Health and Air Pollution in Developing Countries

edited by

Gordon McGranahan and Frank Murray

Background Document for a Policy Dialogue on
Health and Air Pollution in South Asia, EPTRI, Hyderabad, India

16-19 August, 1999

SEI STOCKHOLM ENVIRONMENT INSTITUTE

Sida

MURDOCH UNIVERSITY
PERTH, WESTERN AUSTRALIA

SACEP

EPTRI

UNEP
EAP-AP

July, 1999

Stockholm Environment Institute
Box 2142
S-103 14 Stockholm
Sweden
Tel +46 8 412 14 00
Fax +46 8 723 0348

Desk-top Publishing, Isobel Devane
Cover by Erik Willis
Stockholm Environment Institute

For information concerning this document, contact:

Dr. Gordon McGranahan
Stockholm Environment Institute
Box 2142
S-103 14 Stockholm
Sweden

Tel. +46 8 412 14 23
e-mail: gordon.mcgranahan@sei.se

A/Professor Frank Murray
Environmental Science
Division of Science and Engineering
Murdoch University
Murdoch, WA 6150
Australia
Tel. +61 8 9360 2501
e-mail: murrayf@essun1.murdoch.edu.au

For information concerning the RAPIDC Programme, contact:

Dr. Johan Kuylenstierna
Stockholm Environment Institute at York
Biology Department
University of York
Box 373
York YO10 5YW
UK
Tel. +44 1094 43 2892
e-mail: jck1@york.ac.uk

Preface

The Stockholm Environment Institute (SEI) has been co-ordinating a programme on Atmospheric Environment Issues in Developing Countries, funded by the Swedish International Development Co-operation Agency (Sida) over a number of years. The study of 'Regional Air Pollution in Developing Countries' (RAPIDC) forms a substantial part of this programme. This compilation of papers has been developed to relate air pollution to impacts on human health and is one of the initiatives of RAPIDC. The impact of air pollution on health has been studied substantially in Europe and North America but the research effort in developing countries is less advanced. This document attempts to summarise the current state of knowledge globally, and the applicability of research results from the North to southern conditions. It provides background material for a policy dialogue to be held from 16-19 August 1999 at the Environmental Protection Training and Research Institute (EPTRI) in Hyderabad, India, where it will form the basis for discussions on the issue of air pollution and health in South Asia and other developing countries. Dialogue between scientists and policy makers forms an important focus for the RAPIDC programme and this workshop follows on from a successful policy dialogue held in March 1998 in Bangkok, organised by UNEP-EAP-AP, SEI and SACEP on air pollution in South Asia. A draft declaration concerning air pollution in South Asia resulting from this meeting was later ratified at a SACEP Governing Council meeting in April 1998 and is known as the 'Malé Declaration on the Prevention and Control of Air Pollution in South Asia and their potential Transboundary Effects'.

The workshop in Hyderabad includes participants from the South Asian countries involved in the implementation of the Malé Declaration, scientists studying air pollution, health impacts and local and national policy-makers. It is intended that this document and the workshop will help to develop the process of improving understanding between these groups around this important issue. This forms part of the process upon which appropriate policy responses may be developed.

Acknowledgements

In the development of this document we would like to thank the efforts and responsiveness of the authors for providing the material, Isobel Devane (SEI) for the DTP, Erik Willis (SEI) for the graphic design and to Anders Arvidsson (SEI) and Steve Cinderby (SEI) for use of photographs. In the organisation of the workshop we would like to thank M.G. Gopal (EPTRI), Raghunathan Rajamani, Mylvakanam Iyngararasan (UNEP/EAP-AP), Surendra Shrestha (UNEP/EAP-AP), Pradyumna Kumar Kotta (SACEP) and Ananda Raj Joshi (SACEP). Finally, we would like to thank Sida for funding the activity that developed this document and the workshop in Hyderabad.

Gordon McGranahan (SEI)
Frank Murray (Murdoch University)
Johan Kuylentierna (SEI)
Vikrom Mathur (SEI)

Table of Contents

Chapter 3

Chapter 4

Chapter 5

Chapter 8

Chapter 9

INTRODUCTION

Gordon McGranahan and Frank Murray

1 INTRODUCTION

Air pollution implies different things to different people. Fine particles in the atmosphere can give rise to beautiful sunsets. Odours, such as those from bakeries or breweries, are considered pleasant by some, but obnoxious by others. The adverse effects of air pollution may influence human health, ecosystems, animals, plants, materials or aesthetics.

Air pollution may be defined simply as the presence of substances in air at concentrations, durations and frequencies that adversely affect human health, human welfare or the environment. Air pollution is not a recent phenomenon. The remains of early humans demonstrate that they suffered detrimental effects of smoke in their dwellings (Brimblecombe, 1987). Blackening of lung tissues through long exposure to particulate air pollution in smoky dwellings appears to be common in mummified lung tissue from ancient humans. Classical writers provide evidence of urban air pollution in the cities of Rome and Athens, and the medieval cities of Europe experienced levels of air pollution considered by the citizens to be unhealthy (Brimblecombe, 1987).

It was with industrialization that local impacts of air pollution on human health and the environment became closely documented. Local impacts of air pollution on human health and the environment became apparent in the 18th and 19th centuries and in the early part of the 20th century. Although it generated public complaints, there was generally a passive acceptance of air pollution by the community as a manifestation of progress and opportunity.

Statistics were collected on deaths resulting from air pollution in the 18th and 19th centuries and in the early part of the 20th century, in London; the Meuse Valley, Belgium; Donora USA; New York City, USA; Osaka, Japan; and elsewhere (Table 1).

High air pollution levels in these cities resulted in excess deaths, including more than 4000 excess deaths in London from a stagnant atmosphere of fog, smoke and sulphur dioxide during five days in December 1952 (Brimblecombe, 1987).

In a climate of public pressure for less polluted air in cities, with relatively cheap clean fuels available, strong economic growth and increasing incomes, governments in developed countries slowly introduced measures to improve ambient air quality in cities. In the early stages there was particular emphasis on reducing particulate and sulphur dioxide concentrations in air in cities. These measures included the location of heavy industry outside population centres, and the requirement for major emission sources to discharge from tall chimneys to disperse the emissions and thus reduce ground level concentrations.

Table 1 Some major air pollution episodes and associated deaths (after Elsom, 1992)

Date	Place	Excess deaths
December 1873	London, England	270-700
February 1880	London, England	1000
December 1892	London, England	1000
December 1930	Meuse Valley, Belgium	63
October 1948	Donora, USA	20
December 1952	London, England	4000
November 1953	New York City, USA	250
January 1956	London, England	480
December 1957	London, England	300-800
November-December 1962	New York City, USA	46
December 1962	London, England	340-700
December 1962	Osaka, Japan	60
January-February 1963	New York City, USA	200-405
November 1966	New York City, USA	168

However, some of these measures contributed to regional air pollution, as emissions from urban and industrial areas can travel long distances, crossing national boundaries, and affecting health and environments in rural areas and in other countries. In response, more effective international action has been implemented. International guidelines on ambient air quality have been produced by organisations such as WHO (WHO, 1987; 1999), and international policies are being co-ordinated under conventions such as the Convention on Long-range Transboundary Air Pollution (ECE, 1995).

In the last two or three decades, attention in the cities of developed countries has been broadened to reducing emissions of carbon monoxide, hydrocarbons, nitrogen oxides, toxic compounds, lead and other heavy metals, although the emphasis and success of management activities have varied in different places at different times. There is also increasing action to reduce exposure to indoor air pollutants.

Most of the published studies on the effects on human health of air pollution relate to the effects of outdoor air pollution on residents of North America and Europe of Caucasian descent, usually of good nutritional status, living in uncrowded conditions, without physical stress or untreated chronic diseases. There are relatively few studies on populations of other ethnic backgrounds, nutritional status, living conditions, stress or history of chronic diseases, or of indoor air pollution. These factors may alter the dose-response relations derived for exposure to outdoor air pollutants (WHO, 1999).

Much of the World's population lives in areas where levels of ambient air pollution exceed World Health Organization (WHO) guidelines. More than 1200 million people may be exposed to excessive levels of sulphur dioxide, more than 1400 million people may be exposed to excessive levels of suspended particulate matter and, although data from developing countries are incomplete, approximately 15-20 per cent of the population of Europe and North America could be exposed to excessive levels of nitrogen dioxide (UNEP, 1991).

The relationship between economic development and health-threatening ambient air pollution is somewhat ambiguous. Uncontrolled industrializsation tends to increase the emissions of a wide range of pollutants. Greater affluence, on the other hand, provides an increasing capacity to monitor and control undesirable pollution. Studies have found that urban sulphur dioxide concentrations tend to increase with economic development, up to about $8,000 (in 1990 US$), and then to decline as air pollution controls become more stringent. There are indications that other health-threatening pollutants follow similar patterns. Thus, some of the worst ambient air pollution problems are to be found in industrialized middle-income cities.

Although the data on indoor air quality are considerably weaker than data on outdoor air quality, Saksena and Smith estimate that most of the premature deaths caused by air pollution are due to indoor air pollution (Chapter 7). Indoor air pollution is of particular concern when smoky fuels are used for cooking and/or heating in poorly ventilated rooms. This tends to be more common in low-income rural areas, where fuelwood and biofuels are plentiful and people cannot easily afford cleaner fuels. Thus, economic development tends to be associated with declining indoor air pollution problems.

At every level of economic development, air pollution poses a serious challenge to public authorities concerned with public health. The air pollution produced by human activities, unlike the intentional products, is easily ignored in private negotiations and decision-making. It is no coincidence that economists often use air pollution examples to help describe the different forms of 'market failure', such as failures of information, externalities, public goods. The damaging role of air pollution is often difficult to perceive, even when the effects are substantial. Even if damage being caused by air pollution is recognized, there are no immediate market mechanisms to ensure that polluters take the associated costs into account. And many of the benefits of better air quality are public, so those affected have little incentive to take action privately. Good science as well as effective policies are needed to meet the challenge.

Although significant actions to improve air quality are apparent in some large cities of developing countries, developing countries face very significant challenges to implement effective action to improve air quality. The extent of the air pollution problems is often poorly understood. Other concerns, including especially the pursuit of economic growth, often take priority. The information needed to take effective action is often lacking. It is hardly surprising that air pollution problems are often effectively ignored even when the costs are potentially very high.

2 OBJECTIVE OF THIS PUBLICATION

The purpose of this publication is to synthesize policy-relevant knowledge on air pollution and health, and thereby provide a firmer basis for improving public health in the SACEP region. The emphasis is on scientific knowledge and its use in air pollution management. Air pollution control cannot be based upon science and technical management systems alone. There are too many uncertainties and conflicting interests, and too much is at stake to leave the key decisions to experts. But this makes it all the more important for decision-makers, as well as those who may be affected, to be as well informed as possible, and that the underlying causes be addressed in a systematic and equitable fashion.

The goal is to support locally driven processes of air pollution management, not to summarize current conditions in the region. The chapters draw on a selection of the best international sources and studies available. Studies from Europe and North America, where the research is more advanced, are reviewed to provide insights into the relationships between some of the most critical air pollutants and health. Studies from a wide range of developing countries, where studies are less exhaustive but the results are potentially more comparable, are also reviewed. Various international tools and systems for air pollution management are described, with an emphasis on approaches that can be used when data are scarce. Regional reviews are provided for indoor air pollution and vehicular pollution, both of which have a particular regional significance. In addition, a few cases from the region are summarized.

In the following sections of this Introduction, we describe some of the types and sources of air pollution referred to in a number of the chapters, review some of the policy issues that motivated this publication, and summarize the contents of the later chapters.

3 TYPES AND SOURCES OF AIR POLLUTION

There is a wide range of pollutants present in indoor and outdoor air. They include many types of particulates, sulphur oxides, carbon monoxide, ozone and other photochemical oxidants, nitrogen oxides, toxic compounds, lead and other heavy metals, and a variety of volatile organic compounds. Due to the many differences among the sources, distribution, and effects of these compounds, to avoid over-generalization it is preferable to treat them separately. However, some general comments can be made.

The major sources of air pollution are the combustion of fuels for electricity generation and transportation, industrial processes, heating and cooking. Reactions in the atmosphere among air pollutants may produce a number of important secondary air pollutants responsible for photochemical smog and haze in ambient air.

The spatial distribution and concentrations of the various air pollutants vary considerably. Most air pollutants are a local phenomenon, with concentrations at any particular location varying with local site geography, emission rate and meteorological dispersion factors.

The scales of air pollution are discussed in the chapter by Kirk Smith and Sameer Akbar (Chapter 1).

3.1 Particulates

Particulate air pollution refers to the presence in air of small solid and liquid particles of various physical dimensions and chemical properties. Although it may be convenient to group them as particulates, their sources, distribution and effects can be highly variable. Some particles can be of natural origin, such as biological particles (pollen, fungal spores, etc), fine soil particles, fine marine salts, wildfire smoke particles, volcanic ash, etc. Others can originate from industrial combustion processes, vehicle emissions, domestic heating and cooking, burning of waste crop residues, land clearing, fire control activities, etc. Other fine particulates can be produced in air as a result of slow atmospheric reactions among gases (such as some photochemical smog reactions, or the oxidation of sulphur dioxide and nitrogen dioxide) emitted at distant locations, and transported by atmospheric processes.

The importance of each source varies from place to place, with economic and other conditions. Cities located in low rainfall areas with soils prone to wind erosion may experience periods of high soil particulate levels. In winter, mid temperate cities of the Northern Hemisphere may experience high concentrations of particulates associated with smoke and sulphur dioxide. In summer, many of these cities experience episodes of photochemical smog associated with mixtures of hydrocarbons and nitrogen oxides. Cities in the tropics, particularly those with high vehicle numbers and subject to poor dispersion conditions, are prone to episodes of photochemical smog. Cities that are heavily dependent on solid fuels are prone to smoke and sulphur dioxide pollution, particularly those that use coal products for industrial production, electricity generation and domestic heating, such as some cities in eastern Europe and China. People in rural areas of many developing countries may experience high concentrations of indoor particulate and other air pollution caused by the burning of biomass fuels. (This is discussed further in Chapter 7.)

3.2 Sulphur oxides

The main sources of sulphur dioxide are the combustion of fossil fuels and industrial refining of sulphur-containing ores. Sulphur dioxide is a colourless gas, which can react catalytically or photochemically with other pollutants or natural components of the atmosphere to produce sulphur trioxide, sulphuric acid and sulphates.

Sulphur dioxide is normally a local pollutant, especially in moist atmospheres, but in oxidized forms it can persist and be transported considerable distances as a fine particulate. It is an important component of acid deposition and haze. Gaseous sulphur dioxide can remain in dry atmospheres for many days and be subject to long-range transport processes. As a local pollutant, ambient concentrations of sulphur dioxide may show considerable spatial and temporal variations. Sulphur dioxide concentrations are declining in urban areas of most developed countries, but in many cities of developing countries ambient concentrations continue to increase.

3.3 Ozone and other photochemical oxidants

Ozone and other photochemical oxidants are formed in air by the action of sunlight on mixtures of nitrogen oxides and volatile organic compounds. A complex series of photochemical reactions produce various oxidants, the most important being ozone and peroxyacetyl nitrate (PAN). Ozone is removed from the atmosphere by reactions with nitric oxide. Ozone concentrations vary with factors associated with the processes of formation, dispersion and removal. Concentrations are higher in the suburbs and rural areas downwind of large cities, than in the city centre, due to ozone removal from the air by reactions with nitric oxide and other components. The concentration of ozone often displays a bell-shaped diurnal pattern with maximum concentration in the afternoon, and minimum concentrations before dawn. Depending on meteorological factors, the highest concentrations occur in summer. PAN concentrations may be 5 to 50 times lower than ozone concentrations, but the ratio can be variable.

PAN concentrations demonstrate the same general diurnal and seasonal patterns as ozone concentrations. Indoor concentrations of ozone are normally substantially lower than outdoor concentrations, although indoor concentrations of PAN may be similar to outdoors.

3.4 Carbon monoxide

Carbon monoxide is a gas produced by the incomplete combustion of carbon-based fuels, and by some industrial and natural processes. The most important outdoor source is emissions from petrol-powered vehicles. It is always present in the ambient air of cities, but it often reaches maximum concentrations near major highways during peak traffic conditions. It often reaches maximum concentrations indoors near unvented combustion appliances especially where ventilation is poor. Cigarette smoke contains significant amounts of carbon monoxide.

3.5 Nitrogen oxides

Although many chemical forms of nitrogen oxides exist, the most significant from a human health perspective is nitrogen dioxide. The main source of nitrogen oxides in cities is the combustion of fuels by motor vehicles and stationary sources such as industrial facilities. Other industrial processes produce nitrogen oxides in air, such as nitric acid manufacturing facilities. Urban concentrations tend to be highest near major road during peak traffic conditions, in the vicinity of major industrial sources, and in buildings with unvented sources. Nitrogen oxides are also important indoor air pollutants, as they are produced by domestic and commercial combustion equipment such as stoves, ovens and unflued gas fires. The smoking of cigarettes is an important route of personal exposure.

3.6 Lead and other heavy metals

There are several metals regularly found in air that can present real or potential risks to human health. These are arsenic, cadmium, chromium, lead, manganese, mercury and nickel. On the basis of widespread distribution at concentrations that may damage human health, lead is the most important of these air pollutants on a global basis.

Lead compounds are widely distributed in the atmosphere, mostly due to the combustion of fuels containing alkyl lead additives. As many countries are reducing the lead content of petroleum fuels, or have practically eliminated lead from fuels, this route of exposure is declining. However, high levels of lead in fuels and increasing vehicle numbers are increasing exposure to lead in some countries. Other important sources of lead in air are from the mining and processing of ores and other materials containing lead. Inhalation of lead is a significant source of lead in adults, but ingestion of lead in dust and products such as paint containing lead is a more important route of exposure in children.

Arsenic and its compounds are widespread in the environment. They are released into air by industrial sources which include metal smelting and fuel combustion, by use of some pesticides, and during volcanic eruptions, and by wind-blown dusts. Arsenic can reach high concentrations in air and dust near some metal smelters and power stations, mostly as inorganic arsenic in particulate form.

Cadmium is emitted to air from steel plants, waste incineration, zinc production and volcanic emissions. Tobacco also contains cadmium; smoking, therefore, can increase uptake of cadmium.

Chromium is widely present in nature, but it can be introduced into the atmosphere by mining of chromite, production of chromium compounds, and wind-blown dusts. It is a component of tobacco smoke.

Manganese is a widely distributed element which occurs entirely as compounds that may enter the atmosphere due to suspension of road dusts, soils and mineral deposits. The smelting of ores, combustion of fossil fuels, and emissions from other industrial processes also provide local contributions to the manganese content of the atmosphere.

Mercury enters the atmosphere through natural processes and industrial activities such as the mining and smelting of ores, burning of fossil fuels, smelting of metals, cement manufacture and waste disposal.

Nickel is an element with low natural background concentrations. It enters the atmosphere due to the burning of oils, nickel mining and processing, and municipal waste incineration.

3.7 Air toxics

In addition to the well-recognized air pollutants, there are many tens of thousands of manufactured chemicals that may be present in indoor and outdoor air. They represent a particular challenge due to the wide variety of chemical types and sources, their widespread prevalence although often at very low concentrations, the difficulties they present with routine monitoring and regulation, and the time delay for human response. While effects of acute exposures to these chemicals are quickly recognized, the effects of chronic exposures to toxic compounds in air are difficult to detect and it may take decades before they are unequivocally recognised. Toxic compounds present in air may include carcinogens, mutagens and reproductive toxic chemicals (Calabrese and Kenyon, 1991).

There are numerous sources of these chemicals including industrial and manufacturing facilities, sewage treatment plants, municipal waste sites, incinerators and vehicle emissions. In addition to the toxic metals, toxic compounds in air may include toxic organic compounds such as vinyl chloride and benzene emitted by sources such as chemical and plastic manufacturing plants, dioxins emitted by some chemical processes and incinerators, and various semi-volatile organic compounds such as benzo(α)pyrene and other polynuclear aromatic hydrocarbons, PCBs, dioxins and furans emitted by combustion processes.

They may be introduced into the body by inhalation, and accumulate over time, particularly in human fatty tissue and breast milk, although this may depend on the chemical characteristics of the air toxic.

3.8 Pollutant mixtures

Most of the work on health responses to exposure to air pollutants has been conducted using single pollutants. Indoor and outdoor air usually contains complex mixtures of air pollutants, and it is practically impossible to examine, under controlled conditions, all of the combinations of pollutants, exposure concentrations and exposure patterns. In general, mixtures of air pollutants tend to produce effects which are additive (Folinsbee, 1992). Acute responses to mixtures are similar to the sum of the individual responses. The responses to long-term exposure mixtures of air pollutants at chronic exposure levels are unclear.

A summary of the sources of the various major indoor and outdoor air pollutants is provided in Table 2.

Table 2 Principal pollutants and sources of outdoor and indoor air pollution

Principal pollutants	Sources
	Predominantly outdoor
Sulphur dioxide and particles	Fuel combustion, smelters
Ozone	Photochemical reactions
Pollens	Trees, grass, weeds, plants
Lead, Manganese	Automobiles
Lead, Cadmium	Industrial emissions
Volatile organic compounds, Polycyclic aromatic hydrocarbons	Petrochemical solvents, vaporization of unburned fuels
	Both indoor and outdoor
Nitrogen oxides and carbon monoxide	Fuel burning
Carbon dioxide	Fuel burning, metabolic activity
Particles	Environmental tobacco smoke, resuspension, condensation of vapours and combustion products
Water vapour	Biologic activity, combustion, evaporation
Volatile organic compounds	Volatilization, fuel burning, paint, metabolic action, pesticides, insecticides, fungicides
Spores	Fungi, moulds
	Predominantly indoor
Radon	Soil, building construction materials, water
Formaldehyde	Insulation, furnishing, environmental tobacco smoke
Asbestos	Fire-retardant, insulation
Ammonia	Cleaning products, metabolic activity
Polycyclic aromatic hydrocarbons, arsenic, nicotine, acrolein	Environmental tobacco smoke
Volatile organic compounds	Adhesives, solvents, cooking, cosmetics
Mercury	Fungicides, paints, spills or breakages of mercury-containing products
Aerosols	Consumer products, house dust
Allergens	House dust, animal dander
Viable organisms	Infections

Adapted from WHO (1999)

4 POLICIES AND DEVELOPMENT OF STANDARDS

The information assembled in this publication can be roughly divided into three types: first, there are summaries of the science of air pollution and health; second, there are descriptions of how local air pollution problems can be assessed and managed; and third, there are examples of specific measures that have or could be taken to reduce the health burden of air pollution.

It is clearly preferable to use the best available information, and take difficult decisions, than to use uncertainty as an excuse for avoiding action. Moreover, it is also important to work efficiently to reduce the uncertainties, and provide the basis for more effective decisions in the future. This publication is intended to help with both of these tasks. Given the range of other problems that governments in the region face, and the common perception that economic growth is imperative, it is not surprising that inherently uncertain problems such as air pollution and health tend to be neglected. But there is increasing recognition that continued neglect can impose a great burden in the form of ill health and early mortality.

One of the difficulties inherent in bringing existing scientific evidence to bear on the relation between air pollution and health in the SACEP region is that, even if science is, in its own terms, objective, the historical selection of topics for scientific study has clearly not been impartial. The health effects of air pollution have been far more heavily studied in Europe and North America, largely because of the availability of funds, not because of any prior reason to suspect health effects would be greater than in developing countries. Much the same applies to the relatively large amount of attention given to ambient air pollution as compared to indoor air pollution. Under such conditions, it is inappropriate to require the same standard of scientific justification to motivate policy actions in different areas. The result would be policies favouring the problems of the affluent.

These difficulties could be at least partly overcome by adopting the precautionary principle. The precautionary principle is interpreted in the Rio Declaration on Environment and Development (Principle 15) as meaning that, where there are potentially serious threats due to environmental damage, a lack of scientific certainty is not sufficient justification for delaying cost-effective preventive action. This principle is often taken as relevant to the uncertain effects of global environmental change, but can also be taken to apply to the uncertain but potentially considerable effects of air pollution on human health, and particularly those which have not been adequately researched.

Several recent developments make this publication particularly timely. Recent epidemiological studies in the late 1980s and 1990s based on time-series analyses have raised new concerns about some of the most common air pollutants. The results of these studies have been remarkably consistent and have withstood critical examination (Samet *et al.*, 1995). The methods used in time-series analyses cannot be expected to prove the possible or probable causal nature of the associations demonstrated between levels of air pollution and health impacts. However, detailed examination of the data and application of the usual tests for likelihood of causality have convinced many experts that the findings need to be seriously considered by policy-makers. The results of the various studies in different cities by different research groups are remarkably consistent. They demonstrate associations between air pollutants and health impacts at levels of pollution previously expected to be relatively safe, and below the levels recommended in the 1987 World Health Organization Air Quality Guidelines for Europe. Partly as a result, WHO has developed new air pollution guidelines (WHO, 1999).

New insights into air pollution are also providing the basis for new tools for air pollution management. The recent assessments of WHO show that for particles and ozone there is no indication of any threshold of effect; that is, there are no safe levels of exposure, but risk of adverse health effects increases with exposure. Similar difficulties in identifying a threshold of effect at a population level apply to lead.

This is important to defining an air quality guideline. No single guideline value can be recommended by WHO. It 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.

In addition, there are a number of other issues to be considered when translating the new WHO Air Quality Guidelines, which reflect these new scientific studies into national or state air quality standards (WHO, 1999).

1 The chemical composition of the particles may be substantially different

The mixture of particles in the communities studied in the development of the particulate guideline was dominated by emissions from motor vehicles, power generation, and space heating by natural gas and light oil combustion. The mixtures in communities in developing countries may be different. They may be dominated by different emissions sources with different chemical characteristics, and by wind-blown soil with entirely different toxic properties from those in the studies used by WHO.

2 The concentration range may be substantially different

The WHO response-concentration relationships for particulate matter are based on a linear model of response, within the range of particulate concentrations typically found in the studies used by WHO. There are no grounds for simple extrapolation of the concentration-exposure relationship to high levels of particulate pollution. Several studies have shown that the slope of the regression line is reduced when the concentration of particulates is at high concentration levels. These levels may be observed in urban areas in some developing 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 with access to modern health services. By contrast, the populations exposed to higher concentrations of particles in less developed countries may have lower level of quality of both nutrition and health care. It is currently unclear whether the responsiveness of the populations in other parts of the World differs from those studies in North and South America and Europe.

For these reasons, while providing extremely useful guidance, the WHO Air Quality Guidelines should be used with caution to develop policies to protect human health in developing countries from the impacts of air pollution.

While there is growing concern about the health impacts of air pollution, air pollution management in the SACEP region is constrained by a self-reinforcing combination of inadequate information, political reluctance and scarce resources. Moreover, there is increasing scope, and need, for sharing information on air pollution and health initiatives, not only North-South, but equally important within the SACEP region.

5 SUMMARY OF THE CONTENTS

Following a contextual chapter (1) that situates the air pollution and health problems of the region in the context of long-term development trends and other air pollution problems, the chapters can be roughly divided into three groups:

- **Evidence on the adverse health effects of various types of air pollution** (Chapters 2 and 3). The first of these chapters draws heavily on recent studies in North America and Europe, while the second synthesizes the evidence from studies in developing countries.
- **Tools and approaches to air pollution management** (Chapters 4, 5 and 6). This group includes a chapter describing how international air pollution guidelines and information systems can be used to develop local standards and regulations, a chapter summarizing some of the rapid assessment techniques that can be applied when critical information is lacking, and a chapter on systematic approaches to air quality management.
- **Issues of particular relevance to the region** (Chapters 7 and 8). This includes a chapter on the contribution of transport to health threatening air pollution, and a chapter on indoor air pollution, focusing on the dangers of some highly polluting domestic fuels commonly used in the region.
- **Two case studies** (Chapters 9 and 10). The first case is Hong Kong, where air pollution problems have been severe, but some significant control measures have been taken, while the second case is Sri Lanka, where air pollution is just beginning to emerge as a significant policy issue.

Although we have tried to ensure as much consistency as possible between the different chapters, their styles reflect the discourses from which they emerge. Thus, for example, the review of recent state-of-the-art studies in the North adopts higher scientific criteria in its selection and presentation of results than the review of existing studies of indoor air pollution in developing countries. Similarly, the more policy-oriented chapters are less questioning of the relation between air pollution and health than the more scientific chapters. In short, the chapters are complementary, but they are not uniform, and are not intended to be.

The health problems caused by air pollution are part of a broader set of environmental problems, and it is helpful to see them in this light. In Chapter 1, Kirk Smith and Sameer Akbar situate the air pollution and health problems of the region in the context of a broader environmental risk transition, wherein the prevailing risks shift from local towards regional and even global scales as one moves from poorer to more affluent settings. They also describe environmental pathway analysis, which provides a common framework for understanding a wide range of environmental impacts, including those on human health. In addition, they examine some of the cross-scale effects, and note some of the trade-offs and opportunities that arise as a result. From this broad overview, it is possible to see how, by paying attention to some of these cross-cutting issues, air pollution damage can be controlled more efficiently.

There is also much of relevance to learn from the current state-of-the-art studies relating air pollution to health. In Chapter 2, Mort Lippmann examines recent evidence from studies undertaken primarily in North America and Europe, focusing on ambient air pollutants. This includes in-depth studies of ubiquitous air pollutants, such as ozone and particulate matter, and their adverse effects on a wide range of documented health indicators, such as mortality rates, hospital admissions, time away from school and work, and lung function. The possible health risks of diesel exhaust are also discussed. Since few of the more advanced studies in North America and Europe have been replicated in developing countries, many health assessments in the SACEP region, including those described in Chapter 6, have drawn on these Northern studies to estimate, for example, dose-response functions for particulates.

In Chapter 3, Isabelle Romieu reviews the epidemiological evidence on air pollution and health in developing countries, again focusing on ambient air pollution. Several factors, such as nutritional status and population structure, suggest that the adverse health effects may be even greater than those found in developed countries. The data required for the more in-depth studies are not generally available, but the available evidence tends to confirm the view that residents of polluted cities in developing countries are at considerable risk. For example, appreciable risks were found in studies relating particulate concentrations (PM_{10}) and mortality in Sao Paulo (Brazil), Santiago (Chile), Mexico City (Mexico) and Bangkok (Thailand). This chapter also reviews the evidence on ozone, nitrogen oxide, carbon monoxide and lead. Clearly, more research is needed in developing countries, but the indications are that the effects of air pollution are at least roughly comparable.

WHO air pollution guidelines have long been an important tool for countries developing their own air pollution standards, regulations and policies. In Chapter 4, Dietrich Schwela describes how WHO guidelines can be used to help develop locally appropriate standards, drawing on both local data and modelling and internationally available tools. These guidelines can also be useful for developing the legal framework necessary to translate local standards into effective action. The lack of information can be a significant challenge in many low-income cities, however, and additional data collection and analysis are often needed to translate existing guidelines into effective air pollution management systems.

When information is lacking, decisions must nevertheless be made, and there are a number of relatively rapid techniques to help support local decision-making. Yasmin von Schirnding describes some of the most important techniques in Chapter 5. Rapid assessments may be needed to respond to a particular event (e.g. an air pollution episode or a community concern with regard to the pollution from a certain factory), or to help fill in the gaps in the existing information system. Possible techniques range from rapid epidemiological assessments to rapid source-emissions inventories. The mix of techniques required depends upon local circumstances, but it is important that decision-makers be aware of the range of techniques available. To be used most effectively, rapid assessment should not be a one-off effort, but an integral part of the air quality management system.

The importance of taking a systematic approach to ambient air quality management is noted in a number of chapters. In Chapter 6, Steinar Larssen and colleagues describe how a systematic approach can be implemented, drawing on their experience with the World Bank's URBAIR project, which undertook to help create air quality management systems in Jakarta, Kathmandu, Manila and Mumbai. An air quality management system is an iterative process that can be initiated through the following steps: air quality assessment; environment and health damage assessment; abatement options assessment; cost-benefit of cost-effectiveness analysis; abatement measures selection; design of control strategy. In the participating cities this process helped to identify a number of options whose benefits outweighed the costs.

In large parts of the SACEP region, however, an exclusive focus on ambient air quality is potentially very misleading. As described by Sumeet Saksena and Kirk Smith in Chapter 7, indoor air pollution may be having a large impact on health owing to the use of biofuels, such as fuelwood, to cook (and sometimes heat) in enclosed spaces, especially in rural areas. Among the principal health risks are acute respiratory infection in children, and chronic obstructive lung disease and lung cancer in women. Despite its potentially great importance, most of the research on the health risks of indoor air pollution is recent, with sample sizes and study designs far less sophisticated than those used to study the effects of outdoor air concentrations. In reviewing the evidence, Saksena and Smith argue that there is emerging evidence that indoor air pollution is associated with important health effects. They describe some of the research needed to understand more fully these effects, and some of the actions that can be taken to reduce the risks.

Turning back to issues of outdoor air quality, Michael Walsh reviews the evidence on vehicle emissions and health in the region in Chapter 8. Vehicles are a major contributor to ambient air pollution in many Asian cities, and can lead to particularly high exposures among people situated in heavily trafficked areas. The vehicle fleets of Asia are not as large as in many developed countries. However, the popularity of highly polluting motorcycles and scooters, the age and poor maintenance of the vehicles, the high usage rates, and the continued use of leaded and poor quality fuels, lead to high emissions per vehicle for a number of health-damaging pollutants. On the other hand, there have been effective measures to reduce the vehicular pollution in a number of Asian cities and countries. If appropriate lessons are drawn from the successes of existing programmes (some of which are described in this chapter), the relative contribution of vehicles to health threatening air pollution should decline.

There is, of course, enormous variation in the air pollution problems which different Asian cities face, and the extent to which their health implications have been studied. In Chapter 9, Anthony Hedley and colleagues review recent findings in Hong Kong, whose air quality is not atypical for a large Asian city. The evidence suggests that the relative risks to health from a number of pollutants are higher than in Western European cities, but that recent control measures have had an appreciable effect on air quality and health. They claim that policy-makers can be confident that controls on pollution sources will provide health benefits, but also note that public concerns over the more visible and easily perceived effects of air pollution have been critical to motivating air pollution control measures.

In the final Chapter (Chapter 10), Ramani Ellepola reviews the current situation in Sri Lanka, where urban air quality management is still in its early stages. Continuous air quality monitoring only began in 1996 in Colombo, the capital city. This monitoring indicates excessively high concentrations of particulates, but acceptable levels of other monitored pollutants. Environmental impact statements were among the first tools to be applied to air pollution control. Air pollution standards have been gazetted, and emissions standards for polluting industries will come into force in 1999. A National Policy on Air Quality Management is now being drawn up. But pressures are also growing, as the power sector is moving away from its heavy dependence on hydropower.

Taken together, these chapters both raise very serious concerns about the health hazards of air pollution in the SACEP region, and indicate that much can be done to reduce these concerns. There is, however, a wide range of actors involved, and co-ordination is of critical importance. There is little point in collecting information if it will not be used, and it is unfortunate when actions are taken on the basis of insufficient information. This is one of the justifications from developing air quality management systems at the local level, and involving all stakeholders in the process. National governments need to provide support for local initiatives, as well as an appropriate regulatory and policy framework. In addition, regional and international co-operation can also make important contributions.

Sharing of experiences and information is an important first step in regional co-operation. But in a number of areas, co-operation could go beyond this. In research, for example, the problems of indoor-air pollution clearly deserve more attention, and in-depth studies - too costly to carry out in every country - could be carried out in a selection of locations. Similarly, there are returns to scale in a number of ambient air quality and health research areas, and joint research initiatives could help overcome the current reliance on studies conducted in European and North American countries where conditions are appreciably different.

Regional co-operation on transboundary air pollution could also be linked to air pollution and health concerns. Improved health is one of the most compelling reasons to control air pollution in the SACEP region, as the following chapters demonstrate.

6 REFERENCES

Brimblecombe, P. 1987. *The Big Smoke*. Methuen, London.

Calabrese, E.J. and Kenyon, E.M. 1991. *Air Toxics and Risk Assessment*. Lewis Publishers, Michigan.

Elsom, D. M. 1992. *Atmospheric Pollution: A Global Problem*. Blackwell, Oxford.

Folinsbee, L.J. 1992. Human health effects of air pollution. *Environmental Health Perspectives* **100**: 45-56.

Samet, J.M., Yeger, S.L. and Berhane, K. 1995. The association of mortality and particulate air pollution. In *Particulate Air Pollution and Daily Mortality, Replication and Validation of Selected Studies, the Phase I Report of the Particle Epidemiology Evaluation Project*. Health Effects Institute, August 1995.

UNEP. 1991. *Urban Air Pollution*. United Nations Environment Programme, Nairobi,

WHO. 1987. *Air Quality Guidelines for Europe*. WHO Regional Office for Europe, Copenhagen.

WHO.1999. *World Health Organization Guidelines for Air Quality*. WHO, Geneva.

Chapter 1

HEALTH-DAMAGING AIR POLLUTION: A MATTER OF SCALE

Kirk R. Smith
University of California, Berkeley, and East-West Center, Honolulu
and
Sameer Akbar
World Bank, New Delhi

Abstract

This Chapter presents some key concepts that help to frame the problem of health-damaging air pollution in South Asia and its relationship with other important categories of air pollution. It discusses the risk transition, which places the shift from traditional to modern sources of pollution along the continuum from household, community, regional, to global scale. It describes environmental pathway analysis, as applied to health-damaging pollution, focusing on the concept of exposure assessment, which has become central in recent years. Finally, it briefly outlines some of the major cross-scale effects through which pollution problems at one scale relate to problems at other scales, and the trade-offs and opportunities that arise as a result. It concludes that more efficient control of air pollution damage in South Asia today can be achieved with closer attention to some of these cross-cutting issues.

Later chapters will examine the major sources of air pollution, both indoor and outdoor, in the region. Here, as an introduction, some important cross-cutting conceptual issues are raised which bring out the spatial and temporal nature of air pollutants, followed by a brief description of some of the interactions that reach across scale boundaries. This may help to place later chapters in context.

1.1 INTRODUCTION

As discussed in the Introduction, public concern about air pollution first clearly manifested itself in the context of ambient air quality in urban areas. Indeed, the first public air pollution commission in recorded history was created in 1285 in London. After deliberating for 21 years, it recommended banning of coal burning in urban areas, an action not fully implemented for nearly 700 years (Brimblecombe, 1987). With the growth of urban agglomerations in developing countries, urban air pollution has similarly been recognized as having a serious impact on human health. Cities in South Asia, like many others in the developing world, are paying the social, health and economic costs of elevated levels of air pollution.

In recent decades, however, there has been an expansion of the scale of concerns about urban air pollution, both in terms of its impact at smaller scales such as individual households, as well as larger scales such as an entire region. Accompanying this expansion in scale has been an expansion in the nature of negative health impacts that are of concern.

Examination of air pollution at smaller scales has been necessitated because it has become clear that in some cases potential health impacts are not always well predicted by outdoor measurements. In particular, sources that lead to indoor air pollution may not affect outdoor levels significantly while still resulting in significant ill-health.

Expansion of concern to larger scales has been required because it has become known that some pollutants can travel large distances over time, beyond the site of emissions thus resulting in regional and global impacts. In some cases, the same pollutant can have different kinds of impacts depending on the scale. For example, SO_2 may have a direct health impact at the community scale, but an impact through acid precipitation at a regional scale.

