less polluting technologies (Hall 1995).

Another market-oriented approach is a system of tradeable emission permits. In this system, the regulating authority quantifies the total mass of emissions permitted in an area and issues an equivalent number of tradeable emissions entitlements. These tradeable permits can be freely bought and sold. They have the potential to achieve government policy objectives at the lowest cost to industry, and in some cases to government. A comparison of command-and-control and market-based incentives in Santiago, Chile, found that flexible market-based incentives enabled substantially higher reductions in emissions to be achieved for the same expenditure (O'Ryan 1996).

An Emissions Trading Policy has been adopted in the US, in particular in the 1990 revision of the US Clean Air Act, which enables some trading of emissions permits. The US has established a national cap on SO<sub>2</sub> emissions of 8.9 million tonnes per year, beginning in year 2000. Sources may not emit more SO<sub>2</sub> emissions than their marketable emission allowance. These emissions allowances can be purchased, sold or banked for later use. New or expanding sources must arrange to transfer allowances from existing sources, using pollution control or closure of existing sources to provide the required emissions allowances. Existing industries can reduce or cease emissions from one source within the organization to enable expansion of emissions elsewhere. It is considered that this system provides maximum flexibility to industry to pursue the lowest-cost options, while meeting government policy objectives (Portney 1990).

Systems have been proposed which replace the requirement that every new model of vehicle produced meets a uniform emissions standard. It has been proposed that as the costs of achieving various levels of emission control vary among vehicle types, manufacturers should be required to achieve a weighted emissions level over their fleet (Kling 1994). It was argued that manufacturers who better the standard should be permitted to sell their emissions reduction credits to manufacturers unable to meet the standard. Some limited application of an incentive-based system has been incorporated into the Low Emission Vehicle Program adopted in California.

Emissions trading may be considered to be a government regulated, but private market in tradeable emission permits. While sufficiently developed markets may be a pre-requisite for an emissions trading system in developing countries, some principles of emissions trading may be applied without well-developed markets (Smith 1993).

### ***Co-regulation***

As part of the process of regulatory reform, companies and their industry organisations have been included in discussions of options for regulation reform, and in the review of these options. This pro-active approach by industry organisations has led to a degree of co-regulation in some areas. It has resulted in the adoption of regulations and guidelines considered to be practical and realistic by those affected, and have simplified and reduced the costs of compliance for national governments. The process has resulted in the voluntary adoption of environmental management measures in a collaborative manner.

Corporate environmental management is a combination of public policy and social responsiveness. In the public policy area, the enactment of legislation and regulations has enforced socially responsible behaviour. The role of government has increasingly been to provide guidelines to environmental managers in industry, so that corporate behaviour can be shaped to meet social expectations. The aim is that guidelines should prescribe industrial emissions outcomes, but not the means to achieve these outcomes, and to avoid being too prescriptive, as this can encourage a legalistic approach.

### ***Self regulation***

There is a growing worldwide adoption of environmental management systems. These include the British Standard 7750, the European Union Eco-Management and Audit Scheme, and the environmental management system of the International Organization for Standardization, the ISO 14000 series (ISO 1996a; ISO 1996b; Sheldon 1997). The adoption of environmental

management systems has also influenced the process by which governments define industrial emissions outcomes, while not prescribing to industry how these outcomes should be achieved.

Governments are also using public education strategies to improve the actions of the public that can lead to air pollution. In many cities, area sources of air pollution and vehicle sources together comprise the largest component of emissions, and it is the actions of individuals that decide the scale of these emissions. While technical strategies have a major role, education and public information programmes can also contribute to reducing the magnitude of these sources.

Another recent approach to non-classic air pollutants involves risk assessment. In many cases there is no "safe" level for these air pollutants. They do not follow a threshold-type response, as health and ecological risk can increase with increasing exposure. Consequently, this approach requires an evaluation of health risks for the general or sensitive population, and establishes acceptable levels of health risk for these populations. Sources are required by regulation to implement techniques for reducing the levels of health risk to those prescribed.

Emissions control options can involve broad strategic approaches, such as land use, transportation, energy and industrial development planning. Unless air quality planning has a consistency with these other areas, substantial progress is difficult. Complex models have been developed to assess the interaction and consequences of changes in these areas for air quality. However, changes in land use, transportation, energy, and industrial development planning may take decades to substantially improve air quality, so more specific tactics to control emissions are needed. A decision support system for industrial air pollution control is available which aims to support policy makers and managers in analysing and formulating policy options and control measures (WHO 1995h).

#### **6.1.6 Evaluation of control options**

Unless legal constraints in a country prescribe a particular control option, the evaluation of control options must take into account technical, financial, social, health and environmental factors, as well as the speed with which they can be implemented and whether they are enforceable. Although considerable improvements in air quality have been achieved in some developed countries, the financial costs have been high, and the resource demands of some approaches make them unsuitable for poorer developing countries. Methodologies for evaluating air pollution control strategies have been developed for use in metropolitan areas, both in developed and developing countries, such as the methodology for evaluating options for improving air quality in Mexico City (Hardie et al. 1995).

##### ***Technical***

There needs to be confidence that the selected options are technically practical within the resources of the region. It must be possible to bring a selected option into operation, and maintain the expected level of performance in the long term with the resources available. For some control options in certain regions, this may require regular staff training and other programmes.

### ***Financial***

The selected options must be financially viable in the long term. This may require comparative cost-benefit assessments of options. These assessments must include not only the capital costs of bringing an option into operation, but also the costs of maintaining the expected level of performance in the long term.

### ***Social***

The costs and benefits of each option should be assessed for social equity in its effects on people's lifestyles, community structures and cultural traditions. Considerations may include, disruption or displacement of residents or land uses, impacts on community, culture, and recreation. Some impacts can be managed and resources substituted.

### ***Health and environment***

The costs and benefits of each option should be assessed for health and environmental factors. This may involve use of dose-response relations, or risk assessment techniques.

### ***Effect-oriented and source-oriented principles***

Some countries determine air pollution control requirements on the basis of an assessment of the effects of the pollutants on health and the environment (effect-oriented). Increased emissions may be permitted where the assessment suggests there will be no health or environmental impacts, or ambient air quality standards will not be exceeded. Action may be taken to reduce ambient concentrations where impacts or exceedances are shown to occur. Other countries base their air quality management policies on the requirement for best available technology, or best available techniques not entailing excessive cost (source-oriented). Most developed countries apply a combination of both source-oriented and effect-oriented principles (UNECE 1995; UNECE 1999).

## **6.1.7 Control of point sources**

### ***Siting and planning***

The most powerful and cost-effective air quality management options occur during the planning stages of a new facility, whereas options involving changes in existing production processes or pollution control technology are more limited in scope. Planning options involve careful site selection, to maximize dispersion, and location of the proposed facility away from sensitive receptors, such as residential areas or areas of natural or commercial sensitivity.

### ***Source emissions reduction***

The most cost-effective approaches to controlling existing air pollution sources are those that entail source emissions reduction (Griffin 1994). There are four major approaches, each of which require an understanding of the processes and activities that give rise to the emissions. These source reduction approaches are: management and operational changes; process optimization; combustion modifications; and fuel modifications.



Each approach has a different level of effectiveness on the various air pollutants. For example, process optimization may considerably reduce emissions of volatile and hazardous compounds, but can have little effect on NO<sub>x</sub> and SO<sub>2</sub> emissions. In contrast, fuel modifications can decrease NO<sub>x</sub> and SO<sub>2</sub> emissions but they may have little effect on volatile and hazardous compounds.

### ***Management and operational changes***

Management audits of emissions, sources, and source strength, and subsequent changes in operation to reduce emissions, offer a cost-effective way of reducing emissions. It requires the implementation of good practices in housekeeping and maintenance, to ensure that systems are in place to check that equipment is maintained, and that staff are trained and properly supervised. It aims to minimize fugitive emissions, and losses from stored liquids and solids, by changing the composition of materials used, provided this can reduce emissions while maintaining product quality.

### ***Process optimization***

This approach seeks to achieve emissions reductions by altering the production process without loss of product quality or production volume. It usually involves conducting a series of changes in which a factor involved in the manufacturing process is altered, such as temperature, ventilation or line speed.

### ***Combustion modifications***

Changes to the way in which combustion occurs can substantially reduce emissions. Increasing fuel flow in burners, by taking some burners out of service and increasing fuel flow to those remaining, can substantially reduce emissions of NO<sub>x</sub>. Changes to the geometry of the combustion chamber can reduce emissions of NO<sub>x</sub> without requiring changes to the boiler or fuel. Other techniques that can be applied to reduce nitrogen oxide emissions include tight control over the oxygen feed into a burner, lowering the flame temperature, staged combustion and reburning.

### ***Fuel modifications***

Another alternative for reducing emissions is to reduce the amount of fuel used or to change the type of fuel. The simplest approach is to change the fuel from a relatively dirty fuel, such as coal, to a cleaner fuel such as natural gas. This is usually a cheaper means of reducing emissions than scrubbing SO<sub>2</sub> from emissions. Blending of fuels is also used, such as the blending of low-sulphur coal with high-sulphur coal, and coal/oil blends to reduce emissions of SO<sub>2</sub>. Emissions from processes using coal as a fuel can also be reduced by coal washing, which reduces the proportion of contaminants in coal. In recent years there have been many moves to improve the use of waste flue gases for secondary process such as heating, drying or power. This can reduce the overall requirement for fuel, and consequently reduce emissions.

In Hong Kong a ban on high-sulphur fuel was the best option for reducing SO<sub>2</sub> emissions. The cost of compliance monitoring made the costs of flue gas desulphurization and market-based measures more expensive than a ban on high-sulphur fuels (Barron et al. 1995). Others also doubt the value of flue gas desulphurization and consider that converting to the use of low-polluting fuels and energy conservation are less costly options for Eastern Europe and developing countries (Pearce 1996).

**Emissions control**

Tall stacks have traditionally been used to reduce ground-level concentrations of air pollutants at minimum cost to the producer. Their effectiveness depends on height, the velocity and temperature of the stack gases, and atmospheric conditions such as windspeed and direction, atmospheric stability, local topography and air quality. Stacks of 200-400 metres in height are reasonably effective at reducing ground-level concentrations of air pollutants when they are suitably sited. However, tall stacks do not reduce emissions. They distribute them over a wide area. Where the magnitude of emissions within a region is substantial, or the receiving environment is sensitive, serious environmental effects such as acid deposition and forest decline can occur in remote locations (Wellburn 1988).

Before a system for collecting emissions can be planned, some information is needed to identify, quantify and characterize the chemical and physical properties of the emissions, both under average and extreme conditions. This enables the optimum type and capacity of collection system to be designed. There are many forms of emissions control systems available. Factors involved in the selection of control equipment are discussed by Holmes et al. (1993) and Griffin (1994). Different approaches are usually used for gaseous and particle emissions. The techniques listed in Table 6.3 and 6.4 are not comprehensive and represent only some of the more commonly used methods.

**Table 6.3. Techniques commonly used to control particle emissions**

Particle collection system	Action
Cyclone collectors	The waste gas swirls in a vessel and particles are removed by inertial impaction on the walls of a cylindrical vessel.
Filters	The waste gas is forced through a fabric bag or filter beds on which particles are physically collected.
Electrostatic precipitation	A negative charge is imparted to particles in the waste gas, which are attracted to positively charged collection plates.
Wet scrubbers	Liquids are brought into contact with particles to form agglomerates, which are removed from the waste stream by impaction on plates or on the walls of vessels.

**Table 6.4. Techniques commonly used to control gaseous emissions**

Technique	Action
Combustion	Incineration is used to oxidize combustible air pollutants. It may involve open-ended combustion units such as flares, high-temperature thermal incineration involving specific retention times, and catalytic incineration.
Adsorption	Solid collecting media with large surface-to-volume ratios, such as activated charcoal, are used to remove contaminants from waste gas streams.
Absorption	This involves the use of liquids (commonly water with additives) to scrub contaminants from waste gas streams.
Condensation	Condensers operate by removing heat from the gas stream, enabling the condensation of volatile liquids.

While these control techniques can be very effective, some are expensive in capital and maintenance infrastructure, and may be beyond the resources of some developed and developing countries. However, not all approaches need be expensive. Source reduction techniques are often the most cost-effective and suitable measures for many developing countries. These include fuel modifications, such as the preparation and use of low-sulphur and low-ash fuels, combined with management and operational approaches to reducing emissions.

Another key factor in pollution control and prevention is the designation of responsibility for controlling point source emissions in each facility (Hashimoto 1989). It is a legal requirement in Japan for each factory to designate an individual to be responsible for pollution control. The action of courts in strictly applying concepts of negligence and joint liability has led to an increased focus on due diligence and the application of best practices for controlling emissions. The decisions concerning air quality necessarily take place within the context of each nation, and effective action requires political support assisted by public awareness and demand for change (Hashimoto 1989).

### **6.1.8 Control of mobile sources**

There is considerable variation in the pattern of vehicle emissions at different locations and in different regions of the world. However, considering anthropogenic emissions on a global basis, it has been estimated that motor vehicles can account for about 25-30% of emissions of NO<sub>x</sub>, 50% of HC, 60% of lead and as much as 60% of CO (Faiz and de Larderer 1993). In city centres, vehicles may be responsible for 90-95% of CO and lead and 60-70% of NO<sub>x</sub> and HC. As vehicle emissions usually occur near to the breathing zone of people, exposures can be high and they can represent substantial health risks.

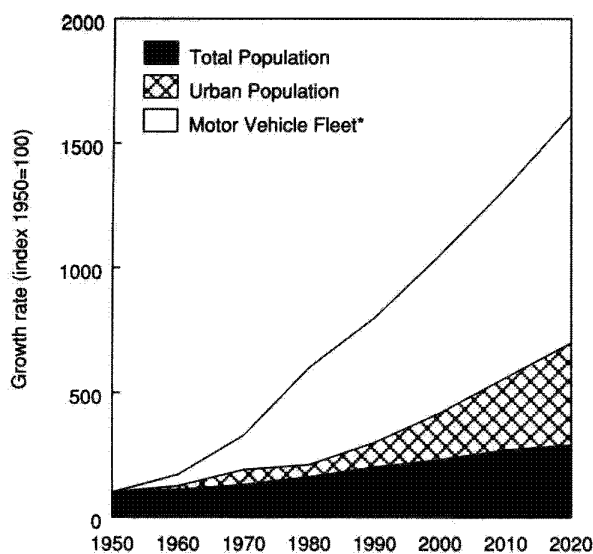
While most of the vehicle population is in developed countries, motor vehicle pollution in developing countries is rapidly worsening due to increasing vehicle fleet growth (Figure 6.3), increasing distances travelled, and high rates of emissions from the vehicle fleets. The causes of the high emissions rates include high proportions of polluting two-stroke engine vehicles, road

congestion which increases emissions per kilometre travelled, poor fuel quality including high lead content, inadequate emissions controls, poor maintenance and high average age of the vehicle fleet (Faiz and de Larderer 1993).

Many countries have acted to regulate and enforce emissions reductions, so ambient concentrations of vehicle-related air pollutants over the last two decades have declined in most developed countries. For example, the decreases in ambient concentrations in the US from 1985 to 1994 were 28% for CO, 86% for lead, and 9% for NO<sub>x</sub> (USEPA 1995). With pressure to improve engine design and operating conditions, and improved tailpipe control technologies, vehicle emissions in many countries have decreased despite increasing number of vehicles and kilometres travelled. For example, while emissions of CO in the period 1980-1990 increased in France from 9216 000 to 10 268 000 tonnes, they decreased subsequently to 8 850 000 in 1996 (UNECE 1999). In the period 1980-1996 CO emissions in Germany decreased from 15 046 000 to 6 717 000 tonnes, and in the European part of the Russian Federation from 13 520 000 to 9 312 000 tonnes (UNECE 1999). Although in the most wealthy of the developing countries significant improvements in air quality are occurring, in most other developing countries for which data are available, both vehicle emissions and ambient concentrations of vehicle-related air pollutants have increased (WHO 1997a). For example it is estimated that emissions of CO in Delhi increased from 140 to 265 tonnes in the period 1980-1990, and are projected to be 400 tonnes in the year 2000 (UNEP/WHO 1992).

