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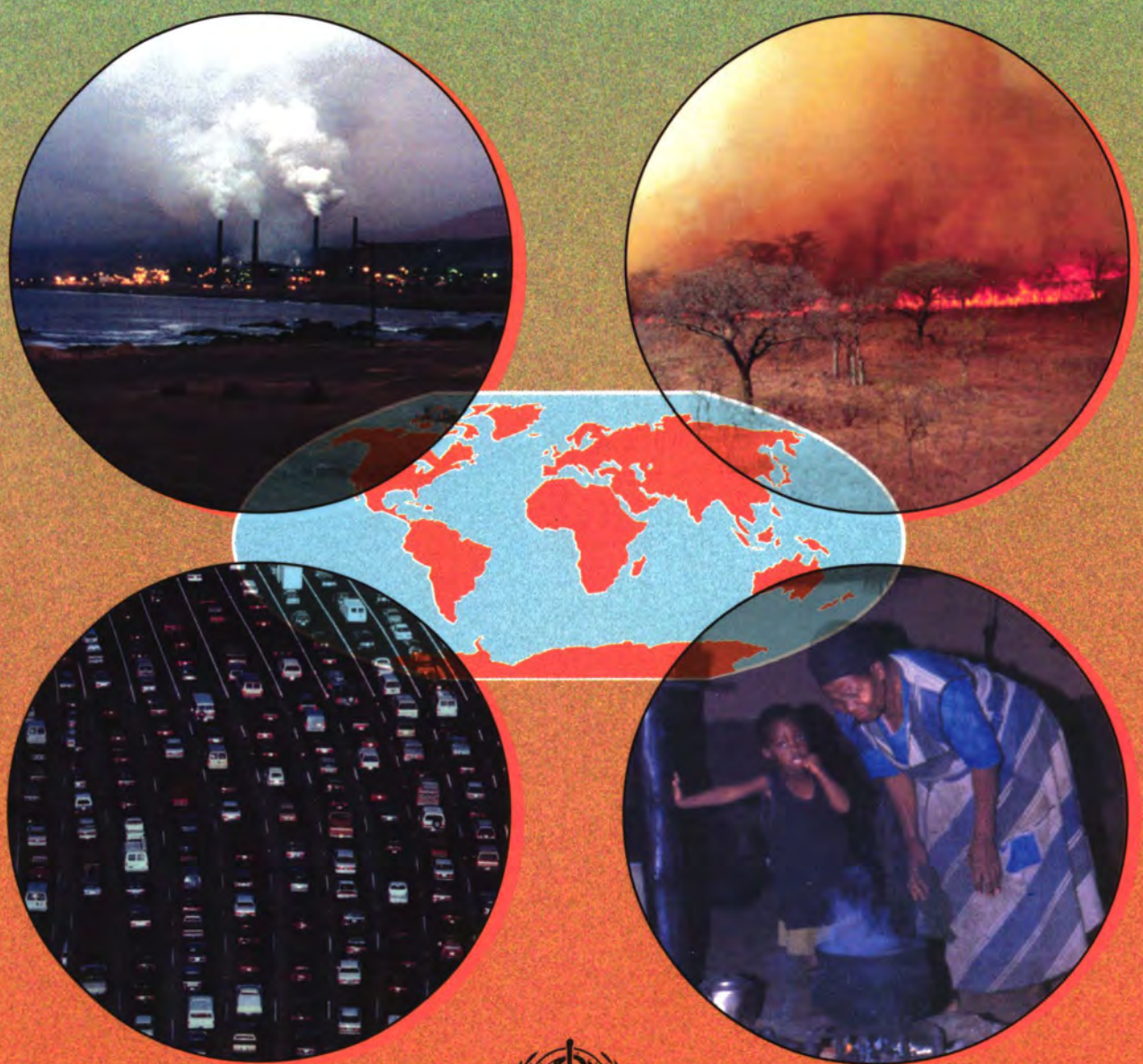
Guidelines for Air Quality



World Health Organization
Sustainable Development and Healthy Environments
Protection of the Human Environment
Occupational and Environmental Health

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:
The electronic form of this document is available on the CD ROM of the Healthy Cities Air Management Information System AMIS and, in part, from the Web site of the World Health Organization (http://www.who.int/peh/).

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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 21st 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).

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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 μm diameter may be determined (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 μm diameter ($PM_{2.5}$) 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_{10}), 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_2, NO_2, CO, O_3, 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_{10}) and < 2.5 μm diameter ($PM_{2.5}$). 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_{10}, (SPM with median aerodynamic diameter less than 10 μm), $PM_{2.5}$ (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_2) and sulphur trioxide (SO_3)), carbon monoxide (CO), nitrogen compounds [e.g. nitric oxide (NO), nitrogen dioxide (NO_2), ammonia (NH_3)], 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_2 can be oxidised to SO_3 which, dissolved in water, gives rise to the formation of sulphuric acid mist (catalysed by manganese and iron oxides). Photochemical reactions between NO_x and reactive hydrocarbons can produce ozone (O_3), 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_2S), carbon disulphide (CS_2) and mercaptans (R-SH, R_1 S R_2), 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_2, nitrogen oxides (NO_x), SPM, CO, O_3, heavy metals, PAH, and VOC. In developing countries the "classical" compounds SO_2, NO_x, SPM, CO, O_3 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_2 and SPM concentrations are decreasing in developed countries while NO_x and O_3 concentrations are either constant or increasing (UNEP/WHO 1992). In many countries in transition and in developing countries, SO_2 and SPM concentrations are increasing as a consequence of increasing combustion, as are NO_x and O_3 due to increasing traffic exhaust and emissions of VOC by industrial sources as precursors of O_3.

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_2) 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_3, SO_2, NO_2, 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 μm) 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 $\mu g/m^3$. 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.

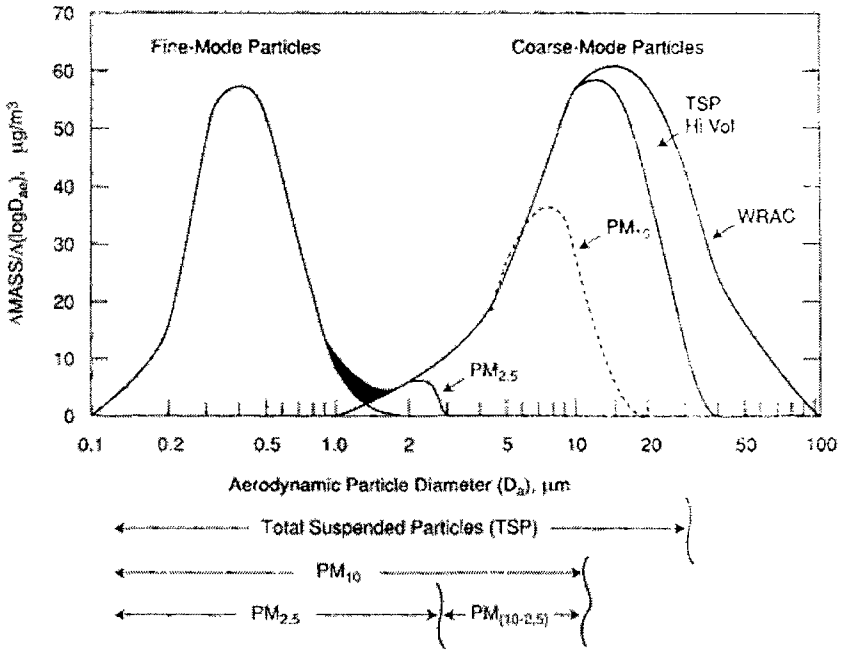
The alternative system, the volume mixing ratio, is applicable only to gases. In this system the concentration of gas is expressed as parts per billion, for example, and assuming ideal gas behaviour, does not depend upon the conditions of sampling because these will affect the air containing the pollutant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

mg/m^3 = ppm x (molecular weight/molar volume)

molar volume = 22.4 x T x 1013/273 x 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_{10} sampler, and the $PM_{2.5}$ 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 µm 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 µm diameter may be determined (PM_{10}) 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 µm in the above example). A PM_{10} sampling head accepts 50% of particles of aerodynamic diameter exactly 10 µm, 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 µm diameter ($PM_{2.5}$) 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_3 formation, as well as sulphate and nitrate particles resulting from the oxidation of SO_2 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

	Fine Mode	Coarse Mode
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, $SO_4^{=}$; nitrate NO_3^{-}; ammonium, NH_4^{+}; hydrogen ion, 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); $CaCO_3$, 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 NO_x, 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 μm, 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 SO_2 to sulphuric acid droplets (H_2SO_4).
Reactions of H_2SO_4 with NH_3 to form ammonium bisulphate (NH_4HSO_4) and ammonium sulphate $(NH_4)_2SO_4$.
The conversion of NO_2 to nitric acid vapour (HNO_3), which reacts further with NH_3 to form particulate ammonium nitrate (NH_4NO_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 μm 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 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 μm) 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" ($PM_{10-2.5}$) 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 μm 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 μm 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 μm 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°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_3 concentrations are generally much lower indoors in the absence of indoor sources, and O_3 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_3 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_2 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_2 exceeded 250 and 500 $\mu g/m^3$, 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_3, SO_2, airborne acidity, NO_2, 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_3 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_3 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_3: 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 ($PM_{2.5}$ 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_2 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_2, NO_2, O_3, 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_2 in residential areas have not exceeded 50 $\mu g/m^3$. Notable exceptions are several cities in China, with the SO_2 concentration of 330 $\mu g/m^3$ in Chongqing and 100 $\mu g/m^3$ 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_2 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_2 was in the range 273 - 350 $\mu g/m^3$ in residential areas of Kathmandu during September - December 1993. In monitoring sites close to main roads, the reported range is 310-875 $\mu g/m^3$, 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_2 concentration was seen over the 1990s. The most dramatic reduction of air pollution with SO_2 was reported from Mexico City, where the concentration in various residential areas dropped from 100-140 $\mu g/m^3$ in 1990-1991 to 32-37 $\mu g/m^3$ 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 $\mu g/m^3$, with the levels exceeding 300 $\mu g/m^3$ 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 $\mu g/m^3$ in 1990-1992 to more than 300 $\mu g/m^3$ in more recent years).