1.2 RISK TRANSITION

There is a tendency, although not an inevitability, that peak environmental risks shift scale from small to large during the economic development process. As shown in Figure 1.1, environmental hazards in the poorest communities tend to be dominated by risks related to poor water, food and air quality at the household level. These hazards still dominate the environmental risks for some 1,000 million people in South Asia. The spatial and temporal dimensions of such hazards are quite short.

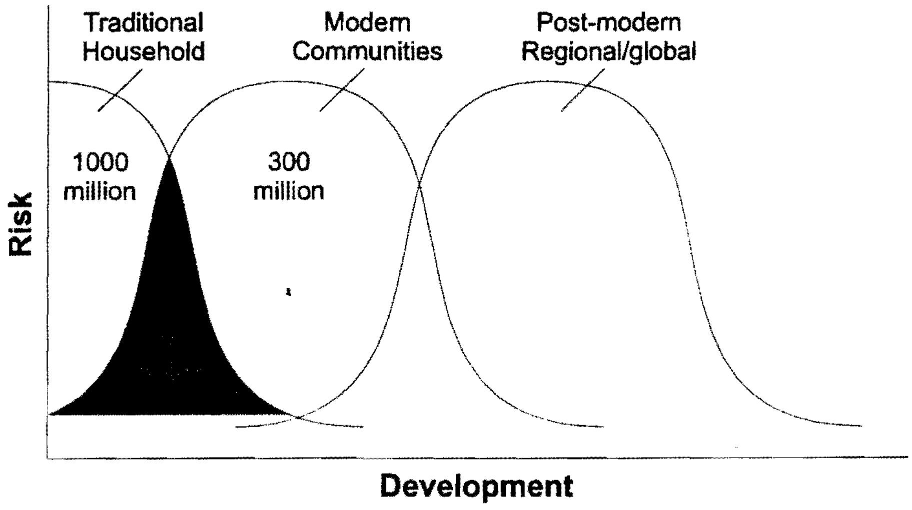


Figure 1.1 Risk Transition. Note how there is a trend in environmental risks during economic development to move from household to community to regional to global scales. The numbers indicate, roughly, how many people are most affected at each scale in South Asia

The solutions that are often implemented to address household problems during development (chimneys, drainage, etc.) tend to shift environmental problems away from households to the community level, i.e. smaller to larger scale. They then join with the new types of environmental risks that are created from agricultural modernization, urbanization, industrialization and other aspects of development. About 300 million people in South Asian live in areas where these risks are likely to dominate.

As these community-scale hazards come under control during further economic development, the peak of impact tends to shift to the regional and global scales through long-term and long-distance transport of pollutants. The spatial and temporal scales shift accordingly, as shown in Figure 1.1. In South Asia, although there are millions of well-to-do people with lifestyles that require the energy and resource use similar to that of people in rich countries, with consequent impacts on the global environment, the percentage of the total population is low.

As in much of the developing world, likewise in South Asia there are large populations at different levels of development living in close proximity to one another. This creates the potential for environmental risk overlap situations. The principal one of concern in South Asia is indicated by the shading in Figure 1.1, which delineates the region between household and community hazards. In this risk overlap region live approximately 100 million of the urban poor who simultaneously confront some of the worst of household environmental hazards from poor food, indoor air and water quality, and the worst of community hazards in the form of outdoor air pollution, hazardous materials, traffic, etc. (Smith, 1997).

It is generally, although not inevitably, true that economic development, in addition to extending the temporal and spatial scales of impacts, tends to shift health risks from the direct to the indirect. Direct health risks, for example, result from inhalation of toxic pollutants. Indirect risks, in contrast, result from processes such as a shift in disease vectors coming from climate change induced by greenhouse gas pollutants that may have no direct health impact.

1.3 ENVIRONMENTAL PATHWAY ANALYSIS

Illustrated in Figure 1.2 is perhaps one of the most basic set of relationships in environmental sciences, those among Sources, Emissions, Concentrations, Exposures and Health Effects. Although concern with air pollution and other environmental hazards is due to the ill-effects they cause, including those on health, waiting until ill-effects can be reliably determined is not an effective means of controlling the impacts. It is more useful to understand the entire environmental pathway from Source through to Health Effects. In this way, the most important sources and best points for control can be determined and ill-effects prevented before they occur. The different steps in environmental pathway analysis can be summarized as follows (Smith, 1993);

Step 1: Sources-Emissions - Although the type of source, dirty *versus* clean fuels, for example, gives some idea of hazard, a more valuable measure is the actual amount of pollution released.

Step 2: Emissions-Concentrations - The most valuable measure, however, is the environmental concentrations of pollution that results, which depends not only by the emissions but also on the transport, transformation and dilution of the pollutant in the environment.

Step 3: Concentrations-Exposures - Environmental contamination, however, is not as reliable an indicator of impact as some measure of exposure, which is the contact of the polluting material with the sensitive system, whether a human, a building, ecosystem or whatever.

Step 4: Exposures-Health Effects - Not all exposures will create the same impact, however, because of differences in the vulnerability of different people or the competing risks that affect them.

Although the pathway in Figure 1.2 refers to ill-health as the endpoint of concern, the same concept can be usefully applied to other important concerns. For example, the endpoint may be ecosystems such as lakes and forests that are vulnerable to acid deposition from regional air pollution. In this case, the important exposures and sources may be entirely different from those that most directly affect health because, among other reasons, the ecosystems are in quite different places than the bulk of the people.

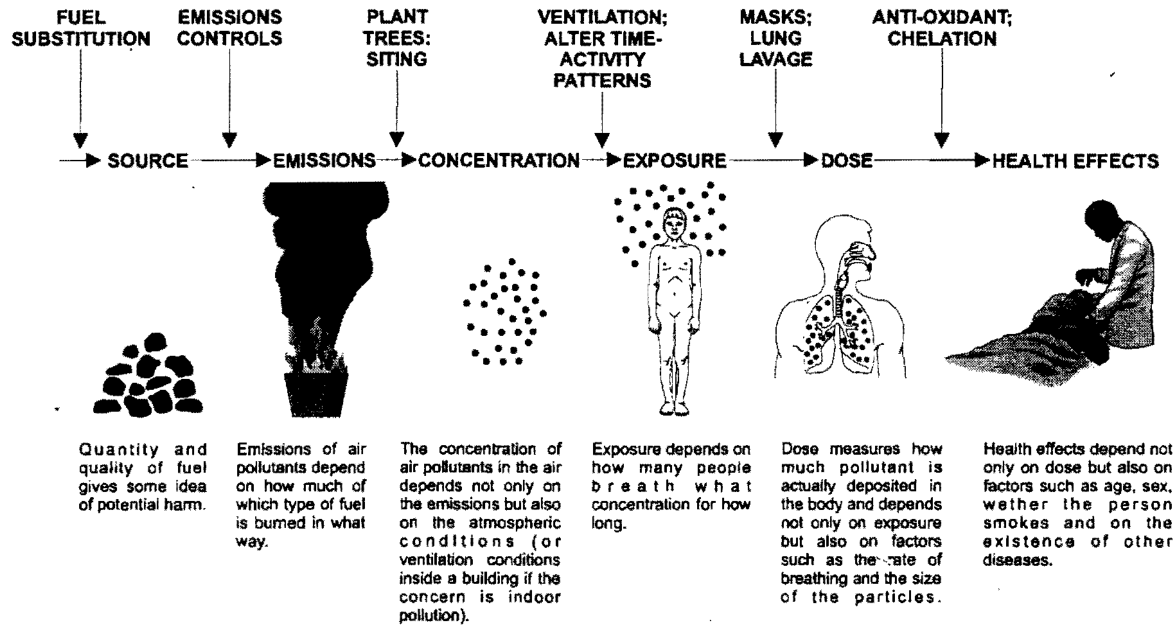


Figure 1.2 Environmental Pathway. To understand the control pollution effectively, it is necessary to understand the entire pathway from source to effect although measurement and control can occur at any number of places along the pathway

1.4 EXPOSURE ASSESSMENT

Most air pollution monitoring and control efforts in South Asia have focused on the emissions and concentrations of pollutants in the outdoor environment (steps 1 and 2 above). In this, they are consistent with the historical development of air pollution management in the currently developed countries. Furthermore, estimation of health impacts has usually been done by extrapolating studies of concentration/health studies carried out in developed countries to the concentrations measured in South Asia. Unfortunately, this is often the only possible approach because of limited data from South Asia itself.

Data from the region itself should be developed, however, because such extrapolation is subject to question for several reasons, including:

(i) Different exposure levels, i.e., the average concentrations of concern in South Asian cities today are many times greater than the levels studied in most recent urban outdoor studies in developed countries and there is evidence of a difference in the effect per unit increase at these higher levels (Lipfert, 1994). Figure 1.3, for example, shows the distribution of urban ambient PM_{10} concentrations in Indian cities containing approximately one-quarter of the national urban population. It should be noted that the mean concentration experienced by the population (194 mg/m^3) is more than six times the US urban mean of approximately 30 mg/m^3 (AMIS, 1998).

(ii) Different populations, i.e. the pattern of disease and competing risk factors differ dramatically between urban developed-country populations, the World's richest and most healthy populations, and people exposed to heavy indoor air pollution in developing countries who tend to be the poorest and most stressed populations in the World. For example, the overall risk of acute respiratory infections in young children, one of the main impacts of air pollution in South Asia, is some 90 times higher in India than in Western Europe and North America where most air pollution epidemiology has been carried out (Murray and Lopez, 1996).

(iii) Different relationship between outdoor concentration and actual human exposures because of differences in behaviour, building construction, climate and the prevalence and strength of local sources not well represented by outdoor monitoring. For example, in tropical climates where housing tends to be well ventilated, in the absence of indoor sources, indoor concentrations may be closer to outdoor levels than in temperate countries where most epidemiological studies are carried out. On the other hand, there may be more indoor and neighbourhood sources that are not well reflected in general ambient levels.

(iv) Different mixtures of pollutants, i.e. although the concentrations of particulate air pollution of certain size ranges are measured in both cases, the chemical nature of the mixtures may be quite different, e.g. higher fractions of diesel exhaust and biomass-fuel particles in South Asian cities.

These concerns cannot be completely resolved except by studies done under South Asian conditions. There is a more fundamental problem, however, with direct application of risk factors derived from outdoor measurements to calculating population impacts, a problem which is also shared in developed countries.

To understand the problem it is important to differentiate the two scales at which typical air pollution health studies operate. Generally, such studies are carried out by examining the way differences in outdoor pollution levels over time or between different populations (cities or parts of cities) correlate with differences in health status in the populations of concern. Thus, the pollution measurements are made at the community level, but the health measurements are made at the individual level. The assumption, therefore, is that the community pollution levels measured accurately represent what individuals experience.

A number of studies carried out around the World, including those in Asia, show that it is often true that the <u>change</u> in outdoor levels is reflected in <u>changes</u> in the level experienced by individuals. Indeed, this is why so many of these studies around the World have shown such high correlations of outdoor pollution differences and differences in ill-health. In addition,

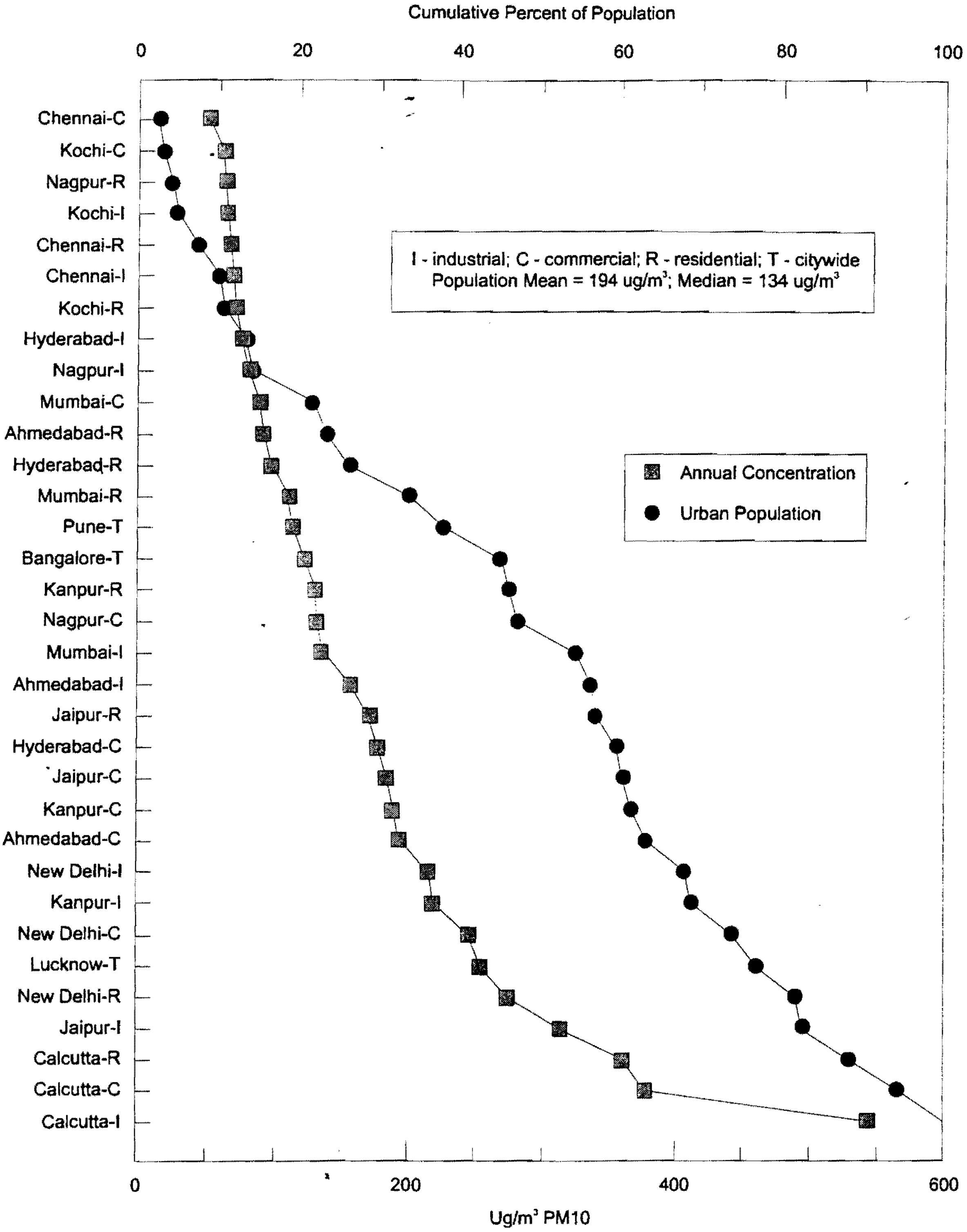


Figure 1.3 **Urban PM_{10} concentrations in Indian cities. These cities contain approximately one-quarter of the 250 million urban dwellers in the country (source: AMIS, 1998)**

however, many studies also show that the absolute levels measured outdoors are often quite different than the absolute levels experienced by individuals, because of a combination of less than perfect penetration indoors by outdoor pollutants or local sources (Janssen, 1998; Tsai *et al.*, 1999).

To determine the total health impact of air pollution, therefore, it is necessary to combine the results of the health studies with estimates of the total exposure to the pollutants from other studies designed specifically to determining where people are in relation to where the pollution is. It may be that the outdoor levels measured in the course of health studies are reliable indicators of total exposure, but it is much more likely that they indicate only part of the exposure - that due to outdoor pollutant levels. Indoor or other localized pollutant sources, which may not affect outdoor levels to any degree, can add substantially to total exposures.

Although, once stated, the idea of looking not only where the pollution is but also where the people are to determine the total health implications of pollution, may seem obvious, it can have a profound impact on how the problem is framed. For example, it alters the relationship between commonly perceived pollution sources, emphasizing how much of their emissions actually reach people's lungs rather than their total emissions. In this way, for illustration, vehicle emissions, which are released in places and times where people are located, may exert a higher health price per unit pollution than emissions from large outdoor sources, such as power plants, which release their pollution relatively far from where people reside. It has been shown, for example, that hourly exposure levels for respirable suspended particulate matter on three-wheeler scooters (782 mg/m^3) in Delhi are almost three times the eight-hourly ambient concentration (275 mg/m^3) (Akbar, 1997).

In addition, exposure assessment often also reveals an entirely new landscape of sources and potential control measures. In South Asia, for example, as discussed in Chapter 7, recognition that a large portion of the entire population's time is spent in households when solid fuels are burned in open stoves reveals a pollution source that probably accounts for a larger total exposure than outdoor sources in the region even though not contributing a majority of total outdoor emissions. It also reveals ways to control exposures that do not rely at all on decreasing outdoor emissions. Indeed, some viable approaches to decreasing exposures, such as disseminating stoves with chimneys, may actually increase outdoor emissions and concentrations.

The concept of exposure is not confined solely to pollutants that produce direct health effects. Consider, for example, pollutants, such as NO_x and SO_x, that can produce acid deposition (rain, snow or particles alone) by conversion through atmospheric chemistry, often far from their source, to acidic aerosols (mixture of liquid and solid particles). Acid deposition is not a problem everywhere. Much of the World's surface is covered with ocean or with soils and vegetation that are little affected by acid deposition. Other areas, however, such as some types of crops, forests and lakes, can be damaged. Whether a certain emission's source creates an exposure of concern, therefore, depends on its orientation and distance from vulnerable ecosystems, local wind patterns, etc. This is exactly parallel to the relationship of health-damaging pollution sources, i.e. some are much more effective at producing exposure than others.

The same concept applies to greenhouse gases, e.g. methane and carbon dioxide, which indirectly affect human health through global warming. In this case, the impact per kilogram of emissions can vary dramatically, depending on the particular physical properties of the substance and its lifetime in the atmosphere. Over the next 20 years, for example, a kilogram of methane emissions will cause approximately 60 times more exposure to Earth in the form of global warming than a kilogram of carbon dioxide. Nitrous oxide, another greenhouse gas, is nearly 300 times as powerful than an equal amount of carbon dioxide.

1.5 MAJOR CROSS-SCALE EFFECTS

The air pollution implications in South Asia at each scale are described briefly in the following sections. The discussions rely on the concepts discussed above related to the risk transition, environmental pathway and exposure assessment. Briefly, implications are discussed for viable air quality management and ways in which actions at each scale may affect other scales. At the end some of the trade-offs and opportunities created by the differences are discussed briefly.

1.5.1 Household/Neighbourhood

As discussed in Chapter 7, there seem to be large health implications of uncontrolled combustion of solid fuels in South Asian homes due to the resulting household exposures to important pollutants. Efforts to reduce household indoor pollution through use of chimneys can act to shift the problem to larger scales. In some villages, household pollution can lead to a "neighbourhood effect" in which outdoor concentrations are elevated in communities with many households using solid fuels. Figure 1.4 shows, for example, outdoor measurements in a village in western India during the evening meal.

In dense urban areas, household fuels can contribute significantly to general ambient pollution. In addition, however, there can also be significant neighbourhood effects in cities. Figure 1.5 shows results from a study of residential areas in Pune, India. It should be noted that the local outdoor levels in the neighbourhood of biomass-using households are much higher than in gas-using areas, which have outdoor levels similar to the citywide concentrations. Kerosene-using areas seem to be intermediate. It should also be noted that the total exposure of people living in biomass-using households is significantly affected by the pollution coming indoors from outside.

Thus, although improved stoves in South Asia are commonly called "smokeless", they actually still produce substantial pollution, although, when operating well, at least the pollution is vented outdoors. It is clear that such stoves can only be considered as an interim solution in such communities, however. If many people use solid fuels, whether with chimneys or not, total neighbourhood exposures can remain high, even for those households not cooking at all or cooking with clean fuels. This situation may also argue for programmes designed to convert whole neighbourhoods at once to clean fuels.

1.5.2 Neighbourhood/Community

To determine total exposure of pollutant emissions, it is necessary to consider possible transformations in the environment. Two such transformations act in cities to create pollution outside the local area where the precursor pollutants are emitted:

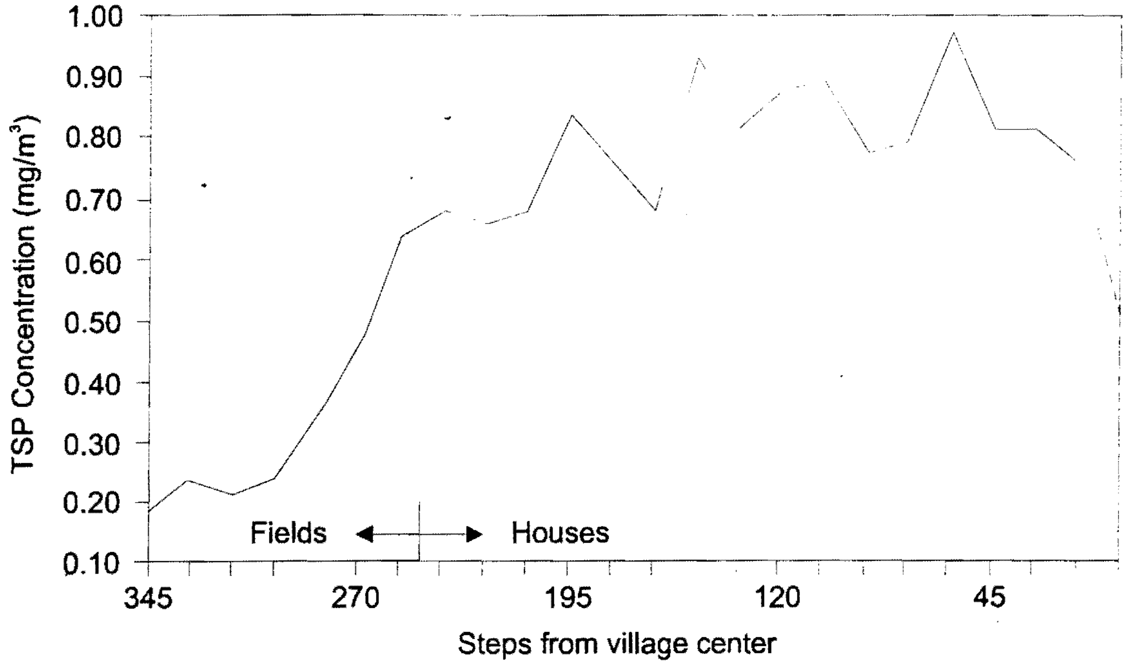


Figure 1.4 Neighbourhood pollution in an Indian village in central Gujarat during the winter (source: Smith, 1987)

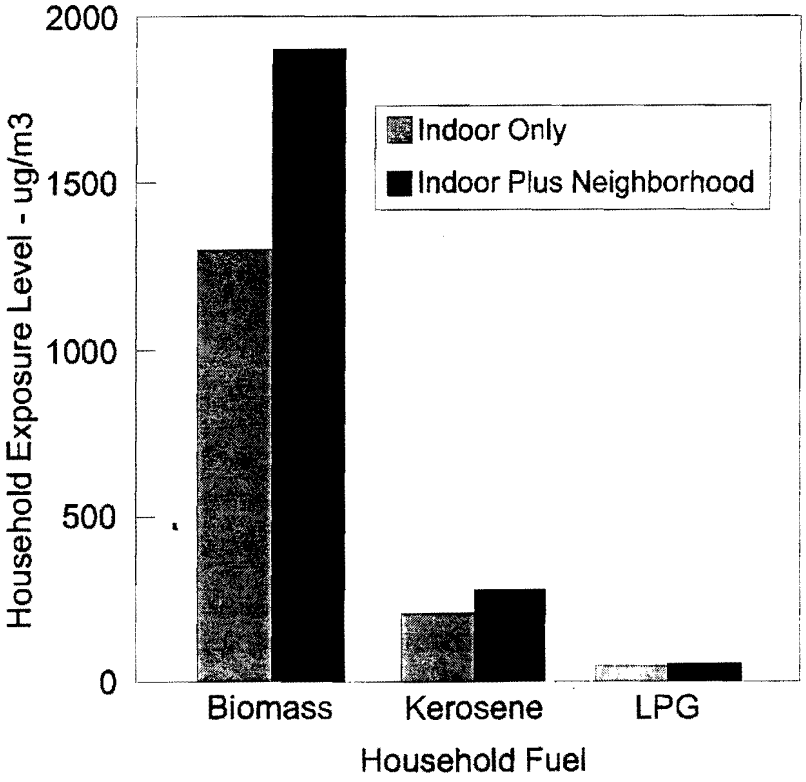


Figure 1.5 Urban neighbourhood pollution measured in Pune, India (source: Smith *et al.*, 1994)

- Urban ozone concentrations are a potentially serious threat to health (see Chapters 2 and 3). It is not emitted directly, however, but is formed by a combination of NO_x and hydrocarbon pollutants in the right conditions of temperature and sunlight; thus it is the principal pollutant in what is often called "photochemical smog". Since it takes on the order of hours to form, it can be a problem relatively far from the points at which the precursors (NO_2 and hydrocarbons) are emitted.
- Particulates are not only emitted directly by fuel combustion and other processes, but are created through chemical reactions in the atmosphere that transform the gaseous pollutants, SO_x and NO_x, into sulphate and nitrate particles. These particles tend to be more acidic than those directly emitted from fuel combustion and other sources and some studies indicate that they may be more toxic by mass than non-acidic particles. Indeed, the new WHO-EURO Air Quality Guidelines specify a separate particle to health relationship for sulphate for this reason. Ammonia emissions from industry or agriculture can also be converted into particles. As with ozone, all these particles can be formed far from the original emitters, depending on wind, temperature, humidity and other factors.

1.5.3 Community/Region

Particles and ozone can also be created outside cities from city sources and impose health risks as well as loss of visibility and damage to crops downwind. In addition, sulphate and nitrate particles can be carried hundreds of kilometres from cities or remotely sited facilities such as power plants to be deposited in dry form or as acid rain/snow (collectively called 'acid deposition'). Such deposition usually imposes little direct health risk, but can damage natural and human-managed ecosystems with indirect impacts on human health and wellbeing. One potential health impact, in selected situations, would be mobilization of toxic metals normally bound up in soil that might be consumed by humans, for example in contaminated fish.

1.5.4 Region/Globe

Combustion-generated particles and those created by downwind transformation of SO_x and NO_x are generally quite small, less than one-millionths of a meter in diameter. Such particles can stay aloft in the atmosphere for months and thus have time to travel around the World and have a global influence. Although the impacts of these particles are not known precisely and are thus the subject of considerable current research, there seem to be two major types of interaction:

- These particles may form the seeds for clouds, causing perhaps earlier and greater cloud formation than would occur without them, leading to more reflected sunlight.
- Depending on the character of Earth's surface below them, the particles can be either darker or lighter, thus leading to changes in the amount of reflected sunlight. In general, it is currently thought that the net effect is to reflect more sunlight than would otherwise occur.

The overall effect of global particles is thought at present to be net cooling of Earth, a conclusion supported by the known impact of the suspended dust from large volcanic eruptions. Indeed, emissions of human-generated particles help researchers to understand the discrepancies in the large computer models designed to "pre-predict" global warming of greenhouse gas emissions over this past century. The models generally predict greater warming than has actually been observed, but come much closer to reality when the cooling from particles is included.

The message for air pollution control policy seems to be that, as emissions of particles and their precursors come under control because of concerns about household, community and regional impacts, their capacity to partly shield humanity from the global warming implications of greenhouse gas emissions will diminish.

1.5.5 Household/Globe

The same incomplete combustion processes in solid-fuel stoves that produce most of the health-damaging pollutants (HDP) such as particles, formaldehyde, benzene, etc., also produce important greenhouse gases (GHG), such as methane. In addition, some of the emitted pollutants, such as carbon monoxide and hydrocarbons, are both HDP and GHG precursors. Typically, 10-20 per cent of the fuel carbon in South Asian solid fuel stoves is diverted to GHG and HDP, instead of carbon dioxide and water, which would be the products of complete combustion (Smith *et al.*, 1999). Carbon dioxide, of course, is a GHG but, if the biomass fuel is harvested renewably, it does not cause a net greenhouse effect because it is captured during regrowth. Because they derive from agriculture, essentially all crop residues and animal dung are renewably harvested on an annual basis. In addition, a significant, although uncertain, fraction of the woodfuel in South Asia is also harvested renewably (Ravindranath and Hall, 1995). Even so, however, because so much of the carbon is diverted to other GHG, particularly methane, which cause greater warming than carbon dioxide, even renewably harvested biomass fuels have significant greenhouse gas potential.

As a result, although individually small, household stoves are numerous enough to contribute significantly to the GHG inventories of South Asian countries. It is estimated, for example, that Indian household stoves emit about three million tons of methane each year, equivalent in global warming to the CO_2 emissions from the entire transport sector (Mitra and Bhattacharya, 1998; Smith *et al.*, 1999).

The significance of GHG as well as HDP from household solid fuel combustion may offer an opportunity for influencing GHG control strategies, and ensuring that international funds and agreements devoted to reducing GHG emissions also contribute to improved health (Wang and Smith, 1999). Figure 1.6 illustrates the GHG and HDP benefits that could occur, for example, by a shift from solid to gaseous or liquid fuels in the household sector.

1.5.6 Community/Globe etc.

Shifts in technology and/or fuel in the power, transport and industrial sectors, as in the household sector, can result in significant health as well as GHG benefits. It should also be noted, however, that there are also shifts in technology that would achieve only one kind of benefit. For example, a shift from natural gas power to hydropower would reduce GHG with little HDP reduction. A shift from coal to hydro (or gas), however, would achieve both. Thus, to assure a win-win result, it is necessary to carefully choose technologies that will achieve both kinds of benefits (Wang and Smith, 1998).

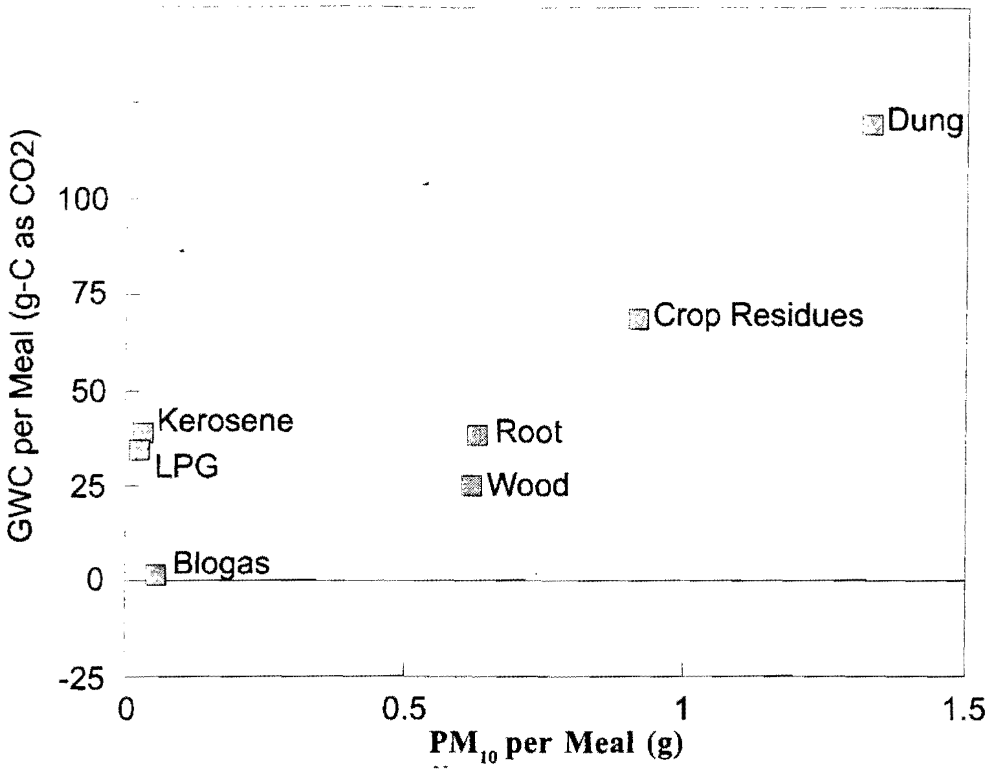


Figure 1.6 **Greenhouse gas and PM$_{10}$ emissions from various houehold fuels illustrating reductions in each that could be attained by fuel switching (source: Smith *et al.*, 1999)**

1.6 CONCLUSION

In South Asia, industrialization, vehicularization and other polluting aspects of modernization are proceeding while many important household and neighbourhood sources still remain important (an example of "risk overlap"). Since these sources are those in and near households, their actual risks may not be well represented by typical ambient urban monitoring schemes, which have been the focus of developed-country control strategies. Thus, to understand the total risk of pollution and the most cost-effective measures available to control its human risk, it is important to consider the exposure implications of different sources, as well as their impacts on urban outdoor pollution levels. Similarly, since pollution can cross boundaries, it is important to consider the relationship among emissions and impacts at household, community, regional and global scales in order to understand the total impact and discover opportunities for efficient control.

1.7 REFERENCES

Akbar, S. 1997. *Particulate Air Pollution and Respiratory Morbidity in Delhi, India.* Ph.D. Thesis. Imperial College of Science, Technology and Medicine, University of London.

AMIS. 1998. *Healthy Cities Air Management Information System.* Version 2.0. World Health Organization, Geneva.

Brimblecombe, P. 1987. *The Big Smoke*. Methuen, London.

Janssen, N. 1998. *Personal Exposure to Airborne Particles*. University of Wageningen, The Netherlands.

Lipfert, F.W. 1994. *Air Pollution and Community Health: A Critical Review and Data Sourcebook*. Van Nostrand Reinhold, New York.

Mitra, A.P and Bhattacharya, S. 1998. *Greenhouse Gas Emissions in India*. Scientific Report No. 11. Centre on Global Change, National Physical Laboratory, New Delhi.

Murray, C.J.L. and Lopez, A.D. 1996. *Global Burden of Disease*. Harvard University, Cambridge.

Ravindranath, N.H. and Hall, D.O. 1995. *Biomass, Energy and Environment: A Developing Country Perspective from India*. Oxford University Press, Oxford.

Smith, K.R. 1987. *Biofuels, Air Pollution and Health*. Plenum, New York.

Smith, K.R. 1993. Fuel combustion, air pollution exposure and health: the situation in developing countries. *Annual Review of Energy and Environment* **18**: 529-566.

Smith, K.R., Apte, M.G., Ma, Y., Wathana, W. and Kulkarni, A. 1994. Air pollution and the energy ladder in Asian cities. *Energy, the International Journal* 19(5): 587-600.

Smith, K.R. 1997. Development, health and the risk transition. In *International Perspectives in Environment, Development, and Health* (ed. by G. Shahi, B.S. Levy, A. Binger, T. Kjellström and R. Lawrence). pp. 51-62. Springer, New York.

Smith, K.R., Uma, R., Kishore, V.V.N., Lata, K., Joshi, V., Zhang, J., Rasmussen, R.A. and Khalil, M.A.K. 1999. *Greenhouse Gases from Small-scale Combustion in Developing Countries: Household Stoves in India*. USEPA, Research Triangle Park NC (forthcoming).

Tsai, F., Smith, K.R., Vichit-Vakakan, N., Ostro, B.D., Chestnut, L.G. and Kungskulniti, N. 1999. Indoor/outdoor $PM_{2.5}$ and PM_{10} in Bangkok, Thailand. *J. Exposure Analysis and Environmental Epidemiology* (forthcoming).

Wang, X. and Smith, K.R. 1998. *Near-Term Health Benefits of Greenhouse Gas Reductions: A Proposed Assessment Method with Application in Two Energy Sectors of China*. WHO EHG/98.12. World Health Organization, Geneva.

Wang, X. and Smith, K.R. 1999. Secondary benefits of greenhouse gas control: health impacts in China. *Environmental Science and Technology* (in press).

Chapter 2

AIR POLLUTION AND HEALTH - STUDIES IN NORTH AMERICA AND EUROPE

M. Lippmann, Professor, New York University School of Medicine,
Tuxedo, NY 10987, USA

Abstract

Studies in the Americas and Europe in recent years on the health effects of ubiquitous air pollutants, such as particulate matter and ozone, have documented responses proportionate to exposures, including excess daily and annual mortality, hospital admissions, lost time from school and work, and reduced lung function. These effects constitute a significant public health challenge in developed countries where more immediate health and safety challenges are under reasonable degrees of control. Ozone levels in many developing countries are currently lower than in the Americas and Europe, and precautionary controls on sources of hydrocarbons and nitrogen dioxide can be instituted to keep them from rising to levels that produce major effects. Levels of particulate matter in the air in cities in developing countries, especially those due to coal smoke, can be high, and the adverse health effects they produce can decrease as emissions are reduced.

2.1 INTRODUCTION

Most of the scientific literature on air pollution and human health has been based on studies of populations exposed to mixtures of ambient air pollutants derived from widely distributed combustion sources, and these will be the focus of this Chapter. There is also an extensive literature on the health effects of air pollutants from specific industrial point sources of toxic and/or cancer-causing chemicals. However, since these are so location and time-specific and since exposures to populations downwind are so variable, it is not possible to offer any meaningful summary of this literature in the space available. There is a similar difficulty in reviewing the health effects of air pollution derived from indoor sources, especially cooking and space heating with non-vented combustion sources, in terms of the highly variable nature and extent of such pollution over space and time in a given community. This discussion is included in Chapter 7 of this publication.

By contrast, the group of air pollutants, known as classical air pollutants by the World Health Organization (WHO) and as criteria air pollutants in the United States, is attributable to relatively large numbers of relatively small sources throughout a community (space heating and motor vehicles) or to power plants with tall stacks whose effluents are more or less uniformly dispersed into the community air. This pollutant group includes some specific primary (directly emitted) gaseous pollutants such as sulphur dioxide (SO_2), nitrogen oxides [(NO_x) emitted primarily as nitric oxide (NO) along with some nitrogen dioxide (NO_2)], and carbon monoxide (CO), as well as lead (in various chemical compounds) as fine particles from the tailpipes of vehicles burning fuel containing organic lead compounds as octane-boosters. The primary particulate matter (PM) category also includes fine carbon particles from vehicles with diesel engines and coarse particles (with aerodynamic diameters > 2.5 μm) from soil and soil-like particles resuspended by winds and motor vehicles, or generated by mechanical forces in operations involving agriculture, construction, demolition and various industries.

The classical pollutant group also covers secondary pollutants that are formed in the ambient air by chemical and photochemical reactions of primary pollutants. These reactions include oxidation of NO to NO_2, reactions of hydrocarbon vapours with NO_2 leading to the formation of oxidant radicals and ozone (O_3), a highly reactive vapour, and the oxidation of SO_2 and NO_2 to produce ultrafine sulphuric acid aerosol (H_2SO_4) and nitric acid vapour (HNO_3). Further atmospheric reactions of the strong acids with ammonia vapour (NH_3) of biogenic origin, followed by aggregation of the ultrafine sulphate (SO_4^+) and nitrate (NO_3^-) particles leads to the accumulation of the fine SO_4^+ and NO_3^- particles that are closely associated with atmospheric haze and acid rain. As secondary pollutants formed continuously and gradually across large geographic areas, O_3 and $PM_{2.5}$ (fine particles with aerodynamic diameters $<$ 2.5 μm) are the most uniformly distributed of the classical pollutants.

Current knowledge about the health effects of the classical air pollutants comes from a variety of sources. The best evidence for SO_2 comes from controlled exposure studies in human volunteers, demonstrating that people with asthma are especially responsive, and may have acute respiratory responses (bronchoconstriction) after brief exposures (as low as 0.25 ppm). For CO, the most sensitive members of the population are cardiovascular patients with angina. For such people, controlled exposures that elevate concentrations of carboxyhemoglobin in the blood to kjroughly 3 per cent have been found to reduce the time to exercise induced angina and to cause characteristic changes in their electrocardiograms.