As the options for controlling vehicle emissions must be considered within the technical, financial, social, health and environmental context of each nation, the challenges and response options in developed and developing countries are different. In some developing countries, financial and regulatory measures to control vehicle emissions impose major economic and social costs, and there may be an uneven distribution of costs and benefits in the community. For example, there may be increased costs but few benefits for people living in rural areas. Capital may be required to alter local vehicle manufacturing and fuel refining processes, and operating costs may increase. This could divert resources from other priority areas, or make local industry uncompetitive.

**Figure 6.3. Estimated and projected increases in the total population, urban population, and vehicle numbers 1950-2020, excluding motorized two- and three-wheeled vehicles (after Faiz et al. 1990).**



The experience with vehicle inspection programmes in developing countries has been reported to be generally poor (Faiz et al. 1990), and the use of sophisticated vehicle control technologies is expected to have greatest utility in only the most advanced of the developing countries. Consequently, the most promising approaches for controlling vehicle emissions in developing countries is likely to be through the use of cleaner fuels, traffic management and administratively simple policies (Faiz et al. 1990). However, many developing countries have found that improving fuel economy and emission standards, as well as encouraging use of fuel-efficient vehicles and clean fuels, have the effect of both reducing costs and contributing to an improvement in air quality. In addition the strengthening of traffic management programmes, improvements in public transport, restrictions on motorized traffic and encouragement of the use of gas-fuelled vehicles in fleets are also cost-effective means of reducing vehicle emissions (Faiz and de Larderer 1993).

Many middle-income countries have introduced most of the above measures. Some have implemented additional measures, including approval standards and testing of new vehicles, exhaust emission controls, fuel improvements, roadside emission checks, replacement of two-stroke engines with four-stroke engines, and use of low- or zero-emission fuels (such as electric light-rail) for public transport.

Most developed countries apply regulations for vehicle emissions as part of an international process, under which vehicles and their component parts are required to be approved before marketing. Some countries also require regular in-service inspection and maintenance for emissions and safety, as a condition for continued operation of vehicles. This includes retrofitting or scrapping of non-conforming vehicles. Technology requirements for new vehicles in most developed countries include three-way catalytic converters, with closed loop and charcoal canister for petrol-fuelled passenger cars. There are also requirements that apply to diesel, light- and heavy-duty trucks and buses. Conventional two-stroke motorcycles are usually prohibited. There are programmes to control fuels losses during refuelling. Most developed countries require use of unleaded fuels for new cars, and encourage their use by economic instruments. In some countries, leaded petrol is banned (UNECE 1995; UNECE 1999). Advanced area-wide traffic management systems may be employed to facilitate vehicle flow and to minimize emissions.

Policy measures to control vehicle ownership and use, and to encourage other forms of transport, are also commonly employed to support vehicle emissions programmes. For example, tight control over vehicle ownership and use in Singapore, especially within the central business district during the day, has contributed to reducing air pollution from motor vehicles (Chin 1996). Coercive programmes such as no-drive days are normally used as a last resort on days when air quality reaches extreme levels, as they are politically unpopular, and they create social costs and enforcement problems. More socially acceptable measures include incentives to develop and use public transportation, such as buses, light rail and bicycles. Land-use planning approaches that encourage public transport and provide disincentives for use of private vehicles are attractive and cost-effective long-term measures.

#### **6.1.9 Control of area sources**

The control of area sources of air pollutants involves a number of strategies, as the characteristics of area sources are highly variable. The sources are often small, such as domestic and light-

industry sources. Area sources include open burning of waste materials from agriculture, forestry and land clearance. Other sources are forest fires, emissions from vehicle refuelling, off-road vehicles and marine craft, and commercial and domestic fuel combustion. Surface mining and overgrazing of land in semi-arid areas can also act as sources of particles.

The options for controlling area sources can be classified as technical, regulatory, educational and market-based strategies. Technical strategies involve investigating alternatives to existing polluting activities, and implementing cleaner production and pollution prevention technologies and best practices. They encourage the replacement of existing technologies with lower- or zero-emission technologies.

Regulatory strategies involve legal enforcement of regulations at local and national government levels. This could involve banning of some emissions, banning of some open burning, or burning of materials during certain periods, increasing penalties, control of fuel quality, and restrictions on the types of combustion equipment available.

Educational strategies involve informing the community about sources of emissions and the impact of air pollution on health and the environment, and informing them about practices such as open burning, use of poor quality fuels etc, which lead to pollution.

Market-based strategies may involve polluter pays concepts. They include changes in cost structures to provide financial incentives for using clean fuels. They also involve reducing the costs of emissions licenses for adopting best practices, load-based emission charges and true cost pricing of resources (Table 6.2).

#### **6.1.10 "Non-classical" air pollutants**

While most attention is given to the relatively small number of well-recognized and often ubiquitous classic air pollutants (Table 6.1), there are thought to be several tens of thousands of synthetic chemicals in use, some of which are known or thought to be highly toxic air pollutants. The US Congress has identified 189 toxic air substances. Although the acute effects of the most common non-classic chemicals are relatively well-known, much less is known about the chronic, long-term or indirect effects of exposure at ambient concentration levels. In addition, exposure to many non-classic air pollutants may occur in small commercial operations, such as in agricultural enterprises where staff may not be well trained, and accidental exposure or release can occur. Some of these air pollutants are emitted at low concentrations from common sources such as vehicle emissions, and from wood fires. Some non-classic air pollutants can be very persistent in the environment, with exposure occurring many years after release.

As many of these chemicals, such as agricultural chemicals, are often traded, international approaches have been developed to maintain registers of their known toxic effects and their legal status. While techniques to manage them have been developed, the effect of increasing the regulation of non-classic air pollutants has been to exert pressure on industry to manage their production, using pollution prevention approaches. This can involve, but not be limited to, replacing them with safer compounds, changing the production process to prevent them from being produced in side reactions, and recycling or destroying them (such as by high temperature incineration) to prevent release into the environment.

### **6.1.11 Education and communication**

Effective education and communication are important tools in raising public awareness of air quality issues. The successes of air quality management strategies have often involved action at all levels in the community. In many cases, central government action is triggered by local complaints from citizens. Actions to control air pollution have sometimes been possible only by establishing communications between local communities, local government and the national government agency responsible for air quality issues (e.g. Hashimoto 1989). Two-way communication between local communities and those responsible for air quality management is essential, and it requires use of many techniques to be successful.

Reporting air quality information in a form that is generally understandable by the public is a difficult problem. One approach is the use of the pollutant standard index, an example of which is explained by Griffin (1994). This system enables a wide range of air quality components, concentrations and averaging times to be reported to the public as one simple normalized figure. Although a pollution index provides a relatively simple and easy way to disseminate information on the level of air pollution, there are difficulties associated with the setting of these indices. Most of these difficulties arise from the fact that the composition of the pollutant mixture varies in both time and space, and that the components of the mixture have different health impacts. Despite these difficulties, they have been successfully used in some countries. In particular, they were used in communicating complex air quality information to the public during the 1997 haze periods that affected several major South East Asian cities.

### **6.1.12 International air quality management**

The recognition that air pollution does not respect national frontiers has led to considerable action to develop international approaches to the management of air quality. Initially, action to control air pollution was conducted only at a national level, ignoring the import or export of air pollution across national frontiers. Acid deposition, photochemical oxidants, and accidental releases of ionising radiation and toxic chemicals first became international issues only during the 1970's. The development of international environmental law to guide air quality management in the area of transboundary air quality matters is still at an early stage of development, although some principles, international agreements and treaties have been formulated.

The Organization for Economic Co-operation and Development (OECD) provided the first multinational co-operative assessments of the long-range transport of sulphur pollutants in Western Europe. It urged member countries to reduce emissions and pressed for the principle of "polluter pays" to be applied internationally (Elsom 1992). Following this, members of the United Nations Economic Commission for Europe adopted the Convention and Resolution on Long Range Transboundary Air Pollution. This convention commits signatories in North America, Western Europe and Eastern Europe to reduce and prevent air pollution and to use the best available technology that is economically feasible. Many nations agreed to cut emissions of SO<sub>2</sub> by thirty percent by 1993, and fifty percent by 1995, based on 1980 emissions levels.

The European Union has also agreed on directives to reduce emissions of SO<sub>2</sub> and NO<sub>x</sub>, has established air quality standards and has limited the sulphur content of some fuels. It has also agreed to apply the "best available technology not entailing excessive costs" and agreed to limits

on emissions from power stations. In other parts of the world there has been action to increase the information flow among nations for air quality management by introducing international reporting of emissions, ambient concentrations, policy directives and tools for strengthening air quality management. This has largely involved UN agencies such as WHO and UNEP (UNEP/WHO 1992; UNEP/WHO 1996; WHO 1997a), the World Bank and Regional Banks (World Bank 1992), OECD (OECD 1991) and the international development agencies.

## **6.2    *Management of indoor air quality***

Most human beings spend most of their time in indoor environments, where they can be exposed to poor air quality. Pollution and degradation of indoor air cause illness, increased mortality, loss of productivity and have major economic and social implications. Indoor air problems can be reduced by better urban planning, by better design, operation and maintenance of buildings, and through the use of less-polluting materials and equipment in buildings. Indoor air quality problems affect all types of buildings, including homes, schools, offices, health care facilities and other public and commercial buildings. Health effects can include increased rates of cancer, lung disease, allergy and asthma, as well as fatal conditions such as CO poisoning and legionnaires' disease (discussed in Section 4.1). The medical and social costs associated with these illnesses, and the related reduction in human productivity, result in staggering economic losses.

This section considers management of indoor air quality in developed countries, and in some situations in developing countries, and then focuses on the important and widespread problems of indoor air quality management associated with biomass fuel combustion in developing countries.

### **6.2.1    *Management of indoor air quality in developed countries***

#### **6.2.1.1    *Strategies for indoor air quality management***

Control and improvement of indoor air quality can be achieved by combining the three main strategies: proper design and construction of buildings; control of indoor air pollution; and adequate management of problems associated with indoor air quality.

#### **6.2.1.2    *Design considerations***

##### ***Site***

Site investigation. Potential sites of buildings need to be evaluated to determine whether they may be prone to indoor air quality problems, or may be in a high-risk area for radon. Assessment of sites includes a consideration of past uses and identification of any contaminants that might remain as a result of previous use. The use of adjacent sites should also be noted, to evaluate the potential for outdoor pollutants being carried to the proposed building.

Site preparation. Accumulation of moisture favours the growth of biological agents and can be prevented by choosing dry and well-drained building sites and properly grading the property.



### ***Building Envelope Design***

*Tightness.* Buildings should be designed to conserve energy and with good control over infiltration of air and movement of pollutants. This requires that adequate outside air be effectively delivered to occupants through the high volume air conditioning system (HVAC). Natural ventilation should be encouraged whenever possible and convenient. Energy conservation can also be achieved by controlling internal loads (e.g. through increased use of natural light).

### ***Ventilation***

*Outside air-flow rates.* Outdoor air-flow requirements are calculated as part of the mechanical design process. Guidelines based on occupancy and space usage are important; however, outdoor air-exchange rates should also take into consideration the total indoor pollution load and the desired quality of air. Adequate outdoor air flows are important in residential as well as commercial properties.

*Breathing space.* A consideration in HVAC design should be the amount of supply air and outside air that actually reaches the occupants of a building. This involves examining the method and efficiency of air distribution. The effectiveness of the HVAC system to dilute and remove indoor pollutants, and to properly distribute outside air throughout the building, is an important aspect of the design. Ventilation rates should be re-evaluated when interior spaces are renovated.

*Mechanical ventilation in houses.* Some houses are designed airtight with insufficient outside air entering through passive infiltration. As a result, mechanical ventilation is necessary to introduce a satisfactory flow of outside air and to provide adequate dilution and removal of pollutants. This enables heat recovery from the ventilation air.

### ***Commissioning***

levels of VOC. New buildings should be commissioned before occupancy. Commissioning should include testing and balancing of the HVAC system, and documentation that the system meets needs both during operations and during potential renovations. Commissioning should also establish responsibilities for maintaining and operating the system, and for training the staff responsible for operating and maintaining the system. In addition, commissioning should include ventilation specifications for use while the building is new, to control

### ***Material selection***

Designers should specify building materials that are minimal sources of indoor emissions. These materials include low-emitting products and materials, which do not generate or store dust particles. In addition, the design should minimize horizontal surfaces on interior finishes and furnishings to reduce the particle levels in buildings.

### ***Combustion appliances***

Designers and builders should specify and install combustion appliances according to manufacturers' specifications, paying special attention to requirements for combustion and for exhaust ventilation.

### **6.2.1.3 Indoor air pollution control**

#### ***Management of pollutant sources***

*Biological contaminants.* Biological contaminants will flourish wherever there is adequate moisture because adequate nourishment is always available on building surfaces. Because so many building materials can serve as a nutrient source for moulds and other biological contaminants, the most practical means for controlling biological contamination is to avoid excess moisture wherever possible. Moisture control can be accomplished by dehumidification, ventilation, and increasing the temperature at the building surfaces to prevent condensation. Dehumidification is most important in humid climates, and ventilation can aid in moisture control by increasing air movement. These techniques may be used individually or in combination.

Biological contamination can be avoided both by sustaining high-quality maintenance and by monitoring the materials and procedures used in operating and maintaining the building components, including the air-conditioning system. Proper maintenance of air-conditioning equipment is critical for preventing microbiological growth and the entry of undesirable micro-organisms into the indoor air. These components include drainage pans, coils, cooling towers, ductwork and humidifiers. Poor filter maintenance is a common problem and a poorly maintained filter can act as a source of fungal spores, and bacterial and other biological particles that can be distributed in air within the building. Routine maintenance schedules are required that include filter checking and replacement and drain pan clearing.

*Volatile Organic Compounds.* The concentration of VOC within the air of a building can be controlled through careful selection of building materials and products. Building managers should become familiar with the VOC found in the building components and products. Designers and building managers should attempt to select the safest, least toxic materials, when they can be identified, or those with the lowest emission rates. Information regarding VOC may be found by reviewing product labels, Material Safety Data Sheets (MSDS), and available compendia (e.g. the American Institute of Architects Environmental Resource Guide).

For building materials, off-gassing is greatest immediately after the installation of VOC sources, when they are new. This off-gassing decreases with age of the VOC sources. To minimize occupant exposure to VOC, areas that have newly-installed VOC-emitting materials, or that have undergone renovation, should receive increased outdoor air ventilation and/or local exhaust. In the initial months after building completion, the ventilation should operate 24 hrs/day and 7 days/week. Installation of new products or renovation work should preferably occur when the space is unoccupied and remain unoccupied until the strongest VOC off-gassing has occurred.

Air clearing is not recommended as a substitute for source control and adequate ventilation for removing VOC. Although VOC can be removed by air cleaners relying on adsorption and absorption methods, care must be taken to avoid re-emission of the collected VOC from the filter medium.

*Radon.* To prevent the migration of radon gas into a structure, any cracks or openings in the foundation of the lowest level of that structure should be tightly sealed. Ventilation can be introduced to the lowest level in a building to dilute and remove the gas. Exhaust fans and piping can create sub-slab depressurization to remove radon and deter it from accumulating in the

building.

*Combustion Gases.* Combustion gases should normally be exhausted to the outdoor air, and prevented from entering occupied spaces. Outdoor air intakes for a building should not be located near exhaust systems or other sources of combustion gases, such as highways. Combustion appliances are a source of combustion gases within a building and should be used and vented in accordance with the manufacturers' recommendations. Adequate general ventilation, as well as exhausts for the appliances, should be provided to minimize the exposure of occupants.