In a limited number of cities reporting data to AMIS levels of PM_{10} are also measured. The most commonly registered annual average PM_{10} levels ranged from 50 - 100 $\mu g/m^3$ in the years 1995-1996. The highest concentrations, exceeding 250 $\mu g/m^3$, were observed in Calcutta and New Delhi. In most towns with high PM_{10} 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_{10} level were seen in the Central and Southern America cities. In Mexico City, the relative decrease in PM_{10} 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_{10}. For most sites and years with data on both indicators, the PM_{10} 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_{10} (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_2 remain moderate or low, not exceeding 40 $\mu g/m^3$. However, in Mexico City and in Cape Town, the annual average of 70 $\mu g/m^3$ has been exceeded regularly in the 1990s. A paper based on data from centrally located monitors in Sao Paulo indicates annual mean of 240 $\mu g/m^3$ 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_2 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_3 in New Delhi (Singh et al 1997). The build-up of O_3 over the day is faster than scavenging of O_3 by the NO_2. In Mexico City, the mixture of high NO_2 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_3 concentration exceeded a concentration of 120 $\mu g/m^3$ in over 300 days a year in 1994-96, and the 95th percentile of maximum daily 1-hour average O_3 concentration was around 500 $\mu g/m^3$. Some decrease was seen, however, in the annual mean O_3 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_x, 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 $\mu g/m^3$ in indoor living areas, whereas smoky houses in Nepal and Papua New Guinea have peak levels of 10 000 $\mu g/m^3$ 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, 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_3 and NO_2. 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_2. 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_3 and SO_2 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_2 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 $\mu g/m^3$ in most cities in developed countries and daily means rarely exceed 125 $\mu g/m^3$.

However the situation is more complex in developing countries. In cities, the annual mean concentrations of SO_2 in ambient air may range from very low levels up to 300 $\mu g/m^3$ (WHO 1998b). Peak concentrations measured as ten-minute averages may exceed 2000 $\mu g/m^3$ 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_2 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_2 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_2 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_2 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_2 in air are variable. Natural background concentrations in ambient air can be less that 1 $\mu g/m^3$ to more than 9 $\mu g/m^3$. In ambient air in cities annual mean concentrations can range from 20-90 $\mu g/m^3$ with hourly maximum concentrations from 75-1000 $\mu g/m^3$ (WHO, 1994a). Concentrations of NO_2 in indoor air can reach average concentrations of 200 $\mu g/m^3$ over several days, with hourly maximum concentrations of 2000 $\mu g/m^3$ where there are unvented gas heating appliances and poor ventilation (WHO, 1994a).

NO_2 is a relatively water-insoluble gas and appreciable amounts of inhaled NO_2 can penetrate to, and elicit biological responses in, small lung airways. As with SO_2, 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^3 (WHO 1994a). In urban environments, mean concentrations over eight hours are usually less than 20 mg/m^3, and one-hour peak levels are usually less than 60 mg/m^3. 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 carparks, 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^3 for several hours. In houses with unflued combustion heaters, peak CO concentrations can reach up to 60 mg/m^3 (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_3 in remote and relatively unpolluted parts of the world are often in the range of 40 to 70 μg/m^3 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^3. High O_3 concentrations can persist for 8 to 12 hours per day for several days, when atmospheric conditions favour O_3 formation and poor dispersion conditions exist (Section 2.2.2). O_3 is normally at higher concentrations in ambient air outdoors than in indoor air.

O_3 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_2 and SO_2, 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_{10} are in the range 20 to 50 μg/m^3 for ambient air (WHO 19994a). However, annual average concentrations in some cities in Eastern Europe and in some developing countries can be above 100 μg/m^3. Concentrations of $PM_{2.5}$ are usually about 45 to 65% of the concentrations of PM_{10}.

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^3 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 $\mu g/m^3$ have been reported in urban areas near smelters, whereas lead levels below 0.1 $\mu g/m^3$ 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 $\mu g/m^3$ with extreme annual mean values between 1.5-2 $\mu g/m^3$.

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 ofTables 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 SO2, 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 guidelines 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 $\mu g/m^3$ 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 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_{10}, 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 O3, nitrogen-containing compounds and SO2 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_3 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_3, 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_{10} and/or $PM_{2.5}$) 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 "Stąkeholder 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_2, NO_2, CO, O_3, SPM and lead - are briefly described with respect to health risk evaluations and recommended guideline values. Particular emphasis is given to PM_{10} and $PM_{2.5}$. 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_2 comes from controlled chamber experiments on volunteers exposed to SO_2 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_1), 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_2 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_1 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_2, 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_2 exceeds 250 $\mu g/m^3$ 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 $\mu g/m^3$; daily levels usually not exceeding 125 $\mu g/m^3$) 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_2 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_2 and SPM, using data from the coal-burning era in Europe. The lowest-observed-adverse-effect level of SO_2 was judged to be at an annual average of 100 $\mu g/m^3$, when present with SPM. More recent studies related to industrial sources of SO_2, 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_2.

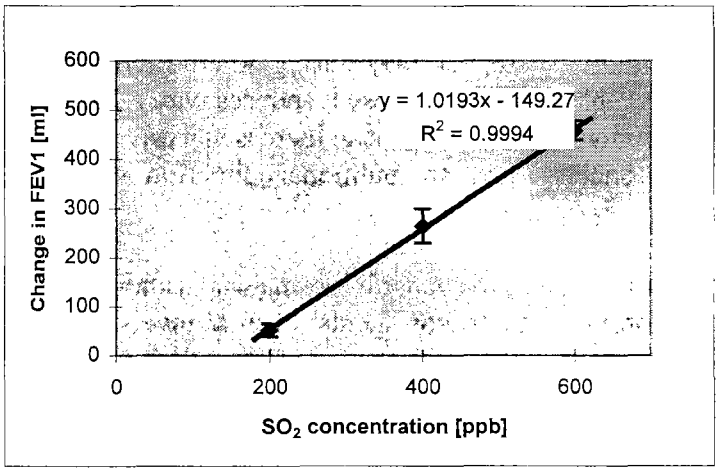


Figure 3.1 Mean change of FEV_1 in asthmatics with changing SO_2 concentrations

Guidelines

Based upon controlled studies with asthmatics exposed to SO_2 for short periods, it is recommended that a value of 500 $\mu g/m^3$ (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_2 are necessarily based on epidemiological studies, in which people are in general exposed to a mixture of pollutants; and guideline values for SO_2 have previously been linked with corresponding values for SPM. This approach led to a previous guideline 24-hour average value of 125 $\mu g/m^3$ (0.04 ppm) for SO_2, 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_2 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_2 and thus 125 $\mu g/m^3$ for an averaging time of 24 hours and 50 $\mu g/m^3$ 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_2 concentrations of less than 1880 $\mu g/m^3$ (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 $\mu g/m^3$ (2.5 ppm), experience pronounced decreases in pulmonary function; generally, normal subjects are not affected by concentrations less than 1880 $\mu g/m^3$ (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 $\mu g/m^3$ (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 $\mu g/m^3$ (0.3ppm) NO_2 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_2 can increase airway reactivity to cold air in asthmatic subjects. At lower concentrations, the pulmonary function of asthmatics was not changed significantly.

NO_2 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_2 levels as low as 376-565 $\mu g/m^3$ (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_2 studies with allergen challenges showed no effects at the lowest concentration tested (188 $\mu g/m^3$; 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_2 concentrations of less than 1880 $\mu g/m^3$ (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 $\mu g/m^3$), to emphysema-like effects. Biochemical changes often reflect cellular alterations, with the lowest effective NO_2 concentrations in several studies ranging from 380-750 $\mu g/m^3$.