For lead, the clearest evidence for adverse health effects at low levels comes from epidemiological studies, and includes elevated blood pressure in adults and developmental abnormalities in children. For NO_2, there are no clearly established health effects associated with ambient levels, only associations between ambient NO_2 and elevated mortality and morbidity in large population studies. Since NO_2 may simply be serving as a surrogate measure of "dirty air" in these studies, the jury is still out on a direct role for NO_2.

On the other hand, NO_2 may well be one of the most important of the classical pollutants in relation to the much more substantial effects that have been attributed to exposures to O_3 and $PM_{2.5}$. This is because NO_2 is an essential ingredient (along with hydrocarbon vapours and sunlight) in the photochemical reaction sequences leading to the formation of both O_3 and organic fine particles. Furthermore, the oxidants produced in the photochemical reactions accelerate the transformation of the weak acid vapours (SO_2 and NO_2) into strong acids and their particulate ammonium salts within the $PM_{2.5}$ fraction.

Most of the following in this Chapter is devoted to a summary review of a broad array of current knowledge on the health effects of the most influential of the classical air pollutants, i.e., O_3 and fine particles.

2.2 HEALTH EFFECTS OF OZONE (O_3)

A great deal is known about some of the health effects of O_3. However, much of what is known relates to transient, apparently reversible effects that follow acute exposures lasting from 5 minutes to 6.6 hours. These effects include respiratory effects (such as changes in lung capacity, flow resistance, epithelial permeability, and reactivity to broncho-active challenges). These effects can be observed within the first few hours after the start of the exposure and may persist for many hours or days after the exposure ceases. Repetitive daily exposures over several days or weeks can exacerbate and prolong these transient effects. There has been controversy about the health significance of such effects and whether such effects are sufficiently adverse to serve as a basis for an air quality standard (Lippmann, 1988, 1991, 1993; EPA, 1996a).

Decrements in respiratory function (such as forced vital capacity (FVC) and forced expiratory volume in the first second of a vital capacity manoeuvre (FEV_1)) fall into the category where adversity begins at some specific level of pollutant-associated change. However, there are clear differences of opinion on what the threshold of adversity ought to be.

It is known that single O_3 exposures to healthy non-smoking young adults at concentrations in the range of 80-200 ppb produce a complex array of respiratory effects. These include decreases in respiratory function and athletic performance, and increases in symptoms (airway reactivity, neutrôphil content in lung lavage, and rate of mucociliary particle clearance) (Lippmann, 1988, 1991). Table 2.1 shows that decreases in respiratory function (mean FEV_1 decrements of greater than 5 per cent) have been seen at 100 ppb of O_3 in ambient air for children at summer camps and for adults engaged in outdoor exercise for only 30 minutes.

Table 2.1 Population-based decrements in respiratory function associated with exposure to ozone in ambient air

	Per cent decrement at 120 ppb O_3			
	Camp children[a]		Adult exercisers[b]	
Functional index	Mean	90th percentile	Mean	90th percentile
Forced vital capacity	5	14	5	16
Forced expiratory volume in 1 s	8	19	4	12
FEF_{25-75}[c]	11	33	16	39
PEFR[d]	17	42	13	36

Source: Lippmann (1991)

[a] 93 children at Fairview Lake, NJ, YMCA summer camp, 1984.
[b] 30 non-smoking healthy adults at Tuxedo, NY, 1985.
[c] Forced expiratory flow rate between 25% and 75% of vital capacity.
[d] Peak expiratory flow rate.

Further research will be needed to establish the interrelationships between small transient decreases in lung function, such as FEV_1, peak expiratory flow rate (PEFR), and mucociliary clearance rates, which may not in themselves be adverse effects, and more clinically relevant changes (symptoms, performance, reactivity, permeability and neutrophil counts). The latter may be more closely associated with the accumulation or progression of chronic lung damage.

The clearest evidence that current US peak ambient levels of O_3 are closely associated with adverse health effects in human populations comes from epidemiological studies focused on acute responses. The 1997 revision to the US O_3 Standard (see Table 2.2) relied heavily (for its quantitative basis) on a study of emergency hospital admissions for asthma in New York City, and its consistency with other time-series studies of hospital admissions for respiratory diseases. However, other acute responses, while less firmly established on quantitative bases, are also occurring. In order to put them in perspective, Dr. George Thurston of NYU prepared a graphic presentation showing the extent of related human responses based on the exposure-response relationships established in a variety of published studies. For New York City, as shown in Figure 2.1, a variety of human health responses to ambient ozone exposures could be avoided by full implementation of the 1997 US O_3 Standard of 80 ppb averaged over 8 hrs. The extent of effects avoided on a national scale would be much larger (Thurston, 1997).

Table 2.2 1997 Revisions: US National Ambient Air Quality Standards (NAAQS)

I. Ozone (Revision of NAAQS Set in 1979 and reaffirmed in 1993)

	1979 NAAQS	1997 NAAQS
Daily Concentration Limit - ppb	120	80
Averaging Time	maximum - 1 hr avg.	maximum - 8 hr. avg.
Basis for Excessive Concentration	4th highest over 3 yr period	3 yr avg. of 4th highest in each yr
Equivalent Stringency for 1 hr max in New Format - ppb	~ 90	
Number of U.S. Counties expected to exceed NAAQS	106	280
Number of People in Counties exceeding NAAQS	74×10^6	113×10^6

II. Particulate Matter (Revision of NAAQS Set in 1987)

	1987 NAAQS	1997 NAAQS	
Index Pollutant	PM_{10}	PM_{10}	$PM_{2.5}$
Annual Avg. Concentration Limit - µg/m³	50	50	15
Daily Concentration Limit - µg/m³	150	150	65
Basis for Excessive Daily Concentration	4th highest over 3 yr period	> 99th percentile avg. over 3 yrs	> 98th percentile avg. over 3 yrs
Number of U.S. Counties expected to exceed NAAQS	41	14	~ 150
Number of People in Counties exceeding NAAQS	29×10^6	$\sim 9 \times 10^6$	$\sim 68 \times 10^6$

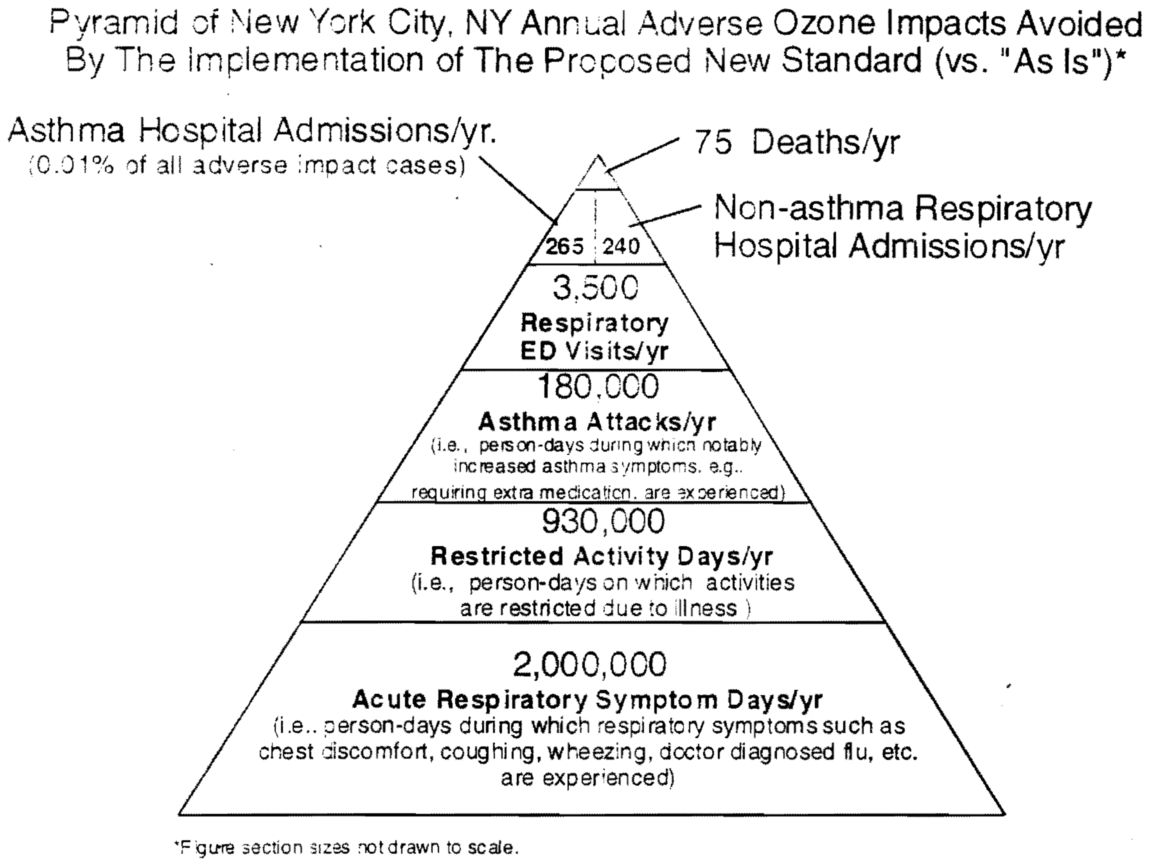


Figure 2.1 **Pyramid summarizing the adverse effects of ambient O_3 in New York City that can be averted by reduction of mid-1990s levels to those meeting the 1997 NAAQS revision (data assembled by Dr. G.D. Thurston for testimony to US Senate Committee on Public Works)**

The plausibility of accelerated ageing of the human lung from chronic O_3 exposure is greatly enhanced by the results of sub-chronic animal exposure studies at near ambient O_3 concentrations in monkeys (Tyler *et al.*, 1988; Hyde *et al.*, 1989). The monkey exposures related to confined animals with little opportunity for heavy exercise. Thus, humans who are active outdoors during the warmer months may have greater effective O_3 exposures than the test animals. Finally, humans are exposed to O_3 in ambient mixtures. The enhancement of the characteristic O_3 responses by other ambient air constituents has been seen in short-term exposure studies in humans and animals. This may also contribute towards the accumulation of chronic lung damage from long-term exposures to ambient air containing O_3.

Although the results of epidemiological and autopsy studies are strongly suggestive of serious health effects, they have been found wanting as a basis for standards setting (EPA, 1996a). Scepticism centres on the uncertainty of the exposure characterization of the populations and the lack of control of confounding factors. Some of these limitations are inherent in large-scale epidemiologic studies. Others can be addressed in more carefully focused study protocols. The lack of a more definitive database on the chronic effects of ambient O_3 exposures on humans is a serious failing. The potential impacts of such exposures on public health deserve serious scrutiny and, if they turn out to be substantial, strong corrective action. Further controls on ambient O_3 exposure in developed countries will be extraordinarily expensive and will need to be very well justified. However, precautionary controls on sources of hydrocarbons and nitrogen dioxide in developing countries can be instituted to keep them from rising to levels that produce major effects.

2.3 HEALTH EFFECTS OF PARTICULATE MATTER (PM)

In Europe, and elsewhere in the eastern hemisphere, particulate pollution has historically been measured as black smoke (BS) in terms of the optical density of stain caused by particles collected on a filter disc. However, it has been expressed in gravimetric terms ($\mu g/m^3$) based on standardized calibration factors. By contrast, US standards have specified direct gravimetric analyses of filter samples collected by a reference sampler built to match specific physical dimensions or performance criteria.

In the US the initial PM Standard, established in 1971, used total suspended particulate matter (TSP) as the index pollutant. The PM Standard was revised in 1987, replacing TSP as the index pollutant with PM_{10}. The 1997 US PM Standard retained somewhat relaxed PM_{10} limits, but added new annual and 24-hour limits for $PM_{2.5}$, as summarized in Table 2.2.

While justifications for the specific measurement techniques that have been used have generally been based on demonstrated significant quantitative associations between the measured quantity and human mortality, morbidity or lung function differences, established biological mechanisms that could account for these associations are lacking, and there is too little information on the relative toxicities of the myriad specific constituents of airborne PM. In addition to chemical composition, airborne PM also varies in particle size distribution, which affects the number of particles that reach target sites as well as the particle surface area. To date, there are no US Standards for the chemical constituents in PM (other than lead) or for the number of particles or surface concentrations of the PM. However, one recent study indicates that number of fine particles per unit volume of air may correlate better with effects than does the mass of fine particulate matter per unit volume of air (Peters *et al.*, 1997a,b).

A broad variety of processes produces PM in the ambient air, and there is an extensive body of literature that demonstrates that there are statistically significant associations between the concentrations of airborne PM and the rates of mortality and morbidity in human populations. In those studies that reported on associations between health effects and more than one mass concentration, the strength of the association generally improves as one goes from total suspended particulate matter (TSP) to thoracic particulate matter, such as PM_{10}, to $PM_{2.5}$. The influence of a sampling system inlet on the sample mass collected is illustrated in Figure 2.2. The different sampling instruments sample different size groups of particulate matter. This Figure also shows that particles have a bimodal distribution in air, with a considerable mass of coarse particles, and fine particles.

The $PM_{2.5}$ distinction, while nominally based on particle size, is in reality a means of measuring the gravimetric concentration of several specific chemically distinctive classes of particles that are emitted into, e.g., diesel exhaust or formed within the ambient air. These include the carbonaceous particles formed during the photochemical reaction sequence that also leads to ozone formation, as well as the sulphur and nitrogen oxide particles resulting from the oxidation of SO_2 and NO_x vapours released during fuel combustion and their reaction products.

The coarse particle fraction is largely composed of soil and mineral ash that are mechanically dispersed into the air. Both the fine and coarse fractions are chemically complex mixtures. To the extent that they are in equilibrium in the ambient air, it is a dynamic equilibrium in which they enter the air at approximately 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 PM are effectively depleted by rainout and washout. Further elaboration of these distinctions is provided in Table 2.3.

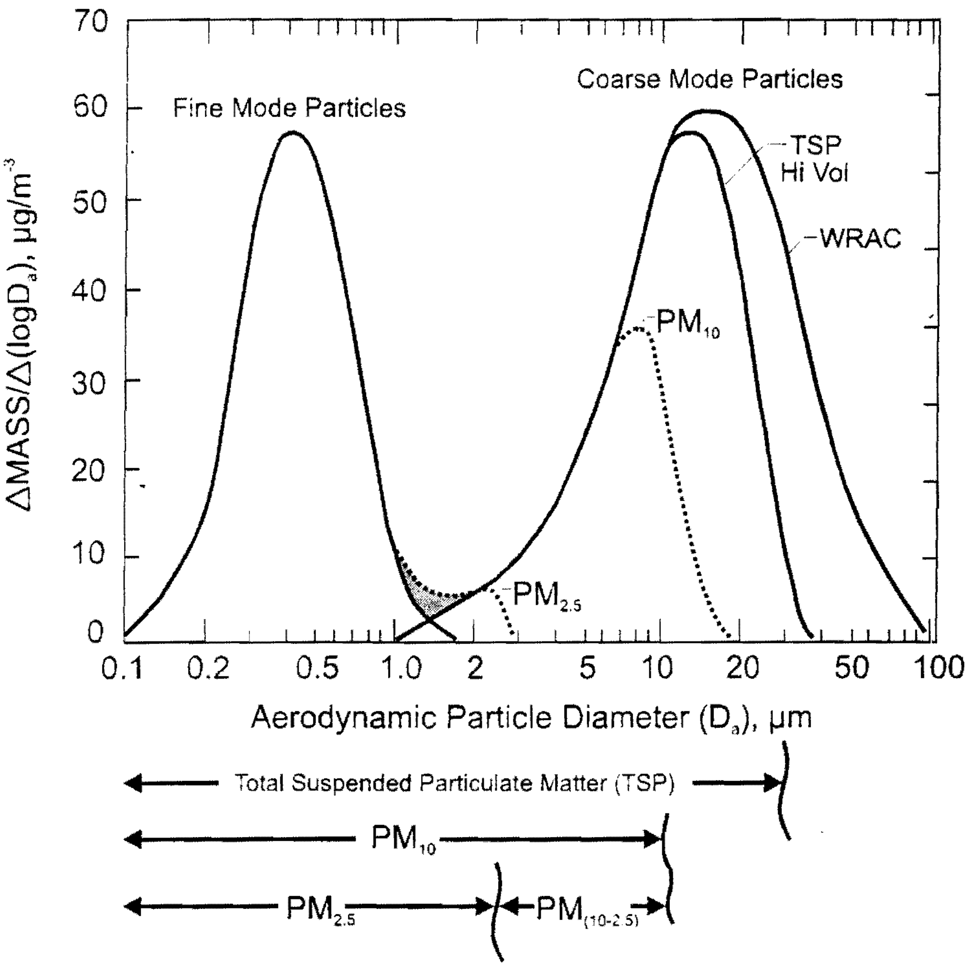


Figure 2.2 Representative example of a mass distribution of ambient PM as function of aerodynamic particle diameter. A wide ranging aerosol classifier (WRAC) provides an estimate of the full coarse mode distribution. Inlet restriction of the TSP high volume sampler, the PM$_{10}$ sampler, and the PM$_{2.5}$ sampler reduce the integral mass reaching the sampling filter

Table 2.3 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, PNAs); metals (e.g., Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); particle-bound water	Resuspended dusts (e.g., soil dust, street dust); coal and oil fly ash; metal oxides of crustal elements (Si, Al, Ti, Fe); $CaCO_3$, NaCl, sea salt; pollen, mold spores; plant/ animal fragments; tyre 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 kilometre

Source: EPA (1996b)

There is an absence of a detailed understanding of the specific chemical components responsible for the health effects associated with exposures to ambient PM. However, there is a large and consistent body of epidemiological evidence associating ambient air PM with mortality and morbidity that cannot be explained by potential confounders such as other pollutants, aeroallergens, or ambient temperature or humidity. Consequently, the US has established standards based solely on mass concentrations within certain prescribed size fractions (see Table 2.2).

As indicated in Table 2.3, fine and coarse particles generally have distinct sources and formation mechanisms, although there may be some overlap. Although some directly emitted particles are found in the fine fraction, particles formed secondarily from gases dominate the fine fraction mass.

The acute mortality risks for PM_{10} are relatively insensitive to the concentrations of SO_2, NO_2, CO, and O_3. The results are also coherent, in that the relative risks for respiratory mortality are greater than for total mortality, and the relative risks for the less serious symptoms are higher than those for mortality and hospital admissions.

While there is mounting evidence that short-term responses are associated with short-term peaks in PM_{10} pollution, the public health implications of this evidence are not yet fully clear. Key questions remain, including:

- Which specific components of the fine particle fraction ($PM_{2.5}$) and coarse particle fraction of PM_{10} are most influential in producing the responses?
- Do the effects of the PM_{10} depend on co-exposure to irritant vapours, such as ozone, sulphur dioxide, or nitrogen oxides?
- What influences do multiple day pollution episode exposures have on daily responses and response lags?
- Does long-term chronic exposure predispose sensitive individuals being "harvested" on peak pollution days?
- How much of the excess daily mortality is associated with life-shortening measured in days or weeks *vs*. months, years or decades?

The last question above is a critical one in terms of the public health impact of excess daily mortality. If, in fact, the bulk of the excess daily mortality were due to "harvesting" of terminally ill people who would have died within a few days, then the public health impact would be much less than if it led to prompt mortality among acutely ill persons who, if they did not die then, would have recovered and lived productive lives for years or decades longer.

Pope *et al.* (1995) linked ambient air pollution data from 151 US metropolitan areas in 1980 with individual risk factors in 552,138 adults who resided in these areas when enrolled in prospective study in 1982. Death records until December, 1989 were analysed. Exposure to SO_4^- and $PM_{2.5}$ pollution was estimated from national databases. The relationships of air pollution to all-cause, lung cancer, and cardiopulmonary mortality were examined using analyses that controlled for smoking, education, and other personal risk factors. Adjusted relative risk ratios (and 95 per cent confidence intervals) of all-cause mortality for the most polluted areas compared with the least polluted equaled 1.15 (1.09-1.22) and 1.17 (1.09-1.26) when using sulphate and $PM_{2.5}$, respectively. Particulate air pollution was associated with cardiopulmonary and lung cancer deaths, but not with deaths due to other causes. The mean life-shortening in this study was between one and one-half and two years. The results were similar to those found by Dockery *et al.* (1993) in a prospective cohort study in six US cities, as well as those of previous cross-sectional studies of Ozkaynak and Thurston (1987) and Lave and Seskin (1970). The Pope *et al.* (1995) and Dockery *et al.* (1993) results thus indicate that the concerns raised about the credibility of the earlier results, based on their inability to control for potentially confounding factors such as smoking and socio-economic variables at an individual level, can be eased.

If mean life span shortening is of the order of two years, then many individuals in the population have lives shortened by many years, and there is excess mortality associated with fine particle exposure greater than that implied by the cumulative results of the time-series studies of daily mortality. Excess mortality is clearly an adverse effect, and the epidemiological evidence is consistent with a linear non-threshold response for the population as a whole.

2.4 HEALTH EFFECTS OF DIESEL ENGINE EXHAUST

Diesel engine exhaust, which contributes a relatively small fraction of the $PM_{2.5}$ in the US, and a larger fraction in Europe, has been of special concern because of its odour and its possible effects on cancer rates. In recent years diesel engines are much more prevalent in light-duty applications in Europe than in the US, largely because of the much higher fuel prices. In addition, European concerns for emissions have focused more on global warming than on particles, and diesels emit less carbon dioxide than equivalent gasoline engines. As a result, the development of light-duty diesel engines with performance characteristics acceptable to individual consumers occurred primarily in Europe. Although still called "diesels," new technology compression ignition engines hardly resemble diesel engines of the past. Tailpipe emissions of soot particles and toxic gases are rapidly approaching the levels of those from gasoline-powered spark ignition engines, while fuel economy and durability continue to increase.

There are concerns for the potential adverse health effects of diesel exhaust because it contains trace amounts of toxic compounds and because occupational exposures are common and environmental exposures are widespread. Diesel exhaust is a ubiquitous component of air pollution. All populations living in developed countries are exposed frequently to diesel exhaust at some concentration. While the potential for diesel exhaust to present a health hazard has been known for several decades, the current emphasis dates to the late 1970s when extracts from diesel soot were found to be mutagenic to bacteria. Research during the last 20 years focused on the potential contribution of diesel exhaust to human lung cancer risk.

Concern for the cancer risk of diesel exhaust has centred on the organic hydrocarbons associated with soot particles. Soot consists of aggregates of spherical primary particles that form in the combustion chamber, grow by agglomeration, and are emitted as clusters having average particle diameters ranging from 0.1 to 0.5 μm (Cheng *et al.*, 1984). As released to the environment, the portion of the mass of diesel soot consisting of adsorbed organic matter can range from 5-90 per cent (Johnson, 1988). Values of 10-15 per cent are representative of modern engines under most operating conditions. The size of diesel soot particles makes it readily respirable. Approximately 20-30 per cent of the inhaled particles in diluted exhaust would be expected to deposit in the lungs and airways of humans (Snipes, 1989).

The partitioning of compounds between the gas and particulate phases of exhaust depends on the vapour pressure, temperature and concentration of each chemical in the exhaust. Although the potential effects of hydrocarbon vapours are not well understood, little concern has been raised for the long-term health effects of these compounds. Lung tumours are not induced in rats by chronic exposure to high concentrations of diesel exhaust if the exhaust is filtered and animals are exposed to only the gas and vapour phases.

The volatile compounds in diesel exhausts are not without adverse effects. People exposed to high concentrations of diesel exhaust complain about objectionable odour, headache, nausea and eye irritation, symptoms thought to be primarily associated with the gas and vapour-phase constituents. It is not known if there is any link between these transient symptoms and other health effects. The US EPA estimated that the US annual average concentration of airborne diesel soot in 1990 was 1.8 $\mu g/m^3$, and that the urban and rural averages were 2.0 and 1.1 $\mu g/m^3$, respectively (EPA, 1993). The urban, rural and nationwide average concentrations were predicted to fall to 0.4, 0.2, and 0.4 $\mu g/m^3$, respectively, by 2010.

The most relevant information on the human health risks from exposures to potential toxicants is information obtained from studies of humans, assuming that the information is adequate for establishing exposure-effects relationships. Numerous epidemiological studies of the relationship between diesel exhaust exposure and lung cancer have been reported, with some studies focusing specifically on diesel exhaust and others on occupations receiving substantial diesel exhaust exposures. This body of information, while large, is weakened by the lack of direct measures of the exhaust exposures of the populations studied. The weight of the epidemiological evidence suggests a positive effect of small magnitude, but confidence in conclusions drawn from this largely circumstantial evidence is eroded by uncertainties regarding exposure and potential confounding by cigarette smoking and other exposures. Contemporary reviews of this information have been published by EPA (1994), HEI (1995) and Cal. EPA (1997).

In the absence of definitive data from humans, hazard characterization and risk assessment typically use data from animals exposed experimentally to the agent in question. Regarding the carcinogenicity of diesel exhaust however, results from animals have not proved to be very helpful because essentially the same lung tumour response is obtained with pure carbon soot and other inert particles as with diesel exhaust at comparable mass concentrations (Mauderly, 1999).

While it is plausible that diesel soot presents a carcinogenic hazard, there is little consensus of opinion regarding the existence or magnitude of lung cancer risk under current occupational or environmental exposure conditions. The aggregate epidemiological data suggest that occupational exposures may slightly increase lung cancer risk, but do not provide a basis for quantitative estimates of risk with confidence. Overall, it is reasonable to assume that, if the inhalation of airborne mutagenic material is attended by cancer risk, then environmental diesel soot contributes in some measure to that pool of material and thus to the risk. It is also reasonable to assume that cancer risk might parallel the deposited dose of inhaled mutagenic material, depending on the bio-availability of the material and its mutagenic potency in humans.

2.5 DISCUSSION AND CONCLUSIONS

Air pollutants can adversely affect human health in a number of ways, both directly and indirectly. Direct effects in downwind populations can range from acute intoxication and prompt mortality from peak point source discharges, as in the Bhopal, India, methyl isocyanate release, to delayed developmental deficits resulting from chronic lead exposure to people living near the Port Pirie lead smelter in South Australia. Indirect effects can include those resulting from a primary pollutant such as NO_x from combustion sources being essential to O_3 formation in the atmosphere, with O_3 having the largest impact on the health effects that result. Indirect affects can also result from acidic sulphur and nitrogen compounds that deposit on soil and leach toxic metals from the soil that bio-accumulate in food crops and flesh that are ingested. Such diverse and complex aspects of air pollution and health are too broad for discussion in this Chapter. Rather, this review has been limited to the effects of the most ubiquitous air pollutants in both developed and developing countries that are attributable to transportation and space heating sources, i.e., ozone (O_3), particulate matter (PM) and diesel engine exhaust.

Ozone, resulting from atmospheric chemical reactions involving hydrocarbon vapours, nitrogen dioxide (NO_2) and sunlight causes reduced lung function, airways' inflammation, increased usage of physicians and hospitals, and lost time from school and work following exposures that frequently occur in major urban areas of North and South America. Concentrations of O_3 in South Asia are lower at the present time, but can be expected to rise in proportion to increasing motor vehicle use. This is further discussed in the following chapter, and in Chapter 8.

The concentrations of thoracic particulate matter (PM_{10}) and fine particulate matter ($PM_{2.5}$) in ambient air are significantly associated with excess daily mortality, annual mortality, and daily hospital admissions for respiratory and cardiovascular causes, as well as increased rates of bronchitis in children, lost time from work and school, and reduced lung function following exposures at current levels in the Americas and Western Europe. Concentrations of PM_{10} and $PM_{2.5}$ are much higher in many parts of South Asia than they are in the US or Europe, and studies of mortality rates and lung function in South Asia have produced concordant findings. There is no evidence for a threshold in any of the PM-associated health effects and it is therefore reasonable to expect that reductions in PM exposures in South Asia will result in proportionate reductions in the health effects associated with PM, as discussed in the following chapter.

The concentrations of diesel exhaust are much more spatially variable than those of O_3 or PM, and their odour and nuisance effects, as well as their potential for producing lung cancer, will be much greater for those living or working near major roadways than for people further away. At this point in time, strategies to reduce diesel exhaust pollution should be incorporated into overall strategies to reduce PM pollution.

2.6 ACKNOWLEDGEMENTS

This research was undertaken, in part, by the author as part of a Centre programme supported by Grant ES 00260 from the National Institute of Environmental Health Sciences. It has also been based, in part, on material contained in the chapters on ambient particulate matter and ozone written by the author and on the chapter on diesel exhaust written by Dr. Joe L. Mauderly for the second edition of Environmental Toxicants-Human Exposures and Their Health Effects (M. Lippmann (ed.)) being published by Wiley in 1999.

2.7 REFERENCES

Cal EPA (California Environmental Protection Agency). 1997. *Health Risk Assessment for Diesel Exhaust*. Office of Environmental Health Hazard Assessment.

Cheng, Y.S., Yeh, H.C., Mauderly, J.L. and Mokler, B.V. 1984. Characterization of diesel exhaust in a chronic inhalation study. *American Industrial Hygiene Association Journal* **45**: 547-555.

Dockery, D.W., Pope, C.A. III, Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G. Jr. and Speizer, F.E. 1993. An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine* **329**: 1753-1759.

EPA (U.S. Environmental Protection Agency). 1993. *Motor Vehicle-Related Air Toxics Study*. EPA 420-R-93-005. Ann Arbor, MI: Office of Mobile Sources, Emission Planning and Strategies Division.

EPA (U.S. Environmental Protection Agency). 1994. *Health Assessment Document for Diesel Emissions*. Volumes **I** and **II**. EPA/600/8-90/057Ba. Office of Research and Development, Washington D.C.

EPA (U.S. Environmental Protection Agency). 1996a. *Air Quality Criteria for Ozone and Related Photochemical Oxidants*. EPA/600/P-93/004F. National Center for Environmental Assessment, Research Triangle Park, NC.

EPA (U.S. Environmental Protection Agency). 1996b. *Air Quality Criteria for Particulate Matter*. EPA/600/P-95/001F. National Center for Environmental Assessment, Research Triangle Park, NC.

HEI (Health Effects Institute). 1995. *Diesel Exhaust: A Critical Analysis of Emissions, Exposure and Health Effects*. A Special Report of the Institute's Diesel Working Group. Health Effects Institute, Cambridge, M.D.

Hyde, D.M., Plopper, C.G., Harkema, J.R., St. George, J.A., Tyler, W.S. and Dungworth, D.L. 1989. Ozone-induced structural changes in monkey respiratory system. In *Atmospheric Ozone Research and Its Policy Implications* (ed. by T. Schneider, S.D. Lee, G.J.R. Wolters and L.D. Grant). pp. 525-532. Elsevier, Nijmegen, The Netherlands.

Johnson, J.H. 1988. Automotive emissions. In *Air Pollution, The Automobile and Public Health* (ed. by A.Y. Watson, R.R. Bates and D. Kennedy). National Academy Press, Washington, DC. pp. 39-75.

Lave, L.B. and Seskin, E.P. 1970. Air pollution and human health. *Science* **169**: 723-733.

Lippmann, M. 1988. Health significance of pulmonary function responses to airborne irritants. *Journal of the Atr Pollution Control Association* **38**: 881-887.

Lippmann, M. 1991. Health effects of tropospheric ozone. *Environmental Science and Technology* **25**(12): 1954-1962.

Lippmann, M. 1993. Health effects of tropospheric ozone: implications of recent research findings to ambient air quality standards. *Journal of Exposure Analysis and Environmental Epidemiology* **3**: 103-129.

Mauderly, J.L. 1999. Diesel exhaust. In *Toxicants: Human Exposures and Their Health Effects* - 2nd Ed. (ed. by M. Lippmann). Wiley, New York.

Ozkaynak, H. and Thurston, G.D. 1987. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. *Risk Analysis* **7**: 449-461.

Peters, A., Döring, A., Wichmann, H.E. and Koenig, W. 1997b. Increased plasma viscosity during an air pollution episode: a link to mortality? *Lancet* **349**: 1582-1587.

Peters, A., Wichmann, H.E., Tuch, T., Heinrich, J. and Heyder, J. 1997a. Respiratory effects are associated with the number of ultrafine particles. *American Journal of Respiratory and Critical Care Medicine* **155**: 1376-1383.

Pope, C.A. III, Thun, M.J., Namboodiri, M., Dockery, D.W., Evans, J.S., Speizer, F.E. and Heath, C.W. Jr. 1995. Particulate air pollution is a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine* **151**: 669-674.

Snipes, M.B. 1989. Long-term retention and clearance of particles inhaled by mammalian species. *Critical Reviews in Toxicology* **20**: 175-211.

Thurston, G.D. 1997. Testimony submitted to U.S. Senate Committee on Environment and Public Works, Subcommittee on Clean Air, Wetlands, Private Property, and Nuclear Safety.

Tyler, W.S., Tyler, N.K., Last, J.A., Gillespie, M.J. and Barstow, T.J. 1988. Comparison of daily and seasonal exposures of young monkeys to ozone. *Toxicology* **50**: 131-144.

Chapter 3

AIR POLLUTION AND HEALTH IN DEVELOPING COUNTRIES: REVIEW OF EPIDEMIOLOGICAL EVIDENCE

Isabelle Romieu, M.D., M.P.H., Sc.D[*]
Mauricio Hernandez, M.D., Sc.D.[**]

[*]Pan American Health Organization
[**]Centro de Investigaciones en Saud Poblacional, Instituto Nacional de Salud Publica, Cuernavaca, Mexico; and Department of Environmental and Occupational Health, The Rollins School of Public Health, Emory University

Abstract

The adverse health effects of air pollution are now well recognized. Review of evidence from developed nations substantiates the harmful effects of air pollutants, even at levels considerably lower than those observed in many megacities in the developed world. Although air pollutants are likely to have similar adverse effects on different populations, differences regarding the level exposure, the population structure, the nutritional status and the lifestyle observed in developing nations suggest that adverse effects of air pollution may be even larger than those observed in developed nations. In this Chapter, the epidemiological studies are reviewed which describe health effects of various air pollutants (including particulate mass, sulphur dioxide, nitrogen dioxide, ozone, carbon monoxide and lead) in different cities of the developing world. Reports from developing countries show similar effects as those observed in other parts of the World, adding support for a causal relationship between air pollution and health effects. Various studies documented increased mortality and visits for respiratory emergencies associated with particulate pollution (particularly with particulate smaller than 10 mm and than 2.5 mm). Higher frequencies of respiratory symptoms and low pulmonary function in subjects exposed to particulate have also been reported. Asthmatic populations appear to be more susceptible to the impact of particulate and SO_2 *exposure. Most of the studies on the health effects of* O_3 *have focused on short-term exposure and have documented increases in emergency visits and hospital admissions due to respiratory diseases, increase in respiratory symptoms and temporary lung function decrements. Time series evaluating associations between* O_3 *and daily mortality have been inconsistent. For CO, few studies have been reported; however, limited data suggest that CO exposure is prevalent and may be associated with intrauterine death. Recent trends indicate that lead exposure is decreasing due to changes in gasoline formulation. However, other sources remain uncontrolled and health effects in cognitive development have been reported.*

There is concern about the health effects of the high levels of air pollution observed in many megacities of the developing world; moreover, because developing countries are trapped in the trade-offs of economic growth and environmental protection, it is likely that this problem will continue to grow. There is an urgent need for the implementation of control programmes to reduce levels of greenhouse gas, particulate and other pollutant emissions. To be effective, these programmes should include the participation of the different stakeholders and initiate activities to identify and characterize air pollution problems, and to estimate potential health impacts. The impact of each pollutant on human health has proved difficult to establish; which pollutant to target and how to reduce exposure should consider local conditions and should be a matter of careful discussion given the high cost associated with environmental interventions.

In many developing countries economic growth without protection of the environment has resulted in widespread environmental damage, creating new emerging environmental problems. Populations in urban areas are at risk of suffering adverse health effects with rising problems of severe air and water pollution.

Although air pollution is recognized as a recent emerging public health problem, most developing nations do not have data to evaluate its real dimension. The fact that air pollution coexists with other important public health problems, such as low immunization coverage, malnutrition or sanitation deficiencies - which are given higher priority in circumstances where economical resources are scarce - has delayed the actions needed to adequately assess, evaluate and control air pollution in most urban conglomerates in the developing world.

Over the past year various health indicators have being used to evaluate the effects of air pollution (Samet and Speizer, 1993). Some indicators, such as lung function tests, require special training and are obtained mostly in research or clinical settings. Other indicators are related to routinely collected health and vital statistics, such as total mortality, mortality by cause, the number of daily visits to emergency services or the number of hospitalizations for air pollution potentially related diseases. Many of these records are collected with reasonable quality and are readily available in most developed countries. However, in developing nations the completeness and accuracy of these records are important limitations for their use as reliable indicators to assess the health effects of air pollution. Nevertheless, the limited data available suggest that air pollution may have serious health effects in many developing countries.

Health effects of air pollution have been broadly studied in developed nations (see Chapter 2). Review of the current evidence substantiates the harmful effects of air pollutants, even at levels considerably lower than those commonly registered in many cities in the developed world (Lippmann, 1993; Dockery and Pope, 1994; Pope *et al.*, 1995; American Thoracic Society, 1996; EPA, 1996). For example, during 1991-1994, concentrations of Total Suspended Particulates (TSP) in Delhi were nearly five times that of the WHO annual average guideline (Cropper *et al.*, 1997). Although air pollutants are likely to have similar adverse effects on different human populations, the range of exposure, co-exposure to different pollutant mixtures, the population structure, the nutritional status, and the lifestyle observed in developing nations, suggest that potential health effects of air pollution may be even greater than those reported for developed nations. In this Chapter the epidemiological studies describing health effects of air pollution in developing nations are reviewed briefly. The review was restricted to studies that have evaluated the health effects in relation to exposure to the criteria air pollutants (particulate matter, sulphur dioxide, nitrogen dioxide, ozone, carbon monoxide, and lead), as information regarding atmospheric levels of these pollutants is becoming available for major cities in the developing world (Romieu *et al.*, 1990). Other air pollutants, such as volatile organic compounds, have adverse health effects, but little is known of their ambient levels in developing countries.

3.1 HEALTH EFFECTS OF PARTICULATE MATTER AND SULPHUR DIOXIDE (SO_2)

Particulates and sulphur oxides result from the combustion of fossil fuel. Depending on the source, the ratio of particulates to SO_2 in the ambient air may vary (American Thoracic Society, 1996). In areas where fossil fuels with high sulphur content are used, especially during the hot season such as in Beijing, China, high levels of SO_2 may be reached.

Particulate matter is a product of many processes: soil erosion, road dust, forest fires, land-clearing fires and agricultural burning (American Thoracic Society, 1996). Particulates range over several orders of magnitude in size and, as discussed in Chapter 2, particulate material of less than 10 microns in size may be inhaled into the respiratory system resulting in adverse health effects. Acute exposure to inhalable particulates can result in loss of lung function, onset of respiratory symptoms, aggravation of existing respiratory conditions and increase susceptibility to infection. These problems may occur to a greater degree in asthmatics, small children and in the elderly with chronic respiratory and cardiovascular diseases.

Exposure to particulates has been consistently associated with: decrease in levels of pulmonary lung function in children and adults with obstructive airways disease; increase of respiratory symptoms; increase in school absenteeism and restricted activity days; increase in physician and emergency room visits for asthma; increase in hospitalizations for respiratory and cardiac conditions; and with premature death in adults with chronic respiratory and cardiovascular diseases (American Thoracic Society, 1996; Dockery and Pope, 1996; Pope and Dockery, 1996).