*Particles.* Particles, including asbestos, tobacco smoke particles, dust and pollen are hazardous or troublesome to occupants when they become airborne. Materials that generate high loads of particles need to be avoided. Proper housekeeping practices should be followed to keep dust levels low. Cleaning activities should be conducted during the off-peak hours to minimize effects of fine particles on sensitive occupants. High-efficiency filtration in the air handling system can also help reduce airborne particle levels.

*Asbestos* Asbestos products must be avoided. When asbestos products are identified in existing buildings, the general recommendation is to minimize disturbance of those materials, which are non-friable or friable, yet in good condition. This also involves training staff on emergency maintenance handling procedures.

*Environmental tobacco smoke.* To eliminate exposure of non-smokers to ETS, organisations should prohibit smoking in buildings. If this is not possible, organisations should provide enclosed, separately-ventilated, negatively-pressurized smoking rooms, with direct external exhaust. These smoking rooms should provide a high volume of outdoor air per smoker.

### ***Operation and maintenance of ventilation systems***

Building maintenance personnel should be trained to understand the indoor air quality aspects of their work. Many maintenance activities directly affect indoor air quality, and some may reveal indicators of potential problems. The staff should be made aware of indoor air quality considerations and how their work can directly impact the health and comfort of occupants.

Preventive maintenance of an HVAC system is essential for it to operate correctly and provide suitable comfort conditions and good indoor air quality. Detailed maintenance logs should be kept for all equipment, including controls and filters. A scheduled program should be developed for a routine check of equipment, calibration of control system components, and necessary filter replacements.

Space is often used for purposes other than those originally intended, especially in older buildings. Changes such as increased occupant density, or altered function of the space, can affect both the required outdoor air supply to the space and the necessary exhaust from the space, and consequently can reduce indoor air quality. When space is reallocated, renovated, or changed from the original design, the use of the space should be re-examined to determine if adjustments to the HVAC system are warranted. The same procedure is required when new sources of contaminants are introduced.

### ***Air cleaning***

Depending on the pollutants of interest, four technologies can be considered for removing contaminants from the air: particle filtration, electrostatic precipitation, negative ion generation and gas sorption. The first three are devised for the removal of particulate matter, while the fourth is designed to remove gases. Air cleaning is most effective when used in conjunction with source control and adequate ventilation. Most air cleaning in large buildings is directed primarily at preventing contaminant accumulation in HVAC equipment and enhancing equipment efficiency.

Filtration is effective only when properly installed and maintained. It is important that filters be changed or cleaned on a regular basis and that leakage around the filters is minimized. High-efficiency filtration is most effective at improving indoor air quality.

## **6.2.4 Resolving indoor air quality problems**

### ***Addressing occupant complaints and symptoms***

When complaints are received from occupants of a building, the building management needs to be responsive to these complaints. The initial investigation into the cause of the complaint may be conducted by the in-house building management staff. Building management should continue an investigation as far as it can, and be responsible for hiring an outside consultant if needed.

### ***Building diagnostic procedures***

*Investigation protocol.* After receiving complaints related to indoor air quality, experienced staff or consultants should investigate the cause of the problem through an iterative process of information collection and hypothesis testing. To begin, a walkthrough inspection of the building is required, including the affected areas and the mechanical systems serving these spaces. A walkthrough can provide information on the occupants, HVAC system, pollutant pathways and contaminant sources. Visual indicators of possible contaminant sources or HVAC system malfunctions should be evaluated first. Measurements of temperature, relative humidity, and air flow should be taken if a walkthrough alone does not provide a solution. Symptom logs and schedules of building activities may provide enough additional information to resolve the problem. When visual inspection and data gathered from the occupants do not identify a possible cause, it may be necessary to sample for suspected contaminants, or compare indoor and ambient levels of pollutants, to ascertain the source of the problem. Whenever a problem is discovered during the investigation, a remedy should be attempted and then a determination made as to whether the complaint has been resolved.

### **6.2.1.5 Government policy**

Many of the problems associated with poor air quality can be prevented at low cost and without compromising energy efficiency if governments develop and implement integrated strategies for the indoor environment, in concert with all social and economic partners.

## ***Guidance/education***

Understanding indoor air quality issues enables a government to focus public education. Both general information, as well as technical training, can be provided for minimizing indoor air pollution. Special focus needs to be given to the design process, so that buildings meet acceptable indoor air standards. Targeted technical guidance and training can be provided for audiences that influence building air quality or occupant health. These include architects; mechanical designers; building owners; facility managers; homebuilders; diagnosis and mitigation professionals; and physicians.

## ***Research support***

*Pollutant source characterization.* Research on indoor air quality can be used to characterize pollutant sources and provide protocols for reducing exposures; it can also provide information on the relationship between health effects and indoor pollutants.

*Health effects.* There are three areas where research can significantly improve our understanding of the health effects of indoor pollution. These are: low-level chemical exposure and pollutant mixtures; allergy/hypersensitivity; and multiple chemical sensitivity, also known as environmental illness.

The effects of low-level exposure to the mixtures of pollutants found in non-industrial environments need to be characterized, since they can induce different health effects from those produced by higher pollutant levels, such as those identified in occupational exposure limits.

Research can be used to better understand the mechanisms causing the health effects, and the different responses of individuals, or groups of individuals. For example, an improved understanding of hypersensitivity associated with allergy and other conditions would help find medical solutions to hypersensitive reactions.

Research to characterize and determine the causes of, and solutions to multiple chemical sensitivity is also needed. Identifying the physiological nature of multiple chemical sensitivity is the first step in understanding whether and how indoor air quality contributes to the syndrome.

*Technology development.* The development of better technologies in diagnosis, mitigation, and control would help to improve indoor air quality. Mitigation and control studies are needed to provide economical and practical alternatives to current technologies. Better means of measuring the effectiveness of ventilation systems are also needed. While the ability to measure individual pollutants often exceeds our knowledge of their health effects at the measured levels, progress is still needed in measurement of pollutant mixtures. There is a need for the development of diagnostic tools that are inexpensive and easy to use.

There is a particular need for improving methods for assessing airborne biological contamination, including both viable and total microorganisms. Research needs to be directed towards the development of immunological and other methods for reliably detecting and quantifying specific organisms, or their allergens. Techniques are also needed for assessing mycotoxins and microbial metabolites that may affect health via non-allergic mechanisms.

*Sick Building Syndrome and Building Related Illnesses.* Efforts to identify causes and solutions

to SBS and BRI are required (for definitions of these terms see chapter 4). Research in health effects and building diagnostics, combined with analyses of data compiled from building investigations, are important for gaining a better understanding of indoor air quality problems.

### ***Problem assessment and surveys***

There is a need to assess the extent of indoor air quality problems, to provide accurate information when setting priorities for public health problems.

*Building surveys/Epidemiology.* Building surveys are necessary to identify building types in which problems occur more frequently. The results of these studies support effective risk reduction programs. Epidemiological studies are needed to characterize indoor air quality-related symptoms, and to distinguish the effects of air pollution from those due to other causes. Epidemiological studies also help to quantify the risk for indoor air pollutants.

*Economics.* Economic studies are needed to measure the costs of indoor air pollution and indoor air quality control strategies to individuals, businesses and society. This research includes developing measures of productivity loss and health cost increases, as well as costing various control strategies, including increasing ventilation, controlling pollutant sources and air cleaning.

### ***Standard/ protocol development***

*Exposure guidelines for indoor air quality.* When the health effects of exposure to pollutants are known, it is important to ensure the protection of workers by setting reasonable exposure limits. In cases where research or risk assessment activities have yet to determine precise dose-response relationships, but where health effects are generally recognized, exposure limits should be set conservatively, weighing risk, economic impact and feasibility. In addition, efforts should be made to develop exposure limits that recognize non-carcinogenic effects.

*Building codes.* Building codes provide an opportunity to incorporate indoor air quality considerations into the design process. There is a need to develop codes for ventilation design, building envelope design, site preparation, materials selection and commissioning.

*Ventilation standards.* Adequate ventilation of occupied spaces with outside air is necessary to ensure good indoor air quality. Research and development is needed for a health-based ventilation standard. Encouraging code-setting bodies to adopt ventilation standards, set by consensus organisations or governmental bodies, will help improve indoor air quality in buildings.

*Maintenance protocols.* Easily implementable guidelines are needed for maintaining HVAC systems and other maintenance activities that affect indoor air quality.

*Product labelling.* As an incentive to industries to develop and market products that emit less pollution, improved product labelling programmes should be implemented. The intent is to provide information to consumers and building designers; it is not intended as a sign of safety approval. Labelling would serve to achieve general reductions in emissions, rather than requiring manufacturers to meet specific guidelines, except for those cases where undesirable chemicals can be identified.

*Accreditation.* By instituting a system of accreditation that recognizes and highlights areas of

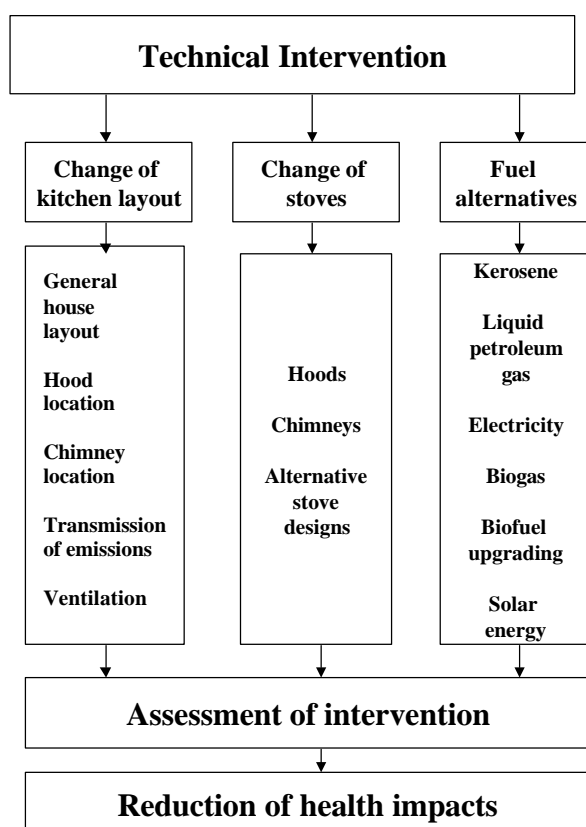
expertise, consumers can be provided with information to make better informed choices when procuring indoor air quality services.

*Emission Standards.* Guidelines for product VOC emissions would provide useful information for manufacturers, architects, design engineers, building managers and others who play a role in selecting products used indoors. However, development of such guidelines is dependent upon additional research establishing a health basis for them.

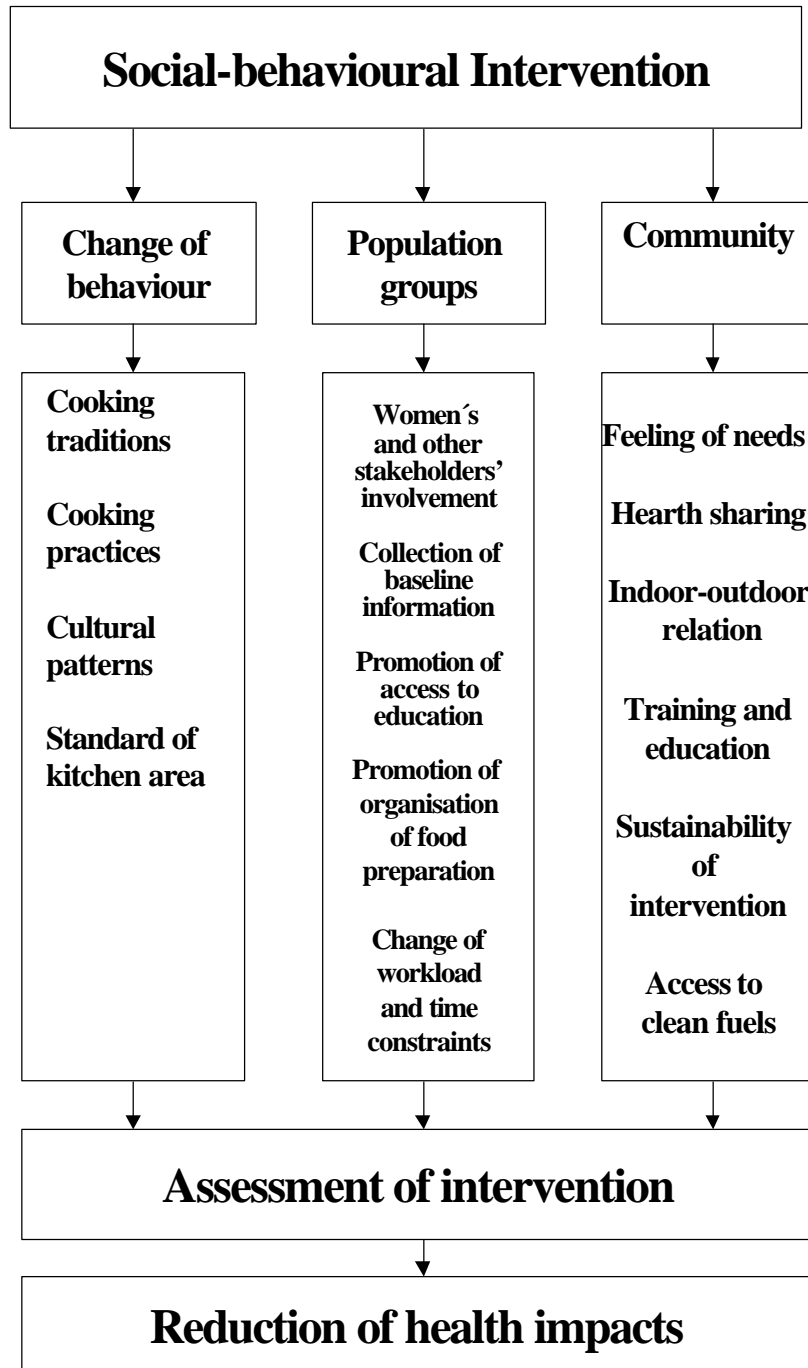
## 6.2.2 Management of indoor air quality in developing countries

The management of indoor air pollution in developing countries is a very important task of the building occupants if adverse impacts from e.g. open stove cooking and heating are to be avoided. Decisions of the building occupants, however, will often be driven by the household economy, convenience or habits rather than by minimal health risk considerations with respect to activities, facilities, and materials used indoors. Legislative and economic mechanisms should encourage individuals to manage the indoor environment in a health promoting way by means of technical and behavioural interventions. WHO has summarized technical and social-behavioural interventions in a publication on indoor air pollution from the use of biomass fuels (WHO 1992). Both types of interventions are depicted in figures 6.4 and 6.5.

**Figure 6.4 Technical interventions for reducing health impacts from use of biomass fuel in households**

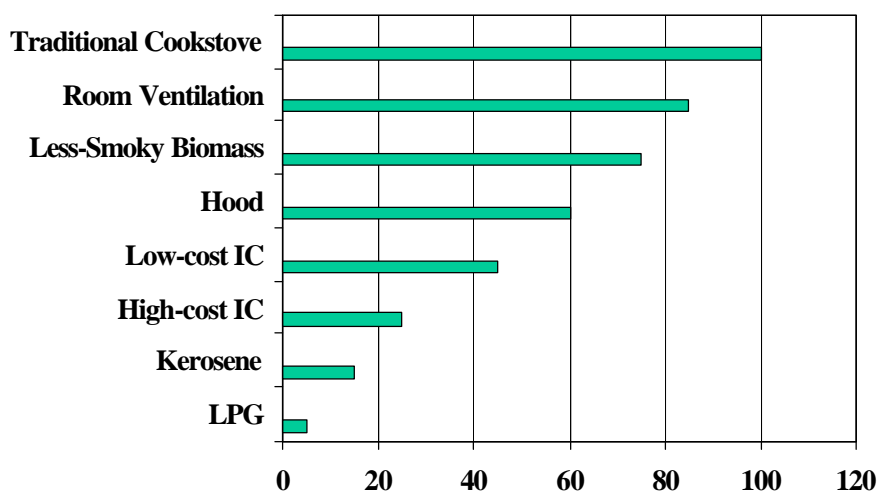


**Figure 6.5 Social-behavioural interventions for reducing health impacts from use of biomass fuels in households**





An overview of the effectiveness of major technical interventions for reducing ill-health from the use of household solid fuel is shown in Figure 6.6. This section concentrates in the following on only the major technical options and provides a brief outline of the issues involved.