NO_2 levels of about 940 $\mu g/m^3$ (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_2 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_2 in the same range as levels causing effects in some animal and human clinical

studies. Epidemiological studies evaluating the effects of NO_2 exposures in such homes have been conducted. In general, epidemiological studies of adults and infants (less than 2years 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 $\mu g/m^3$ NO_2 (2-week average), where the weekly average concentrations are in the range of 15-128 $\mu g/m^3$ 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_2 exposures exhibit increased respiratory symptoms that are of longer duration, and show a decrease in lung function. However, outdoor NO_2 epidemiological studies, as with indoor studies, provide little evidence that long-term ambient NO_2 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_2 concentrations in the range of 50-75 $\mu g/m^3$ 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_2 exposure. For acute exposures, only very high concentrations (>1,000 ppb; 1,990 $\mu g/m^3$) affect healthy people. Based on small changes in lung function, often less than a 5% drop in FEV_1 with NO_2 exposure, and changes in airway responsiveness in studies on asthmatics and patients with chronic obstructive pulmonary disease, a range of 365-565 $\mu g/m^3$ (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 $\mu g/m^3$, and because of a meta-analysis suggesting changes in airway responsiveness below 365 $\mu g/m^3$. However, the significance of the response at 188 $\mu g/m^3$ 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 $\mu g/m^3$ is proposed. At double this recommended guideline (400 $\mu g/m^3$), there is evidence to suggest possible small effects in pulmonary function of asthmatics. Should the asthmatic be exposed either simultaneously or sequentially to NO_2 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_2 exposures. Based on the studies reviewed, it is not currently possible to select a well-supported value; but a previous review on NO_2 recommended an annual value of 40 $\mu g/m^3$ (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 arhythmias 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^3 (90 ppm) for 15 minutes, 60 mg/m^3 (50 ppm) for 30 minutes, 30 mg/m^3 (25 ppm) for 1 hour, and 10 mg/m^3 (10 ppm) for 8 hours.

Ozone and other photochemical oxidants

O_3 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 $\mu g/m^3$ (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_3 concentration of 240 $\mu g/m^3$ (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 $\mu g/m^3$ 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_3 concentrations in the range 120-240 $\mu g/m^3$ and higher. Mobile laboratory studies have observed changes in pulmonary function in children or asthmatics exposed to O_3 concentrations of 280-340 $\mu g/m^3$ (0.14-0.17 ppm) for several hours. Respiratory symptoms, especially coughing, have been associated with O_3 concentrations as low as 300 $\mu g/m^3$ (0.15 ppm). O_3 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_3 (and co-pollutants) and with controlled exposures to O_3 alone. This demonstrates that the functional and symptomatic responses can be attributed primarily to O_3.

A number of studies evaluating animals (rats and monkeys) exposed to O_3 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 $\mu g/m^3$ (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_3 in the range of 240-500 $\mu g/m^3$ (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_3 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_3 levels of 200 $\mu g/m^3$ 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_3 exposure to warrant designation as a group needing protection from exposure to ambient O_3. In the case of respiratory function responses, a judgement could be made that O_3-related reductions of FEV_1 at, for example, less than 10% were of no clinical concern. The balance of evidence indicates that reductions of FEV_1 of more than 10% occurred at O_3 levels of 160 $\mu g/m^3$ and higher. It is generally accepted that the exposure duration to O_3 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 $\mu g/m^3$ 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_3 concentrations that are associated with levels of responses among population subgroups. Although chronic exposure to O_3 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_3 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_3 concentration and hospital admissions (Figure 3.5), the reduction in health risk associated with decreasing one-hour or eight-hour O_3 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_3 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.

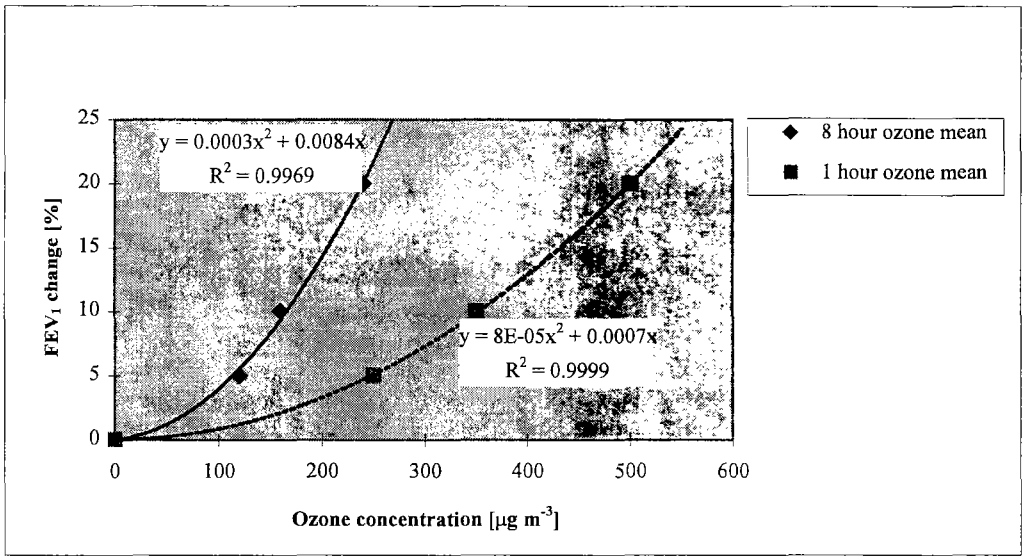


Figure 3.2. Change in FEV_1 as a function of O_3 concentration in the most sensitive 10% of active young adults and children.

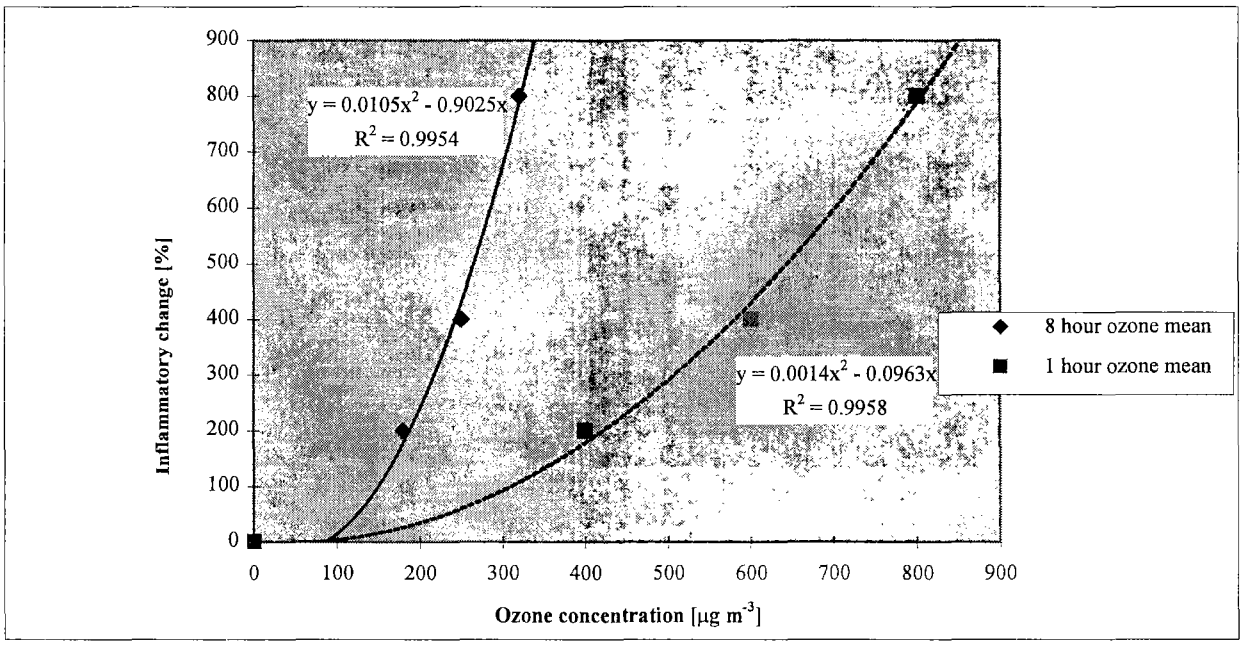


Figure 3.3. Inflammatory change (neutrophil influx in lungs of healthy young adults exercising outdoors at more than 40l/min expiratory volume in the lung) as a function of O_3 concentration.

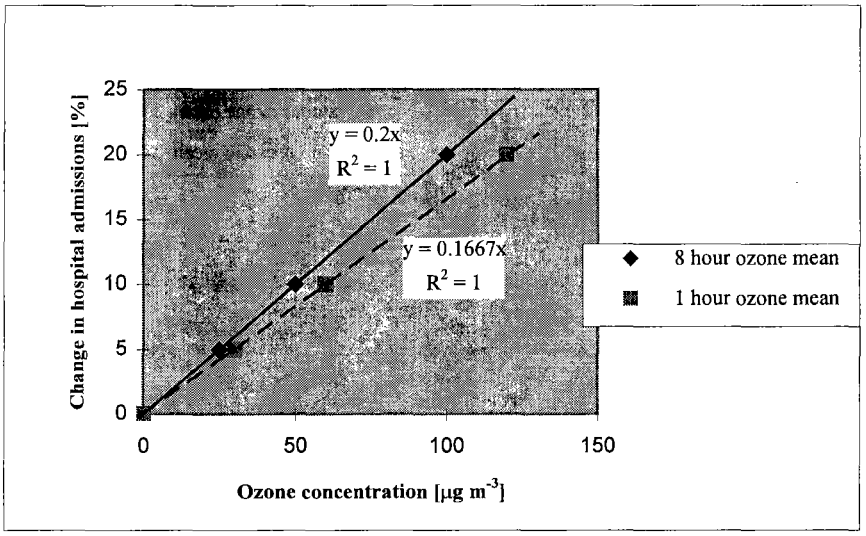


Figure 3.4. Increase in hospital admissions for respiratory conditions as a function of O_3 concentration.