Current concerns about health effects of airborne particles are largely based on results of recent epidemiological studies suggesting an increase in mortality and morbidity at levels below the current standards, and a stronger (roughly twice that previously reported) and more consistent effect of fine particles (smaller than 2.5 mm) that appear to contain more of the reactive substance potentially linked to health effects (EPA, 1996). Particulate matter is one of the major air pollutants in developing countries where levels frequently exceed current guidelines to protect health (HEI, 1988; WHO, 1993; WHO 1997).

3.1.1 Premature mortality

Air pollution may lead to premature death by exacerbating chronic conditions. Short-term acute exposures may increase the probability that a person with a chronic condition, such as chronic obstructive lung disease or cardiovascular disease, will die.

Recent studies relating to the occurrence of daily deaths (total and by cause) to daily changes in air pollution levels have provided strong evidence of the health effects associated with particulate pollution (Pope *et al.*, 1995; Dockery and Pope, 1996; Pope and Dockery, 1996). Premature mortality has being documented in a wide range of exposures. A pooled estimate of major studies, provided by WHO, suggested that a 10 mg/m^3 increase in PM_{10} will be associated with an increase in daily mortality equal to 0.74 per cent (95 per cent CI 0.62

per cent to 0.86 per cent). Similarly, an increase of 10 mg/m^3 in $PM_{2.5}$ levels will increase daily mortality by 1.5 per cent (95 per cent CI 1.1 per cent to 1.9 per cent) (WHO (in press)). A recent estimate for Delhi in India suggests that an annual reduction of 100 mg/ m^3 in Total Suspended Particulates (TSP) could be associated with a reduction of about 1400 premature deaths per year (Cropper *et al.*, 1997). Results from studies reported in other cities with similar problems are in accordance with these estimates and they suggest that large health benefits may be obtained by reducing particle levels in the air (Figure 3.1). However, several factors need to be considered when comparing results of studies conducted in different parts of the World such as particulate composition and air mixture, exposure profiles and the age structure and characteristics of populations studied (Romieu and Borja-Aburto, 1997).

3.1.2 Morbidity

Most of the studies of emergency visits and hospital admissions for respiratory illnesses conducted in Western countries have reported an increase of 1-4 per cent in relation to a 10 mg/m^3 increase in PM_{10} on the day of the visit or 1 to 2 days before the visit (Dockery and Pope, 1994; Pope *et al.*, 1995). Studies conducted in developing countries to determine the impact of particulate pollution on respiratory emergencies and medical visits have also suggested that increases in air pollution are associated with increasing frequency of visits to medical services (Table 3.1). Particulate and SO_2 exposure during the last trimester of pregnancy have also been related to low birth weight, an important predictor of future performance in children (Yang and Yang, 1994).

Studies related to the evaluation of respiratory health, in general, have observed higher frequency of respiratory symptoms and lower pulmonary functions in subjects exposed to particulates. Asthmatics appear to be more susceptible to the impact of particulate and SO_2 exposure, and an increase in respiratory symptoms and decrease in lung function related to exposure to PM_{10} have been documented (Dockery and Pope, 1994; Pope *et al.*, 1995; American Thoracic Society, 1996).

3.2 HEALTH EFFECTS OF OZONE

Ozone (O_3) is a colourless reactive oxidant that occurs with other photochemical oxidants and fine particles in the complex mixture commonly called "smog", as discussed in Chapter 2. O_3 is a strong oxidant, formed in the ground ambient air by a complex series of reaction involving volatile organic compounds, sunlight and nitrogen oxides (WHO, 1987). The main health concern of exposure to O_3 is its effect on the respiratory system (Table 3.2).

Most of the studies on the health effects of O_3 have focused on short-term exposure. Epidemiological studies have documented a number of acute effects including increases in emergency visits and hospital admissions due to respiratory diseases, increase in respiratory symptoms (such as cough, throat dryness, eye and chest discomfort, thoracic pain and headache) and temporary lung function decrements (Lippmann, 1989; American Thoracic Society, 1996; Nyberg and Pershagen, 1996). It is considered that O_3 is not a cause of asthma; however, O_3 exposure is a risk factor for exacerbation of symptoms in asthmatic subjects. Studies conducted in Mexico City, where O_3 levels frequently and by a large margin exceed the WHO guideline, have documented an increase in asthma-related emergency visits, decrease in peak expiratory flow rate and an increase in respiratory symptoms in asthmatic children (Romieu *et al.*, 1997). Results of time series studies of associations between O_3 and daily mortality have been inconsistent (Loomis *et al.*, 1996). However, recent studies have reported a significant association between ozone and daily mortality counts (Hoek *et al.*, 1997; Burnett *et al.*, 1998; Ponka *et al.*, 1998).

Table 3.1 Health outcomes associated with changes in daily ambient levels of particulate

Health effect indicators	$PM_{2.5}$	PM_{10}
Daily mortality (65 years and over)		
Total mortality		
Change of 5%		50[a]
Change of 10%		100
Change of 20%		200
Respiratory mortality		
Change of 5%		25[b]
Change of 10%		50
Change of 20%		100
Daily respiratory morbidity		
(Emergency visits for respiratory causes among children)		
Change of 5%	40[c]	80[c]
Change of 10%	80	160
Pneumonia		
Change of 5%	10	20
Change of 10%	20	40
Exacerbation of respiratory symptoms in children with moderate asthma		
Change of 5%	-	10[d]
Change of 10%	-	20
Change of 20%	-	40
Peak expiratory flow rate in children with moderate asthma		
Change of 5%		140[d]
Change of 10%		280

Source:
[a] Saldiva *et al.* (1995); Ostro *et al.* (1996); Borja-Aburto *et al.* (1997); Tellez-Rojo *et al.* (1997)
[b] Tellez-Rojo *et al.* (1997)
[c] Ilabaca *et al.* (1998)
[d] Romieu *et al.* (1996)

Because O_3 is a potent oxidant, anti-oxidant supplementation could modulate the impact of ozone exposure on the respiratory tract. Results from recent studies suggest that increasing dietary intake of antioxidant vitamins (beta-carotene, vitamin E and vitamin C) may protect against the acute adverse effects of O_3 exposure (Romieu *et al.*, 1998). This finding is important because micronutrient deficiency is prevalent in many developing countries and is likely to enhance the adverse effect of air pollutant exposure, in particular in population chronically exposed.

As for many other pollutants, there is major concern in relation to the long-term effects of O_3. Many children in developing nations are exposed on a daily basis to high levels of O_3. The health implications of this exposure are still unclear but there is good reason for concern.

O_3 exposure induces inflammatory responses with the result that repeated effects on lung tissue due to long-term exposure to high O_3 could lead to chronic impairment of lung function (Lippmann, 1993).

Table 3.2 Health outcomes associated with changes in peak daily ambient ozone concentration in epidemiological studies

Health effect indicators	Changes in 1-h O_3 (ug/m^3)[a]
Daily morbidity (Upper respiratory illnesses)	
Change of 5%	25[b]
Change of 10%	50
Change of 20%	100
Daily morbidity (Emergency visits for asthma among children)	
Change of 5%	20[c]
Change of 10%	40
Change of 20%	80
Exacerbation of respiratory symptoms in children with moderate asthma	
Change of 5%	30[d]
Change of 10%	60
Change of 20%	120
Peak expiratory flow rate in children with moderate asthma	
Change of 2.5 %	185[d]
Change of 5 %	370

Source:
[a] 1 mg/m3= 0.5 ppb
[b] Tellez-Rojo *et al.* (1997)
[c] Castillejos *et al.* (1995)
[d] Romieu *et al.* (1996); Romieu *et al.* (1997)

3.3 HEALTH EFFECTS OF NITROGEN DIOXIDE

Nitrogen dioxide (NO_2) is formed from emissions of oxides of nitrogen (NO_x). The major sources of man-made emission of NO_2 into the atmosphere are motor vehicles and stationary sources, such as electric utilities and industrial boilers. NO_2 is highly reactive and has been reported to cause bronchitis and pneumonia, and increase susceptibility to respiratory infections (Table 3.3). NO_2 has been shown to affect both the cellular and humoral immune system, and impair the immune response. A review of epidemiological studies suggests that children exposed to NO_2 are at increased risk of respiratory illness (Hasselblad *et al.*, 1992). A recent study (Pereira, 1998) correlated daily counts of intrauterine mortality and air pollution levels in Sao Paulo, Brazil. It reported NO_2 was more significantly associated than other pollutants studied. Although it is difficult to establish a direct causal link in this type of study, the findings strengthen evidence of adverse effects of air pollution and suggest a need for additional studies to improve the understanding of toxic effects.

Table 3.3 Health outcome associated with NO_2 exposure in epidemiological studies

Health effect	Mechanism
Increased incidence of respiratory infections	Reduced efficacy of lung defences
Increased severity of respiratory infections	Reduced efficacy of lung defences
Respiratory symptoms	Airways injury
Reduced lung function	Airways and alveolar (?) injury
Worsening clinical status of persons with asthma, chronic obstructive pulmonary disease or other chronic respiratory conditions	Airways injury

Source: Romieu (1999)

3.4 HEATH EFFECTS OF CARBON MONOXIDE

Carbon monoxide (CO) is one of the most common and widely distributed air pollutants. It is a product of incomplete combustion of carbon-containing materials, in particular fuel.

Carbon monoxide enters the blood stream and reduces the delivery of oxygen to the body's organs and tissues. People who suffer from cardiovascular disease, particularly those with angina or peripheral vascular disease, are much more susceptible to health effects of CO.

Carbon monoxide leads to a decreased oxygen uptake capacity with decreased work capacity under maximal exercise conditions. Inhalation of CO leads to an increased concentration of carboxyhemoglobin (COHB) in the blood. According to available data (Table 3.4), the concentration of COHB in the blood required to induce a decreased oxygen uptake capacity is approximately 5 per cent. An impairment in the ability to judge correctly slight differences in successive short time intervals has been observed at lower COHB levels of 3.2 to 4.2 per cent. The classic symptoms of CO poisoning are headache and dizziness at COHB levels between 10 and 30 per cent, and severe headache, cardiovascular symptoms and malaise at COHB levels higher than about 30 per cent. Above roughly 40 per cent COHB levels, there is considerable risk of coma and death (Romieu, 1999).

Table 3.4 Health effects associated with low-level carbon monoxide exposure, based on COHB levels

Carboxyhemoglobin concentration (%)	Effects
2.3 - 4.3	Decrease (3-7%) in the relation between work time and exhaustion in exercising young healthy adults
2.0 - 4.5	Decrease in exercise capacity in patients with angina (cardio vascular impairment)
5 - 5.5	Decrease in maximal oxygen consumption and exercise in young healthy men during strenous exercise
<5	Vigilance decrement
5 - 7.6	Impairment of vigilance tasks in healthy experimental subjects
5 - 17	Decrease of visual perception, manual dexterity, ability to learn, or performance in complex sensorimotor tasks (e.g driving)

Source: Romieu (1999)

Epidemiological studies relating to CO with daily counts of mortality or hospital admissions need to be interpreted with caution. In contrast with other pollutants, CO measurements from fixed monitors (used for air surveillance) correlate poorly with CO levels measured at personal level. However, various studies in developed countries have documented significant association between daily variations in CO and increase in premature mortality or hospitalizations from congestive heart failure (Schwartz, 1995; Burnett *et al.*, 1998). Few studies have been reported from developing countries. However, limited data suggest that CO exposure is prevalent and may be associated with intrauterine death in Sao Paulo, Brazil (Pereira *et al.*, 1998).

3.5 HEALTH EFFECTS OF LEAD

Lead poisoning is one of the most important problems of environmental and occupational origin, because of its high prevalence and persistence of toxicity in affected populations. Lead poisoning may alter virtually all biochemical processes and organ systems in humans. Lead can interfere with the cardiovascular and reproductive systems, with the blood formation process, vitamin D function, and neurological processes, among others (Howson *et al.*, 1995). Of special concern has been the accumulation of experimental and epidemiological evidence suggesting that lead is a neurotoxin that impairs brain development in children, even at levels that were considered as safe (Needleman and Bellinger, 1991). Studies conducted in Mexico and China have documented similar effects (Munoz *et al.*, 1993; Shen *et al.*, 1996).

The toxic effects associated with chronic low level lead exposure are a major concern, especially as there are no clinical symptoms that will allow the prompt recognition of lead intoxication; yet, lead exposure is preventable through the identification and control of sources of exposure. Beginning in the 1970s, many countries initiated regulatory and legislative efforts to prevent lead exposure. Regulatory actions have been targeted to reduce the use of leaded paints, to eliminate lead from gasoline and to control large industrial point source emissions. These interventions have resulted in important reductions in lead exposure. For example, in Mexico City introduction of unleaded gasoline in 1990 was associated with a decline in lead ambient concentrations from an annual average of 1.2 mg/m^3 to an annual average of 0.2 mg/m^3 in 1993 (Hernandez, 1997), as well as with an estimated decline of 7.6 mg/dl in the mean blood lead of children (Rothenberg *et al.*, 1998). In South Africa (Maresky and Grobler, 1993) reduction of lead content of gasoline, from 1984 to 1990, was also reported to be associated with a significant decrease in blood lead levels from 9.7 mg/dl to 7.2 mg/dl.

Although lead exposure is recognized as an important public health problem, there are few studies published from developing countries (Table 3.5). Furthermore, most published studies have not evaluated exposure among children aged 24 months to 6 years, who are at higher risk of exposure and of suffering health effects of lead exposure. Therefore, the real magnitude of the problem remains unknown.

Control of lead exposure in developing countries will require additional efforts and properly targeted interventions to account for the particular condition in which exposure takes place.

3.6 CONCLUSION

Epidemiological data collected in developed countries suggest that air pollution affects both death and illness rates and generates high social costs, associated with premature death and decrease of quality of life. In these countries large quantities of resources are targeted for clean-ups and remedial actions and safety standards and regulations are becoming stricter. In contrast, developing countries are trapped in the trade-offs of economic growth and environmental protection. Air pollution occurs jointly with other important public health problems, a situation that inhibits the adequate targeting of resources for remediation or prevention of environmental problems. Furthermore, policy-makers may favour employment or economic growth over environment.

Direct extrapolation of health effects observed in populations living in urban areas of developed countries to populations living in urban areas of developing countries is difficult. For example, it is likely that the neurotoxic effect of lead will be similar for Mexican or Australian children living in similar conditions. However, the concurrent existence of iron deficiency and lead exposure in Mexican children could increase toxicity of lead. Similarly, other variables such as population structure, as well as exposure to other pollutants, may preclude direct extrapolation. Nonetheless, most available evidence suggests that populations living in cities with high levels of air pollution in developing countries experience similar or greater adverse effects of air pollution. Certainly, more information is needed to assess health effects of air pollution in these countries and efforts should be targeted to increase the number of epidemiological studies.

The World Health Organization has established guidelines for ambient air pollution levels at which the risk of adverse effects is considered acceptable. The use of these criteria may serve as long-term objectives for countries initiating air pollution control programmes and a base for the development of national standards and regulations.

Although the current fossil-fuel use in developing countries is still half that of developed countries, it is expected to increase by 120 per cent by the year 2010. If control measures are not implemented, it has been estimated that, by the year 2020, more than 6.34 million deaths (95 per cent CI 3.5 to 9.4 millions) will occur in developing countries due to ambient concentrations of particulate air pollution (Working Group on Public Health and Fossil-fuel Combustion, 1997).

There is an urgent need for the implementation of control programmes to reduce levels of particulate and other pollutant emissions. To be effective, these programmes should include the participation of the different stakeholders and initiate activities to identify and characterize air pollution problems and to estimate potential health impacts. A full understanding of the problem and its potential consequences for the local setting is essential for effectively targeting interventions to reduce harmful impacts of air pollution in human populations.

Table 3.5 Recent published studies describing blood lead levels in developing countries

Author and year of publication	City and country	Age group (years)	Population studied	Sample size	Sources of exposure identified	Blood levels in µg/dl
Heinze, I. (1998)	Jakarta, Indonesia	6-8	Children	131	Air lead	7.7
Song, H.Q. (1993)	Beijing, China	5-6	Children	128	Air and food	7.7
Hwang, Y.H. (1990)	Taipei, China	At birth	Newborns	205	Air Lead	7.4
Saxena, D.K. (1994)	Lucknow, India	At birth	Newborns		Not identified	16.9
Potula, V. (1996)	Madras, India	26-55	Office workers	10	Gasoline and ambient air	4.1
			Autoshop workers	9		17.5
			Bus drivers	22		12.1
			Traffic police	88		11.2
Counter, S.A. (1997)	Rural communities Ecuador	4-15	Children	82	Recycling of batteries	52.6
Schutz, A. (1997)	Montevideo Uruguay	2-14	Children	96	Exposure to traffic	9.5
Lopez-Carrillo (1996)	Mexico City Mexico	1-5	Children	603	Ambient air Lead Glazed Ceramics	15.0
Romieu, I. (1995)		1-5	Children	200	Ambient air Lead glazed ceramics	9.9
Hernandez-Avila (1998)		At birth	Children	238	Ambient air Lead glazed ceramics	7.1
Farias, P. 1996		13-43	Pregnant women	513	Ambient air Lead glazed ceramics	11.08
Ramirez, A.V. (1997)	Lima, Peru	18-50	Adult	320	Degree of industrialization	26.9
	Huancayo, Peru					22.4
	La Oroya, Peru					34.8
	Yaupi, Peru					14.0

Table 3.5 Recent published studies describing blood lead levels in developing countries (contd.)

Author and year of publication	City and country	Age group (years)	Population studied	Sample size	Sources of exposure identified	Blood levels in μg/dl
Nriagu. J. (1997)	Durban Metropolitan region	3-10	Children	1200	Ambient air	10.0
	Vulamehlo South Africa					3.8

3.7 REFERENCES

American Thoracic Society (ATS). 1996. A committee of the environmental and occupational health assembly of the American Thoracic Society. State of the Art Review: health effects of outdoor air pollution. *Am. Rev. Crit. Care Med.* **153**(1): 33-50 and **153**(2): 477-998.

Borja-Aburto, V.H., Loomis, D.P., Bangdiwala, S.I., Shy, C.M. and Rascón Pacheco, R.A. 1997. Ozone, suspended particulates and daily mortality in Mexico City. *Am. J. Epidemiol.* **145**: 258-268.

Burnett, R.T., Cakmak, S. and Brook, J.R. 1998. The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. *Canadian Journal of Public Health. Revue Canadienne de Sante Publique* **89**: 152-156.

Burnett, R.T., Cakmak, S., Raizenne, M.E., Stieb, D., Vincent, R., Krewski, D., Brook, J.R., Philips, O. and Ozkaynak. 1998. The association between ambient carbon monoxide levels and daily mortality in Toronto, Canada. *Journal of the Air and Waste Management Association* **48**: 689-700.

Castillejos, M., Gold, D.R., Damokosh, A.I., Serrano, P., Allen, G., McDonnell, W.F., Dockery, D., Ruiz, Velasco S., Hernandez, M. and Hayes, C. 1995. Acute effects of ozone on the pulmonary function of exercising schoolchildren from Mexico City. *Am. J. Resp. and Crit. Care Med.* **152**: 1501-1507.

Cropper, M.L., Simon, N.B., Alberini, A. and Sharma, P.K. 1997. *The Health Effects of Air Pollution in Delhi, India.* The World Bank. PRD Working Paper 1860.

Dockery, D. and Pope, A. 1996. Epidemiology of acute health effects: summary of time-series studies. In *Particles in Our Air. Concentration and Health Effects* (ed. by R. Wilson and J. Spengler). Harvard University Press. Harvard School of Public Health. pp. 1-123-148.

Dockery, D.W and Pope, III C.A. 1994. Acute respiratory effect of particulate air pollution. *Annu. Rev. Public Health* **15**: 107-32.

EPA. 1996. National ambient air quality standards for particulate matter: proposed rule. *Federal Register.* Vol. **61**(241). 13 December, 1996. 40CFR part 50.

Hasselblad, V., Eddy, D.M. and Kotchmar, D.J. 1992. Synthesis of environmental evidence: nitrogen dioxide epidemiological studies. *J. Air Waste Manag. Ass.* **5**: 662-71.

Health Effect Institute (HEI). 1988. *Air Pollution, the Automobile and Public Health.* Health Effect Institute. National Academic Press, Washington DC.

Hernandez-Avila M. 1997. El plomo: un problema en la salud pública en México. Intoxicación por plomo en México: Prevención y Control. *Perspectivas en Salud Pública,* Editores: Mauricio Hernandez-Avila y Eduardo Palazuelos Rendon. 13-24.

Hoek, G., Schwartz, J.D., Groot, B. and Eilers, P. 1997. Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands. *Archives of Environmental Health* **52**(6): 455-63.

Howson, P.C., Hernandez-Avila, M. and Rall D.A. (eds.). 1995. *Lead in the Americas: A Call for Action.* Institute of Medicine. National Academy of Sciences and National Institute of Public Health (Mexico).

Ilabaca, M., Olaeta, I., Campos, E., Villaire, J., Tellez, M.M. and Romieu, I. 1998. Association between levels of fine particulate and emergency visits for pneumonia among children in Santiago, Chile. In *Proceedings from $PM_{2.5}$ A Fine Particulate Standard.* Long Beach, CA. January 28-30pp. 920-938.

Lippmann, M. 1989. Effects of ozone on respiratory function and structure. *Annual Review of Public Health* **10**: 49-67.

Lippmann, M. 1993. Health effects of tropospheric ozone: review of recent research findings and their implications to ambient air quality standards. *J. Exposure Analysis and Env. Epidemiol.* **3**: 103-129.

Loomis, D.P., Borja-Aburto, V.H., Bangdiwala, S.I. and Shy, C.M. 1996. Ozone exposure and daily mortality in Mexico City: a time-series analysis. Research Report. *Health Effects Institute* **75**: 1-37; discussion 39-45.

Maresky, L.S. and Grobler, S.R. 1993. Effect of the reduction of petrol lead on the blood lead levels of South Africans. *Science of the Total Environment* **136**: 43-48.

Munoz, H., Romieu, I., Palazuelos, E, Mancillasanchez, T., Menesesgonzalez, F. and Hernandez-Avila, M. 1993. Blood lead level and neurobehavioral development among children living in Mexico City. *Arch. Environ. Health* **48**: 132-139.

Needleman, H.L. and Bellinger, D. 1991. The health effects of low level exposure to lead. *Annual Review of Public Health* **12**: 111-40.

Nyberg, F. and Pershagen, G. 1996. Epidemiologic studies on ozone. *Scandinavian Journal of Work, Environment and Health* **22** (Suppl): 72-98.

Ostro, B., Sanchez, J.M., Aranda, C. and Eskeland, G. 1996. Air pollution and mortality: results from a study of Santiago, Chile. *J. Exp. Environ. Epidemiol.* **6**(1): 97-114.

Pereira, L.A., Loomis, D., Conceicao, G.M., Braga, A.L.F., Arcas, R.M., Kishi, H.S., Singer, J.M., Bohm, G.M. and Saldiva, P.H. 1998. Association between air pollution and intrauterine mortality in Sao Paulo, Brazil. *Environmental Health Perspectives* **106**: 325-329.

Ponka, A., Savela, M. and Virtanen, M. 1998. Mortality and air pollution in Helsinki. *Archives of Environmental Health* **53**: 281-286.

Pope, A. and Dockery, D. 1996. Epidemiology of chronic health effects: cross-sectional studies. In *Particles in Our Air. Concentration and Health Effects*. (ed. by R. Wilson and J. Spengler). Harvard University Press. Harvard School of Public Health. pp. 149-167.

Pope, C.A. 3rd., Thun, M.J., Namboodiri, M.M., Dockery, D.W., Evans, J.S., Speizer, F.E..and Heath, C.W. Jr. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine* **151**: 669-74.

Pope, III C.A, Bates, D.V. and Raizenne, M.E. 1995. Health effects of particulate air pollution: time for reassessment. *Environ. Health Perspect* **103**: 472-480.

Romieu, I. 1999. Epidemiological studies of health effects arising from motor vehicle air pollution. In *Urban Traffic Pollution* (ed. by D. Schwella and O. Zali). Ecotox, WHO. E & FN Spoon, London and New York. pp. 9-49.

Romieu, I. and Borja-Aburto, V. 1997. Particulate air pollution and daily mortality: can results be generalized to Latin American countries. *Salud Pub. Mex.* **39**: 403-411.

Romieu, I., Meneses, F., Ramirez, M., Ruiz, S., Perez Padilla R., Sienra. J.J., Gerber, M., Grievink, L., Dekker, R.; Walda, I. and Brunekreff, B. 1998. Antioxidant supplementation and respiratory functions among workers exposed to high levels of ozone. *Am. J. Resp. Critic Care Med.* **158**: 226-232.

Romieu, I., Meneses, F., Ruiz, S., Huerta, J., Sienra, J.J., White, M., Etzel, R. and Hernandez, M. 1997. Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City. *Arch. Environ. Health* **52**(5): 368-376.

Romieu, I., Meneses, F., Ruiz, S., Sienra, J.J., Huerta, J., White, M and Etzel, R. 1996. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *Am. J. Resp. Crit. Care Med.* **154**: 300-307.

Romieu, I., Weizenfeld, H. and Finkelman, J. 1990. Urban air pollution in Latin America and the Caribbean: health perspectives. *Wld. Hlth. Statist. Quart.* **43**: 153-168.

Rothenberg, S.J., Schnaas, L., Perroni, E., Hernandez, R.M. and Karchmer, S. 1998. Secular trend in blood lead levels in a cohort of Mexico City children. *Arch Environ Health* **53**: 231-235.

Saldiva, P.H.N., Pope, C.A. III, Schwartz, J., Dockery, D.W., Lichtenfelds, A.J., Salge, J.M., Barone, Y. and Bohm, G.M. 1995. Air pollution and mortality in elderly people: a time series study in Sao Paulo, Brazil. *Arch. Environ. Health* **50**: 159-163.

Samet, J.M. and Speizer, F.E. 1993. Assessment of health effects in epidemiologic studies of air pollution. *Environ. Health Perspect.* **101**(suppl.4): 149-154.

Schwartz, J. 1995. Carbon monoxide a risk factor for hospital admission for heart failure? *American Journal of Public Health* **85**(10): 1343-1345.

Shen, X., Yan, C. and Zhou, J. 1996. Relationship between lead content in umbilical blood and neurobehavioral development in infants. *Chinese Journal of Preventive Medicine* [Chinese] **30**: 68-70.

Tellez-Rojo, M., Romieu, I., Lezana, M.A., Meneses, F. and Hernandez, M. 1997. Associacion entre exposicion a contaminantes ambientales y mortalidad por causas respiratorias en la cuidad de Mexico. XXX Congreso de la Sociedad Matematica Mexicana. Aguascaliente, October 2.

Tellez-Rojo, M.M., Romieu, I., Pena, M.P., Ruiz-Velasco, S., Meneses-Gonzales, F. and Hernandez-Avila, M. 1997. Efecto de la contaminacion ambiental sobre las consultas por infectiones respiratorias en ninos de la Ciudad de Mexico. *Salud Publica. de Mexico* **39**(6): 513-522.

Working Group on Public Health and Fossil-fuel Combustion. 1997. Short-term improvements in public health from global-climate policies on fossil-fuel combustion: an interim report. *Lancet* **350**: 1341-1349.

World Health Organization. 1987. *Air Quality Guidelines for Europe.* WHO Regional Office for Europe, Copenhagen. (WHO Regional Publications, European Series No 23.)

World Health Organization. 1993. *The Urban Health Crisis: Strategies for Health for All in the Face of Rapid Urbanization.* Report of WHO Expert Committee (WHO, Geneva).

World Health Organization (WHO). 1997. *Healthy Cities Air Management.* Information System, AMIS 1.0. Geneva.

World Health Organization. 1999. *Air Quality Guidelines for Europe.* WHO Regional Office for Europe, Copenhagen. 2nd edition (in press).

Yang, S.C. and Yang, S.P. 1994. Respiratory function changes from inhalation of polluted air. *Arch. Environ. Health* **49**(3): 182-187.

Chapter 4

LOCAL AMBIENT AIR QUALITY MANAGEMENT

Dr. Dietrich Schwela
World Health Organization, Division of Operational Support in Environmental Health,
Urban Environmental Health, Geneva, Switzerland

Abstract

The aim of local ambient air quality management is to protect public health and the environment from the damaging effects of air pollution, and to eliminate or reduce to a minimum human exposure to hazardous pollutants. In developed countries air quality management has used sophisticated instruments to determine the necessary measures to control polluting sources. This has taken form in clean air implementation plans, based on an evaluation of the most efficient method to reduce air pollution. In contrast, an assessment of the air pollutant reduction measures in developing countries is typically based on more limited information concerning local sources, the dispersion of air pollution, actual air pollutant levels and related adverse effects. The lack of emissions' inventories and air quality standards makes assessment difficult. The World Health Organisation (WHO) has produced guidelines on air quality to assist developing countries to determine the best measures to abate air pollution with limited information. This has been achieved by drawing upon knowledge gained in developed countries and giving practical advice on how to develop legally enforceable air quality standards and simplified clean air implementation plans. The guidelines provide advice on which legal aspects need to be considered, how adverse effects in the population at risk can be defined, how exposure-response relationships can be applied, and how acceptable levels of risks can be assessed. The air quality guidelines provide advice on the health effects of air pollution under different geographical, social, economic and cultural conditions and how the capability for implementing air quality standards may be increased. The Air Management Information System (AMIS), recently set up by WHO, is an additional information source on air pollution concentrations in major and megacities, air quality standards in various countries, on air quality management capabilities and on the instruments used in various cities of developed and developing countries. This Chapter discusses the factors which need to be considered in local air quality management and the guidance provided by the WHO Guidelines for Air Quality and the Air Management Information System.

4.1 INTRODUCTION

This Chapter discusses the various aspects of local air quality management in developing countries, including: the conversion of WHO air quality guidelines to national air quality standards; their use in local air quality management; and the assistance which AMIS can provide (WHO, 1997a; WHO 1998a). The WHO Air Quality Guidelines for Europe (WHO, 1987a) have been of major-support to countries undertaking risk assessment and setting national standards. The guidelines have been updated, revised (WHO, 1994a; WHO, 1995a; WHO, 1996a; WHO, 1998b) and made globally applicable by taking into account factors which might be influential to the health outcome in other regions (WHO, 1998c). The application of the guidelines for the setting of national air quality standards has been extensively discussed in two publications (WHO, 1987b; WHO 1998d).

In order to understand the difference between air quality guidelines and air quality standards, these terms are defined as follows:

An *air quality guideline* is any kind of recommendation or guidance on the protection of a population of human beings or receptors in the environment (e.g. vegetation, materials) from the adverse effects of air pollutants. Air quality guidelines are exclusively based on exposure-response relationships found in epidemiological, toxicological and environment-related studies. An air quality guideline is not restricted to a numerical value, and may express exposure-response information or unit risks in different ways.

An *air quality guideline value* is a fixed numerical value corresponding to a defined averaging time. It is expressed as a concentration in ambient air, a deposition level, or some other physico-chemical value. In the case of human health, the air quality guideline is a concentration below which no adverse effects are expected, although a small residual risk always exists. Compliance of appropriate statistical location parameters with a guideline value does not guarantee that effects do not occur.

An *air quality standard* is a level of air pollutant (concentration, deposition, etc.) that is promulgated by a regulatory authority and adopted as legally enforceable. In addition to the effect-based level and the averaging time of a guideline value, several elements have to be specified in the formulation of a standard. These include the measurement procedure, definition of compliance parameters corresponding to the averaging times, and the permitted number of exceedences.

The WHO air quality guidelines provide 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 to be hazardous to human health and well-being (WHO, 1987a). Air quality guidelines provide background information and guidance to governments in making risk management decisions, particularly in setting standards. They also assist governments to undertake local control measures in the framework of air quality management.

The updated and revised, globally applicable air quality guidelines of WHO are presented in Annex 1 (Tables 4.1 and 4.2) and Annex 2 (Table 4.3).

The Air Management Information System (AMIS), a programme set up by WHO as a successor to the UNEP/WHO GEMS/AIR programme, provides valuable information on air pollutant monitoring and management in major and megacities (WHO, 1997a; WHO, 1998a).

AMIS is a programme developed by WHO under the umbrella of the Healthy Cities Programme. The objective of AMIS is to transfer information on air quality management (air quality management instruments used in cities, ambient air pollutant concentrations, health effects, control actions, air quality standards, emission standards, rapid emission assessment tools, dispersion modelling tools) between countries and cities. In this context, AMIS acts as a global air quality information exchange system. AMIS programme activity areas include:

- co-ordinating databases with information on air quality issues in major and megacities;
- acting as an information broker between countries;
- providing and distributing technical documents on air quality management;
- publishing and distributing Annual Trend Reviews on air pollutant concentrations; and
- providing training courses on air quality monitoring and management.

AMIS provides a set of user friendly Microsoft Access based databases. A core database contains summary statistics of air pollution data such as annual means, 95-percentiles, and the number of days on which WHO guidelines are exceeded. Any compound for which WHO air quality guidelines exist can be entered into the open-ended database. Data handling is easy and data validation can be assured with relatively little means. In the present version, data (mostly from 1986 to 1996) from about 100 cities in 40 countries are represented (WHO, 1998a). Another AMIS database covers the air pollution management capabilities and procedures of cities. Databases on the use and accessibility of dispersion models, control actions, health effects and the magnitudes of their respective costs are also planned.

The following discussion covers legal aspects, exposure-response relationships, the characterisation of exposure, the assessment and acceptability of risks, application of cost-benefit analysis, and the enforcement of air quality standards through the instrument of clean air implementation plans.

4.2 USE OF WHO GUIDELINES FOR AIR QUALITY IN LOCAL AIR QUALITY MANAGEMENT

For air quality management to be effective, goals, policies, strategies and tactics have to be defined. Goals for air quality management can include the elimination or reduction to acceptable levels of ambient air pollutant concentrations or the avoidance of adverse effects on humans and other receptors. Policies for air quality management encompass clean air acts, environmental impact assessments, air quality standards, clean air implementation plans and cost-benefit comparisons. Strategies for air quality management refer to command-and-control procedures and/or the application of market mechanisms. The tactical instruments of air quality management are inventories, dispersion modelling, monitoring and comparison with standards.

A framework for a political, regulatory and administrative approach is required to guarantee a consistent and transparent derivation of air quality standards and to ensure a basis for decisions on risk-reducing measures and abatement strategies. In such a framework, legal aspects, adverse effects on health, the population at risk, exposure-response relationships, exposure characterisation, risk assessment, the acceptability of risk, cost-benefit analysis and stakeholder contribution in standard setting have to be included.

4.2.1 Legal aspects

A legislative framework usually provides the basis for policies in the decision-making process of setting air quality standards at the municipal, regional, national or supranational level. The setting of standards strongly depends on the risk management strategy adopted which, in turn, is influenced by country-specific socio-political and economic considerations and/or international agreements. Legislation and air quality standards vary from country to country but, in general, the WHO Guidelines for Air Quality and the information provided by AMIS can provide guidance on how to consider the following issues in developing countries:

- *Identification of the pollutants to be considered* - Provided the types of sources are known, the guidelines and the rapid assessment procedures of AMIS can identify the most important sources and estimate their emissions.

- *Existing background concentrations of air pollutants* - The knowledge on global concentrations from the AMIS database on air pollutant concentrations and the WMO Global Atmospheric Watch can serve to estimate background concentrations. The Decision Support System for Industrial Pollution Control (DSS IPC) is a useful and user-friendly instrument to estimate concentrations on the basis of initial emissions estimates and simple dispersion models.

- *Applicable monitoring methodology and its quality assurance* - The most appropriate and least-cost means for ground-based monitoring can be selected on the basis of the AMIS-GEMS/AIR Methodology Handbook Review Series (UNEP/WHO, 1994a; UNEP/WHO, 1994b; UNEP/WHO, 1994c). In these publications WHO gives simple advice on monitoring, siting and quality assurance when existing information and means are minimal. Publications from other agencies also provide insight into monitoring strategies (BMU, 1997; AEA, 1996; WHO/PAHO, 1997; WHO/SEARO, 1996).

- *The numerical value of the standards for the various pollutants or the decision-making process* - Air quality standards may be based on WHO air quality guidelines, but other aspects, such as technological feasibility, costs of compliance, prevailing exposure levels, social, economic cultural conditions, are also relevant to the standard setting procedure and the design of appropriate emission abatement measures. Several air quality standards may be set, e.g. effect-oriented standards as a long-term goal and less stringent standards to be achieved within shorter time intervals. As a consequence, air quality standards differ widely from country to country (WHO, 1998d). The Guidelines for Air Quality enable country-specific air quality standards to be derived based on existing or estimated concentrations. Cost of control estimates and the efficiency of controls can be assessed using the DSS IPC (WHO, 1993b; WHO, 1995b).

- *Emission control measures and emission standards* - Given the types of sources and estimations of their emissions via the rapid assessment method and their spatial distribution, the DSS IPC can serve to simulate the efficiency of control measures and help to set appropriate emission standards for the main sources (WHO, 1995b).

- *Identification and selection of adverse effects on public health and the environment to be avoided* - Health effects range from death and acute illness through chronic and lingering diseases, minor and temporary ailments, to temporary physiological or psychological changes. The guidelines advise on the more serious adverse effects of air pollutants in a global frame. Consideration of health effects that are either temporary and reversible, or involve biochemical or functional changes with uncertain clinical significance, need not be considered in the first step of deriving a standard in developing countries. Judgements as to adversity of health effects may differ between countries because of, for example, different cultural backgrounds and different levels of health status.

- *Identification of the population to be protected from adverse effects on health* - The most sensitive subgroups of the population are identified in the guidelines as infants, pregnant women, disabled persons and the elderly. Other groups may be judged to be at higher risk due to enhanced exposure (outdoor workers, athletes and children). The sensitive groups in a population may vary across countries due to differences in medical care, nutritional status, lifestyle and/or prevailing genetic factors, or due to the existence of endemic diseases or the prevalence of debilitating diseases. The air quality guidelines have been set with respect to the sub-groups more sensitive to air pollution. Setting standards on the basis of the guidelines and considering the consequence of uncertainty provide at least some protection for these sub-populations.

Air quality standards strongly influence the implementation of air pollution control policies. In many countries, the exceeding of standards is linked to an obligation to develop action plans at the municipal, regional or national level to abate air pollution (clean air implementation plans).

4.2.2 Exposure-response relationships

In general, there is limited information available on exposure-response relationships for inorganic and organic pollutants, especially at low exposures. The revised air quality guidelines for Europe provide exposure-response relationships for a number of pollutants including detailed tables of the relationships for particulate matter (PM) and ozone. For PM_{10} and $PM_{2.5}$ the changes of various health endpoints such as daily mortality and hospital admissions with each 10 mg/m^3 increase in concentrations are quantified.

If it can be assumed that these relationships apply across the entire range of concentrations between 0 and 200 mg/m^3, then the available data imply that there are linear relationships between various health endpoints and PM concentrations.

For carcinogenic compounds, the quantitative assessment of the unit risks provides an approximate estimate of responses at different concentrations. These relationships, which are extensively discussed in the Guidelines for Air Quality, give guidance to decision-makers to determine the acceptable risk for the population exposure to particulate matter and to carcinogenic compounds and set the corresponding concentrations as standards.

4.2.3 Exposure characterisation

Exposure to air pollution is not only determined by ambient air pollutant concentrations. In deriving air quality standards that protect against the adverse health impacts, the size of the population at risk (i.e. exposed to enhanced air pollutant concentrations) is an important factor to consider. The total exposure of people also depends on the time people spend in the various environments: outdoor, indoor, work place, in-vehicle and other. Exposure also depends on the various routes of intake and absorption of pollutants in the human body: air, water, food and tobacco smoking. Therefore, it should be kept in mind that there is a weak relationship between pollutant concentrations and personal exposures. An example of this weak relationship is provided by indoor air pollution when biomass fuels are used for heating and cooking. However, in developing countries, ambient air concentrations are at present the only readily available surrogate for estimating personal exposures.