IC = Improved cookstove with flue (chimney)

**Figure 6.6 Effectiveness of potential exposure interventions: Percent of exposure compared to using traditional cookstove without flue – typical values.**

#### **6.2.2.1 Improved ventilation**

An obvious solution when observing a village kitchen containing an unvented woodstove is better ventilation - i.e. more windows or other openings. In practice, however, there are often severe constraints because of weather, security or architecture. Thus, while better ventilation can clearly help in many circumstances, it is not a solution applicable to all situations.

#### **6.2.2.2 Improved stoves - chimneys**

Standard industrial hygiene suggests that general ventilation is not always sufficient as a means of exposure control, and that ventilation at the individual workplace is needed. Improved stoves utilizing flues or hoods can reduce indoor exposures substantially, particularly in the vicinity of the stove. Studies in developing countries show that pollution levels in homes can be lowered by nearly a factor of ten in ideal circumstances when well-constructed and maintained improved stoves are used.

Unfortunately, however, such circumstances do not seem to prevail in a large percentage of households. Inexpensive stoves tend to deteriorate through use of poor quality materials, poor construction and poor maintenance, leading at best to a factor of three improvement in exposures

over long periods. In a large number of studies, no statistically significant difference was found between the indoor concentrations of particles in homes using these improved stoves, and those in homes of their neighbours, who used open fires.

Another factor reducing the health benefit of even well-operating chimney stoves is that they exhaust emissions outdoors. In a built-up areas, such as exists in most urban slums and many villages, outdoor levels of air pollutants around the houses can significantly affect indoor levels. This "neighborhood effect" is not well-characterized, but it can limit practical exposure reduction.

If not carefully designed, chimney stoves can decrease fuel efficiency. The natural draft of the flue can so reduce the heat transfer to the cooking pots that overall efficiency is reduced. Perversely, in an attempt to prevent this effect, improved chimney stoves often have dampers to reduce the airflow. While overall efficiency may increase due to increased heat transfer efficiency, combustion efficiency can be impaired by poor air supply. The result, therefore, is paradoxically an increase in both fuel efficiency and emissions, although perhaps also lower exposures since the emissions are released outdoors.

In spite of these problems, chimney stoves seem to offer a short-term solution in many situations, but they probably need to be considered as only a first step in managing indoor emissions from fuel combustion.

#### **6.2.2.3 Improved stoves - combustion**

Wood and most other biomass fuels have few intrinsic contaminants, so that in ideal circumstances, virtually complete combustion can almost eliminate health-damaging emissions. In many developed countries, wood-burning stoves are subject to severe restrictions on emissions. By application of good engineering, remarkable improvements in emission performance has been achieved in such devices. This includes not only the use of catalytic converters, but also clever designs incorporating fluidized beds or secondary combustion.

Unfortunately, it can be difficult to design inexpensive devices that can reliably achieve high combustion efficiency and low emissions. A "low cost" low-emissions metal heating stove in the USA might cost \$500. More typically, costs are twice this or more. Improved stove programmes in many developing countries attempt to keep costs well under \$20. The technical potential seems high for filling this gap and inexpensive devices utilizing downdraft designs, for example, have achieved remarkable combustion efficiency in experimental situations. Much work remains to be done before such devices are practical on a global scale.

#### **6.2.2.4 Fuel**

Although the potential of truly low-emission stoves is alluring, improved fuels are the only proven long-term approach to the indoor air quality problem in developing countries. History has shown that people generally move to higher-quality fuels given access and affordability. This observation has led to the concept of the "energy ladder." At its bottom rung are the lowest-quality biomass fuels, grass, shrubs and roots; next come agricultural residues of dung and crop wastes; and then the highest-quality unprocessed biomass fuel, wood.

Although improved stoves have important roles, the long-term approach to the indoor air quality problem in developing countries is probably to accelerate the natural movement up the energy ladder to liquid and gaseous fuels. These fuels can be made from biomass itself, for example in the forms of alcohol and biogas. Promoting movement up the energy ladder in some cases can be accomplished by changing government policies that restrict access to certain petroleum fuels because of concerns about balance of payments. In many cities, even at international prices, such fuels are often cheaper than buying wood or charcoal when all costs are considered. Here the constraint is often the up-front cost of the stove and storage system (pressurized tank), which could be provided by low-interest loans or other subsidies.

Subsidizing kerosene and bottled gas directly, however, has many problems. When this has been done, usually as a means to reduce the pressure on biomass resources, the poor often do not benefit much, while others benefit by using the fuels for unintended purposes, e.g., irrigation pumps and vehicles. The result is high cost to the society and little shifting of household fuel use. Creative new approaches using vouchers or other types of incentives are needed in this area, to confine the benefits of subsidies to those who need them and are the intended beneficiaries. In addition, much enhanced research is needed on conversion of biomass into liquid and gaseous fuels that are compatible with high-quality household environments.

Other renewable fuels, such as solar energy, have the potential to provide clean fuels at an affordable economic cost, and in some areas to wholly or partially replace those household fuels that create substantial indoor air quality problems.

#### **6.2.2.5 Conclusion: simple exposure indicators**

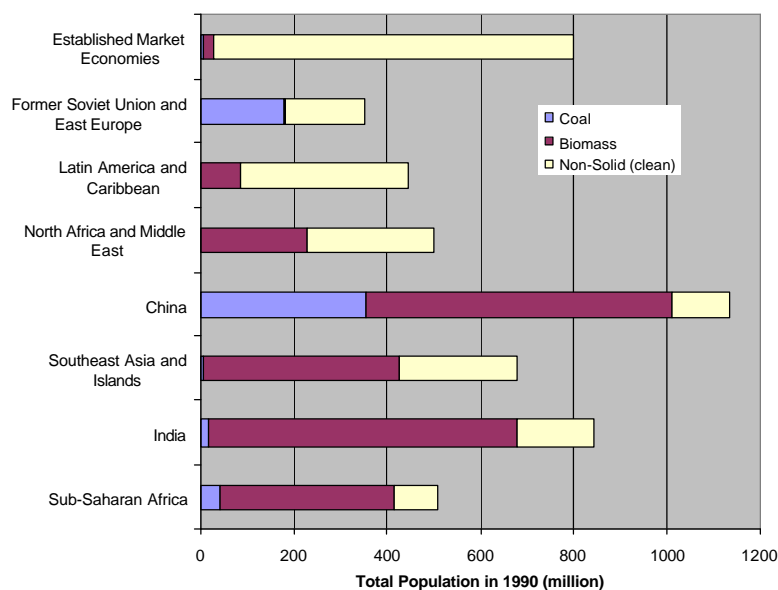
Although work on deriving simple exposure indicators urgently needs to be encouraged, realistically it is likely to be some years before sufficient environmental monitoring can be undertaken in most developing countries. In the interim, simple affordable exposure assessment tools are needed to assist in estimating the scale of the problem in local areas.

Simple indicators of poor water quality and sanitation have been developed and implemented successfully by WHO and others. These indicators are widely used to show trends and locate special problem areas. They do not require actual measurements but are able to be collected from other socio-economic information on households.

It should be possible to collect a set of indicators of the potential for air-pollution-related diseases from the segments of the population with:

Access to clean fuels (defined to be stoves using liquid and gaseous fuels or electricity, or defined as use of the clean fuels themselves) and,  
Access to venting [or ventilation] (defined to be the use of flues, chimneys, or outdoor cooking).

Although both of these indicators are subsets of broad goals related to achieving adequate, healthy and sustainable living environments, such simple indicators have high utility because of their ability to be quantified and easily understood. Trends over time and comparisons among different regions or populations are thus facilitated. Figure 6.7 is a preliminary attempt at reporting the first of these indicators at the global level.



Note: Most use of solid fuels in the first two regions is in vented heating stoves.

**Figure 6.7 World population using clean and potentially dirty (solid) household fuels**

Such indicators will help focus efforts to develop interventions. Eventual control of indoor air exposure from solid fuel cooking and heating is likely to require many years, and the integrated efforts of several sectors, including health, environment, energy and housing. Nevertheless, efforts to accelerate this process are likely to be rewarded with major improvements in human health.

## 7. Priority Setting in Air Quality Management

### 7.1 Introduction

This chapter is intended to give guidance on how to set priorities in rational air quality management. Actual priorities will differ for each country; therefore, each country sets priorities in air quality management according to its policy objectives, needs and capabilities. Priority setting in air quality management refers to prioritizing the health risks of air pollution, with corresponding prioritization of the pollutants, and concentrating on the most important sources of the pollutants. Conceptually, prioritizing health risks is straightforward (WHO 1999a; WHO 1999d). High priority health risks will be given to those compounds for which “high” toxicity and “high” exposure of the population are entailed.

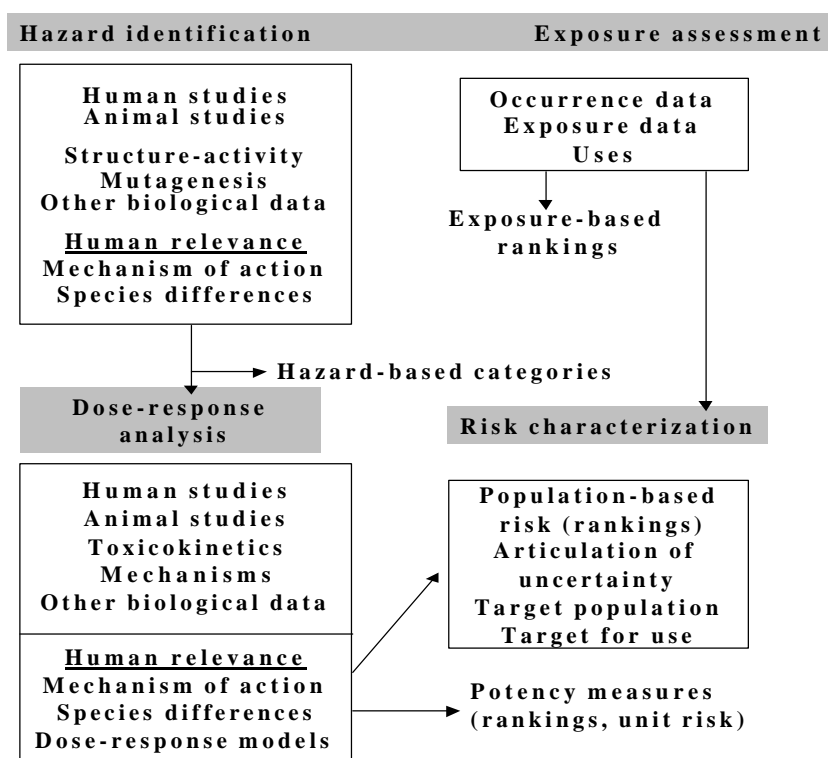


Figure 7.1 Basic elements of the estimation and prioritization of health risks

Conversely, low priority health risks involve agents of “low” toxicity and “low” exposure. “Medium” priority risks include compounds for which toxicity is “low” and exposure is “high,” or vice versa. Basic elements of the estimation and prioritization of health risks is illustrated in Figure 7.1 (Sexton 1993; Younes et al 1998).

For effective air quality management, goals, policies, strategies and tactics need to be defined.

these are discussed in Chapter 6.

A framework for a political, regulatory and administrative approach is required to guarantee a consistent and transparent derivation of air quality standards and to ensure a basis for decisions on risk-reducing measures and abatement strategies. In such a framework the following considerations need to be included:

- legal aspects.
- the potential of air pollution to cause adverse effects on health, taking into account populations at risk.
- exposure-response relationships of pollutants and pollutant mixtures and the actual exposure responsible for related health and/or environmental risks.
- the acceptability of risk.
- cost-benefit analysis.
- stakeholder contribution in setting standards.

## **7.2    *Legal aspects***

A legislative framework usually provides the basis for policies that set air quality standards at the municipal, regional, national or supranational level. The setting of standards strongly depends on the risk management strategy adopted which, in turn, is influenced by country-specific socio-political considerations and/or international agreements. Legislation and air quality standards vary from country to country, but in general the following issues may be taken into consideration:

- Identification and selection of the adverse effects on public health and the environment to be avoided.
- Identification of the population to be protected from the adverse health effects.
- Identification of the pollutants to be considered.
- The numerical value of the standards for the various pollutants or the decision-making process.
- Existing background concentrations of air pollutants.
- Applicable monitoring methodology and its quality assurance.
- Enforcement procedures to implement air quality standards within a defined time frame, to achieve compliance.
- Emission control measures and emission standards.
- Environmental impact assessment procedures.
- Identification of responsible enforcement authorities.
- Resource commitment.

Air quality standards may be based solely on scientific and technical data. However, other aspects such as technological feasibility, costs of compliance, prevailing exposure levels, social, economic and cultural conditions, are also usually considered in setting standards or in designing appropriate emission abatement measures. These are discussed in Section 2.4. As a consequence, air quality standards differ widely from country to country (WHO 1998b).

Air quality standards can set the reference point for emission control and abatement policies at national, regional or municipal levels. The latter two levels are only effective if long-distance transport of air pollution does not influence exposure. In the case of exposure to pollutants from

long-range transboundary transport, however, adequate measures can only be achieved through appropriate international agreements.

Air quality standards strongly influence the implementation of air pollution control policies. In many countries, there is an obligation to develop action plans at the municipal, regional or national level to abate air pollution (clean air implementation plans) if standards are exceeded. Such plans have to address all relevant sources. Air quality standards also play a role in environmental impact assessments and in the provision of public information on the state of the environment.

### **7.3     *Adverse effects on health***

In setting air quality standards on the basis of air quality guidelines, it is necessary to define from which effects the population is to be protected. Health effects range from death and acute illness, through chronic and lingering diseases, to temporary physiological or psychological changes. The distinction between adverse and non-adverse effects poses considerable difficulties. WHO has given a definition of adverse effects as “any effect resulting in functional impairment and/or pathological lesions that may affect the performance of the whole organism, or which contribute to a reduced ability to respond to an additional challenge” (WHO 1987). A more recent definition was given in the framework of the International Programme for Chemical Safety in its Environmental Health Criteria Series (WHO 1994c): “An adverse effect is any change in morphology, physiology, growth, development or life span of an organism which results in impairment of functional capacity, or impairment of capacity to compensate for additional stress, or increase in susceptibility to the harmful effects of other environmental influences.” Even this elaborate definition incorporates significant subjectivity and uncertainty in defining an adverse effect of air pollutants on health.

More serious effects are generally accepted as adverse. But when the health effects are either temporary and reversible, or involve biochemical or functional changes with uncertain clinical significance, a judgement is required on whether these less serious effects should be considered when deriving the standards. Judgements as to whether the health effects are adverse may differ between countries, because of factors including different cultural backgrounds and different levels of health status. The use of biomarkers or other indicators of exposure may provide a basis for setting air quality standards. Changes in such indicators, while not necessarily being adverse effects in themselves, may be harbingers of adverse effects on health. An example is blood lead content as an indicator of likely impairment of neuro-behavioural development.