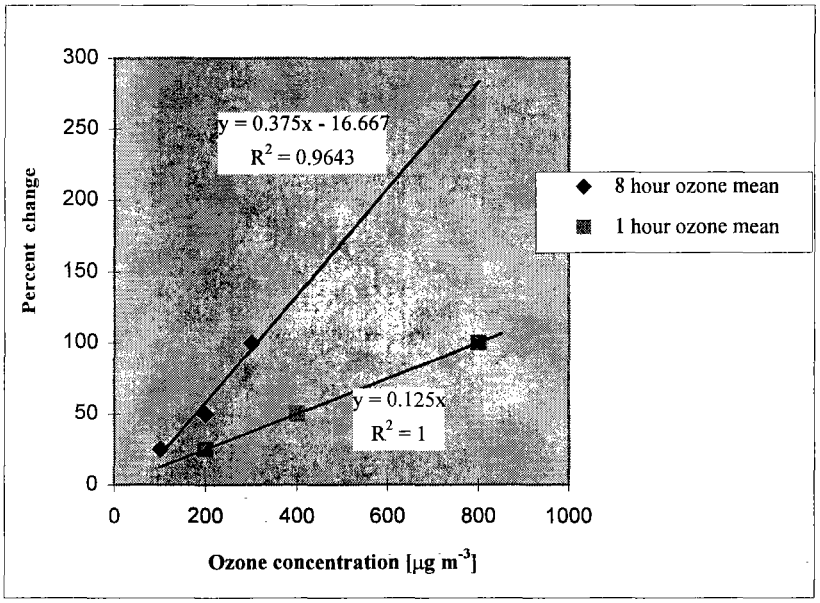


Figure 3.5. Change in symptom exacerbation among adults and asthmatics as a function of O_3 concentration.

Epidemiological data show relationships between changes in various health outcomes and changes in the peak daily ambient O_3 concentration. Two examples of such relationships are shown in Figures 3.4 and 3.5. Short-term increases in levels of ambient 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 O_3 concentration. The dose-response relationships presented in Figures 3.4 and 3.5 assume a linear relationship between 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 O_3 levels. In the event that such relationships are curvilinear (i.e., concave upwards), the benefits of lowering the O_3 concentration are likely to be greater when the average ambient level is higher. Conversely, if the ambient O_3 concentration is already low, the benefits of lowering the concentration may be less than would be suggested

be greater when the average ambient level is higher. Conversely, if the ambient O_3 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 $\mu g/m^3$ for O_3. 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_{10} or $PM_{2.5}$ 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_{10} 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_{10} and $PM_{2.5}$. 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 $\mu g/m^3$), short-term exposure is associated with health effects. At low levels of PM_{10} (0 - 100 $\mu g/m^3$), 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 $\mu g/m^3$ of PM_{10}), 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_{10} that are reasonably

Although many studies have obtained acute effect estimates for PM_{10} 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}.

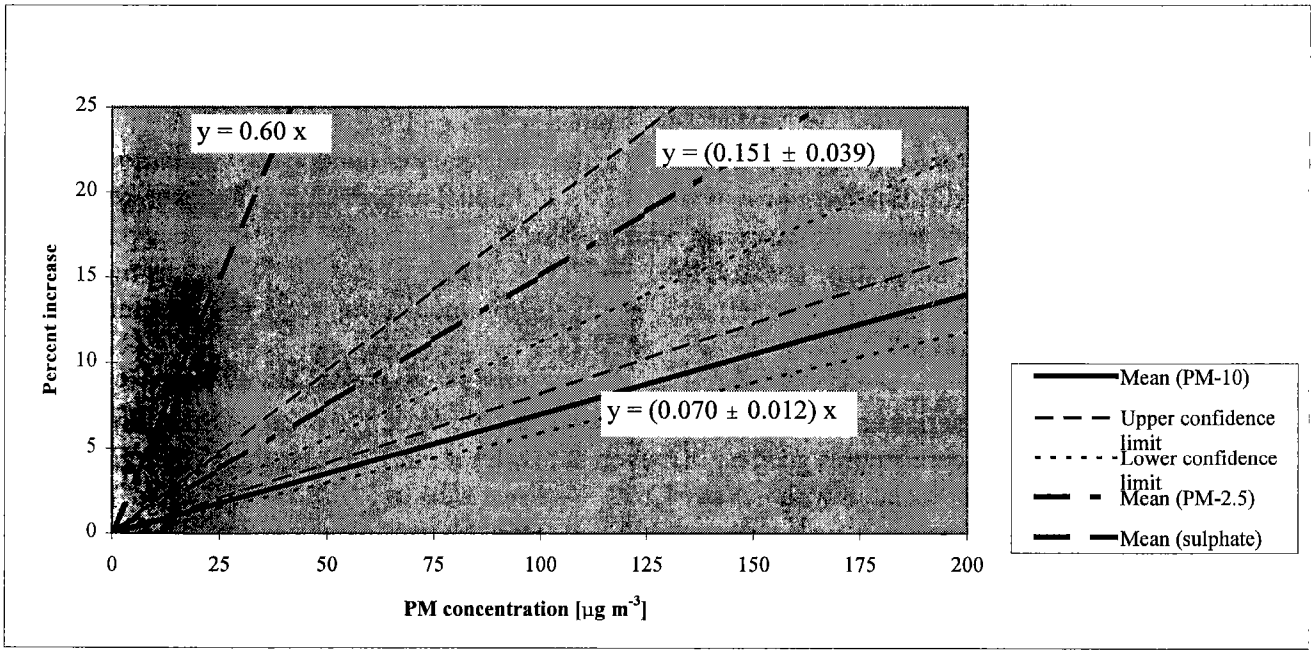


Figure 3.6. Increase in daily mortality as a function of PM concentration.

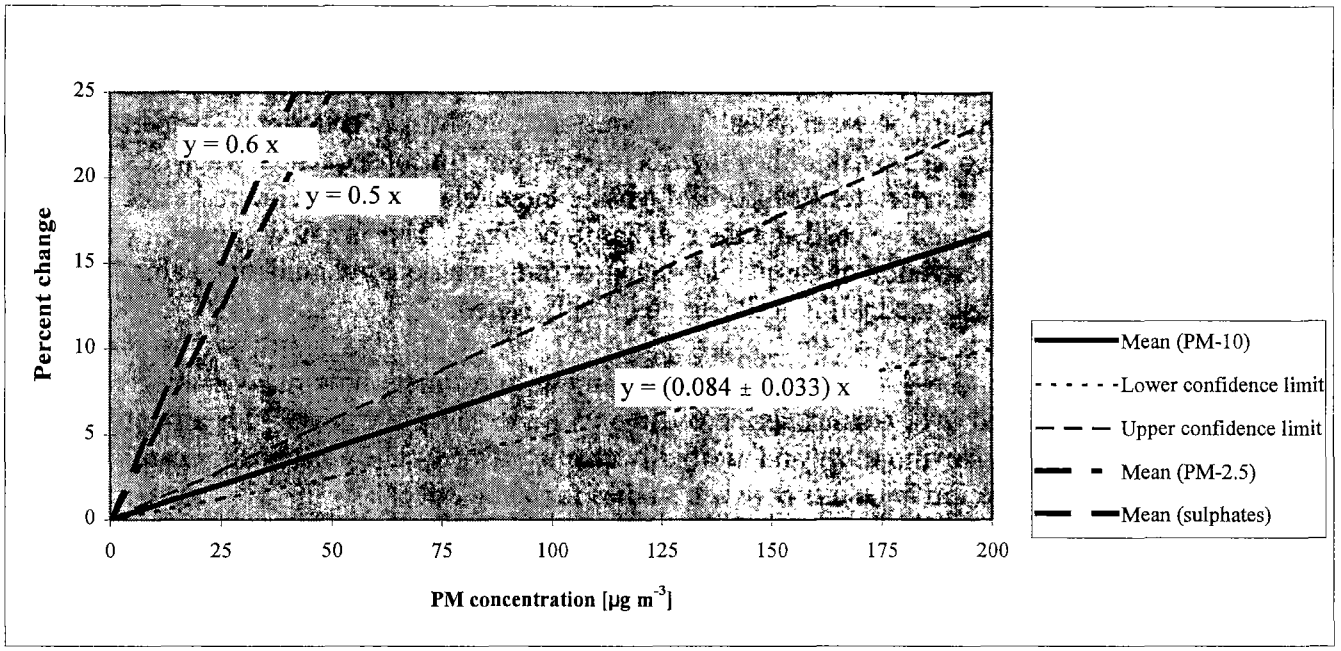


Figure 3.7. Percent change in hospital admissions assigned to PM_{10}, $PM_{2.5}$ and sulphates.