4.2.4 Risk assessment

WHO Air Quality Guidelines are based on health or ecological risk models. These models provide a tool that is increasingly used to inform policy-makers on some of the possible consequences of air pollutants at different pollutant levels which correspond to various options for standards. Using this information, the policy-maker is able to perform a regulatory risk assessment of air pollution induced effects. Regulatory risk assessment in air pollution management includes the following steps: hazard identification, development of exposure-response relationships, exposure analysis and quantitative risk estimation. The first step - hazard identification - and, to some extent, the second step - exposure-response relationships - have already been provided in the air quality guidelines. The third step - exposure analysis - may predict changes in exposure associated with reductions in emissions from a specific source or group of sources under different control options. The final step in regulatory risk assessment - risk analysis - refers to the quantitative estimation of the risk of health effects in the exposed population (e.g. the number of individuals who may be affected). Examples for such estimates were given by Hong (1995); Ostro (1996); Schwela (1996); Schwela (1998); Murray and Lopez (1997). Regulatory risk assessments are likely to result in different risk estimates across countries and economic regions owing to differences in exposure patterns and in the size and characteristics of sensitive groups. In addition, differences in the legislation and availability of information necessary to undertake quantitative risk assessments may affect the results.

4.2.5 Acceptability of risk

In the absence of thresholds for the onset of health effects - as in the cases of fine and ultra-fine particulate matter and carcinogenic compounds - the selection of an air quality standard that provides adequate protection of public health requires the regulator to determine an acceptable risk for his population. Acceptability of the risks, and therefore the standards selected, will depend on the expected incidence and severity of the potential effects, the size of the population at risk, and the degree of scientific uncertainty that the effects will occur at any given level of air pollution. For example, if a suspected but uncertain health effect is severe and the size of the population at risk is large, a more cautious approach would be appropriate than if the effect were less troubling or if the population were smaller.

The acceptability of risk may vary among countries because of differences in social norms, degree of risk aversion and perception in the general population and various stakeholders. Risk acceptability is also influenced by how the risks associated with air pollution compare with risks from other pollution sources or human activities (WHO, 1987b).

4.2.6 Cost-benefit analysis

Two different approaches for decisions can be applied in the derivation of air quality standards from air quality guidelines. In the first approach, decisions are based purely on health, cultural and environmental consequences with little weight to economic efficiency. The objective of this approach is to reduce the risk of adverse effects to a socially acceptable level. The second approach is based on a formal cost-effectiveness or cost-benefit analysis, the objective being to identify the control action that achieves greatest net economic benefit, or is the most economically efficient. The development of air quality standards should take account of both extremes. Cost-benefit analysis (CBA) is a highly interdisciplinary task and, if appropriately applied and not used as the sole and overriding determinant of decisions, can be a legitimate and useful way to provide information for risk managers making decisions that will affect human health and the environment.

The WHO Guidelines for Air Quality describe in some detail the individual steps of a CBA and give advice on which information is needed to undertake CBA. In developed countries, at least part of this information can be made available but, in most developing countries, comprehensive CBA procedures can only be applied in the long term. It would be useful for developing countries to collect data on the use of medication, number of hospital admission, outpatient visits or days of labour lost and relate them to air pollution. This procedure would at least give some indication of the potential magnitude of the benefits of air pollution control (WHO, 1998d).

4.2.7 Review of standard setting

The setting of standards should encompass a process involving stakeholders (industry, local authorities, non-governmental organisations and the general public) that assures - as far as possible - social equity or fairness to all the parties involved. It should also provide sufficient information to guarantee understanding by stakeholders of the scientific and economic consequences. The earlier stakeholders are involved the more likely is their co-operation. Transparency in moving from air quality guidelines to air quality standards helps to increase public acceptance of necessary measures. Raising public awareness of air pollution-induced health and environmental effects (changing of risk perception) serves to obtain public support for the necessary control action, e.g. with respect to vehicular emissions. Information provided to the public with regard to air quality during pollution episodes and the risks entailed lead to a better understanding of the issue (risk communication).

Air quality standards should be reviewed and revised regularly as new scientific evidence on the effects on public health and the environment emerges.

4.3 ENFORCEMENT OF AIR QUALITY STANDARDS: CLEAN AIR IMPLEMENTATION PLANS

It is the enforcement of air quality standards that ensures that actions are taken to control polluting sources in order to comply with the standards. The instruments used to achieve this goal are Clean Air Implementation Plans (CAIPs). The outline of such a plan is usually defined in regulatory policies and strategies. CAIPs were implemented in several developed countries during the 1970s and 1980s. At that time the air pollutant situation was characterised by a multitude of different types of sources leading to an extremely difficult causal assessment of public health risks with respect to single source or group of sources. As a consequence, and on the basis of the polluters pay principle, sophisticated tools were developed to assess the pollution sources, air pollutant concentrations, health and environmental effects and control measures. The tools also made a causal link between emissions, the air pollution situation and the efficiency of the necessary control measures. The CAIP has proved to be a most efficient instrument for air pollution abatement in developed countries (Schwela and Köth-Jahr, 1994; WHO 1997b).

In developing countries, the air pollution situation is often characterised by a multitude of sources of few types and sometimes few sources. Using the experience obtained in developed countries, the control action to be taken is often very clear. As a consequence, lower monitoring would be sufficient, and dispersion models could help to simulate spatial distributions of concentrations in the case where only limited useful monitoring data are available. Only simplified CAIPs would have to be developed for cities of developing countries or countries in transition. The main polluters at present in many cities of the developing world are old vehicles and some industrial sources such as power plants, brick kilns and cement factories.

In such situations, a simplified clean air implementation plan could include:

- a rapid assessment of the most important sources (WHO, 1993a; WHO, 1993b; WHO 1995b);
- a minimal set of air pollutant concentrations monitors (UNEP/WHO, 1994a; UNEP/WHO, 1994b; UNEP/WHO, 1994c);
- simulation of the spatial distribution of air pollutant concentrations using simple dispersion models (WHO, 1995b);
- comparison with air quality standards;
- control measures and their costs (WHO, 1995b); and
- transportation and land-use planning.

Examples of successful simplified CAIP in developing countries are provided in a recent report on air quality management capabilities in 20 major cities (UNEP/WHO/MARC, 1996)

4.4 CONCLUSIONS

This Chapter has presented a simplified procedure for setting air quality standards from guidelines and implementing action plans for air pollution abatement in developing countries. Issues such as rapid emissions assessment and the use of dispersion modelling as a surrogate for extensive air pollutant concentration monitoring and the testing of local air pollutant concentrations against air quality standards for local ambient air quality management have been discussed. Air quality standards are often based on WHO Air Quality Guidelines. In moving from air quality guidelines to air quality standards, several factors have to be considered including the political, regulatory and administrative approaches to control air pollution. The

WHO Guidelines for Air Quality, and the Air Management Information System (AMIS), provide guidance in achieving effective air quality management in developing countries.

4.5 REFERENCES

AEA. 1996. *Air Quality Monitoring: A Handbook for Local Authorities, AEA Technology* (ed. by J. McGinley, J. Vallance-Plews and J. Bower). Atomic Energy Agency, AEA/RAMP/20029001/01, UK.

BMU. 1997. *Determination and Evaluation of Ambient Air Quality - Manual of Ambient Air Monitoring in Germany* (ed. by E. Lahmann). Second Revised Edition. Federal Environmental Agency, Berlin, Germany.

Hong, C.J. 1995. *Global Burden of Diseases from Air Pollution* (unpublished).

Murray, C.J.L. and Lopez, A.D. (eds.).1996. *The Global Burden of Disease.* A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Harvard School of Public Health, Harvard University Press.

Schwela, D.H. 1996a. Exposure to environmental chemicals relevant for respiratory hypersensitivity: global aspects. *Toxicology Letters* **86** (1996): 131-142.

Schwela, D.H. 1998. *Health and Air Pollution - A Developing Country's Perspective.* Keynote address at the 11th World Clean Air and Environment Congress, Durban, South Africa, 13-18.September, 1998.

Schwela, D.H. and Köth-Jahr, I. 1994. *Leitfaden für die Aufstellung von Luftreinhalteplänen* (Guidelines for the Implementation of Clean Air Implementation Plans). Report No. 4. Landesumweltamt Nordrhein Westfalen, Essen, Germany.

UNEP/WHO. 1994a. *GEMS/AIR Methodology Review Handbook Series.* Volume **1**. Quality Assurance in Urban Air Quality Monitoring, United Nations Environment Programme, Nairobi, Kenya; World Health Organization, Geneva, Switzerland.

UNEP/WHO. 1994b. *GEMS/AIR Methodology Review Handbook Series.* Volume **2**. Measurement of Suspended Particulate Matter in Ambient Air, United Nations Environment Programme, Nairobi, Kenya; World Health Organization, Geneva, Switzerland.

UNEP/WHO. 1994c. GEMS/AIR Methodology Review Handbook Series. Volume **3**. Passive and Active Sampling Methodologies for Measurement of Air Quality, United Nations Environment Programme, Nairobi, Kenya; World Health Organization, Geneva, Switzerland.

UNEP/WHO/MARC. 1996. *GEMS/AIR Air Quality Management and Assessment Capabilities in 20 Major Cities.* Environmental Assessment Report, United Nations Environment Programme, Nairobi, Kenya; World Health Organization, Geneva, Switzerland; The Monitoring and Assessment Research Centre, London, UK.

USEPA. 1987. *Draft Regulatory Impact Analysis on the National Ambient Air Quality Standard for Sulphur Dioxide.* United States Environmental Protection Agency, Washington DC.

WHO. 1987a. *Air Quality Guidelines for Europe.* World Health Organization, WHO Regional Publications, European Series No. **23**, Copenhagen.

WHO. 1987b. *Setting Environmental Standards. Guidelines for Decision-making* (ed. by H.W. de Koning). World Health Organization, Geneva.

WHO. 1992b. *Acrolein.* Environmental Health Criteria 127. World Health Organization, Geneva.

WHO. 1993a. *Assessment of Sources of Air, Water and Land Pollution. Part One: Rapid Inventory Techniques in Environmental Pollution* by Alexander Economopoulos. WHO/PEP/GETNET/93.1-A, World Health Organization, Geneva.

WHO. 1993b. *Assessment of Sources of Air, Water and Land Pollution, Part Two: Approaches for Consideration in Formulating Environmental Control Strategies* by Alexander Economopoulos. WHO/PEP/GETNET/93.1-B, World Health Organization, Geneva.

WHO. 1994a. *Updating and Revision of the Air Quality Guidelines for Europe - Inorganic Air Pollutants*. World Health Organisation, Regional Office for Europe, Copenhagen, EUR/ICP/EHAZ 94 05/MT04.

WHO. 1994b. *Chloroform*. Environmental Health Criteria 163. World Health Organization, Geneva.

WHO. 1995a. *Updating and Revision of the Air Quality Guidelines for Europe - "Classical" Air Pollutants*. World Health Organization, Regional Office for Europe, Copenhagen, EUR/ICP/EHAZ 94 05/PB01.

WHO. 1995b. *Decision Support System for Industrial Pollution Control* (DSS IPC). PC Programme for the assessment of air emission inventories, liquid and solid waste inventories, estimation of pollution in air, water, and soil. PAHO/World Bank, 1995.

WHO. 1995c. *Acetaldehyde*. Environmental Health Criteria 167. World Health Organization, Geneva.

WHO. 1996a. *Updating and Revision of the Air Quality Guidelines for Europe - Volatile Organic Compounds*. World Health Organisation, Regional Office for Europe, Copenhagen, EUR/ICP/EHAZ 94 05/MT12.

WHO. 1996b. *Updating and Revision of the Air Quality Guidelines for Europe - Meeting of the Working Group on Certain Indoor Air Pollutants*. World Health Organisation, Regional Office for Europe, Copenhagen, EUR/ICP/EHAZ 94 05/MT09.

WHO. 1996c. *Diesel Fuel and Exhaust Emissions*. Environmental Health Criteria 171. World Health Organization, Geneva.

WHO. 1996d. *Ethylbenzene*. Environmental Health Criteria 186. World Health Organization, Geneva.

WHO. 1997a. *Healthy Cities Air Management Information System*, AMIS 1.0, CD ROM. World Health Organization, Geneva.

WHO. 1997b. *Health and Environment in Sustainable Development - Five Years after the Earth Summit*. World Health Organization, Geneva

WHO. 1997c. *Acrylic Acid*. Environmental Health Criteria 191. World Health Organization, Geneva.

WHO. 1997d. *Di-n-Butyl Phthalate*. Environmental Health Criteria 189. World Health Organization, Geneva.

WHO. 1997e. *Xylenes*. Environmental Health Criteria 190. World Health Organization, Geneva.

WHO. 1998a. *Healthy Cities Air Management Information System*, AMIS 2.0, CD ROM. World Health Organization, Geneva.

WHO. 1998b. *Air Quality Guidelines for Europe*. World Health Organisation, Regional Office for Europe, Copenhagen, WHO Regional Publications, European Series. Internet address: http://www.who.dk

WHO. 1998c. *Guidelines for Air Quality*. World Health Organisation, Geneva. Internet address: http://www.who.ch

WHO. 1998d. *Guidance for Setting Air Quality Standards*. Report of a WHO Working Group, Barcelona, Spain, 12-14 May 1997. EUR/ICP/EHPM 02 01 02. WHO Regional Office for Europe, Copenhagen.

WHO/PAHO. 1997. *Introduccion al Monitoreo Atmosférico* (ed. by A.P. Martinez and I. Romieu). World Health Organization, Pan American Health Organization, Mexico DF, Mexico.

WHO/SEARO. 1996. *Ambient Air Quality Monitoring Networks: Considerations for Developing Countries* (ed. by A.L. Aggarwal and S.K. Gopal). World Health Organization, Regional Office for South-East Asia, New Delhi, India.

ANNEXES

Annex 1

Table 4.1 WHO air quality guidelines for “classical” compounds

Compound	Annual ambient air concentration [μg/m^3]	Health endpoint	Observed effect level [μg/m^3]	Uncertainty factor	Guideline value [μg/m^3]	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 < 25 :g 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	1 000	2	500	10 minutes
		Exacerbations of respiratory symptoms in sensitive individuals	250	2	125	24 hours
			100	2	50	1 year

Table 4.2 WHO air quality guidelines for non-carcinogenic compounds

Compound	Average ambient air concentration [µg/m³]	Health endpoint	Observed effect level [mg/m³]	Uncertainty factor	Guideline value [µg/m³]	Averaging time	Source
Acetaldehyde	5	Irritancy in humans	45 (NOEL)	20	2 000	24 hours	WHO, 1995c
		Carcinogenicity related irritation in rats	275 (NOEL)	1000	300	1 year	WHO, 1995c
Acrolein	15	Eye irritation in humans	130	2.5	50	30 min	WHO, 1992b
Acrylic acid	No data	Nasal lesions in mice	15 (LOAEL)	50	54	1 year	WHO, 1997c
Cadmium	(0.1-20) . 10-3	Renal effects in the population	n.a.	n.a.	5×10^{-3}	1 year	WHO, 1998a
Carbon disulphide	10-1500	Functional CNS changes in workers	10 (LOAEL)	100	100	24 hours	WHO, 1987
		Odour annoyance (Odour threshold)	n.a.	n.a.	20	30 minutes	WHO, 1987
Chloroform	0.3-110	Hepatoxicity in beagles	from TDI	1000	15	24 hours	WHO, 1994b
1,2-Dichloroethane	0.2-6	Inhalation in animals	700 (LOAEL)	1000	700	24 hours	WHO, 1987
Dichloromethane	< 5	COHb formation in normal subjects		n.a.	3 000	24 hours	WHO, 1998a
Diesel exhaust	1.0 - 10.0	Chronic alveolar inflammation in humans	0.139 (NOAEL)*	25	5.6	1 year	WHO, 1996c
		Chronic alveolar inflammation in rats	0.23 (NOAEL)	100	2.3	1 year	WHO, 1996c
Di-n-butyl Phthalate	$(3\text{-}80) . 10^{-3}$	Developmental/Reproductive toxicity	from ADI	1000	14	24 hours	WHO, 1997d
Ethylbenzene	1-100	Biological significance criteria in animals	2150 (NOEL)	100	22 000	1 week	WHO, 1996d
Fluorides	0.5 - 3	Effects on livestocks	n.a.	n.a.	1	1 year	WHO, 1994a
Formaldehyde	(1-20) . 10-3	Nose, throat irritation in humans	0.1 (NOAEL)	n.a.	0.1	30 minutes	WHO, 1998a

Table 4.2 WHO air quality guidelines for non-carcinogenic compounds (contd.)

Compound	Average ambient air concentration [µg/m³]	Health endpoint	Observed effect level [mg/m³]	Uncertainty factor	Guideline value [µg/m³]	Averaging time	Source
Hydrogen sulphide	0.15	Eye irritation in humans	15 (LOAEL)	100	150	24 hours	WHO, 1987
		Odour annoyance (Odour threshold)	n.a.	n.a.	7	30 minutes	WHO, 1987
Manganese	0.01 - 0.07	Neurotoxic effects in workers	0.03 (NOAEL)	200	0.15	1 year	WHO, 1998a
Mercury, inorganic	(2-10) . 10-3	Renal tubular effects in humans	0.020 (LOAEL)	20	1	1 year	WHO, 1998a
Styrene	1.0 -20.0	Neurological effects in workers	107 (LOAEL)	40	260	1 week	WHO, 1998a
		Odour annoyance (Odour threshold)	n.a.	n.a.	70	30 minutes	WHO, 1998a
Tetrachloroethylene	1 - 5	Kidney effects in workers	102 (LOAEL)	400	250	24 hours	WHO, 1998a
		Odour annoyance (Odour threshold)	n.a.	n.a.	8 000	30 minutes	WHO, 1987
Toluene	5 - 150	Effects on CNS in workers	332 (LOAEL)	1 260	260	1 week	WHO, 1998a
		Odour annoyance (Odour threshold)	n.a.	n.a.	1 000	30 minutes	WHO, 1987
Vanadium	0.05 - 0.2	Respiratory effects in workers	0.02 (LOAEL)	20	1	24 hours	WHO, 1987
Xylenes	1 - 100	Neurotoxicity in rats	870 (LOAEL)	1 000	870	1 year	WHO, 1997e
		CNS effects in human volunteers	304 (NOAEL)	60	4 800	24 hours	WHO, 1997e
		Odour annoyance (Odour threshold)	n.a.	n.a.	4 400	30 minutes	WHO, 1997e

Annex 2

Table 4.3 WHO air quality guidelines for carcinogenic compounds

Compound	Average ambient air concentration [µg/m³]	Health endpoint	Unit risk $[\mu g/m^3]^{-1}$	IARC classification	Source
Acetaldehyde	5	Nasal tumours in rats	$(1.5\text{-}9) \times 10^{-7}$	2B	WHO,1995c
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, 1998a
Benzene	5.0 - 20.0	Leukemia in exposed workers	6×10^{-6}	1	WHO, 1998a
ChromiumVI	$(5\text{-}200) . 10^{-3}$	Lung cancer in exposed workers	4×10^{-2}	1	WHO, 1998a
Diesel exhaust	1.0 - 10.0	Lung cancer in rats	$(1.6\text{-}7.1) \times 10^{-5}$		WHO, 1996c
Nickel	1-180	Lung cancer in exposed humans	3.8×10^{-4}	1	WHO, 1998a
PAH (BaP)	$(1\text{-}10) . 10^{-3}$	Lung cancer in exposed humans	8.7×10^{-5}	1	WHO, 1998a
Trichloroethylene	`1-10	Cell tumours in testes of rats	4.3×10^{-7}	2A	WHO, 1998a
Vinychloride	0.1 - 10	Hemangiosarkoma in exposed workers Liver cancer in exposed workers	1×10^{-6}	1	WHO, 1987

Table 4.3 WHO air quality guidelines for carcinogenic compounds (contd.)

Compound	Average ambient air concentration $[\mu g/m^3]$	Health endpoint	Unit risk $[\mu g/m^3]^{-1}$	IARC classification	Source
Fibres	[fibres/l]		$[\text{fibres/l}]^{-1}$		
MMVF (RCF)	$2 - 2 . 10^3$	Mesotheliomas in animal inhalation	1×10^{-6}	2B	WHO, 1998a
	$[Bq/m^3]$		$[Bq/m3]^{-1}$		
Radon	100	Lung cancer in residentials	$(3\text{-}6) \times 10^{-5}$	1	WHO, 1998a

PAH:	Polycyclic aromatic hydrocarbons;	BaP:	Benzo (a) pyrene
MMVF:	Man-Made Vitreous fibres	RCF:	Refractory ceramic fibres

Chapter 5

RAPID ASSESSMENT OF AIR POLLUTION AND HEALTH: MAKING OPTIMAL USE OF DATA FOR POLICY AND DECISION-MAKING

Dr Yasmin von Schirnding
Sustainable Development and Healthy Environments
World Health Organisation

Abstract

In many countries throughout the World air pollution concentrations are increasing, yet information on the associated health effects is lacking. Decision-makers nevertheless need to set policies and control strategies, often on the basis of limited data. Many situations arise in which it may be necessary to conduct rapid appraisals of air pollution and associated health effects. For instance, there may be a toxic spill or environmental disaster/air pollution episode demanding a rapid response; there may be community concern about a potential air pollution risk from a polluting industry; there may be a sudden rise in hospital admissions for air pollution-associated respiratory illness; or there may be a need to make best available use of routinely available data in order to establish air pollution control priorities or plan services.

This Chapter discusses various approaches which can be used to conduct rapid appraisals, recognising that the specific needs of any one particular situation will determine which method or mix of methods will be optimal. Assessment methods which rely on obtaining information at the level of the individual, as well as at the group level, are discussed. In addition, the Chapter discusses collection of aggregate and individual level data needed for rapid appraisals, with emphasis on exposure assessment techniques.

As the relationship between air pollution and health effects is complex, and will depend on the circumstances in any one setting, rapid appraisals should not be considered as a one-off effort, but rather as part of an overall programme on air pollution health effects assessment and control.

5.1 INTRODUCTION

In developing countries throughout the World, especially in Latin America, eastern Europe and Asia, air pollution concentrations are reaching significant levels, yet information on the associated health effects is lacking. As a consequence, there is frequently little basis for decision-makers to prioritise among alternative control strategies and policies, in deciding which pollutants need to be controlled, in what way and to what extent.

The relationship between air pollution and health is complex, and will depend on a variety of factors and circumstances, all of which may vary from setting to setting, and from one population group or area to another. Data and information availability, as well as capacity, will vary from setting to setting.

In one area there may be a limited air pollution monitoring network in place, whilst in another a source emissions' inventory may have been compiled. In one setting there may be scanty health-related information available only from clinic records, whilst in a different setting sophisticated epidemiological studies may have been conducted on the health impact of air pollution. Not only may there be a lack of data on air pollution exposures or on health effects in a particular setting, but also it may be difficult to extrapolate results of studies from one setting to another.

5.1.1 Need for rapid appraisals

Decision-makers are often faced with the need to act on the basis of uncertain knowledge, and to make a rapid appraisal of the situation based on an optimal use of a variety of information and data sources, with a minimal amount of investment in sophisticated research studies or monitoring of air pollution health effects.

There may occur a range of differing circumstances for which a rapid appraisal may be necessary. There may be a need to establish priorities for air pollution control based on a situational analysis of the existing air pollutants in an area, and associated ill-health effects in the population or there may be a spill of toxic substances which requires rapid assessment of the potential exposures and health effects. There may be concern in a particular community about the potential ill health effects of emissions from a factory, causing speculation about an increase in respiratory disorders in young children and the elderly.

There may be a sudden marked increase in the number of hospital admissions for asthma which needs rapid assessment in terms of the potential role of air pollution. There may be an air pollution episode of widespread regional significance, such as the recent forest fires in south-east Asia, which demands immediate, rapid assessment and response. Each situation/ problem is different, and will require its own rapid assessment approach and response mechanism.

Some situations may require an assessment of the current or immediate past situation regarding the impact of air pollution on health, whereas other situations may require some future forecasting of the impact, or of a scenario-based impact of some anticipated future exposure (for example, impacts associated with alternative transport systems, or energy policies, or urban air pollution trends in developing countries).

In general, however, regardless of the precise circumstances for which the rapid assessment is needed, in setting and evaluating policies, standards, control strategies, and in planning for the provision of health services, consideration of a range of rapid assessment methods and approaches is often necessary.

5.2 RAPID EPIDEMIOLOGICAL ASSESSMENT

5.2.1 Environmental epidemiology

Epidemiology, the cornerstone of public health (Lilienfeld and Lilienfeld, 1980; Mausner and Kramer, 1985; Rothman, 1986; WHO, 1993a), is by definition concerned with the distribution and causes of diseases and ill-health effects in human populations. Environmental epidemiology is that sub-specialty of epidemiology which is concerned more specifically with the environmental determinants of diseases and ill-health effects, and in understanding the nature of the relationship between environmental exposures such as air pollution, and ill-health in population groups (WHO, 1983; Goldsmith, 1986; von Schirnding, 1997). Epidemiologic studies provide "real world" evidence of associations between air pollution and health, based on normal living conditions and exposure situations (WHO, 1996).

Environmental epidemiologists have been described as "canaries" (used in bygone days to detect toxic concentrations of carbon monoxide in mines), who are capable of giving warning of impending environmental disaster. Fortunately, their fate is not to die, as the unfortunate canaries of the coal miners did, "but to sing - to call out in clear tones the nature and type of impending health danger that threatens" (Goldsmith, 1988).

5.2.2 Development of rapid appraisal approaches

The US Academy of Sciences Advisory Committee on Health for Medical Research and Development first coined the term "rapid epidemiological assessment" in 1981. It has been described as a collection of methods which provides health information more rapidly, simply and at a lower cost than standard methods of data collection, yet also yields reliable results for use primarily at the local level (Anker, 1991).

The intention is thus to generate as reliable information as possible quickly, which at the same time is accurate and useful. Whilst there is no such thing as a "quick and dirty" method when it comes to obtaining valid information on the health effects of exposures in human populations, nevertheless it is indeed possible to modify and adapt conventional methods, techniques and approaches to make more appropriate use of data for decision-making in special circumstances.

Rapid epidemiological assessment (REA) represents a new approach to epidemiological research, drawing on well-known methods and stressing speed and simplicity, adaptation to local conditions, and the need to obtain information timeously at a level of precision demanded by decision-makers. It is thus a response to the need for timely and accurate information on which to base decisions.

Many of the traditional epidemiological methods are not well suited to an environment in which there are extremely limited financial resources, and in which there is a lack of people skilled in data collection and analysis (Anker, 1991). Epidemiological sampling techniques generally aim to obtain representative samples of fairly large areas. This usually involves many resources and time, and frequently results are not fed back quickly enough to influence action or decision-making processes. Thus, there is a need to find alternatives to the traditional methods.

Considering the implications for government and industry of instituting better control measures and policies to regulate pollutants, it is essential for rapid appraisals of air pollution and health to be conducted in as rigorous and unbiased a way as possible. It is inevitable, however, that some statistical precision will be sacrificed for the sake of speed and simplicity. Thus, strengths and weaknesses of each method need to be made explicit. Rapid assessments should not be considered as a one-off effort, but rather as part of an on-going process to be updated and developed over time.

In addition, it is important to realise that REA methods are frequently goal oriented to services and community needs, and are not necessarily geared to answer more fundamental questions on the nature of relationships between health and exposures. REA methods can be used under a variety of conditions, for example, to evaluate routine environmental health service functioning, or even during emergencies or times of crisis, for example, to target at-risk populations in need of attention (Guha Sapir, 1991) (Table 5.1).

Table 5.1 Rapid epidemiological assessment characteristics

- Rapid
- Simple
- Low-cost
- Minimal data requirements
- Aid in priority setting and planning
- Identify critical issues, problems, hotspots
- Minimal human resource requirements
- Results easily communicated and understood

REA focuses on two aspects of epidemiology - sampling methods which reduce the time and resources required to collect and analyse data from individuals, and methods for the collection, organisation, analysis and presentation of data at the community level (Anker, 1991). In all respects, an important aim is always to obtain information that will shed light on associations between exposures and ill-health effects that are worthy of further investigation, and to minimise the chances of drawing the wrong conclusions based on spurious associations.

A variety of epidemiological methods can be used to assess the relationship between air pollutants and exposures, some of which are sophisticated and time-consuming (not considered here), whilst others can be adapted for use in a rapid appraisal situation, depending on the particular circumstances in question.

Before considering some of the rapid epidemiological appraisal methods which one might use to obtain the necessary information on the relationship between air pollutants and ill-health effects, it is important to appreciate some of the key distinguishing factors relating to ill- health effects in relation to air pollution exposures, which will influence which methods to use in a particular setting, as well as the interpretation of the data.

5.2.3 Characteristics of air pollution-related health effects

Despite the fact that there is now a wealth of information on the health effects of air pollutants (WHO, 1987; WHO 1999a), there is still much uncertainty regarding the contribution of air pollutants, either directly or indirectly, or singly or in combination, to the health of people in differing circumstances.

Ill-health effects of air pollution exposures may occur over short or long periods of time, they may be reversible or irreversible, they may increase or decrease in time, and they may be continuous or temporary. They may be acute, for example, following relatively soon after an exposure (often a single major dose of a substance, such as may occur by accident or due to a chemical spill for example), or they may be chronic, occurring as a result of cumulative exposure to complex mixes over long periods of time.

A long period of time may elapse between the initial exposure and the appearance of an adverse health effect. Dispersal of the population at risk over time and the long incubation period make it difficult to reconstruct exposures. Acute health effects are thus often easier to detect than chronic effects, which may be difficult to relate to exposure to specific hazards or sources.

A hierarchy of effects may occur, ranging from minor, temporary ailments through to acute illness to chronic disease, with relatively resistant and susceptible persons at either extreme of the distribution. Outcomes may include death, specific defined diseases (for example, lung cancer, disease categories (respiratory illnesses such as pneumonia, asthma, bronchitis), symptom complexes (cough, wheezing), and biochemical/physiological changes which may not necessarily result in symptoms (for example, elevated levels of zinc protoporphyrin resulting from lead exposure)).

Infants and young children may be at particular risk, as they take in more of a contaminant relative to their size than do adults, and they have immature and therefore particularly vulnerable physiologies. The unborn foetus is particularly susceptible. Elderly people are also vulnerable from a physiological point of view (for example, they may be more susceptible to lung infections than younger people). The vulnerability of individuals (as opposed to groups) may vary, however, and a range of susceptibilities to hazardous substances may occur.

Linking health outcomes to possible exposures is complex. Most ill-health effects from air pollution are multifactorial insofar as the causal factors are concerned, and it may be difficult to determine the effects of one exposure in the light of the possible existence of simultaneous exposure to other factors. When dealing with low level exposures in particular, one may often be dealing with factors which play a contributory rather than a primary role in the causation of an increased incidence of disease. The co-action of other factors may be needed for effects to occur.

Effects may be interactive, resulting in a reductive, an additive or a synergistic effect where the combined effect is greater than the sum of the individual effects. Combined effects may often arise from the influence of nutritional, dietary and other lifestyle factors such as smoking and alcohol intake.

Air pollution health assessments, regardless of the overall design, need to take into account issues such as confounding (interfering) factors and sources of bias but some designs lend themselves better to dealing with such issues than others. Such designs are normally the more sophisticated and time-consuming to conduct, however.

Normally it would not be possible to assess whether associations between air pollution and ill-health effects were causal, through rapid appraisal methods. Several criteria exist for assessing whether an association is likely to be causal or not (Bradford Hill, 1965, Griffith *et al.*, 1993) (Table 5.2).

Table 5.2 Some criteria for establishing causality

- Strength of the association
- Specificity of the association
- Dose-response relationship
- Consistency of association
- Temporal relationship
- Biologic plausibility

5.3 INDIVIDUAL LEVEL ASSESSMENT METHODS

In this section some methods for assessing health effects in relation to exposure are discussed (for a general discussion of methods see also Lilienfeld and Lilienfeld (1980); WHO (1983); Mausner and Kramer (1985); Rothman (1986); WHO (1993a)). These rely on information at the level of the individual, as opposed to the group. Of importance is that in all studies sources of exposure are well identified, as well as populations at risk such as children and the elderly. The more the data is capable of being analysed in this way, the better the chances of developing control measures which are targeted at high risk population groups and areas.

5.3.1 Intervention study

Occasionally unusual circumstances may present themselves, in which it may be feasible to conduct some form of a rapid intervention study, for example in a situation where a corrective action was tried out in reducing exposure, and it was possible to assess the health effects accordingly. An example would be the addition of scrubbers to an industrial plant, and the monitoring of exposures and ill-health effects in the surrounding area, both prior to the intervention, as well as subsequent to it. This would provide important data on the nature of potential air pollution-related health effects.

5.3.2 Cohort study

The situation could also arise where it was possible to conduct a cohort (longitudinal or follow-up) study, which would involve following up defined population groups (exposed and unexposed respectively) over a certain period of time for a presumed health outcome, and measuring exposures (and other potential influencing factors) along the way. An example would be a study in which two comparable disease-free population groups, one exposed and one unexposed to indoor air pollution, were followed up over a period of time and the incidence of respiratory disorders in the two groups determined (see Armstrong and Campbell (1991) for one example of this type of study design that might be adaptable).

In general, cohort studies allow for a more complete investigation of complex exposures or multiple outcomes, and can be used when detailed information becomes available about the exposure to characterise it effectively. They are, on the other hand, normally costly to conduct and involve a considerable amount of time and resources; therefore, they would not lend themselves easily to adaptation for a rapid appraisal, unless the health outcome of interest occurred relatively quickly, thus limiting the follow-up time necessary. One could also do such a study based on historical records, for example by assembling exposed and unexposed groups on the basis of hospital records and following them up to the present and determining disease status. This would involve considerable time-saving.

5.3.3 Case control study

A case control study can yield important information fairly rapidly, if carefully conducted (Baltazar, 1991). This involves starting with a diseased population (i.e. in this case a population with well-documented air pollution related ill-health effects or symptomatology) and working backwards in time to determine or reconstruct the prior exposure. In pressing environmental problems, where timeliness of findings may be important, the case control study, being relatively quick to conduct in many circumstances, may be appropriate. They can be rapid and efficient and provide reliable results when confounding (the influence of extraneous variables not under prime consideration for the purpose of the study at hand) is properly addressed.

Where it is possible to adequately classify cases and controls, this type of design can be used. An example would be a study which looked at exposure to indoor air pollution among children with pneumonia (cases) compared to children who were disease-free (controls) (see Robin *et al.* (1996) for one example of this type of study design which might be adaptable). An environmental "outbreak" investigation would be a special application of this methodology, for example in a situation where there occurred a sudden release of a toxic substance or spill. Of critical importance in such studies is that the exposure information is accurately assessed. When relying on past exposure information, this may be difficult.

5.3.4 Cross-sectional study

A cross-sectional study is one in which a sample of the study population is investigated and the exposures and outcomes determined almost simultaneously in time. The information sought can be current (for example, relating to prevailing air pollution exposures) or past (relating to exposures to air pollution in the past). Whilst this type of design is of limited use in assessing the nature of potentially causal relationships, due to the problem of the time sequence of events, they nevertheless have the advantage in that the relationship between several exposures and outcomes can be studied. These types of studies are often the first approach used in assessing relationships.

An example would be a study in which a questionnaire is distributed to a cross-section of a community to obtain information on various potential exposures and health outcomes, such as respiratory ill-health symptomatology and exposure to traffic, industry and indoor air pollution. Several communities or locations could be compared in this way. They provide a picture of the overall situation at a point in time, and can be rapid and inexpensive to conduct. If very large areas are to be sampled, areas within the region can be randomly selected, for example using multistage or stratified sampling techniques (see Pope and Dockery (1996) for examples).

5.4 GROUP LEVEL ASSESSMENT METHODS

These methods rely on obtaining information at the level of the group, as opposed to the individual, and therefore can be fairly rapidly conducted.

5.4.1 Ecological study

Ecological studies are weak in determining causality, as they involve measuring exposures and outcomes at the group level, as opposed to the individual level, and making inferences about the relationship of exposures and outcomes in individuals, based on information obtained at the level of the group. Often there may not be homogeneity of exposure of individuals in an area, although all individuals in an assigned exposure category will be assumed to be equally exposed (Greenland, 1992). This could lead to what is referred to as the "ecological fallacy". Thus, an apparent association at the group level may not hold at the individual level. Where air pollution levels are fairly uniformly distributed in an area, and microvariations of little significance, this type of problem is less likely to arise.

Other problems relate to the fact that information on potentially confounding factors is frequently absent, thus the relationship between exposure and outcome may be distorted. In addition, exposure and outcome data are usually not available for exactly corresponding areas. Thus, it may be difficult to convert existing health and environmental data into corresponding units of analysis. Frequently, proxy (substitute) measures of exposures and outcomes are used. Nevertheless, despite their limitations, they are relatively easy and quick to conduct using existing databases.

Studies which have relied on this type of design include cancer studies in which, for example, lung cancer rates in different parts of a region are analysed in relation to air pollution levels estimated on the basis of air monitoring data in the region, or in relation to types of industry in the region. They can often yield useful information and early clues, which can then be further pursued using different study designs.

5.4.2 Geographic Information Systems (GIS)

These can be used to organise, analyse and present data at the community level. They can range from very sophisticated and well developed systems which require substantial inputs in terms of data and equipment, to very simple systems which can be run on microcomputers and economical, user-friendly software (Scholten and de Lepper, 1991). Whilst a large scale GIS is not a rapid assessment method in itself, nevertheless once the initial investment of setting up such a system has been made, information can be retrieved quickly. In addition, in a rapid assessment, it is extremely useful to be able to draw on the facility of a GIS to present data in map form. Maps are easy to understand and use which makes them an attractive communication tool (Anker, 1991).

The GIS can be very useful for providing a method of analysis which relates specifically to the geographical component of the data. At the simplest level, data about different spatial entities such as land use and air pollution can be combined by overlay analysis. At an intermediate level GIS may allow statistical calculations of the relationship between datasets to be computed. The most sophisticated analysis occurs when modelling is introduced.

Atmospheric modelling techniques can be used to discover which areas might be affected by pollution resulting from an explosion at a particular hazardous installation, for example Chernobyl, given certain wind and weather conditions. It can also be used to assess the impact of locating a specific industrial development in different sites in a city or region.

The compilation of maps and atlases can show how patterns of spatial distribution of disease can be revealed. They can be useful for looking for clusters of diseases in relation to industries such as nuclear reprocessing facilities or hazardous waste incinerators. GIS has been used, for example, in looking at the relationship between perinatal mortality and radiation fallout from Chernobyl. It provides a method to perform various tasks more quickly and with less effort, and provides researchers with new, reliable, scientifically valid methods for handling spatial information (Scholten and de Lepper, 1991). Costs of hardware have declined substantially and a range of simple, introductory level systems now exists.

5.4.3 Time-series study

A variation on these designs is the time-series study, in which changes in health outcome in an area are looked at in relation to changing air pollution levels (Dockery and Pope, 1996). For example, daily hospital admissions or emergency room visits could be assessed in relation to daily air pollution levels (for example, particulates) and acute effects such as asthma examined. As these studies are concerned with examining changes in air pollution levels and associated health effects, extraneous or confounding factors (for example, smoking) are more effectively controlled because they would not be expected to vary in the same manner as the exposures under consideration. Whilst these studies are useful for studying the relationship between transient exposures and acute health effects, they are not, however, of use in studying chronic health effects. They can also be statistically and computationally demanding.