### **7.4     *Population at risk***

The population at risk is that part of the population that is exposed to enhanced concentrations of air pollution. Each population has sensitive groups or sub-populations that are at higher risk for developing health effects following exposure to air pollutants. Sensitive groups include individuals impaired by concurrent diseases or other physiological limitations, and those with specific characteristics, which makes them more vulnerable to air pollutants (e.g. infants, elderly people). Other groups may be judged to be at higher risk due to enhanced exposure (outdoor workers, athletes, children). The sensitive groups in a population may vary across countries due to differences in medical care, nutritional status, lifestyle, and/or prevailing genetic factors, or

due to the existence of endemic diseases or the prevalence of debilitating diseases

## **7.5 Exposure-response relationships**

Chapter 3 provides exposure-response relationships for a number of pollutants, including graphs for particulate matter and O<sub>3</sub>. The percent change of various health endpoints, such as daily mortality and hospital admissions, are derived for a 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> and PM<sub>2.5</sub> concentrations. Assuming linearity, the relationships apply from 0 and 200 µg/m<sup>3</sup>. For carcinogenic compounds quantitative assessment of the unit risks provides an approximate estimate of responses at different concentrations. In setting standards, the definition of acceptable risk is related to risk perception and economic and social circumstances.

In developing standards, regulators should consider the degree of uncertainty in the exposure-response relationships provided in the air quality guidelines. Differences in the population structure (age, health status) climate (temperature and humidity), and geography (altitude, environment) can influence the prevalence, frequency and severity of health effects. Consequently, modified exposure-response relationships should be applied when setting standards (see Section 2.4).

## **7.6 Exposure characterization**

When setting standards it is not enough to simply consider the pollutant concentrations in ambient air. Personal exposure of the population should also be considered. As discussed in Section 4.2.3, the total exposure of people to pollutants also depends on the time people spend in the polluted environments, i.e. outdoor, indoor, workplace, in-vehicle etc. Exposure also depends on the various routes of intake of the pollutants into the human body, for example air, water, food and tobacco smoking. Multiple exposures may vary across these routes which should be considered in the standard setting procedure. In deriving air quality standards, the size of the population at risk (i.e. exposed to enhanced air pollutant concentrations) is also an important factor to consider. Models of exposure estimates should be used in addition to ambient and indoor concentration monitoring.

## **7.7 Risk assessment**

The development of air quality standards should be based on health and ecological risk models. Increasingly, these models are used to inform policy makers on some of the possible consequences of air pollutants at levels corresponding to various options for standards. Using this information, the policy maker can better assess the effects of air pollution.

Regulatory risk assessment in air pollution management includes a consideration of hazard identification, exposure-response relationships, exposure assessment and quantitative risk characterization. The first step, hazard identification, and to some extent exposure-response relationships, have already been provided in the *Guidelines for Air Quality*. Exposure assessment may predict changes in exposure associated with reductions in emissions from a specific source or group of sources. When using ambient air concentrations in the assessment of exposure, the issues discussed in Section 7.4 have to be taken into consideration. The final



step in regulatory risk assessment, risk characterization, refers to the quantitative estimation of the health effects in the population at risk. Examples for such estimates were given by Hong 1995; Ostro 1996; Schwela 1996a; Schwela 1996b; Schwela 1998; Murray and Lopez 1996. Regulatory risk assessments are likely to result in different risk estimates across countries and economic regions, owing to differences in exposure patterns, and in the size and characteristics of sensitive groups. Differences in the legislation and availability of information may also lead to differing results. There are many uncertainties at each step of a regulatory risk assessment. Therefore, the methods used to conduct the risk assessments should be clearly described and the limitations associated with the analysis discussed. A sensitivity and uncertainty analysis should be performed to characterize the major uncertainties of the risk estimates.

## **7.8     *Acceptability of risk***

In the absence of thresholds for the onset of health effects - as in the cases of fine and ultra-fine particulate matter and carcinogenic compounds - the selection of an air quality standard requires that the regulator determine an acceptable risk for the population. This also applies in cases where thresholds are present, but it would not be feasible to adopt air quality guidelines as standards because of economic or technical constraints. The acceptability of the risks and, therefore, the standards selected, depends on the expected incidence and severity of the potential effects, the size of the population at risk, the perception of related risks and the degree of scientific uncertainty that the effects will occur at a specific level of air pollution. For example, if a suspected but uncertain health effect is severe, and the size of the population at risk is large, a more cautious approach would be appropriate than if the effect were less severe, or if the population were smaller.

The acceptability of risk may vary among countries because of differences in social norms, the degree of adversity and risk perception in the general population, and because of the influences of various stakeholders. Risk acceptability is also influenced by how the risks associated with air pollution compare with risks from other pollution sources or human activities.

## **7.9     *Cost-benefit analysis***

In the derivation of air quality standards from air quality guidelines two different approaches for decision making can be applied. Decisions can be based purely on health, cultural and environmental consequences with little weight to economic efficiency. This approach would have the objective of reducing the risk of adverse effects to a socially acceptable level. The second approach would be based on a formal cost-effectiveness or cost-benefit analysis (CBA), with the objective of identifying the control action that achieves greatest net economic benefit, or is the most economically efficient. The development of air quality standards should account for both extremes, and encompass a process that involves stakeholders and assures social equity to all involved. It should also provide sufficient information to guarantee that the stakeholders understand the scientific and economic consequences. Cost benefit analysis is discussed in Section 2.4.7.

The steps in a cost-benefit analysis include:

Identification and cost analysis of control action (emission abatement strategies and tactics).

Assessment of air quality and population exposure, with and without the control action.  
Identification of benefit categories (health effects, material damage, damage to ecosystems).  
Comparison of health and environmental effects, with and without control action.  
Comparison of the estimated costs of control action and benefits.  
Sensitivity and uncertainty analysis.

*Cost analysis of control action.* To determine the financial burden of control action, cost assessment should include all costs of investment, operation and maintenance. This is usually not a problem for direct abatement measures at the source, which can be monetarized. It may be more difficult to determine the costs of indirect measures, such as alternative traffic plans or change in behaviour of individuals. Even when secondary air pollutants are not monitored they should be included in the CBA.

*Assessment of air quality.* An assessment of air quality includes information about expected air quality, both with and without control measures. Typically, the assessment is based on air quality monitoring data and dispersion modelling. The types of data requested in a CBA include pollutant concentrations (evaluated for relevant averaging times), site classification, emission data (with sufficient temporal and spatial resolution), and meteorological and topographical data relevant to the dispersion of emissions. The air quality guidelines are based on a set of health and environmental effect endpoints determined by consensus and scientific judgement. Other effects that were not included in the air quality guidelines may occur in a special local situation and may be considered in an analysis of costs and benefits.

*Identification of benefit categories.* Relevant benefit categories defined in existing CBAs include: mortality and morbidity due to long- and short-term exposures, climate and visibility effects, non-human biological effects, soiling and material damage (USEPA 1987a,b); total premature mortality and mortality due to respiratory and cardiovascular diseases, hospital admissions, upper and lower respiratory symptoms, symptom exacerbation among asthmatics and reduced activity days (EC DG XII 1995; GVF 1996). The quantification of benefit categories included in a CBA is a difficult task. Some indicators of diseases can be quantified, such as the use of medication, number of hospital admissions, outpatient visits or days of labour lost. Other effects, such as premature death of the elderly or excess mortality present more difficult problems. Well-being, the quality of life or the value of ecosystems may be difficult or even impossible to monetarize. The values assigned to benefit categories might differ substantially among countries due to different cultural or social attitudes. It is better, however, to include the relevant benefit categories, even if the economic assessment is uncertain or ambiguous.

*Comparison of health and environmental effects.* A comparison of the health and environmental benefits with and without control action, and information on exposure-response relationships, should be combined with information on air quality assessment. The combined information is applied to the population at risk. To assess the influence of air pollution, knowledge is needed of the prevalence of different health effects in the population at risk and the percent increase of health effects with one unit of pollutant concentration.

*Comparison of costs and benefits.* The CBA should provide a benefit-cost ratio based on monetarized costs and benefits, and be accompanied by a description of the non-monetarized items that also should be considered. Monetary valuation of control actions, and of the effects on health and the environment, may be different in concept and vary substantially from country

to country. There may be differences in assessing costs, and the relative value of benefit categories can vary. The costs of environmental policy action may also vary according to the scale and level of decision making, e.g. with respect to transfer costs (taxes, subsidies aimed at redistribution of costs). Benefits may also be transferable between groups of the population. Furthermore, action taken to reduce one pollutant may increase or decrease the concentration of other pollutants. These additional effects should be considered, as well as pollutant interactions, which may lead to double counting of costs or benefits, or to disregarding some costly but necessary action. Due to different levels of knowledge about the costs of control action and the costs of health effects there is a tendency to overestimate the cost of control action and underestimate the benefits. Thus, CBAs in two areas with otherwise similar conditions may differ significantly.

*Sensitivity and uncertainty analysis.* In a CBA, sensitivity analysis provides valuable insight into the properties and assumptions underlying the results of the CBA. Sensitivity methods include comparison with other CBA studies, recalculation of the whole chain of CBA using other assumptions, or ranges around a central value. Sensitivity analysis has to be carefully designed and requires considerable resources.

In conclusion, CBA is a highly interdisciplinary task. Appropriately applied, CBA is a legitimate and useful way to provide information for risk managers making decisions that will affect public health and the environment. CBAs should be peer-reviewed and not be used as the sole and overriding determinant of these decisions.

## **7.10 Review of standard setting**

The setting of standards should involve stakeholders (industry, local authorities, non-governmental organisations and the general public) that assures, as far as possible, social equity or fairness to all the parties involved. It should also provide sufficient information to guarantee that stakeholders understand the scientific and economic consequences. The earlier stakeholders are involved the more likely is their Cupertino. Transparency in moving from air quality guidelines to air quality standards helps to increase public acceptance of necessary measures. Raising public awareness of air pollution-induced health and environmental effects (changing of risk perception) serves to obtain public support for necessary control action. Information to the public about the air quality during episodes, as well as the risks entailed, lead to a better understanding of the issue (risk communication).

Air quality standards should be regularly reviewed and revised as new scientific evidence on the effects on public health and the environment emerges.

## **7.11 Enforcement of air quality standards: clean air implementation plans**

The aim of enforcement is to attain compliance with the standards. The instruments used to achieve this goal are Clean Air Implementation Plans (CAIPs). The outline of such a plan should be defined in regulatory policies and strategies. Clean air implementation plans were formulated in several developed countries during the 1970s and 1980s. Air pollution was characterized by many sources and many different types of air pollutants. Consequently it was extremely difficult

to assess the public health risks associated with a single source or group of sources. As a consequence, on the basis of the polluters pay principle (Chapter 6), sophisticated tools were developed to assess the sources (e.g. air pollutant concentrations, health and environmental effects, control measures) and to make a causal link between emissions, air pollution and the necessary and efficient control measures. A typical clean air implementation plan (CAIP) includes:

Description of area.

- Emissions inventory.
- Air pollutant concentrations inventory - monitored and simulated.
- Comparison of emissions and air quality standards or guidelines.
- Inventory of effects on public health and the environment.
- Causal analysis of effects and attribution to individual sources.
- Control measures and their costs.
- Transportation and land-use planning.
- Enforcement procedures.
- Resource commitment.
- Projections for the future.

Costs of public health and environmental effects have not been included in published clean air implementation plans. However, the CAIP has been a very efficient instrument of air pollution abatement in developed countries (Schwela and Köth-Jahr 1994, WHO 1997a). In the cities of developing countries, or countries in transition, much simplified CAIPs would have to be developed. The main sources of emissions in many cities of the developing world are old vehicles and some industrial sources such as power plants, brick kilns, cement factories and a few others. Their relative contribution to air pollution could be determined by use of rapid emission inventories. The emission factors used in such inventories are published (WHO 1993a 1993b), and a PC programme is available (WHO 1995; WHO 1997b; WHO 1998) that enables emissions and ambient air concentrations to be estimated, and the impact of possible control measures to be evaluated. Projections for the future can also be evaluated by the programme. By using the experience obtained in developed countries, the control action to be taken is very often obvious. As a consequence less monitoring could be sufficient, and dispersion models could help simulate spatial distributions of concentrations when little useful monitoring data are available.

## Appendix 1

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## Appendix 2

### Acronyms

ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Acceptable daily intake
ALA	-Levulinic Acid
ALAD	Amino-Levulinic Acid Dehydratase
AMIS	Air Management Information System (WHO, Healthy Cities)
AMRO	WHO Regional Office for the Americas
AR	Allergic Rhinitis
ARI	Acute Respiratory Infection
ATDSR	Agency for Toxic Substances and Disease Registry (USA)
BMEPB	Beijing Municipal Environment Protection Bureau
BRI	Building Related Illness
BS	Black Smoke
bw	body weight
CaCO <sub>3</sub>	Calcium carbonate
CAIP	Clean Air Implementation Plan
CBA	Cost-benefit analysis
Cd	Cadmium
CEN	European Committee for Standardization
CFC	ChloroFluoroCarbon
CFR	Code of Federal Regulations
CH <sub>4</sub>	Methane
CI	95% Confidence Interval
CMD	Cyclopedic Medical Dictionary
CMD	Count Median Diameter
CNS	Central Nervous System
CO	Carbon monoxide
CO <sub>2</sub>	Carbon dioxide
COHb	Carboxyhemoglobin
COPD	Chronic Obstructive Pulmonary Disease
CS <sub>2</sub>	Carbon disulphide
Cu	Copper
DOAS	Differential Optical Absorption System
DQOs	Data Quality Objectives
EA	Environment Agency of Japan
EC DG	European Commission Directorate General
ECA	European Concerted Action
ECE	Economic Commission for Europe
ECEH	WHO European Centre of Environment and Health
EHC	Environmental Health Criteria
ETS	Environmental Tobacco Smoke
EURO	WHO Regional Office for Europe
FEF <sub>25-75</sub>	Forced Expiratory Flow – interquartile range
FEP	Free Erythrocyte Protoporphyrin
FEV <sub>1</sub>	Forced Expiratory Volume in first second of expiration
FVC	Forced Vital Capacity
GEMS	Global Environmental Monitoring System (UNEP/WHO)



GC/FID	Gas Chromatography/Flame Ionization Detector
GIS	Geographic Information System
GVF	Dienst für GesamtVerkehrsFragen des Eidg. Verkehrs- und Energiewirtschafts-departementes (Switzerland)
H <sup>+</sup>	Hydrogen ion
H <sub>2</sub> S	Hydrogen sulphide
H <sub>2</sub> S O <sub>4</sub>	Sulphuric acid droplets
HC	Hydrocarbons
HCl	Hydrochloric acid
HF	Hydrogen fluoride
HNO <sub>3</sub>	Nitric acid
HOP	Hydroxyproline
HVAC	High Volume Air conditioning System
IARC	International Agency for Research on Cancer
ICRP	International Commission on Radiological Protection
IgE	Immunglobulin E
IgG	Immunglobulin G
IOMC	Inter-Organization Programme for the sound Management of Chemicals
IPCS	International Programme on Chemical Safety
IR	Infrared Radiation
ISO	International Standards Organization
ISO/DIS	ISO Draft International Standard
ISO/FDIS	ISO Final Draft International Standard
KI	Potassium iodide
KOH	Potassium hydroxyde
LOAEL	Lowest-Observed-Adverse-Effect-Level
LOEL	Lowest-Observed-Effect-Level
LPG	Liquefied petroleum gas
µm	micrometer
µg	microgram
MMD	Mass median diameter
MMVF	Man-Made Vitreous Fibres
Mn	Manganese
MSDS	Material Safety Data Sheets
Na <sub>2</sub> CO <sub>3</sub>	Sodium carbonate
NaCl	Sodium chloride
NaNO <sub>2</sub>	Sodium nitrite
NH <sub>3</sub>	Ammonia
NH <sub>4</sub> <sup>+</sup>	Univalent ammonium radical
NH <sub>4</sub> HSO <sub>4</sub>	Ammonium bisulphate
(NH <sub>4</sub> ) <sub>2</sub> HSO <sub>4</sub>	Ammonium hydrogen sulphate
NH <sub>4</sub> NO <sub>3</sub>	Ammonium nitrate
Ni	Nickel
NO	Nitrogen oxide
NO <sub>2</sub>	Nitrogen dioxide
NO <sub>x</sub>	Nitrogen oxides
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
O <sub>3</sub>	Ozone
OECD	Organization for Economic Cupertino and Development
OEH	Occupational and Environmental Health, WHO, PHE, Geneva
OH	Odds Ratio
PAH	Polycyclic (Polynuclear) Aromatic Hydrocarbons