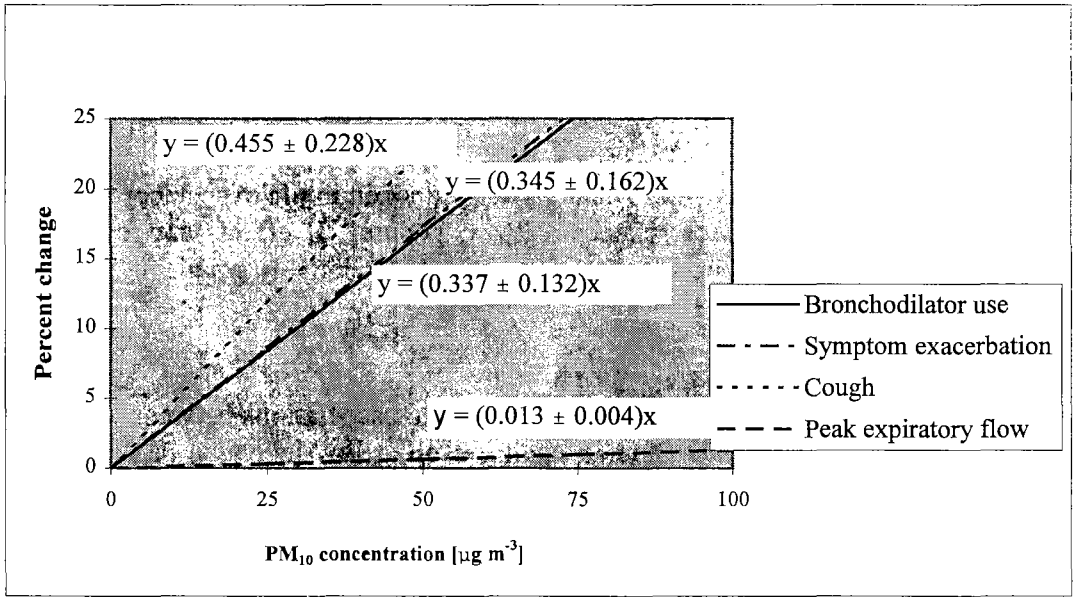


Figure 3.8. Change in health endpoints as a function of PM_{10} concentration.

The following issues should be considered when using these graphs:

(1) The graphs should not be used for PM_{10} concentrations below 20 μg/m^3, or above 200 μg/m^3; or for $PM_{2.5}$ concentrations below 10 μg/m^3, or above 100 μg/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_{10} or $PM_{2.5}$ and the

(3) There is a fundamental difference between the guidelines for PM_{10} or $PM_{2.5}$ and the guideline values for respirable particulate matter derived in the WHO *Air Quality Guidelines for Europe* (WHO 1987). The guidelines for PM_{10} and $PM_{2.5}$ 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_{10} or $PM_{2.5}$ 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_{10}, or $PM_{2.5}$. 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_{10}. There is now also an increasing body of information on $PM_{2.5}$, and the most recent studies show that, in general, $PM_{2.5}$ is a better predictor of health effects than PM_{10}. Evidence is also emerging that constituents of $PM_{2.5}$, such as sulphates and strongly acidic particles, are sometimes better predictors of health effects than $PM_{2.5}$.

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 coproporyphyrin 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 200-300μg/l, and in female adults and children above 150-200 μg/l. A reduction in vitamin D3 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^3 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^3 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^3 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^{-2}/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 [μg/m³]	Health endpoint	Observed effect level [μg/m³]	Uncertainty factor	Guideline Value [μg/m³]	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 μg 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[VI] 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_2 and H_2S 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, WHO1998d); 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^3).

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 [µg/m³]	Health endpoint	Observed effect level [mg/m³]	Uncertainty factor	Guideline Value (GV) or Tolerable Concentr-ation (TC) [µg/m³]	Averaging time	Source	
Acetaldehyde	5	Irritancy in humans	45 (NOEL)	20	2 000 (TC)	24 hours	WHO 1995d	EHC 167
		Carcinogenicity related irritation in rats	275 (NOEL)	1000	50 (TC)	1 year		
Acetone	0.5-125	Odour annoyance	240 (OT)	n.a.	n.p.	-	WHO 1998c	EHC 207
Acrolein	15	Eye irritation in humans	0.13	n.p.	50 (GV)	30 min	WHO 1992b	EHC 127
		Odour annoyance	0.07	n.a.	-	30 min	WHO 1992b	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	Haematoxicity in rats	242 (NOAEL)	10	13100 (TC)	1 week	WHO 1998d	CICAD 10
Cadmium	$(0.1\text{-}20).10^{-3}$	Renal effects in the population	n.a.	n.a.	5×10^{-3} (GV)	1 year	WHO 1999a	
Carbon disulphide	10-1500	Functional CNS changes in workers	10 (LOAEL)	100	100 (GV)	24 hours	WHO 1987	
		Odour annoyance	0.2 (OT)	n.a.	20 (GV)	30 min	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	0.139 (NOAEL)*	25	5.6 (GV)	1 year	WHO 1996b	EHC 171
		Chronic alveolar inflammation in rats	0.23 (NOAEL)*	100	2.3 (GV)	1 year		

* For diesel exhaust two approaches were applied, which based on a NOAEL of 0.41 mg/m3 in rats. The corresponding levels were converted to a continuous exposure scenario. n.a. not applicable; n.p. not provided; OT Odour treshold.

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

Compound	Average Concentration [µg/m³]	Health endpoint	Observed effect level [mg/m³]	Uncertainty factor	Guideline Value (GV) or Tolerance Concentration (TC) [µg/m³]	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 livestocks	n.a.	n.a.	1 (GV)	1 year	WHO 1999a	
Formaldehyde	$(1\text{-}20) \cdot 10^{-3}$	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) $(0.2\text{-}2.0) \times 10^{-3}$ (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	$(2\text{-}10) \cdot 10^{-3}$	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	2.4×10^{-4}	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; OT Odour treshold.

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

Compound	Average ambient air concentration [µg/m³]	Health endpoint	Observed effect level [mg/m³]	Uncertainty factor	Guideline Value (GV) or Tolerance concentration (TC) [µg/m³]	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	107 (LOAEL)	40	260 (GV)	1 week	WHO 1999a	
		Odour annoyance	0.07 (OT)	n.a.	7 GV)	30 minutes	WHO 1987	
Tetrachloroethylene	`1 - 5	Kidney effects in workers	102 (LOAEL)	400	250 (GV)	24 hours	WHO 1999a	
		Odour annoyance	8	n.a.	8000 (GV)	30 minutes	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	332 (LOAEL)	1260	260 (GV)	1 week	WHO 1999a	
		Odour annoyance	1 (OT)	n.a.	1000 (GV)	30 minutes	WHO 1987	
1,3,5 Trichlorobenzene	0.5-0.8	Metaplasia and hyzperplasia of respiratory epithelium in rats	100 (NOEL)	500	200 (TC)	1 year	WHO 1991a	EHC 128
1,2,4 Trichlorobenzene	0.02-0.05	Increae 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	304 (NOAEL)	60	4800 (GV)	24 hours	WHO 1997g	EHC 190
		Neurotoxicity in rats	870 (LOAEL)	1000	870 (GV)	1 year	WHO 1997g	EHC 190
		Odour annoyance	4.35 (OT)	n.a.	-	30 minutes	WHO 1997g	EHC 190

n.a. not applicable; n.p. not provided; OT Odour treshold.

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

Compound	Average ambient air concentration [µg/m³]	Health endpoint	Observed effect level [mg/kg bw d]	Uncertainty factor	Tolerable Daily intake (TDI orADI) [µg/kg bw d]	Averaging Time (over lifetime)	Source	
Chloroform	0.3-10	Hepatoxicity 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) . 10^{-3}	Developmental/Reproductive toxicity	66 (LOAEL)	1000	66 (ADI)	24 hours	WHO 1997e	EHC 189
			Estimated human daily intake [pg/kg bw d]		[TEQ/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	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

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

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

Compound	Average ambient air concentration [$\mu g/m^3$]	Health endpoint	Unit risk $[\mu g/m^3]^{-1}$	IARC classification	Source	
ETS	1-10	Lung cancer in exposed humans	10^{-3}		WHO 1999a	
Nickel	1-180	Lung cancer in exposed humans	3.8×10^{-4}	1	WHO 1999a	
PAH (BaP)	$(1\text{-}10) \cdot 10^{-3}$	Lung cancer in exposed humans	8.7×10^{-2}	1	WHO 1999a	
1,1,2,2-Tetrachloroethane	0.1 - 0.7	Hepatocellular carcinomas in mice	$(0.6\text{-}3.0) \times 10^{-6}$	3	WHO 1998j	CICAD 3
Trichloroethylene	1 -10	Cell tumours in testes of rats	4.3×10^{-7}	2A	WHO 1999a	
Vinychloride	0.1 - 10	Hemangiosarkoma in exposed workers Liver cancer in exposed workers	1×10^{-6}	1	WHO 1987	

For the compounds noted inTable 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:

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

In this relationship the potencies of the mixture and the potency of "coke oven top" are taken fromTable A.I.17 of EHC 202 (WHO 1998i); $UR_{mixture}$ denotes the unit risk of the mixture and UR_{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
Petrol exhaust	0.736
Roofing tar	0.145
Smokeless coal	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) x 10^{-7} $[\mu g/m^3]^{-1}$. 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 AI.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)

$UR_{compound}$ = (potency of compound)/(potency of BaP) x UR_{BaP}

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

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

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^3) in ambient air and 570 F/m^3 in indoor air. Maximum values were 2400 F/m^3 in ambient air and 5600 F/m^3 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^3 was estimated to be 10^{-6} per fibre/m^3 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^3. 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.