Such studies have been used to assess the relationship between daily mortality, air pollution and weather (Dockery and Pope, 1996; WHO, 1996), and were used to assess the impact of major air pollution episodes such as the London smog disaster in the 1950s. They also have application in studying the effects of the recent air pollution episodes in Asia and Latin America, caused by forest fires.

5.4.4 Sentinel surveillance

Surveillance refers to the need for continual monitoring and observation of the distribution and trends of selected health outcomes and exposures, with a view to acting when certain limits are passed. It is needed to continually monitor change. This is particularly important during a period of rapid urbanization and industrial development, for example when there could be significant impacts on health arising from air pollution. Frequently, however, the data collected are not presented in a way which facilitates rapid action or which informs decision-making - on occasion too much data are collected, with loss of quality and accuracy, or too little data are collected, or data are collected too infrequently, or at inappropriate sites, or in such a way that an analysis or action is not timely. Frequently, the best surveillance is found where the risk is least.

Because of the lack of resources and the burden of routine surveillance, sentinel surveillance sites can be identified in order to measure the health status of the population without studying the entire population, or the concept could be expanded to include a list of tracer health conditions which could trigger a study. This could be useful in emergency situations, where time constraints are critical (Guha-Sapir, 1991) and early warning signs of pending disasters are needed, based on key indicators and sentinel surveillance systems.

5.5 RISK ASSESSMENT

Occasionally, there may be no health data or possibility of obtaining such data; in this case a risk assessment may need to be conducted. Risk assessment has been widely used as a basis for setting standards, and has primarily involved three major categories of human health effects, namely carcinogenicity, developmental toxicity and neurotoxicity. The methodology, however, has broad relevance and applicability to other situations in which there is a lack of data on health effects in a particular setting.

Risk assessment has been defined by Griffith *et al.* (1993) as "the characterization of potential adverse health effects of human exposures to environmental hazards". It involves the following steps, as first recommended by the US National Academy of Sciences (National Research Council, 1983): 1) Hazard identification; 2) Dose-response assessment; 3) Exposure assessment; 4) Risk characterisation.

Hazard identification is concerned with establishing whether an agent actually causes a specific effect. Dose-response assessment is concerned with establishing what the relationship is between the dose or exposure, and the incidence of ill-health effects in humans, whilst exposure assessment is concerned with identifying what exposures are currently experienced or anticipated under different conditions. Risk characterisation involves the determining of the estimated incidence of the adverse effect in a given population.

5.5.1 Hazard identification

Here one is concerned with establishing whether causal relationships exist between various exposures and ill-health effects. As already discussed, this is a complex process. There are many factors that characterise the nature of the relationship between exposure to air pollution and health effects, which should be taken into account in trying to impute the nature of associations. On occasion there may be very little epidemiological information available, and therefore reliance must be placed on toxicological studies, or on a combination of both epidemiological and toxicological studies.

5.5.2 Dose-response

One is concerned here in establishing whether at increasing levels of exposure or dose there is a corresponding increase in the frequency of disease, or in the severity of disease. This step involves the quantitative aspect of the assessment. For example, lung cancer incidence is known to rise in relation to cigarette smoking. Another example is lead exposure, where it may be possible to show a rise in the prevalence of raised blood lead levels with increasing levels of lead in petrol, or an increase in the incidence of neurobehavioural disorders in infants with increases in cord blood lead levels.

Information is usually obtained on the basis of the epidemiological literature, supported by animal toxicological and clinical data. Those scientific studies which are likely to show the best evidence of an effect would be selected for assessment. Whilst no single study is likely on its own to be definitive, duplication of results across several studies and a range of exposures and health outcomes are strong evidence of a causal relationship (see earlier discussion on this aspect), and can be used for establishing the nature of the dose-response effect.

There is a wealth of scientific literature which can be consulted for this purpose, including the WHO air quality guidelines (WHO, 1987; WHO 1999a) and various environmental health criteria documents on specific pollutants. Unfortunately, most of the data is derived from studies carried out in developed countries; nevertheless, there are now several studies from developing countries which can also be consulted (see also accompanying Chapters). Extreme caution needs to be exercised in extrapolating results from developed countries, as factors such as susceptibility of groups at risk, influencing factors such as diet and nutrition, and the role of background factors in the home, work and community environment, are likely to differ significantly.

5.5.3 Exposure assessment

Here one is concerned with providing an estimate of human exposure levels from all potential sources for particular population groups under consideration in the assessment. Of critical importance is that major pollution sources in terms of population exposures are well identified and characterised in order that control strategies can be developed. One would need to provide an assessment of the size and composition of the population groups potentially exposed, and the types, magnitude, frequency and duration of exposure to the various agents of concern. All pathways of exposure would need to be assessed, for example not only the direct inhalation pathway, but also, in some cases, indirect pathways of air pollution exposure such as via food, water or the skin. This is one of the most challenging issues in air pollution epidemiology, due to the complexities involved in estimating exposures, particularly personal exposures.

5.5.4 Risk characterisation

This involves estimating what the incidence of an adverse health effect is likely to be that can be expected from exposure to a specific air pollutant. The information obtained on people's exposures can be used to determine how much of an agent will reach people, and, combining this information with what is known of the dose-response relationship, based for example on scientific assessments and reviews, can be used to characterise people's risks. Essentially, in this step the information from hazard identification, dose-response relationships and exposure estimates is integrated to determine the probability of risk to humans.

5.6 COLLECTION OF INDIVIDUAL AND AGGREGATE LEVEL DATA

In all of the above assessments, data need to be collected on health outcomes and exposures, either at the individual or at the group level. If there is an interest in morbidity, in cases where formal disease registries exist, for example for cancer, these can be utilised. Other routine health surveillance and recording systems could also be used, for example hospital records (admission or discharge records), clinic or health service records, school and workplace records, or routine data on infectious diseases such as pneumonia. If there is an interest in mortality, most countries have routine data on causes of death, although cause-specific mortality may be subject to misclassification.

In situations where such data sources are limited, special surveys may be needed. The particular methods and techniques used to assess health effects would depend on the health effect of interest.

5.6.1 Focus group discussions and questionnaires

Questionnaires and focus group discussions may be used to obtain a quick impression of potential health effects (and exposures) in communities fairly rapidly. Data can be collected on an aggregate level in many ways, including using groups of experts, community leaders, individuals from the community, in-depth interviews with selected individuals, etc. Focus group interviews can be used to obtain important in-depth qualitative data, for example to examine local perceptions. Simple self-administered questionnaires can also be distributed, which can form the basis for more substantial quantitative studies. They can be very useful for investigating people's knowledge, attitudes and behaviour, and can be important to conduct prior to designing interventions.

In addition, they can be useful for pinpointing at-risk areas or populations at risk, or issues in need of further study. Often they are used as a complement to a quantitative study. They are useful also in providing background information and to generate hypotheses for field testing, but would rarely be used as a stand-alone method (Khan *et al.*, 1991).

Questionnaires are also an invaluable method in obtaining information from key informants in the community (Lengeler *et al.*, 1991), and can be a useful alternative to the direct interview approach. Self reports of symptoms are often used as a health outcome in studies with uncertain exposures and few or no objectively measurable health effects. However, over-reporting of symptoms may occur in groups who are aware of their exposure to air pollution (Mendell, 1990). Examples of such situations include communities worried about toxic waste site exposures, or office workers concerned about indoor air pollution for example.

5.6.2 Exposure assessments

5.6.2.1 Multiple sources and pathways

There are normally multiple sources and pathways of exposure which would need to be assessed. For example, people could become exposed to lead in petrol via the air they breathe,

the food they eat, or via the soil and dust that may get ingested. Often one pathway contributes the major proportion of the pollutant. Should this critical pathway not be identified, the multiple pathways contributing to the total exposure must be carefully assessed. Adequate control measures can only be applied when the relative importance of the various routes of exposure has been established.

People may be exposed to various sources of the same agent, in addition to the various pathways of uptake. For example, lead may be present in petrol, paint or drinking water. Often there may be multiple sources in differing environments that may contribute to the same health outcome, for example in the domestic (home) environment, the local or community environment, the school or work environment, etc. (von Schirnding *et al.*, 1990). For young children and women, the indoor environment may be a particularly important source of exposure, especially in developing countries where exposures to biomass and coal may be significant (Smith, 1987); for adults, the workplace may be an important source of exposure. Thus, it is important to assess an individual's total exposure in the various environments in which exposure may take place, as well as to identify other substances which may modify its effects.

5.6.2.2 Variations in time and space

Exposures may also vary considerably in time and space. For example, for many pollutants there is a sharp decrease in concentration level as one moves away from a source. There may also occur significant vertical variations in the concentration level of a pollutant. For example, air sampling points placed considerably above breathing level may be safe from vandalism but are inappropriate to use in population exposure studies.

Similarly, there may occur temporal variations (seasonal, daily and diurnal) in the level of pollution. For example, pollutant levels may vary throughout the day with respect to particulate levels associated with biomass burning for cooking and heating. These may reach peak levels in the early morning and early evening when cooking activities take place.

There may also be long-term variations in exposure over time, during which sources (and pathways) may have changed. In investigating acute effects (see earlier discussion on this aspect), the current exposure level may be adequate, but in studying chronic effects (after a long exposure period or latency period) past exposure concentration levels are important, as well as the duration of exposure.

Problems in relying on both historical and current environmental measurements may arise due to the fact that the measurement technology may have changed over time, with current technology increasingly able to measure at lower detection levels.

5.6.2.3 Techniques for assessing exposure to air pollutants

There are many ways, both directly and indirectly, of classifying a person's exposure - it could be on the basis of the nearest air pollution monitor, on a weighted average of all the monitors in an area, on some dispersion modelling scheme in which different areas are designated different values, on the basis of source emissions data, on the basis of personal exposure measures, or merely on the basis of a residential classification scheme. It can be done at the aggregate level or at the individual level.

The specifics of an exposure assessment will depend on its purpose, i.e. the nature of the information required, and the quantity and quality of the data required, will depend on the context for which it is needed. Whilst here the main concern is in obtaining an overall assessment of exposure as rapidly as possible, nevertheless the way in which air pollutants are monitored will always be a very critical aspect to take into account (UNEP, 1994). A great deal of monitoring information is of limited use, due to the fact that it may not be relevant to where people are exposed, or pollutants may not be measured frequently enough.

The problems of relying on stationary environmental monitoring schemes include: pollutants are typically measured at only a limited number of sites; and often the schemes are designed to determine compliance with air quality standards and not to assess exposure. Thus, they may not provide estimates of average pollution levels to which people are typically exposed, but would be useful for other purposes, for example in assessing long-term trends and emissions from point sources. Dispersion modelling can also be used to obtain more reliable estimates of air pollution exposures.

5.6.2.4 Personal sampling

Where microvariations in pollutant levels are considerable it may be more appropriate to use personal air samplers or filter badges rather than stationary air samplers. These have the advantage of being mobile and have the potential to measure an individual's total exposure. Examples of personal monitoring include diffusion tubes for passive sampling of gases, or filters with battery-operated pumps for active sampling of aerosols.

If large populations are being monitored however, such samplers may not be practicable to use on a large scale and exposures might be better characterised at group level using stationary samplers. They could be very useful, however, in small area studies or in studies of particular risk groups such as young children or workers. Radiation dosimeters are an example of this type of monitoring. In general, however, they tend to be relatively expensive and labour intensive, sometimes requiring fairly sophisticated analytical procedures and laboratory facilities, as well as detailed information on time activity patterns (WHO, 1999b).

5.6.2.5 Proxy measures and source emissions inventories

Where no air pollution measurements exist, proxies can be used, such as place of residence (for example, urban, sub-urban, inner city, industrial zone). Dispersion modelling can also be used, provided there are relatively accurate inventories of emission sources. In Jakarta, for example, dispersion models, taking into account local meteorological and topographical features, have been used to determine ambient concentrations throughout the region, and individuals' assigned exposures based on their place of residence (WHO, 1996). Weighted exposures might also be assigned, based on residence and workplace for example.

Many methods for assessing sources of air pollution exist, for example in calculating pollution and waste loads. Whilst detailed and precise source emission inventories can be resource intensive, involving also sophisticated monitoring and data processing systems, by using limited existing information, it is possible to make fairly accurate emission inventories at fairly low cost (WHO, 1982).

The methods involve obtaining information on types and sizes of waste and pollution sources, as well as information on their location (for example, in relation to population centres); pollution and waste loads can then be calculated on the basis of pollution and waste factors for the various sources. Many factors need to be taken into account including, for example, the source type, age, technological sophistication, process or design particularities, source maintenance and operating practices, raw materials used, control systems employed etc. (WHO, 1993b).

Separate inventories could be made for areas with point sources and areas with mobile sources. Estimated emissions for stationary combustion sources, mobile combustion engine sources, industrial processes, waste disposal processes, etc. could be tabulated, and contributions of sources to air pollution loads estimated, taking into account meteorological conditions and location of sources. This can be conducted fairly crudely, or it can involve very sophisticated source apportionment studies. Decisions need to be made in relation to whether data are required for individual sources, or for groups of sources - in situations where there are a few large sources such as electric power plants, individual level data are probably required, whereas in situations where there are numerous small sources such as space heating furnaces, joint calculations will be necessary (WHO, 1993b).

Various tools for rapid assessment of air pollutants have been developed (see also WHO's Air Management Information System (AMIS), available on CD Rom; and the Decision Support System for Industrial Pollution Control (DSS IPC)) and mostly include information on emission factors for various sources, models for pollution dispersion taking into account meteorological data (for example wind speed and direction, temperature), and methods for estimating pollutant concentrations. Thus, the steps involved would include making an inventory of key air polluting activities, compiling an inventory of emissions of pollutants based on emission factors, and calculating ambient concentrations based on dispersion modelling for example. Where direct emissions data are missing, various sources can be consulted on industrial emission factors, or on transport emission factors for example, which are based on data for various fuels and vehicles.

5.6.2.6 Biological markers of exposure

There is growing interest and increasing research into biological and biochemical markers of exposure (WHO, 1993c) which should improve the effectiveness of exposure assessments in the future. Concentrations of air pollutants in body fluids or excreta may be measured, for example biological monitoring of blood, urine and hair can be done. These can also be useful for estimating past exposure levels, for example measurement of lead levels in bone or hair. The advantage is they provide integrated measures of exposures from all sources and pathways. Those most suitable would be ones that are chemical specific, detectable in trace quantities, available by non-invasive techniques (e.g. sampling of urine), and inexpensive to assay (WHO, 1999b). Sources of biological variability need to be taken into account in interpreting the data (age, sex, body size, fat distribution, lifestyle factors, other sources of exposure).

5.6.2.7 Summary

In summary, exposure information provides the critical link between sources of contaminants, their presence in the environment, and their health impacts. Assessments can be direct or indirect, based on monitoring, and interpolation of data from monitoring sites, source emissions inventories and dispersion modelling, or can even be based on questionnaire data at the individual level (see earlier discussion on this aspect) or on biological markers in individuals.

Ultimately the exposure data must be summarised - the choice of an appropriate summary measure may be critical to the ultimate understanding of the exposure. In one instance average exposure level may be appropriate, whilst in another the use of peak values may be important. Cumulative exposures may be of significance in the assessment of, for example, radiation exposure where multiple exposures may be largely cumulative. Thus, one might choose the average, peak, percentile, frequency of exceedance of a specified level, or cumulative duration of exceedance.

5.7 CONCLUSIONS

The relationship between air pollution and health is complex. A wide variety of factors influence the association between exposures and health effects in human populations in any one setting or at-risk group. Decision-makers are frequently faced with the need to make rapid appraisals of situations, often based on sparse data, either on exposure, on health effects, or on their associations.

This Chapter has discussed the need for rapid appraisals, the circumstances in which they may be necessary, their distinguishing characteristics, and some of the assessment methods which can be used, relying on information either at the level of the individual or at the group level. The specifics of the situation will determine the method(s) to be used.

Ultimately, there is no replacement for well-designed epidemiological or risk assessment studies. Rapid appraisals should thus never be considered in isolation, but should rather be seen as part of an overall air pollution health effects assessment programme which is updated and developed over time.

5.8 REFERENCES

Anker, M. 1991. Epidemiological and statistical methods for rapid health assessment. *World Health Statistics Quarterly* **44**(3).

Armstrong, J. and Campbell, H. 1991. Indoor air pollution exposure and lower respiratory infections in young Gambian children. *International Journal of Epidemiology* **20**(2).

Baltazar, J. 1991. The potential of the case control method for rapid epidemiological assessment. *World Health Statistics Quarterly* **44**(3).

Bradford Hill, A. 1965. The environment and disease: association or causation? *Proceedings Royal Society of Medicine* **58**.

Dockery, D. and Pope, A. 1996. Epidemiology of acute health effects: summary of time-series studies. In *Particles in Our Air. Concentration and Health Effects* (ed. by R .Wilson and J. Spengler). Harvard University Press, Geneva, Switzerland.

Goldsmith, J. 1986. *Epidemiological Investigation of Community Environmental Health Problems*. CRC Press Inc, USA.

Goldsmith, J. 1988. Improving the prospects for environmental epidemiology. *Archives of Environmental Health* **43**(2).

Greenland, S. 1992. Divergent biases in ecologic and individual-level studies. *Statistics in Medicine* **11**.

Griffith, J., Aldrich, T. and Drane, W. 1993. Risk assessment. In *Environmental Epidemiology and Risk Assessment* (ed. by T. Aldrich and J. Griffith). Van Nostrand Reinhold, New York.

Guha-Sapir, D. 1991. Rapid assessment of health needs in mass emergencies: review of current concepts and methods. *World Health Statistics Quarterly* **44**(3).

Khan, M., Anker, M., Patel, B., Barge, S., Sadhwani, H. and Kohle, R. 1991. The use of focus groups in social and behavioural research: some methodological issues. *World Health Statistics Quarterly* **44**(3).

Lengeler, C., Mshinda, H., de Savigny, D., Kilima, P., Morona, D. and Tanner, M. 1991. The value of questionnaires aimed at key informants, and distributed through an existing administrative system, for rapid and cost-effective Health Assessment. *World Health Statistics Quarterly* **44**(3).

Lilienfeld, A. and Lilienfeld, D. 1980. *Foundations of Epidemiology*. Oxford University Press, UK.

Mausner, J. and Kramer, S. 1985. *Epidemiology: An Introductory Text*. W.B. Saunders and Co., USA.

Mendell, M. 1990. *Data in Setting with Likely Over-reporting due to Environmental Worry*. 2nd Annual Meeting of the International Society for Environmental Epidemiology, USA, 12-15 August.

National Research Council. 1983. *Risk Assessment in the National Government: Managing the Process*. National Academy Press, USA.

Pope, A and Dockery, D. 1996. Epidemiology of chronic health effects: cross-sectional studies. In *Particles in Our Air. Concentration and Health Effects* (ed. by R. Wilson and J. Spengler). Harvard University Press.

Robin, L., Less, P.,Winget, M., Steinhoff, M., Moulton, L., Santosham, M. and Correa, A. 1996. Wood-burning stoves and lower respiratory illnesses in Navajo children. *Paediatric Infectious Disease Journal* **15**(10).

Rothman, K. 1986. *Modern Epidemiology*. Little, Brown and Co., USA.

Scholten, H. and de Lepper, M. 1991. The benefits of the application of geographical information systems in public and environmental health. *World Health Statistics Quarterly* **44**(3).

UNEP/WHO. 1994. *GEMS/AIR Methodology Review Handbook Series*. Vols. **1**, **2** and **3**. Nairobi, Kenya.

von Schirnding, Y. 1990. Methodological issues in air pollution epidemiology. *Proceedings 1st IUAPPA Regional Conference on Air Pollution*. South Africa, 24 - 26 October.

von Schirnding, Y. 1997. Environmental epidemiology. In *Epidemiology: A Manual for South Africa* (ed. by J. Katzenellenbogen, G. Joubert and S.S. Abdool Karim). Oxford University Press, Southern Africa.

von Schirnding, Y., Bradshaw, D., Fuggle, R;. and Stokol, M. 1990. Blood lead levels in inner-city South African children. *Environmental Health Perspectives* **94**: 125-130.

WHO. 1982. Rapid assessment of sources of air, water and land pollution. *WHO Offset Publication* No. **62**. Geneva, Switzerland.

WHO. 1983. Guidelines on studies in environmental epidemiology. *Environmental Health Criteria* **27**. Geneva, Switzerland.

WHO. 1987. Air quality guidelines for Europe. *European Series* **23**. Copenhagen, Denmark.

WHO. 1993a. *Basic Epidemiology*. Geneva, Switzerland.

WHO. 1993b. *Assessment of Sources of Air, Water and Land Pollution*. WHO/PEP/GETNET/93.1-A.P

WHO. 1993c. *Biomarkers and Risk Assessment: Concepts and Principles*. EHC 155. Geneva, Switzerland.

WHO. 1996. *A Methodology for Estimating Air Pollution Health Effects*. Geneva, Switzerland.

WHO. 1998. *Methods for Health Impact Assessment in Environmental and Occupational Health*. Geneva, Switzerland.

WHO. 1999a. (Revised) *Air Quality Guidelines for Europe* (2nd Edition) (in press). Copenhagen, Denmark.

WHO. 1999b. *Human Exposure Assessment* (Final Unedited Version). Geneva, Switzerland.

Chapter 6

A SYSTEMATIC APPROACH TO AIR QUALITY MANAGEMENT IN ASIAN CITIES: EXAMPLES FROM THE URBAIR CITIES

Steinar Larssen
Norwegian Institute for Air Research

Huib Jansen, Xander A. Olsthoorn
Institute for Environmental Studies, The Free University,
Amsterdam, The Netherlands

Jitendra J. Shah
The World Bank, Washington, D.C.

Abstract

Urban air quality management in megacities is a complex task, which to be effective requires a sound understanding of the causes and effects of air pollution and its various components. As indicated in Chapter 4, the factual basis should ideally include data on emissions, actual air quality, source-exposure-effects relationships and assessment of damage and its costs. Such information can be used to construct a coherent action plan, with control measures prioritized according to cost-effectiveness or cost-benefit ratios. With continued monitoring it is possible to assess the results of the measures selected and implemented. The URBAIR project, financed through the World Bank, employed an Air Quality Management System approach to help develop action plans in four large cities in Asia (Jakarta, Kathmandu, Manila and Mumbai). This Chapter draws on the experience of the URBAIR project, and describes the procedures involved and the policy recommendations.

6.1 URBAN AIR QUALITY MANAGEMENT AND THE URBAIR PROJECT

The Urban Air Quality Management Strategy (URBAIR) project developed a systematic approach for the design and implementation of policies, monitoring systems and management mechanisms to restore ambient air quality in metropolitan areas. This approach has been applied and tested in Jakarta, Mumbai and Metro Manila, as well as in several non-Asian cities. Other metropolitan authorities in South Asia can learn from this experience.

The URBAIR project adopted an air quality management strategy (AQMS) involving the following steps:

- air quality assessment;
- environment and health damage assessment;
- abatement options assessment;
- cost-benefit analysis or cost-effectiveness analysis;
- abatement measures selection (action plan);
- design of optimum control strategy.

The main elements of the approach can be grouped as follows:

Assessment: Air quality assessment, environmental damage assessment and abatement options assessment provide input to the cost analysis, which is also based on established air quality objectives (e.g. air quality standards) and economic objectives (e.g. reduction of damage costs). The analysis leads to an Action Plan containing abatement and control measures for implementation in the short, medium, and long term. The goal of this analysis is an optimum control strategy.

The AQMS depends on the following set of technical and analytical tasks, which can be undertaken by the relevant air quality authorities:

- creating an inventory of polluting activities and emissions;
- monitoring air pollution and dispersion parameters;
- calculating air pollution concentrations with dispersion models;
- assessing exposure and damage;
- estimating the effect of abatement and control measures;
- establishing and improving air pollution regulations and policy measures.

These activities, and the institutions necessary to carry them out, constitute the prerequisites for establishing the AQMS.

Action plans and implementation: Categories of "actions" include the following:

- technical abatement measures;
- improvements of the factual data base (e.g. emission inventory, monitoring, etc.);
- institutional strengthening;
- implementing an investment plan;
- awareness raising and environmental education.

Monitoring: A third essential component of AQMS is continued monitoring, or surveillance. Monitoring is essential to assessing the effectiveness of air pollution control actions. The goal of an Air Quality Information System (AQIS) is, through thorough monitoring, to keep authorities, major polluters and the public informed about the short- and long-term changes in air quality, thereby helping to raise awareness; and to assess the results of abatement measures, thereby providing feedback to the abatement strategy.

Figure 6.1 describes how the necessary activities of an AQMS system should be linked together in an integrated system that enables abatement measures to be prioritized on the basis of cost-efficiency or cost-benefit analysis.

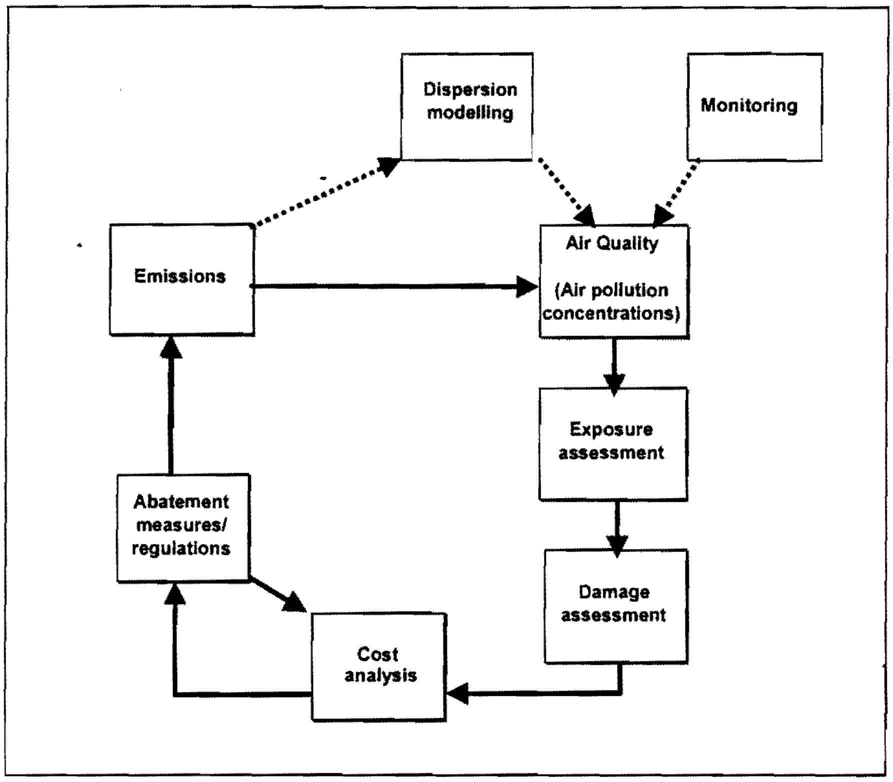


Figure 6.1 The system for developing an Air Quality Management Strategy (AQMS) based upon assessment of effects and costs

The URBAIR Guidebook (WB, 1997a) gives a detailed description of the methodologies that can be used to carry out these activities. Where a methodology is described in this chapter, without a reference, please refer to the URBAIR Guidebook.

In the URBAIR project, which was carried out in 1993-1996, action plans for improved air quality were developed for four Asian cities: Jakarta, Kathmandu, Manila and Mumbai (WB, 1997 b, c, d, e). In the following, the process of developing an air quality management strategy is described briefly, using examples from the four URBAIR cities.

6.2 PHYSICAL ASSESSMENT

6.2.1 Assessment of present air quality, and choice of air quality indicators

The starting point for the air quality improvement study is to assess present air quality. If data of adequate quality are not available, a monitoring programme must be established (see e.g. WB 1997a; Larssen, 1998). It should be emphasized that it is very important that the data are of known and acceptable quality (Larssen and Helmis, 1998b). The choice of pollutants to be used as indicators of the air quality situation depends upon the composition and extent of sources in the city. Experience with air quality assessment in Asian cities indicates that, in general, SO_2, NO_x and NO_2, ozone and particulate matter (PM) are the urban pollutants responsible for most of the potential damage (WHO, 1992). Air quality guidelines are available for these compounds, and much effort has been put into developing dose-response relationships for damage assessment. Another pollutant given increasing attention is benzene.

For the URBAIR cities there were data available from various measurement campaigns, and monitoring systems were in routine operation in all of the cities except Kathmandu. The URBAIR project concentrated on the assessment of damage to health, and on the compounds SO_2, NO_x, NO_2 and PM.

The air quality assessment indicated that the PM problem was the most important in all cities. Table 6.1 gives a brief overview of the total suspended particles (TSP) concentrations measured in each city. In Mumbai, Manila and Jakarta, TSP measurements are made typically every 6th day at a number of stations, while in Kathmandu the data are from a 3-month measurement campaign at several stations in 1993.

For assessment of health effects, PM_{10} is a more appropriate measure of suspended particles than TSP. PM_{10} measurements were scarce in these cities. Using a commonly applied 'rules of thumb', involving ratios of PM_{10} to TSP of between 0.5 and 0.6, it was found that annual PM_{10} concentrations in the cities would be up to 80-140 $\mu g/m^3$. Maximum 24-hour PM_{10} concentrations would run as high as about 400 $\mu g/m^3$.

As indicated in Chapter 4, recent evidence indicates that there may be no concentration level below which there are no health effects of PM_{10} (WHO, 1994; 1996). Nevertheless, the European Union (EU) has target values (EU, 1998). (The Directive was adopted on 22 April, 1999, and given the number 1999/30/EC.) The target for annual average of PM_{10} is 30-40 $\mu g/m^3$ (to be reached in 2005 and 2010 respectively), while the 24-hour target is 50 $\mu g/m^3$, which can be exceeded a certain number and times per year. The 24-hour target value corresponds to a maximum 24-hour value of some 80-100 $\mu g/m^3$. The US EPA has proposed similar standards (US EPA, 1997).

The prevailing PM guideline and target values indicated that PM was the most serious ambient air pollution problem in these cities, and this was used as indicator for assessing the potential health damage caused by air pollution.

Table 6.1 Summary of measured TSP concentrations ($\mu g/m^3$) in four URBAIR cities

	Mumbai 1992/93	Manila 1990-92	Jakarta 1991	Kathmandu 1993
Annual average All stations	223 (118-265)	174 (114-255)	291 (159-648)	253 (87-430)
24-hour average Max. at any station	-	823	840	867
Range of max. at each station	-	247-823	540-840	102-867

6.2.2 Emissions to air

It is important to put sufficient resources into the establishment of emission inventories in cities. This is one of the main pillars of a thorough air quality assessment. For the four URBAIR cities, it turned out that the inventory of vehicle exhaust emissions and the contributions from various vehicle categories could be made fairly complete, because all cities had reasonable data on the vehicle fleet and fuel consumption. In addition, some traffic data were available from the main road network. This is likely to be the situation in a number of South Asian cities. A first estimate of emission factors can be selected from the literature (e.g. Faiz *et al.*, 1996; WB, 1997b), and the same factors were used in all the URBAIR cities. The basis for calculating emissions from industry is less likely to be complete. Even if industrial fuel combustion emissions can be reasonably well estimated, the spatial distribution is likely to be difficult to establish due to lack of data. Moreover, process emissions are difficult to estimate with reasonable accuracy in many cities. In the URBAIR study, only in Kathmandu valley, where the main process industry is brick production, could these emissions be estimated with any completeness and accuracy.

Figure 6.2 shows the relative contributions to PM emissions from road vehicles, other fuel combustion and refuse burning in the URBAIR cities. The total PM emissions from these sources which were estimated, as tons per year/mill. inhabitants, is considerably different between cities, being highest in Manila and lowest in Mumbai. This may, to some extent, reflect different levels of completeness and quality of the emission data given and collected for each city. The need for quality assurance of emission inventories should be acknowledged. In the URBAIR study, this could be accomplished only to some degree.

The total *road traffic* (including the vehicle-induced resuspension from roads) accounts for 45-60 per cent of the emissions from the mentioned sources in Mumbai, Manila and Jakarta, and only 35 per cent in Kathmandu. There are marked differences in the contribution from *industrial fuel*: this is large in Manila, 39 per cent, where the industry uses large amounts of heavy fuel oil. In the other cities, industrial fossil fuel contributes only about 10 per cent. Regarding *domestic fuel*, wood contributes significantly in Kathmandu and to some extent in Mumbai (for cremation). *Domestic refuse* emissions are based on rather rough estimates, varying slightly between the cities. The source is estimated to contribute significantly.

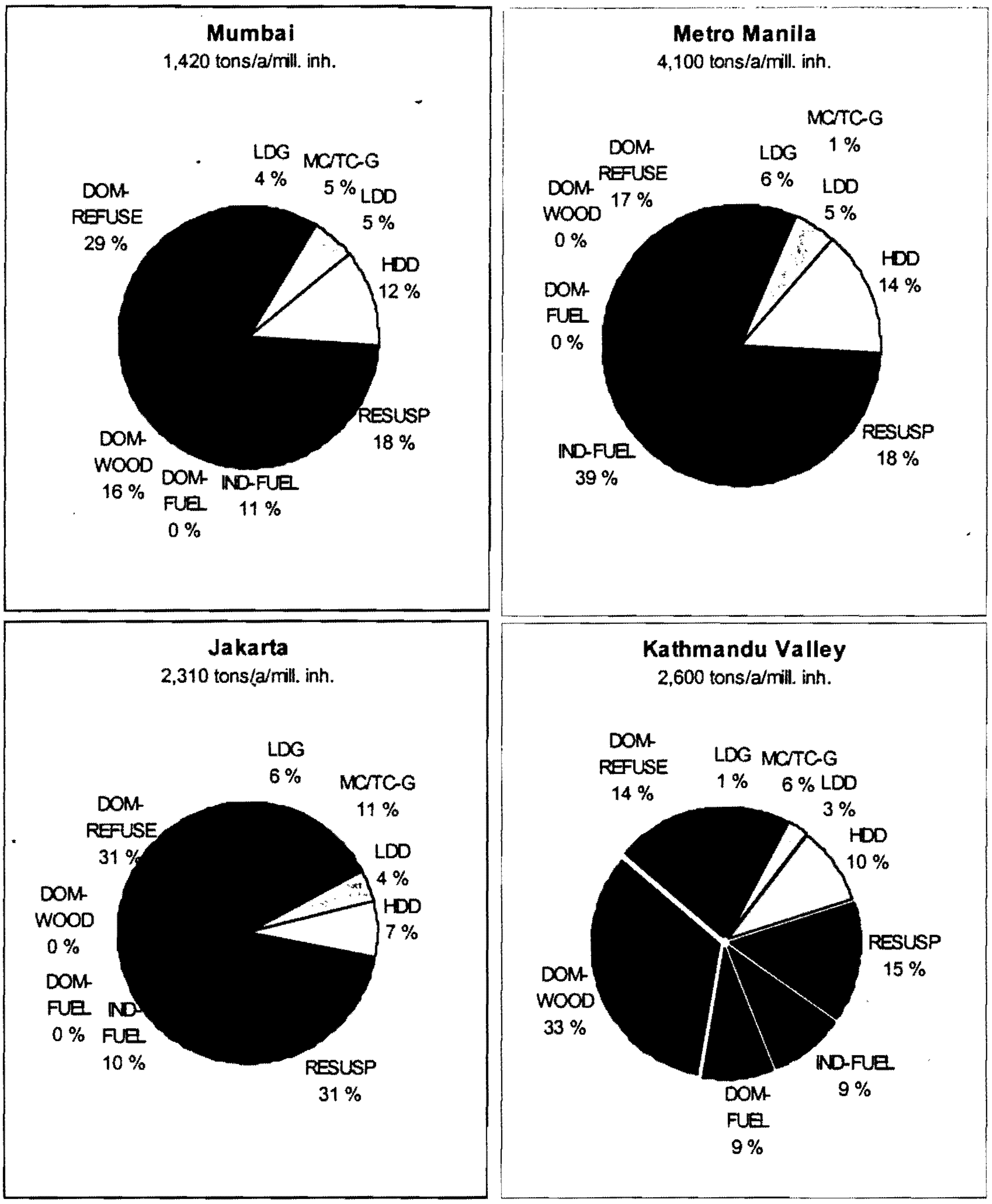
The emissions from *industrial processes* must be added to the numbers in Figure 6.2. In Kathmandu, the brick industry adds emissions amounting to about 80 per cent of the emissions included in Figure 6.2.

6.2.3 Population exposure and assessment of health damage and its costs

The emissions should be distributed spatially according to the locations of the road network and other sources, and the population distribution. This, together with meteorological/dispersion parameter data, is then used as input to dispersion models, to calculate the spatial distribution of concentrations and population exposure.

In the URBAIR study, a climatological, gaussian multi-source model was used to calculate annual average concentrations in km^2 grids over the cities. It is also possible to use more advanced dispersion models which are increasingly available.

Using calculated population exposure distributions and dose-response relationships, the damage to the health of the population and its costs can be estimated. Many of the costs stem from increased incidence of pollution-related illness and reduced life expectancy. The former is valued in terms of medical care costs and lost daily wages, as well as expenses undertaken to prevent illness. The latter is more difficult to evaluate in economic terms. The cost of increased mortality is based on "value of statistical life (VSL)" estimates, either the willingness to pay (WTP) method, or the human capital approach (see World Bank, 1997a).



LDG:	Light duty vehicles, gasoline	Ind-fuel:	Industrial fossil fuel comb.
LDD:	Light duty vehicles, diesel	Dom-fuel:	Domestic fossil fuel comb.
HDD:	Heavy duty vehicles, diesel	Dom-wood:	Domestic wood comb.
MC/TC-G:	Motorcycles/tricycles, gasoline	Dom-refuse:	Domestic refuse burning

Figure 6.2 Emission contributions to PM from various combustion source categories, plus road dust resuspension (RESUS), in four URBAIR cities

In the URBAIR study, the dose-response relationships developed by Ostro (1992; 1994) were used. For mortality and various morbidity indicators related to PM_{10} exposure, estimates of the extent of health damage were made. The morbidity indicators considered were chronic bronchitis, restricted activity days (RAD), emergency room visits (ERV), bronchitis in children, asthma attacks, respiratory symptom days (RSD), and respiratory hospital admissions (RHA). The costs were calculated from specific costs per case of mortality (premature death), using the human capital approach, and of the morbidity indicators. These specific costs were estimated for each city, based upon input from local consultants. The mortality cost is calculated as the discounted value of expected (average) future income at the average age of the population.

Table 6.2 shows the calculated health impact from PM_{10} in the cities, in terms of number of cases per million inhabitants, and the total annual costs associated with the entire impact in each city. The differences reflect the size of the population affected, the air pollution levels, and the "cost per case" estimate made in each city, as well as the rate of the local currency relative to the US dollar.

The estimated costs of the health effects were substantial: more than US $100 million per year in Mumbai, Manila and Jakarta.

In all cities, the costs related to sickness were higher than those estimated for mortality. This relation depends on the method used to value the costs of lives lost. For example, much higher mortality costs than those presented would result if United States 'willingness to pay' estimates were applied.

6.3 COST-BENEFIT ANALYSIS OF SELECTED MEASURES

After having established an estimate of the damage associated with the present pollution level in a city, a comparison of the economic costs and benefits associated with the introduction of selected pollution control measures can be carried out:

- select feasible measures which, evaluated from the emission inventory and other information, has a potential for significant reduction of the pollution level, the population exposure and thus the damage;
- estimate the costs related to the implementation of the measure, implemented to the extent feasible and necessary;
- estimate the reduction in the damage (the benefit in monetary terms) associated with the implementation of the measure, by running the health damage calculations (emissions, dispersion, exposure) with the reduced emissions implemented.