PAHO	Pan American Health Organization
PAN	PeroxyAcetyl Nitrate
Pb	Lead
PBPK	Physiologically Based Pharmacokinetic model
PCDDs	Polychlorinated dibenzodioxins
PCDFs	Polychlorinated dibenzofurans
PCBs	Polychlorinated biphenyls
PEF	Peak Expiratory Flow
PEFR	Peak Expiratory Flow Rate
PHA	PhytoHemAgglutinin
PHE	Department for Protection of the Human Environment, WHO, Geneva
PM	Particulate matter with no regard to size of particles
PM <sub>10</sub>	Concentration of particles with aerodynamic particle diameters of less than 10 micrometers.
PM <sub>2.5</sub>	Concentration of particles with aerodynamic particle diameters of less than 2.5 micrometers.
2.5	
QA/QC	Quality Assurance/Quality Control
QAP	Quality Assurance Programme
R-SH	Mercaptan
RSP	Respirable Suspended Particles
SBS	Sick Building Syndrome
SERPLAC	Secretarias Regionales de Planificación y Coordinación
Si	Silicium
SO <sub>2</sub>	Sulphur dioxide
SO <sub>3</sub> <sup>-</sup>	Sulphur trioxide ion
SO <sub>4</sub> <sup>2-</sup>	Sulphate ion
SPM	Suspended particulate matter
STPD	Standard Temperature and Pressure Dry
sRAW	Specific AirWay Resistance
TEA	Triethanolamine
TC	Tolerable concentration
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
TCM	Tetrachloromercurate
TDI	Tolerable daily intake
TEOM	Tapered Element Oscillating Microbalance
TEQ	Toxicity Equivalent concentration or uptake
TI	Tolerable intake
TSP	Total Suspended Particles
UK	United Kingdom
UN	United Nations
UNEP	United Nations Environment Programme
UR	Unit Risk
USEPA	United States Environmental Protection Agency
USA	United States of America
UV	Ultra-Violet Fluorescence
V	Vanadium
VOC	Volatile Organic Compounds
WHO	World Health Organization
WRAC	Wide Ranging Aerosol Classifier
Zn	Zinc

## Appendix 3

### ***Glossary***

Acidity	The quality of possessing hydrogen ions (CMD 1997).
Adverse effect	Change in morphology, physiology, growth, development or life span of an organism exposed to air pollution, which results in impairment of functional capacity or impairment of capacity to compensate for additional stress or increase in susceptibility to the harmful effects of other environmental influences (WHO 1994a).
Aerosol	A suspension in a gaseous medium of solid particles, liquid particles or solid and liquid particles having a negligible falling velocity (ISO 1994).
Airway permeability	Capability of allowing the passage of air through the natural passageway for air to and from the lungs (CMD 1997).
Allergen	Any substance that causes manifestations of allergy. Among common allergens are inhalants, foods, drugs, infectious agents, contactants, and physical agents (CMD 1997).
Allergic	Pertaining to, sensitive to, or caused by an allergen (CMD 1997).
Anemia	A reduction in the number of circulating red blood cells (CMD 1997).
Asthma	A disease caused by increased responsiveness of the tracheobronchial tree to various stimuli, which results in paroxysmal constriction of the bronchial airways (CMD 1997). Also see paroxysm.
Atherogenic	Pertaining to the formation of degenerated or thickened walls of the larger arteries, marked by cholesterol-lipid-calcium deposits (CMD 1997).
Biomarker	Any parameter that can be used to measure an interaction between a biological system and an environment agent, which may be chemical, physical or biological (WHO 1993).
Biomass	Organic substance of biotic origin: either living organisms or dead substances such as wood, crop residues, or animal dung.
Biomass smoke	Term used for convenience for the smoke generated by burning biomass.
Biotic	Of or relating to life (Webster 1994).
Bronchi	The two main branches leading from the trachea to the lungs, providing a passageway for air (CMD 1997).

Bronchiole	One of the smaller subdivisions of the bronchial tube (CMD 1997).
Bronchiolitis	Inflammation of the bronchioles (CMD 1997).
Bronchitis	Inflammation of the mucous membrane of the bronchial airways (CMD 1997).
Bronchoconstriction	Constriction of the bronchial tubes (CMD 1997).
Bronchodilator	A drug that expands the bronchial tubes by relaxing bronchial muscle (CMD 1997).
Building related illness	Illness related to indoor exposures to biological agents (e.g. fungi, bacteria), biological and chemical substances (e.g. endotoxins, mycotoxins, radon, carbon monoxide, formaldehyde) which is experienced by some people working or living in a particular building and it does not disappear after leaving it.
Carbon dioxide	A colourless, odourless, non-combustible gas, formula CO <sub>2</sub> . It is approximately 50% heavier than air, of which it is a normal constituent. It is formed by certain natural processes (see carbon cycle) and by the combustion of fuels containing carbon, and it has been estimated that the amount in the air is increasing by 0.27% annually. Only in the most exceptional circumstances do local concentrations of carbon dioxide in air rise to levels that are dangerous to health, but it plays a significant role in the decay of building stones and in corrosion (WHO 1980).
Carbon monoxide	A colourless, almost odourless, tasteless, flammable gas, formula CO. It is produced, <i>inter alia</i> , by the incomplete combustion of organic materials (e.g. in automobile engines) and normally occurs in trace amounts in the atmosphere. At concentrations exceeding about 100 cm <sup>3</sup> /m <sup>3</sup> (0.01%) it is highly toxic. Its affinity for hemoglobin (with which it forms carboxyhemoglobin) is between 200 and 300 times that of oxygen, and it has the effect of reducing the oxygen-transport capacity of hemoglobin and leading to death by asphyxiation. Concentrations of carbon monoxide in city streets (arising mainly from motor vehicle exhausts) can be sufficiently high to cause concern, as can those resulting from tobacco smoking in unventilated rooms (WHO 1980).
Carcinogenicity	The production of cancer, equivalent to carcinogenesis (CMD 1997)
Cardiovascular	Pertaining to the heart and blood vessels (CMD 1997).
Centri-acinar	Pertaining to the central terminal respiratory gas exchange unit of the lung, composed of airways and alveoli distal to a terminal bronchiole (CMD 1997).
Chemiluminiscence	Cold light or light resulting from a chemical reaction and without heat

	production (CMD 1997).
Chromatography	The separation of two or more chemical compounds in solution by their removal from the solution at different rates (CMD 1997).
Chronic obstructive Pulmonary disease (COPD)	A disease process that decreases the ability of the lungs to perform ventilation. Diagnostic criteria include a history of persistent dyspnea on exertion, with or without chronic cough, and less than half of normal predicted maximum breathing capacity. Diseases that cause this condition are chronic bronchitis, pulmonary emphysema, chronic asthma, and chronic bronchiolitis (CMD 1997).
Coagulation, blood	The process of clumping together of blood cells to form a clot (CMD 1997).
Cognitive	Adjective to cognition, the awareness with perception, reasoning, judgement, intuition and memory, the mental process by which knowledge is acquired (CMD 1997).
Collagen	A strong, fibrous insoluble protein found in connective tissue (CMD 1997)
Combustion	A chemical reaction in which a material combines with oxygen with the evolution of heat: "burning". The combustion of fuels containing carbon and hydrogen is said to be complete when these two elements are all oxidized to carbon dioxide and water. Incomplete combustion may lead to (1) appreciable amounts of carbon remaining in the ash; (2) emission of some of the carbon as carbon monoxide; and (3) reaction of the fuel molecules to give a range of products of greater complexity than that of the fuel molecules themselves (if these products escape combustion they are emitted as smoke) (WHO 1980).
Contagion	A disease that is easily transmitted from host to host by casual dermal contact or respiratory droplets (CMD 1997).
Coproporphyrin	A porphyrin present in urine and feces (CMD 1997).
Cor pulmonale	Hypertrophy or failure of the cavity of the heart that receives blood from the right atrium and pumps it into the lungs via the pulmonary artery (CMD 1997).
Cough	A forceful and sometimes violent expiratory effort preceded by a preliminary inspiration (CMD 1997).
Cytochrome	An iron-containing protein found in the mitochondria (cell parts of rod or oval shape that perform a distinctive function) of eukaryotic cells (CMD 1997).
Cytochrome oxidase	An enzyme complex of two cytochromes and two copper atoms found in the mitochondria of eukaryotic cells (CMD 1997).

Cytochrome P-450	A group of enzymes, called hemethiolate protein P450, present on every type of cell in the body except red blood cells and skeletal muscle cells (CMD 1997).
Diesel exhaust	<p>Diesel exhaust emissions contain hundreds of chemical compounds, which are emitted partly in the gaseous phase and partly in the particulate phase of the exhaust. The major gaseous products are carbon dioxide, oxygen, nitrogen, and water vapour; carbon monoxide, sulphur dioxide, nitrogen oxides, and hydrocarbons and their derivatives are also present. Benzene and toluene are present in the lower range (percentage weight) in the gaseous part of the hydrocarbon fraction. Other gaseous exhaust compounds are low-relative-molecular-mass polycyclic aromatic hydrocarbons.</p> <p>A main characteristic of diesel exhaust is the release of particles at a rate about 20 times greater than that from gasoline-fuelled vehicles. The particles are composed of elemental carbon, organic compounds adsorbed from fuel and lubricating oil, sulphates from fuel-sulphur, and traces of metallic components. Most of the total particulate matter appears to occur in the submicrometre range, between 0.02 and 0.5 <math>\mu\text{m}</math> (WHO 1996b).</p>
Disease	A pathological condition of the body that presents a group of clinical signs, symptoms, and laboratory findings peculiar to it and setting the condition apart as an abnormal entity differing from other normal or pathological condition (CMD 1997).
Dry deposition	Removal of contaminants of air onto a substrate without involvement of rain, clouds or fog.
Dust	Small solid particles, conventionally taken as those particles below 75 $\mu\text{m}$ in diameter, which settle out under their own weight but which may remain suspended for some time (ISO 1994). National standards may be more specific and include particle diameters or a definition in terms of a sieve of specified aperture. Dust occurs in the atmosphere both naturally and as a result of the activities of man (Willeke 1993).
Dyspnea	Air hunger resulting in laboured or difficult breathing, sometimes accompanied by pain (CMD 1997).
Effect	Change in morphology, physiology, growth, development of life span of an organism exposed to air pollution. It might be either an adverse effect or an alteration, which is not distinguishable from the range of a target variable observed in not exposed organisms of the same species (WHO 1994c).
Emergency Department	The portion of a hospital that treats patients experiencing an emergency (CMD 1997).
Emphysema	A chronic pulmonary disease marked by an abnormal increase in the size of air spaces distal (farthest from the centre) to the terminal bronchioles

	with destructive changes in their walls (CMD 1997)
Encephalopathic	Pertaining to any dysfunction of the brain (CMD 1997).
Endogenous	Produced or originating from within a cell or organism (CMD 1997).
Endometriosis	The presence of functioning ectopic (in an abnormal position) endometrial (pertaining to the lining of the uterus) glands and stroma (foundation-supporting tissues of an organ) outside the uterine cavity (CMD 1997).
Endotoxin	A lipopolysaccharide (linkage of molecules of lipids with polysaccharides) that is part of the cell wall of gram-negative bacteria (CMD 1997).
Environmental Tobacco Smoke (ETS)	ETS is generated by the combustion of tobacco products. ETS is a complex mixture of over 4000 compounds. These include over 40 known or suspected human carcinogens, such as 4-aminobiphenyls, 2-naphthylamine, benzene, nickel, and a variety of PAH and N-nitrosamines. A number of irritants, such as ammonia, nitrogen oxides, sulphur dioxide, various aldehydes, and cardiovascular toxicants, such as carbon monoxide and nicotine are also present (WHO 1999a).
Enzyme	An organic catalyst produced by living cells but capable of acting outside cells. Enzymes are proteins that change the rate of chemical reactions without needing an external energy source or being changed themselves (CMD 1997).
Epithelioma	A malignant tumour consisting primarily of epithelial cells (epidermis of the skin or a mucous membrane) (CMD 1997).
Epithelium	The layer of cells forming the epidermis of the skin and the surface layer of mucous and serous membranes (CMD 1997).
Erythrocyte	A mature red blood cell (CMD 1997).
Expiration	Expulsion of air from the lungs in breathing. Normally the duration of expiration is shorter than that of inspiration. In general, if expiration lasts longer than inspiration, a pathological condition such as emphysema or asthma is present (CMD 1997).
Exposure	Exposure to a chemical is the contact of that chemical with the outer boundary of the human body. The outer boundary of the human body is the skin and the openings into the body such as the mouth, the nostrils, and punctures and lesions in the skin (WHO 1999).
Exposure assessment	Quantitative or qualitative evaluation of the contact of a chemical with the outer boundary of the human body, which includes consideration of the intensity, frequency and duration of contact, the route of exposure (e.g. dermal, oral or respiratory), rates (chemical intake or uptake rates), the resulting amount that actually crosses the boundary (a dose), and the

	amount absorbed (internal dose) (WHO 1999).
Fibrotic	Marked by or pertaining to abnormal formation of fibrous tissues (CMD 1997).
Fine particles	Particles with aerodynamic diameters below 2.5 micrometer.
Fog	As international standard fog is a general term applied to a suspension of droplets in a gas. In meteorology, it refers to a suspension of water droplets resulting in a visibility of less than 1 km (ISO 1994). WMO defines fog as a suspension of very small, usually microscopic water droplets in the air, generally reducing the horizontal visibility at the earth's surface too less than 1 km (WMO 1992).
Folliculi	Small secretory sacs or cavities (CMD 1997).
Forced expiratory Volume (FEV)	The volume of air that can be expired after a full inspiration. The expiration is done as quickly as possible and the volume measured at precise times; at ½, 1, 2 and 3 seconds. This provides valuable information concerning the ability to expel air from the lungs (CMD 1997).
Fume	Aerosol of solid particles, usually from metallurgical processes, generated by condensation from the gaseous state, generally after volatilisation from melted substances and often accompanied by chemical reactions such as oxidation (ISO 1994). By extension, also the gases charged by particles resulting from a chemical process or a metallurgical operation (WHO 1980). Often used in the plural, <i>fumes</i> for visible clouds of gases, vapours, or aerosols that have an unpleasant and malodorous smell (WHO 1980; ISO 1994).
Function	The act of carrying on or performing a special activity. Normal function is the normal action of an organ. Abnormal activity or the failure of an organ to perform its activity is the basis of disease or disease processes (CMD 1997).
Genotoxic	Toxic to the genetic material in cells (CMD 1997).
Gestational	Pertaining to the length of time from conception to birth (CMD 1997).
Gram-negative	Losing the crystal violet stain and taking the colour of the red counterstain in Gram's method of staining bacteria (CMD 1997).
Gram-positive	Retaining the colour of the crystal violet stain in Gram's method of staining bacteria (CMD 1997).