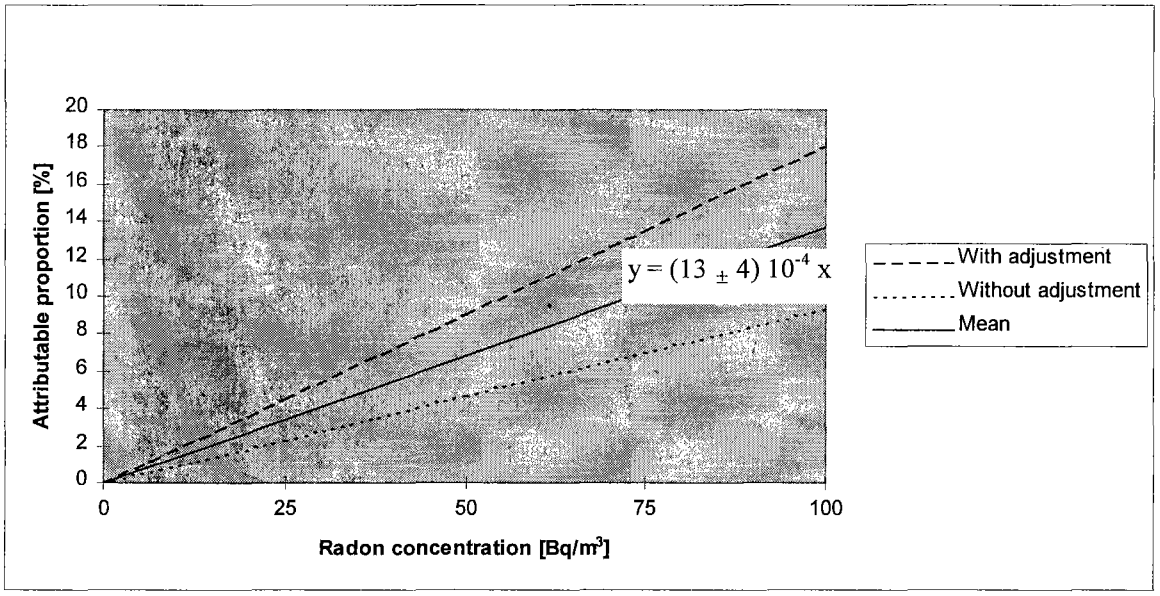


Figure 3.9. The proportion of lung cancers attributable to radon exposure.

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_3, all levels of exposure above zero are associated with effects on health. That pollutants such as sulphur and NO_2 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_2 on health. In a study conducted in Chile close to an industrial area where SO_2 annual means ranged from 101-145 $\mu g/m^3$, and maximum daily averages from 405-1230 $\mu g/m^3$, an increase of 50 $\mu g/m^3$ in the SO_2 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 $\mu g/m^3$ (the WHO guideline) among susceptible children. However, SO_2 may have interacted with PM_{10} levels, which ranged from 5 to 125 $\mu g/m^3$ in this study.

In the same study, when areas with different long-term ambient levels of SO_2 were compared (70 $\mu g/m^3$ vs. 130 $\mu g/m^3$ annual mean over 3 years), the prevalence of chronic respiratory symptoms was higher in the area with the higher SO_2 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_{10} 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_{10} 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 $\mu g/m^3$ SO_2. Suburban area levels ranged from 20.5-23.9 $\mu g/m^3$ and background levels from 13.3-22.9 $\mu g/m^3$. Comparison of the effect of 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 SO_2.

China

Epidemiological investigations in China show short-term exposure to 280 $\mu g/m^3$ 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 SO_2 was 260 $\mu g/m^3$, 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 $\mu g/m^3$ SO_2 (with 550 $\mu g/m^3$ 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 SO_2 pollution on lung function of children and women (Chen et al. 1993). It found that at annual average concentration of 140 $\mu g/m^3$ (with 150 $\mu g/m^3$ particulate matter), SO_2 is associated with lower levels of lung function in children at the ages of 10-12, with major decreases in FVC and FEV_1. For each 60 $\mu g/m^3$ increment in annual average concentration of SO_2, there was an average 99 ml drop in the children's FVC and a 70 ml drop in FEV_1. The FVC of women was decreased by 57 ml under the same conditions. In addition, it was found that 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 $\mu g/ml$ and specific immuneglobuline by 32 $\mu g/ml$ (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 SO_2 at an annual average concentration of 40 $\mu g/m^3$. 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_2 levels			
	Low (<50 μg/m^3)	Intermediate (51-100 μg/m^3)	High (>100 μg/m^3)	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_2, NO_2 and SPM air monitoring was carried out in Parel, Maravali, Deonar and Dadar. Air pollutant levels in winter were higher particularly for SO_2 in Parel (up to 584 μg/m^3) 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_1) were lowest in Parel for males and in Maravali for females. Expiratory flow rates were lower at Dadar, followed by Maravali. Despite lower SO_2 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_2, SPM) or other unmeasured chemicals.

Nitrogen dioxide

Latin America

There are few data from Latin America on the impact of outdoor sources of NO_2 exposure on health. As in many Latin American cities, NO_2 levels are usually low (WHO 1998b). However, in a preliminary study conducted in Sao Paulo, Brazil (Saldiva et al. 1995), a 75 μg/m^3 increase in NO_2 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_2 daily levels correlated with upper respiratory illnesses (Tellez-Rojo et al. 1999). Stronger associations were observed during the winter months, when NO_2 levels ranged from 40-160 μg/m^3 (mean 90 μg/m^3), and O_3 levels from 82-740 μg/m^3 (mean 368 μg/m^3). The correlation coefficient between pollutants and illness was 0.44. The highest indicated effect of NO_2 was observed with a two-day lag. A 56 μg/m^3 increase in daily NO_2 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_2 levels observed in this study, it is not possible to ascertain that NO_2 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_2 levels above 37.6 $\mu g/m^3$ than below this level. In general, however, the levels of NO_2 in Japan are not high enough to demonstrate a clear cause-effect relationship between the prevalence of asthmatic symptoms and NO_2 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_2 sources except for gas cooking stoves, both indoor and individual levels of NO_2 were attributable primarily to automobile exhaust (Nakai et al. 1995).

It has been reported that an interaction between air pollution, especially NO_2, 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_2 and temperature (Choi et al. 1997).

China

In recent years, epidemiological studies examined NO_2 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_2 peak concentrations can reach 990-1,809 $\mu g/m^3$ at the moment of ignition, 17-37.5 -fold higher than the daily average concentration of 50 $\mu g/m^3$ (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_2 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_2 concentration was 53 $\mu g/m^3$, and elevated levels of SO_2, 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_2 at a daily average level of 70-110 $\mu g/m^3$, with the peak values of 150-260 $\mu g/m^3$ for two months, reported a negative correlation between NO_2 concentration and peak expiratory flow rates (PEFR). The results indicate that increased NO_2 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 $\mu g/m^3$ NO_2 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_2 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_2 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_2 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^3 and the COHb blood levels in these children was 4.17%. In rooms with central heating, the CO concentration was 6.4 mg/m^3 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 hypoimmunity 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_3 concentration with respiratory health. A study conducted among children reported both acute and subacute effects of O_3 on lung functions (Castillejos et al. 1992). A 106 $\mu g/m^3$ rise in the mean 48-hour O_3 levels was associated with a decrease of 2% in FEV_1, and a 7.4% decrease in the forced expiratory flow FEF_{25-75}. 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_3 concentration, a decrease of 1.43% in FVC and 2.85% in FEV_1 was reported in the highest quintile of O_3 concentration (364-730 $\mu g/m^3$) (Castillejos et al.1995). This change in FEV_1 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_3 concentrations (Romieu et al. 1992). Children exposed for two consecutive days to peak daily O_3 levels above 260 $\mu g/m^3$ had a 20% increase in risk of respiratory illness. For children exposed for 2 consecutive days to high O_3 levels (above 260 $\mu g/m^3$) 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 $\mu g/m^3$ are frequently reached on several consecutive days.

O_3 exposure has also been related to emergency department visits for acute upper respiratory illness among children in Mexico City. An increase of 100 $\mu g/m^3$ 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 $\mu g/m^3$ 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_3 levels. The upper respiratory illnesses increased linearly from 160-300 $\mu g/m^3$ and then tended to level off. A further increase in risk was observed at levels close to 440 $\mu g/m^3$. Effects at low concentrations of O_3 could not be studied.

Asthmatic children may be more susceptible than others to the effects of O_3 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_3 level, with a 1-day lag (Romieu et al. 1995). In this study, peak O_3 concentrations ranged from 20-500 $\mu g/m^3$, with a mean of 180 $\mu g/m^3$.