For principles of cost-benefit analysis, the reader is referred to the URBAIR Guidebook (WB, 1997a) and to Mishan (1988).

This analysis was carried out for each of the URBAIR cities. The results of cost-benefit analysis of selected measures in Manila are shown in Table 6.3. Table 6.4 summarizes the cost-benefit analysis results for some of the measures, in Mumbai, Manila and Jakarta. For Kathmandu, a cost-benefit analysis in monetary terms was not carried out.

Both costs and benefits of the selected measures vary between the cities (see Section 6.4). However, for a number of the abatement measures in every city, the benefits exceed the costs, in some cases substantially.

Table 6.2 Estimated annual health impacts and its costs related to the PM_{10} pollution in the four URBAIR cities. Mortality (premature death) and selected morbidity indicators

	Mumbai **1991**	**Metro Manila** **1992**	**Jakarta** **1990**	**Kathmandu Valley** **1993**
Exposure (% of pop.)[1]				
TSP>90 $\mu g/m^3$	97%	67%	>99%	50%
TSP>180 $\mu g/m^3$	5%	15%	~50%	3-4%
Health impact from PM_{10} (#/mill. inh.)				
Mortality	279	155	459	79
Morbidity				
Chronic Bronchitis	2 000	1 430	n.c.	477
Restricted Activity Days (10^3)	1 870	1 310	3 265	448
Emergency Room Visits	7 600	5 360	13 370	1 835
Bronchitis in Children	19 000	13 330	33 000	4 575
Asthma attacks	74 100	51 900	130 000	17 800
Respiratory Symptom Days (10^3)	6 000	4 170	10 410	1 430
Respiratory Hospital Admissions	400	238	714	93
Monetary value of health impact, Total city	Mill. US$	Mill. US$	Mill. US$	Mill. US$
Mortality[2]	22.7	18.8	49.7	0.57
Morbidity[3]				
Restricted Activity Days	17.2	67.7	69.4	0.53
Asthma attacks	24.3	19.8	6.9	0.23
Respiratory Symptom Days	39.0	59.1	1.6	1.51

[1] Exposure at residences. The high end of the population exposure, near roads and other sources, and its effects comes in addition.

[2] The human capital approach was employed to estimate the value of a statistical life.

[3] Selected morbidity indicators which represent the largest health costs (monetary value).

n.c. not calculated.

6.4 ACTION PLANS

The development of an action plan for air pollution abatement involves several steps:

The first step is to identify the pollution abatement measures that are available, given the location and source composition of the city. This list of measures should be established early in a study, when an overview of sources and emissions has been made. In the URBAIR study, these abatement measures where categorized in 5 categories:

1. Improved fuel quality;
2. Technology improvement;
3. Fuel switching;

4. Traffic management;
5. Transport demand management.

The second step is to analyse each measure in terms of its costs, effectiveness (potential benefits), feasibility, and any other factors of concern. The analysis is done according to the steps laid out in Section 3. In practice, only selected measures with a large potential and reasonable costs need to be analysed, at least in the first round.

In the URBAIR study, each city built its action plan in somewhat different ways, but for each measure the following characteristics were described:

• What	Description;
• How	Policy instruments to instigate and implement the measure;
• When I	When actions should be implemented;
• When II	When results can be expected;
• Who	Institutions/organizations responsible or affected;
• Effects	Reduced emissions/exposure/damage costs;
• Cost	Cost of measure;
• Feasibility	of the measure;
• Other	significant factors.

In addition to the measures that relate directly to reduced pollution, the action plan should also address the need to improve the basis for the air quality improvement strategy: improving the database (monitoring, emission inventory, modelling, etc.) and the regulatory and institutional basis for establishing an operational air quality management system in the city.

The third step is to make a list of the selected measures, prioritized according to their cost-effectiveness or cost-benefit ratio, their feasibility and availability of policy instruments to facilitate their implementation. This selected list of measures, accompanied by an investment plan, forms the basis for the "Action Plan". Summing the measures in order of priority indicates how many of the measures need to be implemented in order to reach the air quality target. Figure 6.3 gives a visualization of how to rank measures according to their cost effectiveness (and their feasibility), i.e. the cost per ton of reduced emissions *versus* the total potential for population exposure reduction. The Figure also indicates that some measures may give a net saving as well as pollution reduction (e.g. if the measure saves fuel). The "first" measures give significant effects at relatively low cost, and then costs increase and effects decrease as less efficient measures are chosen.

For the URBAIR cities, the analysis was carried as far as is shown in Table 6.4, where costs and benefits of selected, feasible measures are compared. The list reflects the importance of the pollution associated with road traffic in these cities (Mumbai, Manila and Jakarta). In Kathmandu, the industrial brick production was of equal importance to road traffic.

Substantial benefits, larger than the estimated costs, were found for the following measures: unleaded gasoline, low-smoke lubrication oil (for 2-stroke MCs), inspection/maintenance of vehicles (although considered more costly in Jakarta), and the control of gross polluters (e.g. vehicles with visible exhaust). Substantial benefits were also found for the following measures, but here the costs were estimated to be higher than the benefits: clean vehicle standards (state-of-the-art), and cleaner fuel oils.

Table 6.3 Cost-benefit analysis of selected abatement measures in Manila, 1992 (annual costs)

	Benefits			Time frame	
Abatement measure	**Avoided emissions[2], tons PM_{10}/yr**	**Avoided health damage**	**Costs of measure**	**Introduction of measure[1]**	**Effect of measure**
Vehicles Addressing gross polluters:					
Effective smoke-belching campaign	2,000	US $16-20 million 158 deaths, 4 million RSD	US $0.08 million	Immediate	Short-term
Improving diesel quality	1,200	US $10-12 million 94 deaths, 2.5 million RSD	US $10 million	Immediate	2-5 years
Inspection/maintenance	4,000	US $30-40 million 316 deaths, 8 million RSD	US $5.5 million	Immediate	2-5 years
Fuel switching: diesel → gasoline in vehicles	2,000	US $59-73 million 600 deaths, 15 million RSD		Immediate	5-10 years
Clean vehicle standards (cars/vans)	7,000	US $94-116 million 895 deaths, 24 million RSD	US $5-20 million	Immediate	5-10 years
Fuel combustion Cleaner fuel oil	5,000	US $10-20 million 100 deaths, 2.5 million RSD	US $10-20 million	Immediate	1-2 years

Table 6.3 Cost-benefit analysis of selected abatement measures in Manila, 1992 (annual costs) (contd.)

Abatement measure	Benefits		Costs of measure	Time frame	
	Avoided emissions[2], tons PM_{10}/yr	Avoided health damage		Introduction of measure[1]	Effect of measure
Power plants					
Clean fuel	500	small	US $10 million	Immediate	1-2 years

Notes:

1 Time frame for starting the work necessary to introduce measure.

2 The various abatement measures are not necessarily independent of each other. Thus, the "avoided emissions" stated in this table for each measure separately may not simply be added, if one wants an estimate of the total effect of packages of measures.

Table 6.4 Summary of cost-benefit analysis results, three URBAIR cities

Abatement measure	Mumbai (1991)		Manila (1992)		Jakarta (1990)	
	Benefits	Costs	Benefits	Costs	Benefits	Costs
	Mill. US $		Mill. US $		Mill. US $	
Unleaded gasoline	NQ	NQ	NQ	NQ	146	24
Low-smoke lubrication oil for 2-stroke MCs	4.9	1.0	n.a.		16	1-5
Inspection/ maintenance of vehicles	8.2	4.9-9.8	30-40	5.5	15	33
Control gross polluters	4.1	NQ (small)	16-20	0.1	12	Low
Clean vehicle standards:						
- Cars/vans	4.1	24.6	94-116	5-20	33	41
- MC/TC	7.8	19.7	n.a.		NQ	NQ
Improved diesel quality	2.6	9.8	10-12	10	2.9	Low
50% CNG	2.5	NQ	NQ	NQ	6.7	NQ
Cleaner fuel oil	1.6	14.8	10-20	10-20	NQ	NQ

NQ: Not quantified.

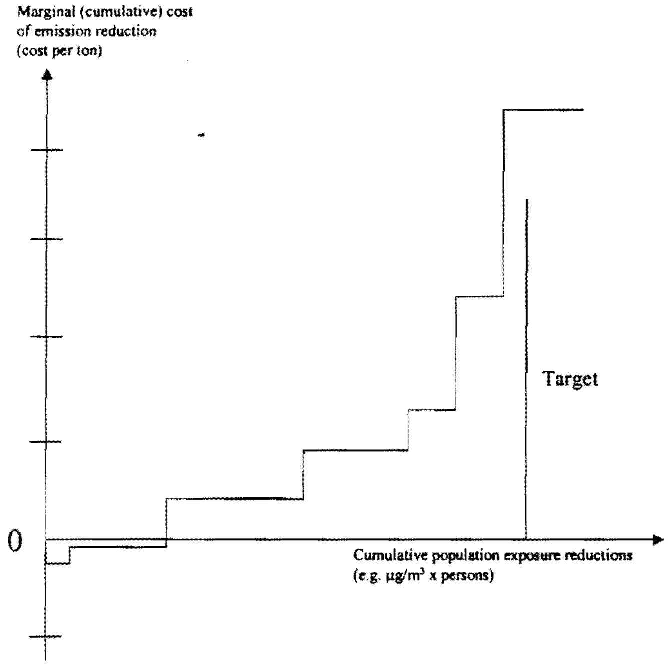


Figure 6.3 Visualization of ranking of measures to reduce population exposure, and thus health damage

One potentially important measure is improved diesel quality. The costs are estimated to be much higher than the benefits in Mumbai, roughly the same as the benefits in Manila, and probably less than the benefits in Jakarta. These differences probably reflect different availability and pricing policies of fuels in the countries.

6.5 POLICY INSTRUMENTS AND PLANS FOR AIR QUALITY IMPROVEMENT IN ASIAN URBAIR CITIES

The implementation of plans and strategies for air quality improvements, in Asian cities as elsewhere, is done through the use of policy instruments by ministries, regulatory agencies, law enforcers and other institutions. Indeed, some of these institutions may well be the same institutions as those which must be in place to carry out the AQMS analysis described here, which ideally is the basis for the plans and strategies. Thus, the existence of relevant institutions, and an organizational institution structure, is part of the basis for AQMS work.

Institutions on different government levels: Different levels of government - national, regional and local - have different roles and responsibilities in the environmental sphere. Air quality standards or guidelines are usually set at the national level, although local government may have the legal right to impose stricter regulations. National governments usually assume the responsibility for scientific research and environmental education, while local governments develop and enforce regulations and policy measures to control local pollution levels.

In the context of an AQMS, local authorities have the most significant responsibilities. These include the following:

- developing and running the monitoring programme;
- assessing the air quality;
- determining the impacts of air pollution;
- setting goals for the quality of air; and
- developing future scenarios and action plans to achieve those goals.

National or state authorities may assume the responsibility for mandating controls, such as regulating fuel quality, setting emissions standards, and providing financial resources, or incentives for the private sector reduce emissions.

Institutional arrangements, laws and regulations are important parts of an AQMS. Some roadblocks to successful air quality management in Asia are weak institutions that lack technical skills and political authority; enforcement agencies that often lack both the necessary information and the means to implement policy, and unclear legal and administrative procedures. Countries have their own political and administrative hierarchies and technical expertise that affect institutions, laws and regulations related to air pollution control.

Laws and regulations on air quality are generally in place in three of the URBAIR cities: Mumbai, Metro Manila and Jakarta. In Kathmandu, the regulatory framework for controlling air pollution was still in an early phase. National Clean Air Acts had been adopted. Standards for air quality and emissions, specifications of maximum amount of pollutant in fuel (e.g. sulphur and lead) were also typically in place, as well as procedures of Environmental Impact Assessments (EIA) for new establishments.

The analysis of institutions involved in air quality control and management in the four URBAIR cities revealed that national, regional (provincial) and local institutions were involved in all cities, to different degrees and with different tasks and division of responsibilities in the cities. The importance of clarity in the organizational structures and the division and description of responsibilities and "lines-of-command" must be stressed.

Various policy instruments available are described in the URBAIR Guidebook (WB, 1997a). It is important that the selected instruments do not have significant negative side effects, within the environmental sphere or elsewhere. Social conditions and characteristics particular to a society must also be kept in mind. Within the local context, one can try to find the most effective or the most efficient instruments. An instrument that maximizes the effect (given a certain budget) or minimizes costs (given a certain environmental objective) should be chosen.

Policy instruments may be grouped into *Direct Regulation* (e.g. guidelines and standards for emission and air quality, enforcement of compliance, spatial planning and zoning, traffic regulations), *Economic Instruments* (e.g. emission charges, taxies or subsidies, emission trading), as well as instruments related to *Communication and Awareness-building*. Effective dissemination of information about pollution levels, contributions and effects is important in creating a sense of responsibility for environmental quality and the results of individual actions and practices. This is relevant for industry, product designers, and other private sector actors, as well as individual citizens.

Clean air policy programmes: Two of the four URBAIR cities - Metro Manila and Jakarta - formulated policy programmes during the early 1990s for air quality improvement. The "OPLAN Clean Air Metro Manila" was a 5-year programme starting in January 1993, to culminate in a "Clean Air 2000 Action Plan". The results from the URBAIR project for Manila has fed into this process.

The national "Blue Sky Programme" (Langit Baru) of Indonesia was launched in 1991, with control plans for selected stationary source categories, and motor vehicles: control of black smoke and introduction of unleaded gasoline. The URBAIR analysis for Jakarta provided an impetus to increase the efforts in this programme. The national plan was paralleled by Jakarta's "Clean Air Programme" (Prodasih).

6.6 REFERENCES

EU. 1998. Common Position (EC) No 57/98 adopted by the Council on 24 September, 1998 with a view to adoption of Council Directive 98/_/EC relating to limit values for sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter and lead in ambient air (98/C360/04). Official Journal of the European Communities C360/99.

Faiz, A., Weaver, C.S. and Walsh, M.P. 1996. *Air Pollution from Motor Vehicles: Standards and Technologies for Controlling Emissions*. The World Bank, Washington, D.C.

Larssen, S. 1998a. Monitoring networks and air quality management systems. In *Urban Air Pollution. European Aspects* (edited by J. Fenger, O. Herteland and F. Palmgren). Kluwer Academic Publishers.

Larssen, S. and, Helmis, C. 1998b Quality assurance and quality control. In *Urban Air Pollution. European Aspects* (edited by J. Fenger, O. Hertel and F. Palmgren). Kluwer Academic Publishers.

Mishan, E.J. 1988. *Cost Benefit Analysis*. Fourth Edition. Unwin Hyman, London.

Ostro, B. 1992. *Estimating the Health and Economic Effects of Air Pollution in Jakarta: A Preliminary Assessment*. Paper presented at the Fourth Annual Meeting of the International Society of Environmental Epidemiology. August 1992. Cuernavaca, Mexico.

Ostro, B. 1994. *Estimating the Health Effects of Air Pollutants: A Method with an Application to Jakarta*. Policy Research Working Paper 1301. The World Bank, Washington, D.C.

US EPA. 1997. *Federal Register* (July 18, 1997). Volume **62**, Number 138. Rules and Regulations. pp. 38651-38701.

WB. 1997a. *Urban Air Quality Management Strategy in Asia: Guidebook* by Larssen, S., Grønskei, K.E., Hanegraaf, M.C., Jansen, H., Kuik, O.J., Oosterhus, F.H. and Olsthoorn, F.H. (edited by J.J. Shah, T. Nagpal and C.J. Brandon). The World Bank, Washington, D.C.

WB. 1997b. *Urban Air Quality Management Strategy in Asia, Greater Mumbai Report* by Larssen, S., Gram, F., Hagen, L.O., Jansen, H., Olsthoorn, X., Mehta, K.H., Joglekar, U., Aundhe, R.V. and Mahashur, A.A. (edited by J.J. Shah and T. Nagpal). World Bank Technical Paper No. 381. The World Bank, Washington, D.C.

WB. 1997c. *Urban Air Quality Management Strategy in Asia, Metro Manila Report* (by Larssen, S., Gram, F., Hagen, L.O., Jansen, H., Olsthoorn, X., Lesaca, R., Anglo, E., Torres, E.B., Subida, R.D. and, Fransisco, H.A. (edited by J.J. Shah and T. Nagpal. World Bank Technical Paper No. 380. The World Bank, Washington, D.C.

WB.1997d. *Urban Air Quality Management Strategy in Asia, Jakarta Report* (by Grønskei, K.E., Gram, F., Hagen, L.O., Larssen, S., Jansen, H., Olsthoorn, X., Soedomo, M. and, Achmadi, U.F. (edited by J.J. Shah and T. Nagpal). World Bank Technical Paper No. 379. The World Bank Washington, D.C.

WB. 1997e. *Urban Air Quality Management Strategy in Asia, Kathmandu Valley Report* (by Larssen, S., Gram, F., Haugsbakk, I., Jansen, H., Olsthoorn, X., Giri, A.S., Shah, R., Shrestha, M.L. and, Shrestha, B. (edited by J.J. Shah and T. Nagpal). World Bank Technical Paper No. 378. The World Bank Washington, D.C.

WHO. 1992. *Urban Air Pollution in Megacities of the World.* (World Health Organization (WHO)/United Nations Environment Programme (UNEP)). Blackwell Publishers, Cambridge, MA.

WHO. 1995. *Update and Revision of the Air Quality Guidelines for Europe*. Meeting of the Working Group "Classical" Air Pollutants, Bilthoven, The Netherlands 11-14 October, 1994. WHO Regional Office for Europe (EUR/ICP/EHAZ 94 05/PB01).

Chapter 7

INDOOR AIR POLLUTION

Sumeet Saksena[1] and Kirk R. Smith[2]
[1]Fellow, Centre for Environmental Studies, TERI, New Delhi, India
[2]Professor, School of Public Health, University of California, Berkeley, USA

Abstract

This Chapter examines the potential impact of indoor air pollution on health in Asia. It begins by reviewing the evidence on the emissions, concentrations and populations exposed to indoor air pollution from traditional cooking fuels. Given the magnitudes involved, and despite considerable uncertainty, the chapter argues that the scale of exposures and health effects are likely to be large. The chapter presents the emerging scientific evidence that supports the numerous anecdotal accounts relating high biomass smoke levels to important health effects. These are principally: acute respiratory infection in children, and chronic obstructive lung disease, adverse pregnancy outcomes and lung cancer in women. The chapter concludes that more research is sorely needed, however, before reliable estimates can be made of the burden of disease associated with indoor air pollution (rough estimates indicate it to be one of the largest single risk factors for mortality - roughly 6 per cent globally) and how much ill health would be reduced by smoke reduction activities such as the promotion of improved stoves.

7.1 INTRODUCTION

Power production, urbanization and rapid industrialisation have generally been regarded as the primary causes of deteriorating air quality. Policy-makers and environmental managers tend to ignore the role of small sources of air pollution, particularly when they do not contribute substantially to ambient emissions. Small sources can be very important, however, when they have a high exposure effectiveness, defined as the fraction of the emitted pollution from a source that actually enters people's breathing zones.

The health damage produced by air pollution is dependent on the dose received by the population in question (see Chapters 2 and 3). Because dose is difficult to determine for large numbers of people, however, air pollution studies have tended to emphasise exposure, which is usually assumed to be closely proportional to dose. In practice, a surrogate for exposure, ambient concentration, has actually been measured in most instances. This has been done, for example, by placing monitoring instruments on the roofs of public buildings in urban areas.

This practice assumes that overall ambient concentration is well characterised by the particular choice of places and times that measurements are made, and that actual human exposures nearby are proportional to the ambient concentration so determined. In fact, Smith (1993) has estimated that only 2 per cent of all people's time is spent outdoors in developed country cities where the bulk of air pollution control efforts have taken place.

Apart from the exposure implications, indoor air pollution is likely to remain a problem because of the patterns of household fuel use. Traditional fuels play a vital role in the developing world. More than 2 billion people rely on them to meet the majority of their energy needs, obtaining these fuels from the same natural environment on which people also depend for food crops and grazing for their animals.

The term 'traditional fuels' is used here to refer to biomass fuels used mainly for domestic energy, including wood, charcoal, agricultural residues and animal waste. Traditional fuels are the dominant source of energy in the developing world. It is estimated that they account for roughly 20-35 per cent of the total energy consumption in the developing countries and 25 per cent in South Asia (WRI, 1998). The regional database on number of houses using a particular type of fuel is very weak. In India, it is estimated that about 62 per cent of the households use firewood, 15 per cent use animal wastes and 3 per cent use coal or coke. There is some uncertainty regarding the use of agricultural wastes (GOI, 1992).

When people are no longer able to rely on an abundance of good quality firewood, they are gradually forced to exercise care and frugality in the use of a variety of lower quality fuels. As a result, a new equilibrium in fuel use is eventually reached. Even if people are still able to meet their household energy needs, there is no question that for many it represents a lowering in the quality of their daily lives.

Figure 7.1 illustrates the evolutionary path for cooking fuels and stoves in South Asia. In some cases, changes in income or availability of other resources may force some groups back down this path but, in general, people prefer, if possible, to move upwards (Smith, 1990).

In summary, biofuels are the most important fuels globally in terms of the number of people affected. In energy content, they are the most important fuels in many poor countries, although second to the fossil fuels on a global basis. They are used principally by households for cooking and space heating. Furthermore, they are likely to remain important for much of South Asia for many decades.

7.2 CONCENTRATIONS AND EXPOSURES

Although there are many hundreds of separate chemical agents that have been identified in biofuel smoke, the four most emphasised pollutants are: particulates, carbon monoxide, polycyclic organic matter and formaldehyde. Unfortunately, relatively little monitoring has been done in rural and poor urban indoor environments in a manner that is statistically rigorous. The results nevertheless are striking (Table 7.1). The concentrations found are 10-100 times higher than typical health-related standards/guidelines. The rest of the discussion will be restricted to particulate matter, because of data gaps regarding other pollutants, and the fact that particulate matter is one of the most common and harmful pollutants.

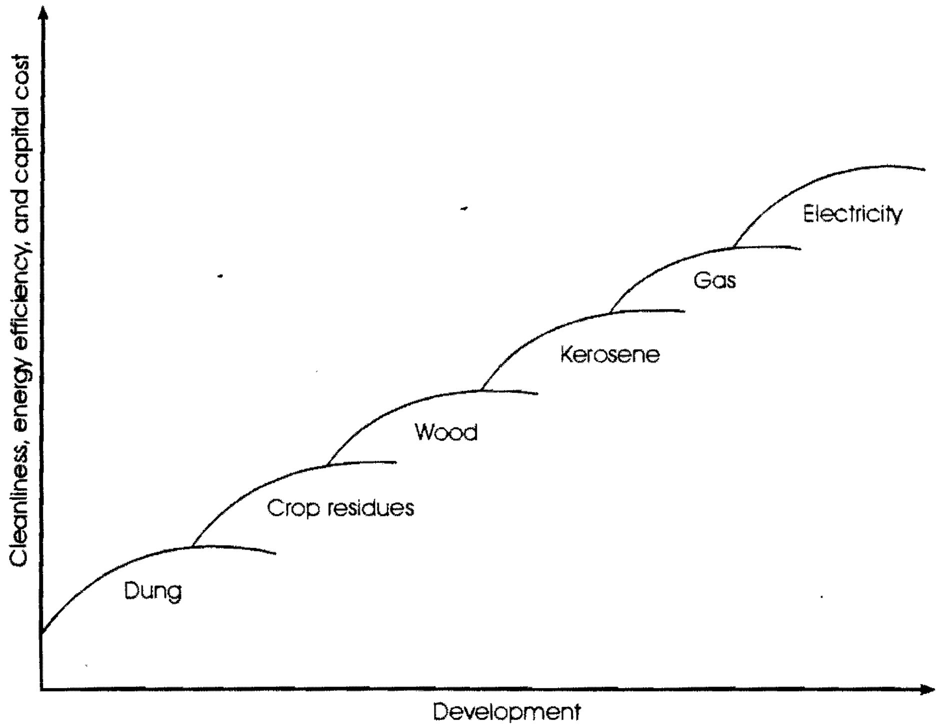


Figure 7.1 The energy ladder (source: Smith *et al.*, 1994)

Table 7.1 Typical concentration levels of total suspended particulate (TSP) matter indoors from biofuel combustion in South Asia measured through area and personal sampling

Country	Year	Sample size	Conditions	Concentration (mg/m^3)
I Area Monitoring				
India	1982	64	30 min, wood/dung/charcoal	15800/18300/5500
	1988	390	Cooking, 0.7m/ceiling	4000/21000
	1992	145	Cooking/non-cooking/living	5600/820/630
	1994	61	24h, ag-resid/wood	2800/2000 (I)
	1995	50	Breakfast/lunch/dinner	850/1250/1460 (I)
	1996	136	urban, cooking/sleeping	2860/880 (I)
Nepal	1986	17	2 h	4400 (I)
II Personal Monitoring				
India	1983	65	4 villages	6800
	1987	165	8 villages	3700
	1987	44	2 villages	3600
	1988	129	5 villages	4700
	1991	95	winter/summer/monsoon	6800/5400/4800
	1996	40	two urban slums, infants, 24 h	400/520 (I)
Nepal	1986	49	2 villages	2000
	1990	40	traditional/improved stove	8200/3000

Note: Unless noted otherwise, figures refer to Total Suspended Particulates (TSP, often called SPM in South Asia); I = Inhalable Particulate Matter (less than 10-15 microns), R= Respirable Particulate Matter (less than 2.5-5 microns); Daily Indian standard for residential areas is 100 mg/m^3 .

Source: Smith (1996)

There are few micro-levels studies that have attempted to measure total exposure levels in communities using biofuels. The first study that measured levels of pollutants in various micro-environments (cooking and non-cooking; indoor and outdoor) and the time spent in each of these by different population groups was conducted in a rural hilly area of India. The study concluded (see Table 7.2) that the daily exposure levels of women and children far exceed comparable Indian or international standards and those of youth and men (Saksena *et al.*, 1992), and that cooking was the major contributor to daily exposure for women and children.

Table 7.2 Mean daily integrated exposure to TSP (mg/m^3) in a rural hilly area of India

Season	Women	Children	Youth	Men
Winter	1.96	1.04	0.79	0.71
Summer	1.13	0.54	0.33	0.25

Note: Daily Indian ambient standard for residential areas is 0.1 mg/m^3; the WHO guideline was 0.10-0.15 mg/m^3.
Source: Saksena *et al.* (1992)

The few studies that enable a comparison of daily levels across various fuel groups confirm the concept of the energy ladder (as one moves up the ladder, the cleanliness, efficiency and convenience of the fuels tend to increase, along with their costs), as indicated in Table 7.3.

Table 7.3 Estimated daily exposures to PM$_{10}$ (mg/m^3) from cooking fuel along energy ladder in two Asian cities

Fuel	Pune, India	Beijing, China
Biomass	0.71 - 1.08	
Coal (vented)		0.10 - 0.15
Kerosene	0.1 - 0.15	
LPG	0.02	0.06
National ambient standard (for residential areas)	0.10	0.15

Source: adapted from Smith *et al.* (1994)

An estimate of exposure on a global level has ascribed approximately 77 per cent of the global exposure to particulates in indoor environments of the developing world (WHO, 1997). Such estimates are based on pollutant levels (concentrations) considered typical in different micro-environments, estimates of the time spent by various population groups in these micro-environments, and the size of these groups. Reliable estimates would require a good database of time-activity patterns, linked to location specific exposure estimates. This does not exist at the present time. Indoor exposure estimates depend heavily on the relatively few surveys that have been conducted in rural areas. However, if these results are representative, the overall health burden is likely to be extremely high.

7.3 HEALTH EFFECTS

The total human exposure to many important pollutants is much more substantial in the homes of the poor in developing countries than in the outdoor air of cities in the developed world, because of the high concentrations and the large population involved. It is, however, the outdoor problem that has received the vast majority of attention in the form of air pollution research and control efforts (Smith, 1993). As a result, it has been necessary to extrapolate from the urban studies to estimate what the health effects might be in biomass-using households (de Koning *et al.*, 1985; Smith, 1987). In recent years, however, there have been a number of studies which directly focus on these households and which generally confirm what has been extrapolated (Chen *et al.*, 1990). Following are brief summaries of the major health effects, as initially documented by WHO (1992). It must be remembered that the quality of all these studies is not as high as desirable, mainly because of a) inappropriate choice of exposure and health outcome indicators; b) poor study design; c) weak statistical foundation; and d) confounding factors not considered.

7.3.1 Acute Respiratory Infections in children (ARI)

ARI, particularly as acute lower respiratory infection (ALRI) such as pneumonia, is one of the chief killers of children in developing countries. At 4-5 million deaths per year, it now exceeds deaths from diarrhoea. In South Asia ARI is responsible for 1.4 million deaths per year (Murray and Lopez, 1997). ARI is known to be enhanced by exposures to urban air pollutants and indoor environmental tobacco smoke at levels of pollution some 10-30 times less than typically found in village homes.

Some of the most suggestive studies available were undertaken in Nepal, Zimbabwe and Gambia. A Nepal study examined approximately 240 rural children under 2 years each week for 6 months for incidence of moderate and severe ARI (Pandey *et al.*, 1989). They found a strong relationship between the maternally reported number of hours per day the children stayed by the fire and the incidence of moderate and severe cases. In Zimbabwe, 244 children under 3 years reporting at hospitals with ARI were compared to 500 similar children reporting at clinics (Collings *et al.*, 1990). Presence of an open wood-fire was found to be a significant ARI risk factor. In a study of 500 children in Gambia, girls under 5 years carried on their mothers' back during cooking (in smoky cooking huts) were found to have a 6 times higher risk of ARI, a substantially higher risk factor than parental smoking. There was no significant risk, however, in young boys (probably because they are kept for shorter periods near the fire) (Armstrong and Campbell, 1991).

A study in Buenos Aires (Cerqueiro *et al.*, 1990) used a matched case-control method to identify risk factors for ALRI in 670 children. The results of the study indicated a high risk from indoor contaminants.

400 children under 5 years of age in South Kerala, India, were studied to identify risk factors for severe pneumonia. Cases were in-patients with severe pneumonia as ascertained by WHO criteria, while controls were outpatients with non-severe ARI. There was no association with presence of an improved "smokeless" cookstove in the children's homes (Shah *et al.*, 1994). This is consistent with other studies in India that often show no significant difference in indoor pollution in households with and without improved stoves (Ramakrishna *et al.*, 1989).

O'Dempsey *et al.* (1996) investigated possible risk factors for pneumococcal disease among children living in a rural area of the Gambia. A prospective case-control study was conducted. The study indicated that there is an increased risk of pneumococcal diseases associated with children being carried their mothers' back during cooking.

A recent study of 642 infants conducted in urban slums of New Delhi, India examined the relationship between incidence of ALRI and indoor air pollution. The study was conducted in 2 slums - one in a highly polluted area and the other in a low polluted area. In each slum, infants from wood using and kerosene using households were chosen. The incidence of ALRI in wood using households was not found to be significantly higher than in kerosene using households (Sharma *et al.*, 1998).

Overall, the studies conducted to date are extremely suggestive and, with a few exceptions, reasonably consistent. Being quantitative, they can be used to calculate health effects (see below). They do not fulfil all the strict scientific requirements for demonstrating causality because ARI has so many other risk factors for which it is difficult to account in studies that observe pre-existing differences in exposure conditions. Randomised trials are needed, in which exposure-reduction technologies, for example improved fuels or stoves, are applied to half the households in a population, which is then followed to see if ARI rates diverge. In this way, one can be fairly certain that other ARI risk factors, for example socio-economic or nutritional, are not also different between the groups using one type of fuel or stove and those using another. Until such studies are conducted, it is necessary to relay the results of less rigorously designed studies.

7.3.2 Adverse pregnancy outcomes

Low birth-weight, a chronic problem in developing countries, is associated with a number of health problems in early infancy, as well as other negative outcomes such as neonatal death. Several risk factors are associated with low birth-weight, most notably poor nutrition. Since active smoking by the mother during pregnancy is a known risk factor and exposure is suspected, there is also reason to suspect biomass smoke as it contains many of the same pollutants. Carbon monoxide, which studies in Guatemala (Dary *et al.*, 1981) and India (Behera *et al.*, 1988) found in substantial amounts in the blood of women cooking with biomass, is the most probable cause. Another study in India found that pregnant women cooking over open biomass stoves had almost a 50 per cent greater chance of stillbirth (Mavalankar, 1991).

7.3.3 Chronic Obstructive Pulmonary Disease (COPD) and cor pulmonale

COPD, for which tobacco smoking is the major risk factor remaining in the developed countries, is known to be an outcome of excessive air pollution exposure. It is difficult to study because the exposures that cause the illness may occur many years before the symptoms are seen. Nevertheless, studies in Nepal (Pandey, 1984) and India (Malik, 1985; Behera and Jindal, 1991) led the investigators to conclude that non-smoking women who have cooked on biomass stoves for many years exhibit a higher prevalence of this condition than might be expected for similar women who have had less use of biomass stoves. In rural Nepal, nearly 15 per cent of non-smoking women (20 years and older) had chronic bronchitis, a high rate for non-smokers.

Cor pulmonale (heart disease secondary to chronic lung disease) has been found to be prevalent and to develop earlier than average in non-smoking women who cook with biomass in India (Padmavati and Arora, 1976) and Nepal (Pandey *et al.*, 1988).

A population-based cross sectional survey was conducted to determine the prevalence of chronic bronchitis and associated risk factors in an urban area of southern Brazil where 1053 subjects aged 40 years and over were interviewed. High levels of indoor air pollution were found to be associated with increased (nearly a doubling) prevalence of the disease (Menezes *et al.*, 1994).

7.3.4 Cancer

There are many chemicals in biomass smoke which are known to cause cancer (Cooper, 1980). In the 1970s, based on a small study in Kenya, it was thought that naso-pharyngeal cancer might be associated with biomass smoke (Clifford, 1972), but more recent studies in Malaysia (Armstrong *et al.*, 1978) and Hong Kong (Yu *et al.*, 1985) have failed to confirm this. Based on risk extrapolations from animal studies, lung cancer, which might be expected to be common in biomass-using areas, is relatively rare (Koo *et al.*, 1983). Indeed, some of the lowest lung cancer rates in the World are found in rural non-smoking women in developing countries. This is somewhat of an anomaly, and can only be partly explained by poor health records. A recent study in Japan (Sobue *et al.*, 1990), on the other hand, found that women cooking with straw or wood-fuel when they were 30 years old have an 80 per cent increased chance of having lung cancer in later life (cancer, as in the case of chronic lung disease, takes many years after exposure to develop).

Compared to biomass, there are many studies of the air pollution levels and health impacts of cooking with coal on open stoves, almost all undertaken and published in China, where coal use for cooking is common (Hong, 1991). A range of effects is found, including quite strong associations with lung cancer (Smith and Liu, 1994). Even in China, however, biomass use is much more prevalent, and yet has not received adequate scientific and policy attention.

7.3.5 Tuberculosis

One of the most disturbing implications of recent research is that wood smoke may be potentially associated with tuberculosis. A large-scale survey (89,000 households) in India found that women over 20 years old in households using biofuels were nearly three times more likely to report having TB than those in households using cleaner fuels, even after accounting for a range of socio-economic factors (Mishra *et al.*, 1998). A study with clinically confirmed TB in Lucknow, India, found a similar risk, but was not able to correct for socio-economic factors (Gupta *et al.*, 1998).

7.3.6 Blindness

A case-control study in India found an excess cataract risk of about 80 per cent among people using biofuels (Mohan *et al.*, 1989). Cataract is the main cause of blindness in India and is known to be caused by wood smoke in laboratory animals. The same large family survey mentioned above found a somewhat lower rate (77 per cent) for partial blindness in adults living in Indian households with clean fuels, and a significant difference for total blindness in women (Mishra *et al.*, 1999).

7.4 HEALTH IMPACTS

Based on extrapolations of health effects in developed countries, WHO has estimated that 2.7- 3.0 million premature deaths (5-6 per cent of annual global deaths) occur due to outdoor and indoor air pollution. Using one methodology it has been estimated that of 3 million premature deaths due to air pollution, 2.8 million are due to indoor pollution (Smith, 1996). It has been estimated that the highest number of deaths will occur in India, followed by Sub-Saharan Africa (WHO, 1997). As regards deaths due to indoor exposure, 90 per cent by a factor of two in either of them will occur in developing countries. These estimates are very uncertain, and could be off direction, but it should be kept in mind that, given the low level of research funding devoted to indoor air pollution in developing countries, such uncertainty is to be expected.

A recent evaluation of the National Burden of Disease in India from indoor air pollution relied only on studies done in biomass-using households (many discussed above) instead of trying to extrapolate from developed-country urban settings that have been the basis of most air pollution health effects studies (Smith, 1998). Using national census data on the distribution of biomass use, the study estimated that some 400-600 thousand premature deaths per year among Indian women and children under 5 can be attributed to indoor air pollution. (Data were too poor to estimate the lower risks experienced by men and older children.) Extrapolating the Indian estimates to the entire South Asia region would indicate some 500-700 thousand premature deaths each year from indoor air pollution, considering only among women and young children

Although based on the best available evidence, it is emphasised that such estimates must be considered preliminary. Too little effort has gone into conducting either the health-effects or exposure-assessment studies in biomass-using populations. Nevertheless, it seems clear that the potential magnitude is substantial.

7.5 KNOWLEDGE GAPS AND NEEDED RESEARCH

A high research priority is to conduct epidemiological studies to identify the relationships between ARI, indoor air quality (biomass smoke, tobacco smoke), nutritional status, other infections, family/household composition, variables and so on.

Possible research strategies for epidemiological studies have been identified by WHO (1992) and these include the following:

1. case-control studies to establish relationships, identify dose-response and dose-effect relationships: some studies have been done but more are needed;
2. studies of "natural experiments" in which incidence rates of episodes might be examined longitudinally in relation to such changes as introducing stoves with chimneys in dwellings previously lacking chimneys;
3. randomised intervention studies in which health status is assessed with and without interventions such as improved stoves.

Intervention studies are suitable only for health effects that occur relatively quickly after exposure. The highest priority for intervention studies would thus seem to be:

- ARI in young children
- Adverse pregnancy outcomes for women exposed during pregnancy

Since the association of chronic obstructive lung disease and biomass smoke is fairly well established and lung cancer is not a major problem among non-smoking South Asian women, highest priority for case-control studies would seem to be:

- TB in adult women
- Heart disease in adult women

TB is of special interest because it is increasing in South Asia due to the growing HIV epidemic. Heart disease, which is one of the chief outcomes of urban air pollution studies in developed countries, has apparently never been studied in biomass-using households.

All such studies, of course, should meet accepted scientific standards for quality control and ethical conduct. In order to maximise scarce resources, research should be linked where possible to existing research projects that have already gathered some of the crucial information on ARI or other outcomes in the target groups (examples include vitamin A deficiency and family planning projects).

Work is needed to improve the quality of existing data and to facilitate the collection of new data. The most accurate available indicator of indoor air pollution is personal and area monitoring for respirable suspended particulates (RSP). Further work is required, however, to improve the capacity to simply and quickly assess exposure to indoor air pollution. It is difficult to assess exposure in children 2-5 years old with existing methods; time-weighted area monitoring might be the best choice.

7.6 INTERVENTIONS

This is a two-fold challenge facing most developing societies attempting to sustainably manage the biomass energy transition. Firstly, there is need to find sustainable means to harvest biofuels for the needs of that majority of humanity now relying upon them. Secondly, there is a need to develop high-grade biomass fuels (liquid and gaseous) that can meet development requirements, and the need to improve the efficiency, controlability and cleanliness of end-use devices.