Guideline	Any kind of recommendation or guidance on the protection of human beings or receptors in the environment from the adverse effects of air pollutants. As such, it is not restricted to a numerical value but might also be expressed in a different way, for example as exposure-response information or as a unit risk estimate (WHO 1998a).
Guideline value	A particular form of a guideline. It has a numerical value expressed either as a concentration in ambient air, a tolerable intake, or as a deposition level, which is linked to an averaging time (WHO 1998a). In the case of human health, the guideline value defines a concentration below which the risk for the occurrence of adverse effects is negligibly low. It does, however, not guarantee the absolute exclusion of effects at concentrations at or below the guideline value. For odorous compounds the guideline value represents an odour threshold.
Haze	A suspension in the atmosphere of extremely small (dry) particles, individually invisible to the naked eye, but which are numerous enough to give the atmosphere an appearance of opalescence together with reduced visibility (ISO 1994, WMO 1992).
Heat	Means both thermal energy and thermal energy transfer.
Hemangiosarkoma	A malignant neoplasm (new and abnormal formation of tissue) originating from the blood vessels (CMD 1997).
Hematological	Pertaining to the science concerned with blood and blood-forming tissues (CMD 1997).
Heme	An iron-containing non-protein portion of the hemoglobin molecule (CMD 1997).
Hemoglobin	The iron-containing pigment of the red blood cells which carries oxygen from the lungs to the tissues (CMD 1997).
Hepatocellular	Concerning the cells of the liver (CMD 1997).
Hepatotoxic	Toxic to the liver (CMD 1997).
Hydrocarbon	An organic compound containing only the elements carbon and hydrogen. The carbon atoms may be arranged either in open-ended chains, which may or may not be branched or in closed rings. There are two types of ring hydrocarbons: <i>alicyclic compounds</i> , consisting of three or more carbon atoms arranged in a closed ring (and whose properties are similar to those of the open-chain compounds of the same molecular mass), and aromatic compounds. The molecular structure of aromatic compounds is based on that of benzene, the simplest member of the class, which

contains six carbon atoms joined by three single and three double carbon-carbon bonds. Such compounds are described as *polycyclic* if they contain two or more rings; the term “polynuclear” (as in “polynuclear aromatic hydrocarbon”, frequently abbreviated as PAH) is also used. The major constituents of gasoline and other petroleum fuels are hydrocarbons of the open-chain type. These compounds are not considered to be a hazard to health even at the concentrations at which they are encountered in city air. Many aromatic hydrocarbons, on the other hand, are highly toxic (WHO 1980; WHO 1997). Well known examples of polycyclic aromatic hydrocarbons are anthracene, naphthalene, and benzo[a]pyrene (WHO 1980).

Hydroxyproline	An amino acid found in collagen (CMD 1997).
Hyperplasia	Excessive proliferation of normal cells in the normal tissue arrangement of an organ (CMD 1997).
Hypoimmunity	Diminished immunity.
Hypoxia	An oxygen deficiency (CMD 1997).
Hypoxic	Oxygen deficient (CMD 1997).
Illness	The state of being sick (CMD 1997).
Immune function	Function of being protected from or resistant to a disease or infection by a pathogenic organism as a result of the development of antibodies or cell-mediated immunity (CMD 1997).
Immunoglobulin	One of a family of closely related though not identical proteins capable of acting as antibodies, abbreviation Ig (CMD 1997).
Immunoglobulin A	The principal immunoglobulin in external gland secretions such as respiratory and intestinal mucin (mucus glycoprotein), saliva, and tears (CMD 1997).
Immunoglobulin E	An immunoglobulin that attaches to mast cells in the respiratory and intestinal tracts and plays a major role in allergic reactions, abbreviation IgE (CMD 1997).
Immunoglobulin G	The principal immunoglobulin in human serum, important in producing immunity in the infant before birth, abbreviation IgG (CMD 1997).
Inflammation	The non-specific immune response that occurs in reaction to any type of bodily injury (CMD 1997).
Influenza	An acute, contagious respiratory infection characterized by the

	sudden onset of fever, chills, headache, tenderness or pain in the muscles, and sometimes absolute exhaustion (CMD 1997).
Interstitial	The space or gap in a tissue or structure of an organ (CMD 1997).
Ischemic	Pertaining to a local and temporary deficiency of blood supply due to obstruction of the circulation to a part (CMD 1997).
Legionnaires' disease	A severe, often fatal disease characterized by pneumonia, dry cough, tenderness or pain in the muscles, and sometimes gastro-intestinal symptoms (CMD 1997).
Leukemia	A malignancy of the blood-forming cells in the bone marrow (CMD 1997).
Life expectancy	The number of years that an average person of a given age may be expected to live, according to mortality tables (CMD 1997).
Low birth weight	Abnormally low weight of a new-born, usually below 2000 g (CMD 1997).
Lower respiratory symptom	Symptom in the lower respiratory tract (i.e. the respiratory tract from trachea to bronchioles).
Lowest-observed-adverse-effect level	Lowest concentration or amount of a substance, found by observation or experiment, which causes an adverse effect (WHO 1994c).
Lowest-observed-effect level	Lowest concentration or amount of a substance, found by observation or experiment, which causes an effect.
Lung cancer	Cancer that may appear in the trachea, air sacs and other lung tubes. It may appear as an ulcer in the windpipe, as a nodule or small flattened lump, or on the surface blocking air tubes. It may extend into the lymphatic and blood vessels (CMD 1997).
Lysozyme	An enzyme found in white blood cells and in body secretions that destroys bacteria by breaking down their walls (CMD 1997).
Malaise	Discomfort, uneasiness, or indisposition, often indicative of infection (CMD 1997).
Metaplasia	Conversion of one kind of tissue into a form that is not normal for that tissue (CMD 1997).
Mist	Loose term applied to a suspension of droplets in a gas. In meteorology it relates to visibility of less than 2 km but greater than 1 km (ISO 1994). See also fog.

Morbidity	The number of sick persons or cases of disease in relationship to a specific population (CMD 1997).
Morphological	Pertaining to the science of structure and form of organisms without regard of function (CMD 1997).
Mortality	The death rate; the ratio of the number of deaths to a given population (CMD 1997).
Mutagenic	Pertaining to an agent that causes genetic mutations (CMD 1997).
Myoglobin	The iron-containing protein found in muscle cells that stores oxygen for use in cell respiration (CMD 1997).
Mycotoxin	Substance produced by mould growing in food or animal feed and causing illness or death when ingested by humans or animals (CMD 1997).
Nausea	An unpleasant sensation usually preceding vomiting (CMD 1997).
Neurological	Pertaining to the branch of medicine that deals with the nervous system and its diseases (CMD 1997).
Neurotoxicity	Having the capability of harming nerve tissue (CMD 1997).
Neutrophil	A granular white blood cell (CMD 1997).
Nitrate	See <i>nitric acid</i> .
Nitric acid	A colourless or yellowish fuming liquid, formula $\text{HNO}_3$ . It is highly corrosive and the vapour is very hazardous. Nitric acid and nitrates (mainly ammonium nitrate) occur in the atmosphere in the form of aerosols: the acid is formed from oxides of nitrogen and then reacts with ammonia to form ammonium nitrate (WHO 1997c).
Nitric oxide	See <i>nitrogen oxides</i> .
Nitrogen	A gaseous element, atomic number 7, relative atomic mass 14.0067, symbol N. It is the principal constituent of air (78% by volume).
Nitrogen dioxide	See <i>nitrogen oxides</i> .
No-observed-adverse-effect Level	Greatest concentration or amount of a substance, found by observation or experiment, which causes no detectable adverse effect (WHO 1994c). Effects may be detected at this level, which

	are not judged to be adverse.
No-observed-effect level	Greatest concentration or amount of a substance, found by observation or experiment, which causes no detectable effect (WHO 1994c).
Nucleation	The process of forming a central point about which matter is gathered (CMD 1997).
Outpatient	One who receives treatment at a hospital, clinic, or dispensary but is not hospitalised (CMD 1997).
Oxidant (in atmospheric chemistry)	A very qualitative term which includes any and all trace gases which have a greater oxidation potential than oxygen (for example O <sub>3</sub> , peroxyacetyl nitrate, hydrogen peroxide, organic peroxides, NO <sub>3</sub> , etc.). It is recommended that alternative, more definitive terms be used which define the specific oxidant of interest whenever possible (IUPAC 1997).
Oxidant defense	Protective action against harm or injury from oxidants.
Oxygen	A gaseous element, atomic number 8, relative atomic mass 15.9994, symbol O. Oxygen is a colourless, odourless gas which supports combustion in air. Molecular oxygen (O <sub>2</sub> ) constitutes 20.95% by volume of dry air in the lower part of the atmosphere. O <sub>2</sub> is essential for the maintenance of almost all forms of life. Above an altitude of 20 km atomic oxygen appears in significant amounts and at 100 km it is in the predominant form. For the tri-atomic form of oxygen, see <i>ozone</i> .
Ozone	The tri-atomic allotrope of oxygen; a pale blue gas with a distinctive pungent odour, formula O <sub>3</sub> . It is a highly reactive oxidising agent and is very poisonous, and is considered a serious pollutant at concentrations much in excess of 125 µg/m <sup>3</sup> (WHO 1980). It is naturally occurring in the atmosphere. It occurs at large concentrations in the upper atmosphere, where it is formed by the action of solar ultraviolet radiation. In the troposphere, O <sub>3</sub> is mostly formed by photochemical reactions involving hydrocarbons and nitrogen oxides.
Paroxysm	A sudden, periodic attack or recurrence of symptoms of a disease; an exacerbation of the symptoms of a disease (CMD 1997).
Particle	Small discrete mass of solid or liquid matter (ISO 1994).
Particle aerodynamic diameter	Diameter of a sphere of density 1 g/cm <sup>3</sup> with the same terminal velocity due to gravitational force in calm air as the particle, under the prevailing conditions of temperature, pressure and relative humidity (ISO 1995).

Particle size distribution	The distribution of equivalent diameters of particles in a sample or the proportion of particles for which the equivalent diameter lies between defined limits (Willeke 1993).
Peak expiratory flow rate	See rate.
Perinatal	Concerning the period beginning after the 28 <sup>th</sup> week of pregnancy and ending 28 days after birth (CMD 1997).
Pharyngitis	Inflammation of the passageway for air from the nasal cavity to the larynx (CMD 1997).
Phytohemagglutinin	A protein substance derived from red kidney beans that agglutinates red blood cells, used to study the proliferation of lymphocytes, abbreviation PHA (CMD 1997).
Phlegm	Thick mucus, especially that from the respiratory passages (CMD 1997).
Photochemical smog	Result of reactions in the atmosphere between nitrogen oxides, organic compounds and oxidants under the influence of sunlight, leading to the formation of oxidising compounds or possibly causing poor visibility, eye irritation or damage to material and vegetation if sufficiently concentrated (ISO 1994).
Pneumonia	An inflammation of the alveoli, interstitial tissue, and bronchioles of the lungs due to infection by bacteria, viruses, or other pathogenic organisms, or to irritation by chemicals or other agents (CMD 1997).
Pneumonitis	Inflammation of the lung, usually due to hypersensitivity (allergic) reactions to organic dust, such as wheat or other grains, or chemicals (CMD 1997).
Polycyclic aromatic Hydrocarbon	See hydrocarbon.
Polynuclear aromatic hydrocarbon	See hydrocarbon.
Protoporphyrin	A derivative of hemoglobin containing four pyrole nuclei (CMD 1997).
Rate	The speed or frequency of occurrence of an event, usually expressed with respect to time or some other known standard (CMD 1997). <i>Death rate</i> or <i>mortality rate</i> is the number of deaths in a specified population, usually expressed per 100 000 population, over a given period, usually 1 year. <i>Morbidity rate</i> is

	the number of cases per year of certain diseases in relation to the population in which they occur. <i>Infant mortality rate</i> is the number of deaths per year of live-born infants less than 1 year of age divided by the number of live births in the same year. <i>Peak expiratory flow rate</i> is the maximum rate of exhalation during forced expiration, measured in litres per second or litres per minute.
Renal	Pertaining to the kidney (CMD 1997).
Respiration	The act of breathing (i.e. inhaling and exhaling) during which the lungs are provided with air through inhaling and carbon dioxide is removed through exhaling (CMD 1997).
Respiratory	Pertaining to respiration (CMD 1997).
Retropharyngeal	Behind the passageway for air from the nasal cavity to the larynx (CMD 1997).
Rhinitis	Inflammation of the mucous membrane of the nose. Symptoms include nasal congestion, thin watery discharge from the nose, sneezing and itching of the nose (CMD 1997).
Rhino-conjunctivitis	Rhinitis and inflammation of the mucous membrane that lines the eyelids and is reflected onto the eyeball.
Sampling	The collection of a representative portion for analysis and testing (WHO 1980). <i>Continuous sampling</i> is sampling, without interruptions, throughout an operation or for a predetermined time. <i>Grab sampling</i> or <i>spot sampling</i> is the taking of a sample in a very short time (ISO 1994).
Scavenging by precipitation	The process of removing pollutants from the atmosphere by precipitation (WMO 1992).
Sick building syndrome	Specific symptoms with unspecified aetiology which are experienced by a proportion of people working or living in a particular building and disappear after leaving it.
Spectrophotometry	An estimation of colouring matter in a solution (CMD 1997).
Standard	A level of an air pollutant, e.g. a concentration or a deposition value, which is adopted by a regulatory authority as enforceable. Unlike a guideline value, a number of elements in addition to the effect-based level and the averaging time must be specified in the formulation of a standard. These elements include the measurement strategy, data handling procedures, statistics used to derive, from measurements, the value to be compared with the standard. The numerical value of a standard may also include the

	permitted number of exceedings (WHO 1998a).
Symptom	Any perceptible change in the body or its functions that indicates disease or the kind or phases of disease (CMD 1997).
Teratogenicity	Causation of abnormal development of the embryo (CMD 1997).
Tolerable intake	An estimate of the intake of a substance over a lifetime that is considered to be without appreciable health risk (WHO 1994c).
Tonsillitis	Inflammation of a tonsil (CMD 1997).
Trachea	A cylindrical tube from the larynx to the primary bronchi (CMD 1997).
Tubular	Relating to or having the form of a tube (CMD 1997).
Ultra-fine particles	Particles with aerodynamic diameters below 0.1 micrometer.
Uncertainty factor	A factor that allows for a variety of uncertainties, for example, possible undetected effects on particularly sensitive members of the population, synergistic effects of multiple exposures, the adequacy of existing data, the extrapolation from animals to humans and the extrapolation from a small group of individuals to a large population. Uncertainty factors are based on scientific judgements in a complex decision process, involving the transformation of mainly non quantitative information into a single number (WHO 1987).
Unit risk	The additional lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 µg/m <sup>3</sup> of the agent in the air they breathe (WHO 1987).
Viable organisms	An organism that is able to live outside a host (CMD 1997).
Vital capacity	The volume of air that can be quickly and forcibly breathed out (CMD 1997).
Vitamin D3	One of several vitamins having anti-rachitic activity (CMD 1997).
Wet deposition	Removal of pollutants from the air through the processes of wash-out, rain-out, fog, and dew
Wheeze	A continuous musical sound caused by narrowing of the space of a respiratory passageway (CMD 1997).

For references see the bibliographical reference list in Appendix 1.