In panels of asthmatic children, O_3 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 $\mu g/m^3$ of daily peak O_3 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_3 in Mexico City seems to be smaller than that observed in children who are not chronically exposed to high levels of O_3, 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_3 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_3 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_3 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 ± 40 $\mu g/m^3$ is the threshold concentration for acute lung dysfunction; and 100$\mu g/m^3$ 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_3 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_3 levels (maximum daily O_3 levels were about 240 $\mu g/m^3$) were significantly associated with total daily mortality. There was little evidence of interaction between the O_3 effects (mainly in summer) and particles or with SO_2 and NO_2. The associations between O_3 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_3 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 $\mu g/m^3$ PM_{10} 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 $\mu g/m^3$ PM_{10} (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_{10} and $PM_{2.5}$ during the winter months. In this study, PM_{10} levels ranged from 16-270 $\mu g/m^3$ and $PM_{2.5}$ levels from 10-156 $\mu g/m^3$. It was observed that an increase of 63.5 $\mu g/m^3$ in PM_{10} (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 $\mu g/m^3$ in $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ daily mean ranged from 10-156 $\mu g/m^3$, 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 PM_{10} and $PM_{2.5}$, had smaller slopes than those provided in the WHO Guidelines (Figure 3.7). In fact, for PM_{10} the slope fell below the lower confidence limit provided. For $PM_{2.5}$, the slope was considerably smaller than that shown in the WHO Guidelines. However, when the relationship of $PM_{2.5}$ with pneumonia-related emergency department visits, a severe respiratory illness, was considered, the slope was larger and above the upper limit of the PM_{10} effect predicted by the WHO Guidelines.

Results from a panel study conducted in Punchucavi, 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 $\mu g/m^3$ in the 24-hour average levels of 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 PM_{10}. During the study, daily PM_{10} ambient levels ranged from 29-363 $\mu g/m^3$, with a mean of 167 $\mu g/m^3$, and daily $PM_{2.5}$ levels ranged from 23-177 $\mu g/m^3$, with a mean of 86 $\mu g/m^3$. The results suggested that an increase of 10 $\mu g/m^3$ in 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 $\mu g/m^3$ in the $PM_{2.5}$ 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 PM_{10} and 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 $\mu g/m^3$ 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 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_2 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 $\mu g/m^3$, 36.5-43.3 $\mu g/m^3$ and 27.8-32.4 $\mu g/m^3$, 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 $\mu g/m^3$ 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 $\mu g/m^3$.

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_2.

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_{10} pollution in urban districts and in control districts were 460 $\mu g/m^3$ and 220 $\mu g/m^3$, 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 $\mu g/m^3$ 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 $\mu g/m^3$ of TSP can cause upper-respiratory diseases in children; and that 290-470 $\mu g/m^3$ of TSP significantly depressed immune functions in children. TSP concentrations less than 160 $\mu g/m^3$ 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 $\mu g/m^3$) 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 $\mu g/m^3$. When TSP concentration were higher than 750 $\mu g/m^3$, 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 $\mu g/m^3$. 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 $\mu g/m^3$ of lead in ambient air, the concentration of lead in blood would increase by 1 $\mu g/dl$.

Africa

Studies conducted in Johannesburg indicated that approximately 60% of children have blood lead levels exceeding 10 $\mu g/dl$. 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 $\mu g/dl$ (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_2, NO_x, 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

Principal pollutants	**Sources, predominantly outdoor**
SO_2,, SPM/RSP	Fuel combustion, smelters
O_3	Photochemical reactions
Pollens	Trees, grass, weeds, plants
Pb, Mn	Automobiles
Pb, Cd	Industrial emissions
VOC, PAH	Petrochemical solvents, vaporization of unburned fuels
Principal pollutants	**Sources both indoor and outdoor**
NO_x, CO	Fuel burning
CO_2	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
Principal pollutants	**Sources, predominantly indoor**
Radon	Soil, building construction materials, water
HCHO	Insulation, furnishing, environmental tobacco smoke
Asbestos	Fire-retardant, insulation
NH_3	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_2, 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_2 concentrations (over 2-7 days) were in the range of 20-40 $\mu g/m^3$ in living rooms and 40-70 $\mu g/m^3$ in kitchens, for dwellings with gas equipment and 10-20 $\mu g/m^3$ 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_2 levels under certain circumstances, such as in dwellings equipped with unvented cooking ranges. In addition, short-term measurements reveal NO_2 concentrations that may be five-fold higher than those averaged over several days. Peak values of up to 3800 $\mu g/m^3$ 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^3 for 30 minutes or 30 mg/m^3 for 1 hour. In kitchens with gas stoves, short-term values of up to 15 mg/m^3 have been measured. High values were also measured in bars and pubs, where smoking is common, with average concentrations of 10-20 mg/m^3 and peak levels up to 30 mg/m^3 (Seifert 1993).

In five developed European countries HCHO concentrations in indoor air were reported to range from 9-70 $\mu g/m^3$. 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^3 (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_{10}. 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 $\mu g/m^3$ 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, hepatoxic 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_2, NO_2 and O_3.

- 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, polycylic 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, polycylic 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 particule 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 $\mu g/m^3$ PM_{10} 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 he 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 [μg/m^3]
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 [μg/m^3]
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).

	Concentrations			Exposures		
Region	Indoor (μg/m^3)	Outdoor (μg/m^3)		Indoor (%)	Outdoor (%)	TOTAL (%)
Developed						
Urban	100	70		7	1	7
Rural	80	40		2	0	2
Developing						
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_2., NO_2, CO, O_3, 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_2) to one year (NO_2, SO_2, 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).

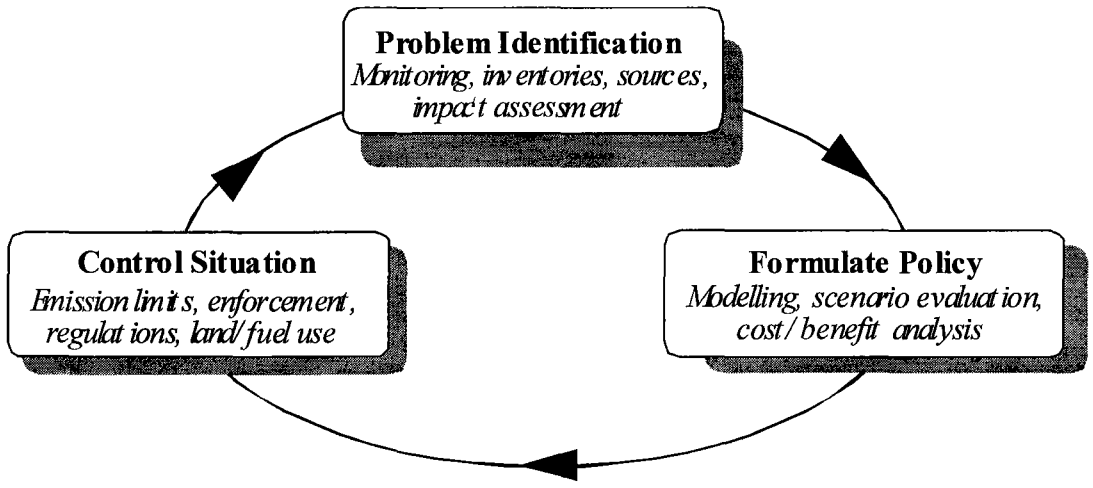


Figure 5.1. The Role of Monitoring in Air Quality Management

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.

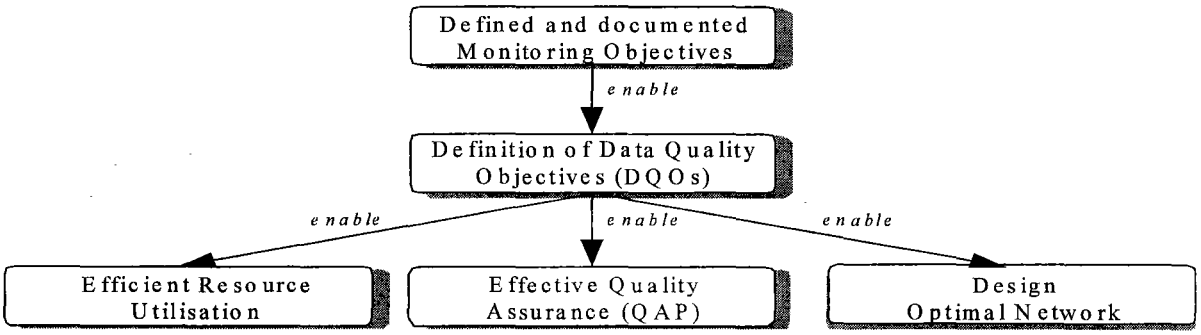


Figure 5.2 The Importance of Objective Setting

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

	• Definition of monitoring and data quality objectives.
Quality Assurance	• Network design, management and training systems. • Site selection and establishment. • Equipment evaluation and selection. • Routine site operations.
Quality Control	• Establishment of calibration/traceability chain. • Network audits and inter-calibrations. • System maintenance and support. • Data review and management.

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.