## Appendix 4

### *Environmental Health Criteria documents*

Environmental Health Criteria	Volume number	Year
Acetaldehyde	167	1995
Acetone	207	1998
Acetonitrile	154	1993
Acrolein	127	1991
Acrylamide	49	1985
Acrylic acid	191	1997
Acrylonitrile	28	1983
Aged population principles for evaluating the effects of chemicals in the	144	1992
Aldicarb	121	1991
Aldrin and dieldrin	91	1989
Alkylbenzene sulphonates, linear and related compounds	169	1996
Allethrins	87	1989
Aluminium	194	1997
Amitrole	158	1994
Ammonia	54	1986
Anticoagulant rodenticides	175	1995
Arsenic	18	1981
Asbestos and other natural mineral fibres	53	1986
Barium	107	1990
Benomyl	148	1993
Benzene	150	1993
Beryllium	106	1990
Biomarkers and risk assessment: concepts and principles	155	1993
Biotoxins, aquatic (marine and freshwater)	37	1984
Boron	204	1998
Brominated diphenylethers	162	1994
Butanols - four isomers	65	1987
Cadmium	134	1992
Cadmium - environmental aspects	135	1992
Camphechlor	45	1984
Carbamate pesticides: a general introduction	64	1986
Carbaryl	153	1994
Carbendazim	149	1993
Carbon disulphide	10	1979
Carbon monoxide	13	1979
Carbon Tetrachloride	208	1999
Carcinogens, summary report on the evaluation of short-term in vitro tests	47	1985
Carcinogens, summary report on the evaluation of short-term in vivo tests	109	1990
Chlordane	34	1984
Chlordecone	43	1984
Chlordimeform	199	1998
Chlorendic acid and anhydride	185	1996
Chlorinated paraffins	181	1996
Chlorine and hydrogen chloride	21	1982
Chlorobenzene other than hexachlorobenzene	128	1991
Chlorofluorocarbons, fully halogenated	113	1990

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Chlorofluorocarbons, partially halogenated (ethane derivatives)	139	1992
Chlorofluorocarbons, partially halogenated (methane derivatives)	126	1991
Chloroform	163	1994
Chlorothalonil	183	1996
Chlorphenols	93	1989
Chromium	61	1988
Chrysotile Asbestos	203	1998
Copper	200	1998
Cresols	168	1995
Cyhalothrin	99	1990
Cypermethrin	82	1989
Cypermethrin, alpha	142	1992
DDT and its derivatives	9	1979
DDT and its derivatives - environmental aspects	83	1989
Deltamethrin	97	1990
Diaminotoluenes	74	1987
Diazinon	198	1998
Dibromoethane, 1,2-	177	1996
Dibromopropyl (2,3-) phosphate	173	1995
Dichloroethane, 1,2- (1st edition)	62	1987
Dichloroethane, 1,2- (2nd edition)	176	1995
Dichloropropene, 1,3-, 1,2-dichloropropane and mixtures	146	1993
Dichlorophenoxyacetic acid, 2,4-	29	1984
Dichlorophenoxyacetic acid, 2,4-, - environemntal aspects	84	1989
Dichlorvos	79	1988
Diesel fuel and exhaust emissions	171	1996
Diethylhexyl phthalate	131	1992
Di lubenzuron	184	1996
Dimethoate	90	1989
Dimethyl sulfate	48	1985
Dimethylformamide	114	1991
Dimeton-S-methyl	197	1997
Di-n-butyl phthalate	189	1997
Diseases of suspected etiology and their prevention, principles of studies on	72	1987
Dithiocarbamate pesticides, ethylenethiourea, and propylenethiourea: a general introduction	78	1988
Electromagnetic fields	137	1992
Endosulfan	40	1984
Endrin	130	1992
Environmental epidemiology, guidelines on studies in	27	1983
Epichlorohydrin	33	1984
Ethylbenzene	186	1996
Ethylene oxide	55	1985
Extremely low frequency (ELF) fields	35	1984
Fenitrothion	133	1992
Fenvalerate	95	1990
Flame retardants: a general introduction	192	1997
Flame Retardants: Tris (chloropropyl) Phosphate and Tris 2-chloroethyl) Phosphate	209	1998
Fluorine and fluorides	36	1984
Food additives and contaminants in food, principles for the safety assessment of	70	1987
Formaldehyde	89	1989
Genetic effects in human populations, guidelines for the study of	46	1985

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Glyphosate	159	1994
Health Effects of Interactions between Tobacco Use and Exposure to other Agents	211	1999
Heptachlor	38	1984
Hexachlorobenzene	195	1997
Hexachlorobutadiene	156	1994
Hexachlorocyclohexanes, alpha- and beta-	123	1992
Hexachlorocyclopentadiene	120	1991
Hexan, n-	122	1991
Human exposure limits, guidance values	170	1994
Hydrazine	68	1987
Hydrogen sulfide	19	1981
Hydroquinone	157	1994
Immunotoxicity associated with exposure to chemicals, principles and methods for assessments	180	1996
Infancy and early childhood, principles for evaluating health risks from chemicals during	59	1986
Isobenzan	129	1991
Isophorone	174	1995
Kelevan	66	1986
Lasers and optical radiation	23	1982
Lead	3	1977
Lead, environmental aspects	85	1989
Lead, inorganic	165	1995
Lindane	124	1991
Magnetic fields	69	1987
Manganese	17	1981
Man-made mineral fibres	77	1988
Mercury	1	1976
Mercury - environmental aspects	86	1989
Mercury, inorganic	118	1991
Methanol	196	1997
Methomyl	178	1996
Methoxyethanol, 2-, 2-ethoxyethanol, and their acetates	115	1990
Methyl bromide	166	1995
Methyl ethyl ketone	143	1992
Methyl isobutyl ketone	117	1990
Methyl parathion	145	1992
Methyl tertiary-Butyl Ether	206	1998
Methylene chloride, 1st edition	32	1984
Methylene chloride, 2nd edition	164	1996
Methylmercury	101	1990
Mirex	44	1984
Morpholine	179	1996
Mutagenic and carcinogenic chemicals, guide to short-term tests for detecting	51	1985
Mycotoxins	11	1979
Mycotoxins, selected: ochratoxins, trichothecenes, ergot	105	1990
Nephrotoxicity associated with exposure to chemicals, principles and methods for the assessment of	119	1991
Neurotoxicity associated with exposure to chemicals, principles and methods for the assessment of	60	1986
Nickel	108	1991
Nitrates, nitrites, and N-nitroso compounds	5	1978

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Nitrogen oxides, 1st edition	4	1977
Nitrogen oxides, 2nd edition	188	1997
Nitropropane, 2-	138	1992
Noise	12	1980
Organophosphorus insecticides: a general introduction	63	1986
Paraquat and diquat	39	1984
Pentachlorophenol	71	1987
Permethrin	94	1990
Pesticide residues in food, principles for the assessment of	104	1990
Petroleum products, selected	20	1982
Phenol	161	1994
Phenothrin, d-	96	1990
Phosgene	193	1997
Phosphine and selected metal phosphides	73	1988
Photochemical oxidants	7	1978
Platinum	125	1991
Polybrominated biphenyls	152	1994
Polybrominated Dibenzo-p-dioxins and Dibenzofurans	205	1998
Polychlorinated biphenyls and terphenyls, 1st edition	2	1976
Polychlorinated biphenyls and terphenyls, 2nd edition	140	1992
Polychlorinated dibenzo-p-dioxins and dibenzofurans	88	1989
Principles of the Assessment of Risks to Human Health from Exposure to chemicals	210	1999
Progeny, principles for evaluating health risks associated with exposure to chemicals during pregnancy	30	1984
Propachlor	147	1993
Propanol, 1-	102	1990
Propanol, 2-	103	1990
Propylene oxide	56	1985
Pyrrolizidine alkaloids	80	1988
Quality management for chemical safety testing	141	1992
Quintozone	41	1984
Radiofrequency and microwaves	16	1981
Radionuclides, selected	25	1983
Resmethrins	92	1989
Selected Chloroalkyl Ethers	201	1998
Selected Non-heterocyclic Polycyclic Aromatic Hydrocarbons	202	1998
Selenium	58	1986
Styrene	26	1983
Sulfur oxides and suspended particulate matter	8	1979
Synthetic organic fibres, selected	151	1993
Tecnazene	42	1984
Tetrabromobisphenol A and derivatives	172	1995
Tetrachloroethylene	31	1984
Tetradifon	67	1986
Tetramethrin	98	1990
Thallium	182	1996
Thiocarbamate pesticides: a general introduction	76	1988
Tin and organotin compounds	15	1980
Titanium	24	1982
Toluene	52	1986
Toluene diisocyanates	75	1987
Toxicity of chemicals (Part 1), principles and methods for evaluating the	6	1978

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Toxicokinetic studies, principles of	57	1986
Tributyl phosphate	112	1991
Tributyltin compounds	116	1990
Trichlorfon	132	1992
Trichloroethane, 1,1,1-	136	1992
Trichloroethylene	50	1985
Tricresyl phosphate	110	1990
Triphenyl phosphate	111	1991
Ultrasound	22	1982
Ultraviolet radiation, 1st edition	14	1979
Ultraviolet radiation, 2nd edition	160	1994
Vanadium	81	1988
Vinylidene chloride	100	1990
White spirit	187	1996
Xylenes	190	1997

## Appendix 5

### *Participants list*

#### WHO GUIDELINES FOR AIR QUALITY

##### List of participants of Task Group Meeting WHO Headquarters, Geneva, 2-5 December 1997

Dr Amrit Aggarwal  
Deputy Director & Head  
Air Pollution Control Division  
National Environmental Engineering Research Institute  
Nehru Marg  
Nagpur - 440 020  
India  
Tel: (+91 712) 226 071 to 75  
Fax: (+91 712) 230 673 or + (91 712) 222 725  
E-mail: root%neeri@sirnetd.ernet.in

Mr Jonathan Bower  
Air Pollution Monitoring  
AEA Technology, E5 Culham  
GB - Abingdon, Oxfordshire OX14 3DB  
Tel: (+ 44 1235) 463 067  
Fax: (+ 44 1235) 463 011  
E-mail: jon.bower@aeat.co.uk

Dr Mostafa El-Desouky  
Technical Advisor  
Occupational & Environmental Health Department  
Ministry of Health  
P.O. Box 10098 Shuaiba  
65451 Kuwait  
Tel & Fax (+965) 261 51 36/326 19 66  
Fax: (+965) 32 62 045

Dr Hidekazu Fujimaki  
Section Chief  
National Institute for Environmental Studies  
16-2, Onogawa, Tsukuba  
Ibaraki 305  
Japon  
Tel: (+81 298) 50 2518  
Fax: (+81 298) 50 2518 or 50 25 74  
E-mail: fujimaki@nies.go.jp

Professor Morton Lippmann  
Department of Environmental Medicine  
New York University Medical Centre  
57 Old Forge Road  
Tuxedo, New York 10987  
USA  
Tel (+1 914) 351 2396  
Fax (+1 914) 351 5472  
E-mail: lippmann@charlotte.med.aryu.edu

Ms Angela Mathee  
Executive Officer  
Eastern Metropolitan Substructure (Johannesburg)  
Sandton Administration Building (Room 310)  
Corner of West and Rivonia Roads  
Sandton 2196  
Afrique du Sud  
Tel: (+27 11) 881 6911  
Fax (+27 11) 881 6071  
E-mail: comam@emss.org.za

Dr Robert L. Maynard  
Head, Air Pollution Section  
Department of Health  
Skipton House, Room 658C  
80 London Road, Elephant & Castle  
London SE1 6LW  
UK  
Tel (+ 44 171) 972 5118 or 972 2000  
Fax (+ 44 171) 972 5156  
E-mail: rmaynard@hefm.demon.co.uk

Professor Frank Murray  
Murdoch University  
Environmental Science Division  
Murdoch WA 6150  
Australie  
Tel (+61 89) 360 2501/6000  
Fax (+61 89) 310 4997  
E-mail: murray@essun1.murdoch.edu.au

Professor Mahmood Nasralla  
Chairman  
Air Quality Improvement Unit  
National Research Centre  
Dokki, Cairo  
Egypte  
Tel (+20 2) 353 7299  
Fax (+20 2) 337 0931

Dr Isabelle Romieu  
Medical Epidemiologist  
2595 Woodwardia Rd.  
Atlanta GA 30345  
USA  
Tel (+1 770) 488 7649  
Fax (+1 770) 488 7335  
E-mail: iar9@cdc.gov

Professor Bernd Seifert  
Director  
Department of Air Hygiene  
Institute for Water, Soil & Air Hygiene  
Federal Environmental Agency  
Corrensplatz 1  
14195 Berlin  
Allemagne  
Tel (+49 30) 8903 1320  
Fax (+49 30) 8903 1830  
E-mail: Bernd.Seifert@uba.de

Dr Bimala Shrestha  
c/o WHO Representative's Office  
or GPO Box 5627, Kathmandu, Nepal  
P.O. Box 108  
Kathmandu  
Nepal  
Tel (+ 977 1) 52 16 62 (private)  
Fax (+ 977 1) 52 77 56  
speets@whonep.mos.com.np

Professor Kirk Smith  
Associate Director for International Programmes  
Centre for Occupational and Environmental Health  
University of California  
Warren Hall, MC-7360  
Berkeley, CA 94720-7360  
USA  
Tel (+ 1 510) 643 0793  
Fax (+ 1 510) 642 5815  
E-mail: KRKSmith@UCLink4.Berkeley.edu

Professor Gerhard Winneke  
Auf'm Hennekamp 50  
40225 Düsseldorf  
Allemagne  
Tel (+49 211) 33 89 291  
Fax (+49 211) 33 89 331  
E-mail: Gerhard.winneke@uni-duesseldorf.de



Dr Ruqiu Ye  
Deputy Administrator  
National Environmental Protection Agency  
N°. 115 Xizhimennei Nanxiaojie  
Beijing 100035  
République populaire du Chine  
Tel (+ 86 10) 66 15 17 54 (direct); (home) 6491 5281  
Fax (+86 10) 66 15 17 62/68  
E-mail: yerq@hotmail.com OR yeruqiu@cenpok.net

## **Observers**

Prof. Dr Ursula Ackermann-Liebrich  
Head of Institute of Social & Preventive Medicine  
University of Basel  
Steinengraben 59  
4051 Basel  
Tel: (061) 267 6066  
Fax: (061) 267 61 90  
E-mail: ackermann@ubaclu.unibas.ch

Mr Gerhard Leutert  
Head  
Air Pollution Control Division  
Federal Office of Environment, Forests and Landscape  
3003 Berne  
Tel: (031) 322 93 33  
Fax (031) 324 01 37

Dr Ruth Etzel  
National Centre for Environmental Health  
Centres for Disease Control and Prevention  
4770 Buford Highway  
Atlanta, Georgia 30341-3724  
USA  
Tel: (+1 770) 488 7321  
Fax: (+1 770) 488 7829  
E-mail: RAE1@CDC.GOV

## **WHO Secretariat**

Dr Bingheng Chen  
International Programme on Chemical Safety  
World Health Organization  
Avenue Appia  
1211 Geneva 27  
Tel: (+41 22) 791 3571  
Fax: (+41 22) 791 4848  
E-mail: chenbh@who.ch

Dr Richard Helmer  
Chief, Urban Environmental Health  
World Health Organization  
Avenue Appia  
1211 Geneva 27  
Tel: (+41 22) 791 3761  
Fax: (+41 22) 791 4127  
E:mail: helmerr@who.ch

Dr Michal Krzyzanowski  
Environmental Epidemiologist  
WHO European Centre for Environment & Health (ECEH)  
P.O. Box 10  
NL - 3730 AA De Bilt  
Tel: (+31 30) 229 5323  
Fax: (+31 30) 229 4120  
E:mail: mkr@who.nl

Dr Rolaf van Leeuwen  
Manager, Chemical Safety  
WHO European Centre for Environment & Health (ECEH)  
P.O. Box 10  
NL - 3730 AA De Bilt  
Tel: (+31 30) 229 5307  
Fax: (+31 30) 229 4252  
E:mail: rle@who.nl

Dr Roberto Romano  
Regional Adviser  
Environmental Quality Programmes  
Division of Health & Environment  
PAHO/AMRO  
525, 23rd Street, N.W.  
Washington, DC 20037-2895, USA  
Tel (+1 202) 974 3865  
Fax (+1 202) 974 3988  
E:mail: romanoro@paho.org

Dr Yasmin von Schirnding  
Scientist  
Office of Global & Integrated Environmental Health (EHG)  
World Health Organization  
Avenue Appia  
1211 Geneva 27  
Tel: (+ 41 22) 791 35 33  
Fax: (+41 22) 791 41 23  
E:mail: vonschirnding@who.ch

Dr Dieter Schwela  
Air Pollution Scientist  
Urban Environmental Health/EOS  
World Health Organization  
Avenue Appia  
1211 Geneva 27  
Tel: (+41 22) 791 4261  
Fax: (+ 41 22) 791 4127  
E:mail: schwelad@who.ch

Dr Maged Younes  
Chief, Assessment of Risk and Methodologies (ARM)  
World Health Organization  
Avenue Appia  
1211 Geneva 27  
Tel: (+41 22) 791 3574  
Fax: (+41 22) 791 4848  
E:mail: younesm@who.ch