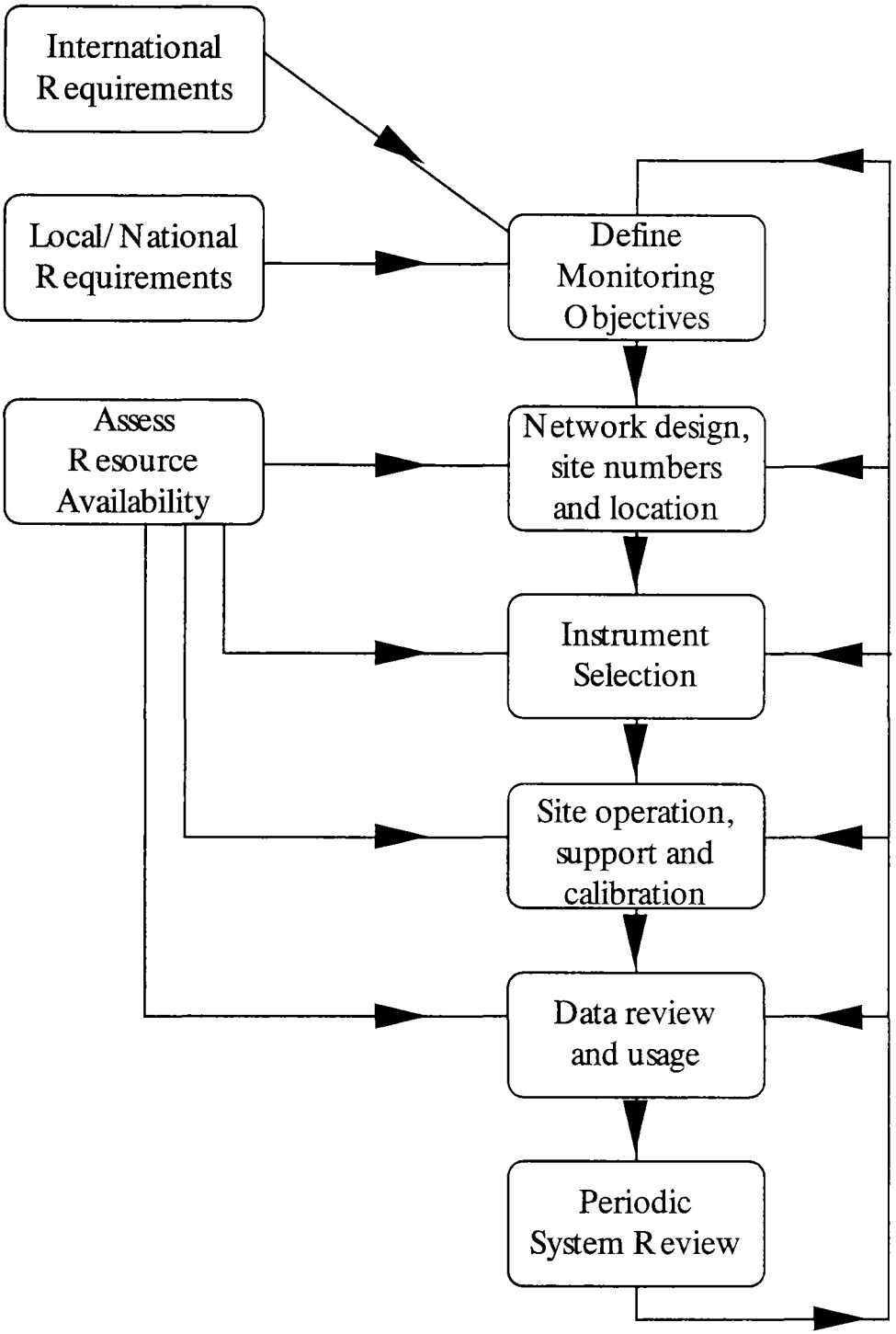


Figure 5.3. QA/QC for Air Monitoring: a Step-by-step Approach

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.

Box 5.9 – Possible Monitoring Locations Relevant to Exposure Assessment	
Site Classification	**Description**
• 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_3 levels have higher spatial uniformity but will be lowest in near-road locations due to scavenging by vehicle NO_x 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_2 and O_3, tends to be more homogeneous than for primary pollutants such as CO and SO_2. 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_2 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.

Box 5.12 - Air Monitoring Techniques

Method	Advantages	Disadvantages	Capital Cost
Passive Samplers	• Very low cost. • Very simple. • No dependence on mains electricity. • Can be deployed in very large numbers • Useful for screening, mapping and baseline studies.	• Unproven for some pollutants. • In general only provide monthly and weekly averages. • Labour-intensive deployment/analysis. • Slow data throughput.	US$10-70 per sample.
Active Samplers	• Low cost. • Easy to operate. • Reliable operation/ • performance. • Historical dataset.	• Provide daily averages. • Labour-intensive sample collection and analysis. • Laboratory analysis required.	US$1000-3000 per unit.
Automatic Analysers	• Proven. • High performance. • Hourly data. • On-line information.	• Complex. • Expensive. • High skill requirement. • High recurrent costs.	US$10 000-15 000 per analyser.
Remote sensors	• Provide path or range-resolved data. • Useful near sources. • Multi-component measurements.	• Very complex and expensive. • Difficult to support, operate, calibrate and validate. • Not readily comparable with point data. • Atmospheric visibility and interferences.	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_2, NO_2, 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_x 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.

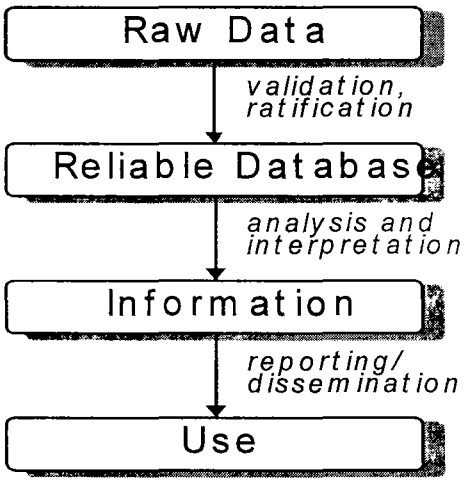


Figure 5.4 Data throughput from a monitoring programme

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_2, NO_2, CO, O_3, 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_2 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_2CO_3)/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_2 diffusion tube analysis. The typical sensitivity of this hybrid technique is ±8.5 $\mu g/m^3$: 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_2 measurements available for trend assessment.

There are several methods of SO_2 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_2 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_2, 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 ±10% has been estimated for SO_2 measurements using the total acidity method, taking account of all contributory factors. A precision of ±4 $\mu g/m^3$ 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_2 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_2 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_2 measurement is ultraviolet fluorescence (UVF). SO_2 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_2 in the sample.

The accuracy of data from automatic SO_2 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 ±10% has been estimated for SO_2 measurements in UK national automatic networks, taking account of all contributory factors. The precision of SO_2 measurements, determined from long-term variations in baseline response of in-service analysers, is estimated to be ±3 $\mu g/m^3$ (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_2, 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_2 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_2 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_2 diffusion tube preparation and analysis.

Recent comparisons of NO_2 diffusion tube measurements with co-located chemiluminescent NO_x analysers show good agreement (Smith et al. 1997; Gerboles and Amantini 1993). Over the range of concentrations generally encountered in urban areas (20-80 $\mu g/m^3$), it was found that on average NO_2 diffusion tubes, exposed for one month, tended to overestimate ambient NO_2 by approximately 10% compared with a chemiluminescent NO_x 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_3 in an evacuated chamber to form an excited energy state of NO_2. The chemiluminescent reaction is:

$$NO + O_3 = NO_2^* + O_2$$

Emitted light from the excited NO_2^* is converted to an output voltage by a photomultiplier tube and amplifier.

Automatic NO_2 analysers based on liquid-phase chemiluminescence, produced by reacting NO_2 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_2 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_2 measurements in well-run automatic networks, taking account of all contributory factors (AEA 1996). The precision of NO_2 measurements is estimated to be ±6.5 $\mu g/m^3$, 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_2, 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 ± 8% and a precision of ±0.5 mg/m^3 may be achieved using this technique in well-managed and quality-assured programmes.

Ozone

O_3 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_x and VOC (see Section 2.1.2). These reactions are not immediate, but may take from hours to days to complete. O_3 is chemically scavenged by primary NO_x emissions from traffic, and is also removed from the atmosphere by deposition to the ground.

Both spatial and temporal distributions of O_3 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).
$NaNO_2/Na_2CO_3$/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_2 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_2 and SO_2). 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_3 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_3-removing scrubber is used to provide a zero reference light intensity, I_0. The analyser alternately measures the absorption of air in the cell with no O_3 present and the absorption in the experimental sample cell, I_s. The ambient O_3 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 ±11% and a precision of ±4 $\mu g/m^3$ should be readily achievable in well-run automatic networks.

Remote sensors

Open-path optical remote sensing techniques such as DOAS are available for 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 $PM_{2.5}$, 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), β-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 m^3/hour) or low-volume (about 1 m^3/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 PM_{10} particulate matter using gravimetric samplers, although these are under development at the present time. An ISO standard for evaluating PM_{10} inlet heads is, however, available (EN 1999). A United States Environmental Protection Agency procedure for 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 $\mu g/m^3$ and a precision of <5 $\mu g/m^3$ (for daily average concentrations <100 $\mu g/m^3$) are given for 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 β-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_{10} inlet head and an accurate flow control system. The PM_{10} 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 $<10 \mu g/m^3$ and precision of $<5 \mu g/m^3$ (for daily average concentrations $<100 \mu g/m^3$) 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^3/day, and Millipore Aerosol Field Monitor filters are exposed and changed weekly.Passive sampling methods are not applicable.