7.6.1 Fuels

In the past, rural development was typically accompanied by a transition from traditional biofuels to fossil fuels, particularly kerosene, diesel oil and LPG (liquified petroleum gas). In recent years, however, it is often concluded that the cost and insecurity of petroleum supplies will delay or deny this transition in many of the now developing countries. Given that the international price of petroleum is lower than it has been for many decades, however, it is no longer clear that this conclusion is valid. Furthermore, reserving sufficient clean-burning gaseous and liquid fuels for household applications where pollution has such important health implications would seem an appropriate policy objective.

Although substantial, the cost of importing sufficient clean fossil fuels for South Asian households would not necessarily be prohibitive (Parikh *et al.*, 1999). For example, it has been estimated that the petroleum demand for powering the South Asian light-vehicle (mainly auto) fleet will grow at an average annual rate of 7.6 per cent until 2020 (WEC, 1998). If the annual growth rate were lowered slightly to 6 per cent, by 2020 the difference in vehicle fuel use would amount to some 40 million litres a day of kerosene equivalent. This quantity could supply most of the region's current biomass-using households and entail no more oil imports than is now being planned for vehicles. Recognising that directing this fuel solely to households is no easy task, it is nevertheless clear that the issue is to a great degree one of social priorities.

In the short term, however, it appears that the economic and logistics barriers to meeting rural energy needs solely by fossil fuels will be unsurmountable. This implies that biofuels will have a continuing role for many decades and must be taken seriously by policy-makers if the behaviour of the entire national energy system is to be understood and manipulated. If biofuels are to provide the type of energy services previously accomplished by the petroleum fuels, there must be substantial changes in the form and use of these fuels. So dramatic are these changes that it is appropriate to call them part of the post-biofuel transition or, more accurately, the post-traditional biofuel transition. This transition is occurring at every stage of the biofuel cycle - from harvesting through conversion to end use (Smith, 1986).

At the production stage, a change is occurring from unplanned and unscientific practices of gathering biofuel to sustainable harvesting. At the conversion stage, a number of processes are becoming available to upgrade the relatively low-grade natural solid biofuels into high-grade solid, liquid and gaseous fuels that can fuel a wide range of tasks beyond the basic necessities of cooking and space heating that biofuel now provides. More importantly, equipment is now being developed to accomplish these conversions at village and household scale. Examples are biogas and producer gas devices, alcohol fermentation and charcoal manufacture. Some of these conversion processes have the additional advantage that they remove the most polluting step out of the household to a village-level or otherwise more centralised location. This can greatly reduce individual exposures even if total emissions are not reduced.

With a view to reducing the pressure on forests and other biomass resources, countries such as India and Pakistan are trying to promote coal as a cooking fuel. Such a move has its merits and demerits. Many studies in China and South Africa have indicated that household coal leads to significant health hazards. China has the highest lung cancer rate for non-smoking women who use coal. Emissions from coal contain the very same pollutants as in biomass emissions; in addition, they contain toxic substances such as sulphur, arsenic and fluorine (Parikh *et al.*, 1999).

7.6.2 Stoves

In the short- and medium-term improving the use of biofuels seems to be the only feasible option for many households. Until recently, most stove research and implementation programmes considered fuel savings as the primary goal. The relationship between various parameters that govern stove performance are complex and surprisingly ill understood. Nevertheless, it seems likely that research can lead to the development of cooking systems that are so efficient that overall exposure can be reduced, while ensuring an optimal thermal performance and social acceptability.

Large-scale acceptance of improved stoves would require a concerted effort on the part of local organisations as well as government in individual countries to mass-produce them, in an appropriate manner, and overcome the social resistance to change. Many of the most important barriers to new stove introductions are not technical but relate to social and marketing questions (Smith, 1987). Currently, nearly all the improved stove designs in South Asia are aimed either at increasing fuel efficiency or at removing smoke from the house via a chimney or flue. Some try to accomplish both goals. Few, however, attempt to modify the combustion conditions in such a way that efficiency and low emissions are both achieved. From a health perspective providing a flue to take the smoke from the room is often sufficient. In urban areas, however, such measures are likely to have less of an impact on exposure, and achieving low emissions is of more critical importance.

Unfortunately, experience with the large-scale Indian improved stove programme indicates that locally made stoves have quite short lifetimes in households, perhaps less than one year on average (Kishore and Ramana, 1998). Preliminary cost-benefit analysis in India, on the other hand, indicates that it is difficult to justify many improved stoves on health grounds unless lifetimes exceed ten years (Smith, 1998). Such lifetimes seem only likely with stoves manufactured with durable materials. One approach is to manufacture the crucial combustion chamber components of high-quality materials under good quality control, ship them to the household, and have the householders construct the outer, less critical, parts of the stoves using local materials. This is the approach taken by the highly successful Chinese improved stove programme which has introduced more than 150 million improved stoves since 1980 (Smith *et al.*, 1993).

7.6.3 Housing improvements

In addition to changing the fuel or the stove, another option for reducing air pollutant levels is to improve the ventilation where the fuel is being used. The easiest solution in principle would be to move the cooking activity outdoors and for the stove to be located downwind from the cook or other persons nearby.

If biofuels are to be in use in rural areas for many years, then consideration should be given to changing the designs for new rural housing units to improve ventilation in the kitchen area. Although it may seem obvious that such ventilation ought to be included in new designs, there are many instances where it is not. Some designs promoted by the rural housing extension programmes of some of the major Asian countries, for example, do not explicitly include such features (Smith, 1987).

7.6.4 Improved awareness

Although scientific and medical experts seem to realise that indoor air pollution is potentially a significant problem, the people who are really affected by it - the poor women of developing countries - do not seem to be aware at all of the hazards they and their children face. Two surveys, one in Jakarta, Indonesia, and another in Accra, Ghana, highlighted the fact that such households rank indoor air pollution as one of the least priority problems in comparison to other problems such as water, wastes and pests (Surjadi *et al.*, 1994; Benneh *et al.*, 1993). Logically then, even their willingness to pay for improvements in indoor air quality was found to be low. This was found to be true across all income groups.

In South Asia there is perhaps a slightly higher awareness of the potential risks. A household survey conducted in north and south India indicated that only 17-24 per cent of the people perceive that indoor air pollution is not a problem (Ramakrishna, 1988). Between 35-52 per cent of the households responded that they had not taken any ameliorative measures. Although people are obviously aware of the relative smokiness of various fuels, they did not appear to make conscious efforts to procure and use less smokey fuels. In fact, many people actually see a benefit in the smoke - repelling mosquitoes and preserving the roof.

Clearly, there is a need for better education, awareness and risk communication programmes.

7.7 CONCLUSION

Given the enormous emissions, concentrations and populations involved with the use of traditional cooking fuels, the scale of exposures and health effects is likely to be large. There is growing scientific evidence to support the numerous anecdotal accounts relating high biomass smoke levels to important health effects. These are principally: Acute Respiratory Infection in children, Chronic Obstructive Lung Disease, Adverse Pregnancy Outcomes, and Lung Cancer in women. Of late, there are indications that Tuberculosis and Blindness may be associated with indoor air pollution. More research is sorely needed, however, before reliable estimates can be made of how much of the global burden of disease can be attributed to indoor air pollution (rough estimates indicate it to be one of the largest single risk factors for mortality - approximately 6 per cent) and how much ill-health would be reduced by smoke reduction activities such as the promotion of improved stoves.

The key areas for intervention are: developing high-grade biomass fuels; improving stove designs and dissemination approaches; improvements in housing; and improving awareness and education.

7.8 REFERENCES

Armstrong, R.W., Kutty, M.K. and Armstrong, M.J. 1978. Self-specific environments associated with nasopharyngeal carcinoma in Selangor, Malaysia. *Social Science and Medicine* **12**(3Do4D): 149-156.

Armstrong, J.R.M. and Campbell, H. 1991. Indoor air pollution exposure and lower respiratory infection in young Gambian children. *International Journal of Epidemiology* **20**(2): 424-428.

Behera, D., Dash, S. and Malik, S.K. 1988. Blood carboxyhaemoglobin levels following acute exposure to smoke of biomass fuel. *Indian Journal of Medical Research* **88**: 522-524.

Behera, D. and Jindal, S.K. 1991. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* **100**(2): 385-388.

Benneh. G., Songsore. J., Nabila J S., Amuzu, A.T., Tutu, K.A., Yangyuoru, Y. and McGranahan, G. 1993. *Environmental Problems and the Urban Household in the Greater Accra Metropolitan Area (GAMA), Ghana.* Stockholm Environment Institute, Stockholm. 126 pp.

Cerqueiro, M.C., Murtagh, P., Halac, A., Avila, M. and Weissenbacher, M. 1990. Epidemiologic risk factors for children with acute lower respiratory tract infection in Buenos Aires, Argentina: a matched case-control study. *Reviews of Infectious Diseases* **12**(8): S1021-S1028.

Chen, B.H., Hong, C.J., Pandey, M.R. and Smith, K.R. 1990. Indoor air pollution in developing countries. *World Health Statistics Quarterly* **43**(3): 127-138.

Clifford, P. 1972. Carcinogens in the nose and throat: nasopharyngeal carcinoma in Kenya. *Proceedings of the Royal Society of Medicine* **65**(8): 682B686.

Collings, D.A., Sithole, S.D. and Martin, K.S. 1990. Indoor woodsmoke pollution causing lower respiratory disease in children. *Tropical Doctor* **20**(4): 151-155.

Cooper, J.A. 1980. Environmental impact of residential wood combustion emission and its implications. *Journal of Air Pollution Control Association* **30**: 855-861.

Dary, O., Pineda, O. and Belizan, J.M. 1981. Carbon monoxide contamination in dwellings in poor rural areas of Guatemala. *Bulletin of Environmental Contamination and Toxicology* **26**(1): 24-30.

de Koning, H.W., Smith, K.R. and last, J.M. 1985. Biomass fuel combustion and health. *Bulletin of World Health Organization* **63**(1): 11-26.

GOI. 1992. Housing and Amenities. Occasional Paper No. **5** of 1994. *Census of India, 1991*. Government of India, New Delhi.

Gupta, B.N., Mathur, N., Mahendra, P.N. and Srivastava, A.K. 1997. A study of household environmental risk factors pertaining to respiratory diseases. *Energy Environment Monitor* **13**(2): 61-68.

Hong, C.J. 1991. *Health Aspects of Domestic Use of Biomass and Coal in China*. WHO/ PEP 92.3B. World Health Organization, Geneva. 23 pp.

Kishore, V.V.N. and Ramana, P.V. 1998. Improved cookstoves in rural India: How improved? *Natural Resources Forum* (in press).

Koo, L.C., Lee, N. and Ho, J.H. 1983. Do cooking fuels pose a risk for lung cancer? A case-control study of women in Hong Kong. *Ecology and Diseases* **2**(4): 255-265.

Malik, S.K. 1985. Exposure to domestic cooking fuels and chronic bronchitis. *Indian Journal of Chest Diseases and Allied Sciences* **27**(3): 171-174.

Mavalankar, D.V. 1991. Levels and risk factors for perinatal mortality in Ahmedabad, India. *Bulletin of the World Health Organization* **69**(4): 435-442.

Menezes, A.M., Victora, C.G. and Rigatto, M. 1994. Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brazil: a population-based study. *Thorax* **49**(12): 1217-1221.

Mishra, V., Retherford, R.D. and Smith, K.R. 1997a. Effects of cooking smoke on prevalence of Tuberculosis in India. Working Papers. *Population Series* No. **92**. Honolulu: East-West Centre. 26 pp.

Mishra, V., Retherford, R.D. and Smith, K.R. 1997b. Effects of cooking smoke on prevalence of blindness in India. Working Papers. *Population Series* No. **91**. East-West Centre, Honolulu. 23 pp.

Mohan, M., Sperduto, R.D., Angra, S.K., Milton, R.C., Mathur, R.L., Underwood, B.A., Jaffery, N., Pandya, C.B., Chhabra, V.K. and Vajpayee, R.B. 1989. India-US case-control study of age-related cataracts. *Archives of Opthalmology* **197**: 670-676.

Murray, C.J.L. and Lopez, A.D. 1996. *The Global Burden of Disease*. Harvard University Press, Cambridge. 990 pp.

O'Dempsey, T.J., McArdle, T.F., Morris. J., Lloyd-Evans, N., Baldeh, I., Laurence, B.E., Seeka, O. and Greenwood, B.M. 1996. A study of risk factors for pneumococcal disease among children in a rural area of west Africa. *International Journal of Epidemiology* **25**(4): 885-893.

Padmavati, S. and Arora, S.S. 1976. Sex differences in chronic cor pulmonale in Delhi. British *Journal of Diseases of the Chest* **70**(4): 251-259.

Pandey, M.R. 1984. Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* **39**(5): 337-339.

Pandey, M.R. 1988. *Chronic Bronchitis and Cor Pulmonale in Nepal.* Mrigendra Medical Trust, Kathmandu. 182 pp.

Pandey, M.R., Neupane, R.P., Gautam, A. and Shrestha, I.B. 1989. Domestic smoke pollution and acute respiratory infections in a rural community of the hill region of Nepal. *Environment International* **15**: 337-340.

Parikh, J., Smith, K.R. and Laxmi, V. 1999. Indoor air pollution: a reflection on gender bias. *Economic and Political Weekly*, February 1997: 539-544.

Ramakrishna, J. 1988. *Patterns of Domestic Air Pollution in Rural India.* Ph.D. Thesis. University of Hawaii, Honolulu.

Ramakrishna, J., Durgaprasad, M.B. and Smith, K.R. 1989. Cooking in India: The impact of improved stoves on indoor air quality. *Environment International* **15**(1-6): 341-352.

Saksena, S., Prasad, R., Pal, R.C. and Joshi, V. 1992. Patterns of daily exposure to TSP and CO in the Garhwal Himalaya. *Atmospheric Environment* **26A**(11): 2125-2134.

Shah, N., Ramankutty, V., Premila, P.G and Sathy, N. 1994. Risk factors for severe pneumonia in children in south Kerala: a hospital-based case-control study. *Journal of Tropical Pediatrics* **40**(4): 201B206.

Sharma, S., Sethi, G.R., Rohtagi, A., Chaudhary, A., Shankar, R., Bapna, J.S., Joshi, V. and Sapir, D.G. 1998. Indoor air quality and acute lower respiratory infection in Indian urban slums. *Environmental Health Perspectives* **106**(5): 291B297.

Smith, K.R. 1986. The biofuel transition. *Pacific and Asian Journal of Energy* **1**(1): 17B32.

Smith K R. 1987. *Biofuels, Air Pollution and Health: A Global Review.* Plenum Press, New York and London. 452 pp.

Smith, K.R. 1993. Fuel combustion, air pollution exposure, and health: the situation in developing countries. *Annual Review of Energy and Environment* **18**: 529B566.

Smith, K.R. 1996. Indoor air pollution in developing countries: growing evidence of its role in the global disease burden. pp. 33B34. Volume **3**. In *Proceedings of Indoor Air '96: The 7th International Conference on Indoor Air Quality and Climate.*. Institute of Public Health, Tokyo.

Smith, K.R. 1998. *Indoor Air Pollution in India: National Health Impacts and the Cost-effectiveness of Intervention.* Indira Gandhi Institute for Development Research, Mumbai.

Smith, K.R., Shuhua, G., Kun, H. and Daxiong, Q. 1993. 100 million improved stoves in China: How was it done? *World Development* **21**(6): 941-961.

Smith, K.R., Apte, M.G., Ma, Y.Q., Wongsekiarttirat, W. and Kulkarni, A. 1994. Air pollution and the energy ladder in Asian cities. *Energy* **19**(5): 587-600.

Smith, K.R. and Liu, Y. 1994. Indoor air pollution in developing countries. In *The Epidemiology of Lung Cancer* (ed. by J. Samet). Marcell Dekker, New York.

Sobue, T. 1990. Association of indoor air pollution and lifestyle with lung cancer in Osaka, Japan. *International Journal of Epidemiology* **19** (Supplement 1): s62Bs66.

Surjadi, C., Padhmasutra, L. and Wahyuningsih, D. et al. 1994. *Household Environmental Problems in Jakarta.* Stockholm Environment Institute, Stockholm. 64 pp.

WEC. 1998. Global Transport and Energy Development: Scope for Change. World Energy Council, London.

WHO. 1992. *Indoor Air Pollution from Biomass Fuel.* WHO/PEP/92-3 A. World Health Organization, Geneva. 64 pp.

WHO. 1997. *Health and Environment in Sustainable Development: Five Years after the Earth Summit.* World Health Organization, Geneva. 242 pp.

WRI. 1998. *World Resources: 1998-99*. World Resources Institute. Oxford University Press, Oxford. 369 pp.

Yu, M.C., Ho, J.H., Henderson, B.E. and Armstrong, R.W. 1985. Epidemiology of nasopharyngeal carcinoma in Malaysia and Hong Kong. *National Cancer Institute Monograph* **69**: 203-207.

Chapter 8

MOTOR VEHICLE POLLUTION AND ITS CONTROL IN ASIA

Michael P. Walsh
3105 North Dinwiddie Street, Arlington, VA 22207, USA

Abstract

Increasing prosperity and population growth in many developing countries are resulting in accelerated growth in vehicle population and vehicle miles travelled. While most developing countries currently have very few motorized vehicles per capita compared to the OECD countries, the vehicle population is growing very rapidly. In South and South-East Asia the popularity of motorcycles and scooters which have highly polluting two stroke engines, and other characteristics of the vehicle fleet, such as vehicle age and maintenance, fuel type, etc., lead to substantially more emissions per kilometre driven than in the developed countries. As most of the current vehicle population is concentrated in the major cities, these cities usually have poor air quality. The poor air quality in these cities can cause serious health problems, especially with the very old, the very young and those with pre-existing respiratory diseases.

Motor vehicles emit large quantities of carbon monoxide, hydrocarbons, nitrogen oxides, and toxic substances including fine particles and lead. Each of these, along with secondary by-products such as ozone, can cause adverse effects on health and the environment. However, significant improvements in air quality are being achieved in some parts of Asia. Taiwan has implemented a motorcycle control programme expected to eliminate new two stroke motorcycles by about 2003, and to encourage users to convert to electric motorcycles. Some other Asian countries including Thailand and China are expected to implement similar programmes to reduce emissions from motorcycles. Singapore has developed one of the pre-eminent land transport planning programmes in the World serving as a model to its neighbours.

Most Asian countries have made or will soon make progress to reduce vehicles emissions as a major source of air pollution in cities. For example, Thailand banned all sales of leaded gasoline in 1996 and China will do so by 2000. India and the Philippines are expected to follow suit. All new cars sold in Hong Kong, Singapore, Taiwan, Thailand and South Korea have been required to be equipped with catalysts for several years and China, India and the Philippines will require these controls within the next few years.

8.1 BACKGROUND AND INTRODUCTION

Over the past 50 years, the World's vehicle population has grown fifteen-fold. As a result, the global motor vehicle population today - including passenger cars, trucks, buses, motorcycles and three-wheeled vehicles (Tuk Tuks) - exceeds 700 million units and is projected to reach 1 billion shortly after the turn of the century. As illustrated in Figure 1, most of these vehicles are concentrated in the highly industrialized countries of the OECD. However, that is now changing rapidly. As a result, an increasing number of urbanized areas in developing countries are experiencing accelerated growth in vehicle population and vehicle miles travelled. Nowhere is this truer than in South-East Asia.

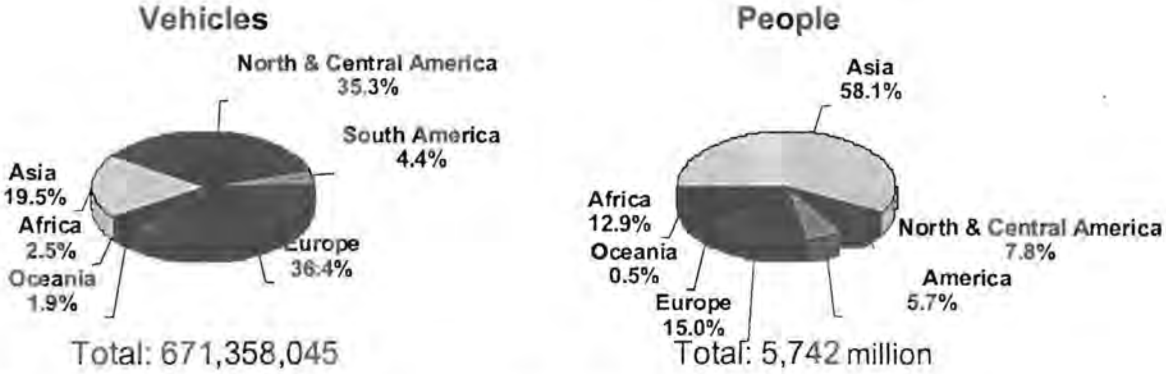


Figure 8.1 Global distribution of vehicles and people - 1996

These vehicles have brought many advantages, including increased mobility, economic flexibility, efficiency improvements, more jobs, and other quality of life enhancements. However, the benefits have been at least partially offset by excessive pollution and the adverse health and environmental effects which result from air pollution.

Motor vehicles emit large quantities of carbon monoxide, hydrocarbons, nitrogen oxides, and such toxic substances as fine particles and lead. Each of these, along with secondary by-products such as ozone, can cause adverse effects on health and the environment. Because of the growing vehicle population and the high emission rates from many of these vehicles, serious air pollution and related health effect problems have been increasingly common phenomena in modern life.

Reducing the pollution that comes from vehicles will usually require a comprehensive strategy as shown in Figure 8.2. Generally, the goal of a motor vehicle pollution control programme is to reduce emissions from motor vehicles in-use to the degree reasonably necessary to achieve healthy air quality as rapidly as possible or, failing that for reasons of impracticality, to the practical limits of effective technological, economic and social feasibility. A comprehensive strategy to achieve this goal includes four key components: stringent emissions standards for new vehicles, clean fuels, programmes to assure proper maintenance of in-use vehicles, and traffic and demand management. The emission reduction goal should be achieved in the most cost- effective manner available.

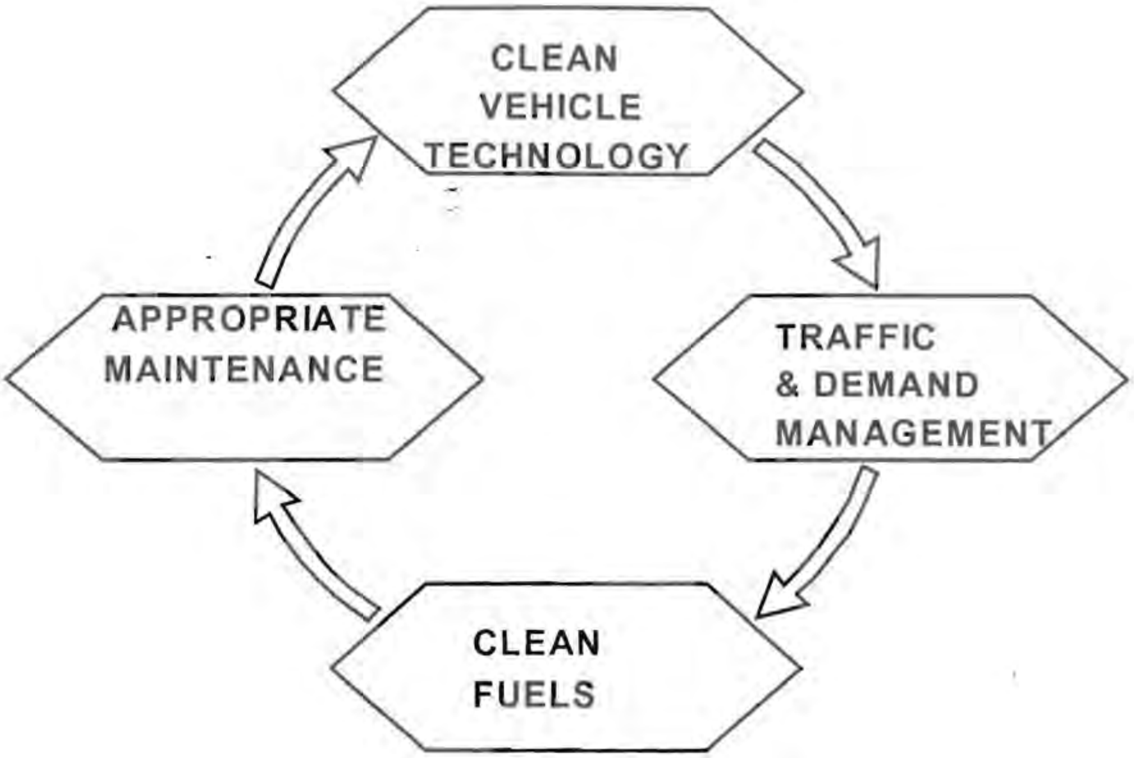


Figure 8.2 Elements of a comprehensive vehicle pollution control strategy

This Chapter will focus on recent developments in Asia. Initially, the vehicle population and characteristics will be summarized. Thereafter, the current role of motor vehicles as a source of air pollution in the region will be reviewed. Finally, some of the more significant efforts to reduce vehicle pollution in Asia will be discussed.

8.2 VEHICLE POPULATION TRENDS AND CHARACTERISTICS

As illustrated in Figure 8.3, led by Japan, Asia has become an equal partner with Europe and North America in terms of annual production of new cars, trucks and buses. While an economic slump is underway at the present time, it is clear that South Korea, Taiwan, Malaysia, China and India are increasingly important producers of new vehicles. Further, motorcycle production in China, Japan, India and Taiwan is higher than anywhere else in the World (Figure 8.4). Consequently, it is not surprising that the fraction of vehicles which are two and three wheelers is much higher throughout Asia than anywhere else in the World (Figure 8.5). Many of these vehicles are powered by two-stroke engines which burn both gasoline and lubricating oil; hydrocarbon and smoke emissions from these engines are very high.

Focusing on India as an example, Figure 8.6 illustrates that the production of all vehicle categories has grown rapidly throughout the 1990s. Figure 8.7 shows that production of almost all vehicle categories grew by double digits in the past year, especially motorcycles and three wheelers. Recent studies carried out in Beijing indicate that, between 2000 and 2010, the motor vehicle population will approximately double (Figure 8.8).

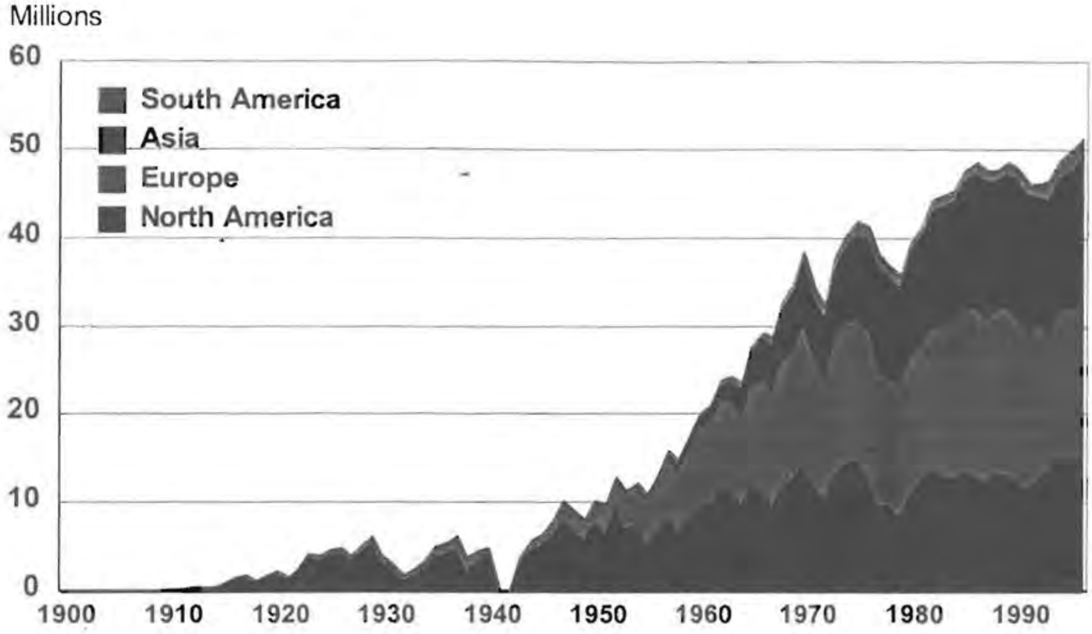


Figure 8.3 Global trends in motor vehicle production

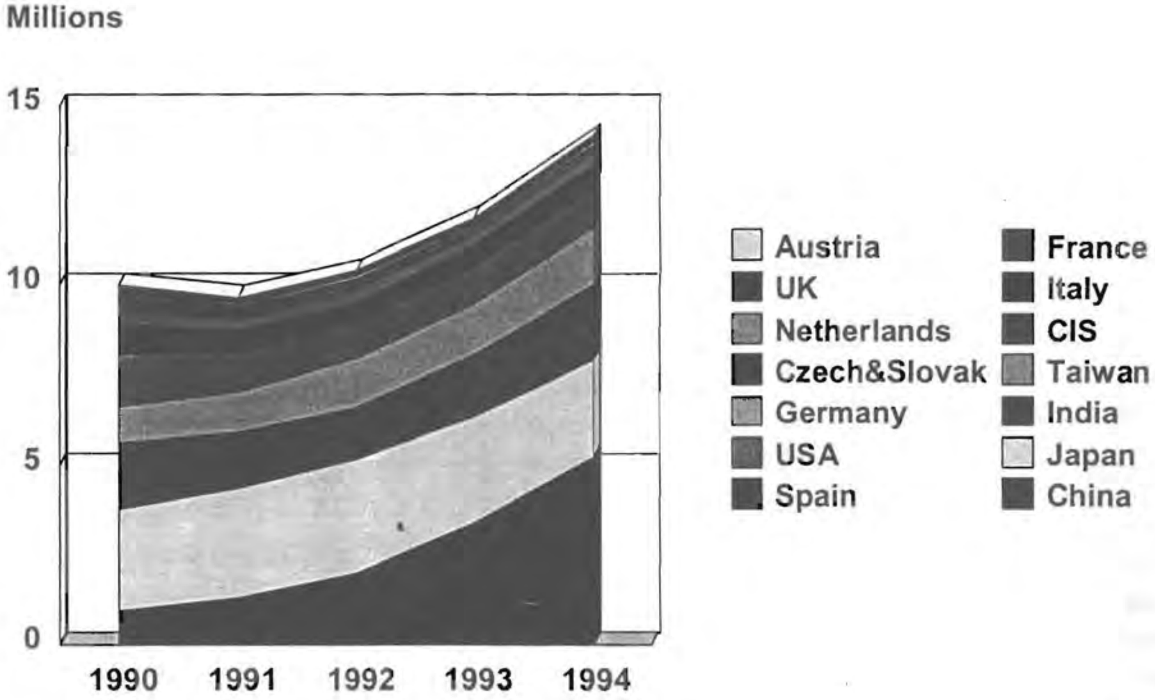


Figure 8.4 Motorcycle production in selected countries

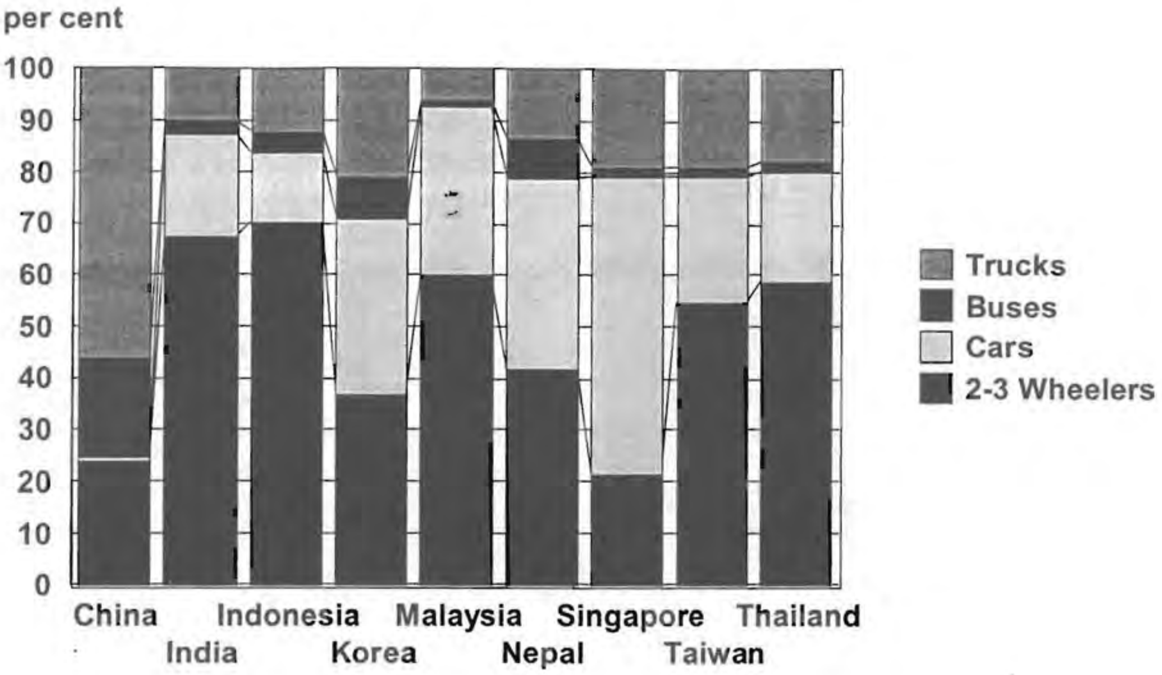
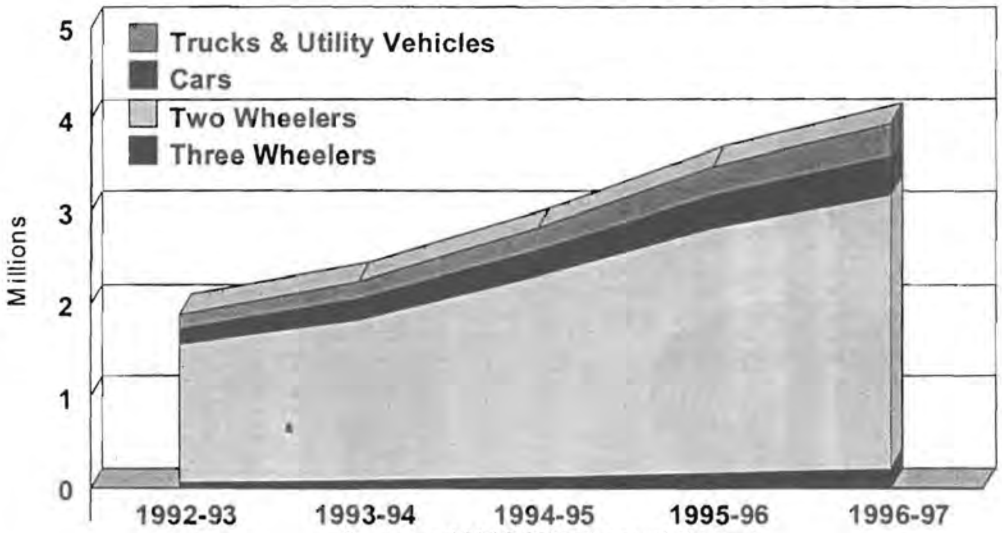


Figure 8.5 Vehicle types throughout Asia



Source: AIAM 37th Annual Report

Figure 8.6 Vehicle production trends in India

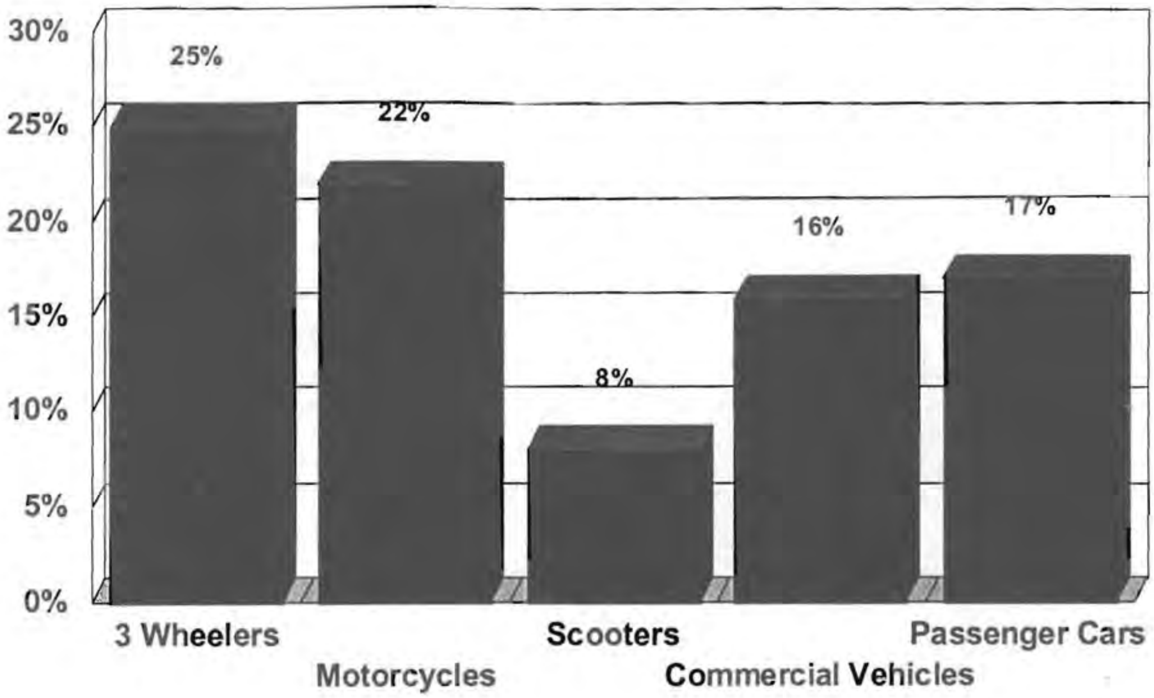


Figure 8.7 Annual average growth in vehicle production - India

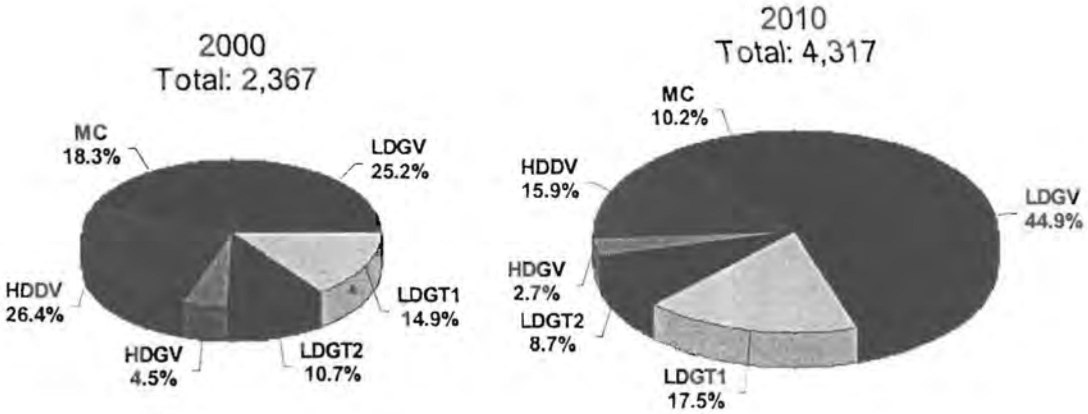


Figure 8.8 Projected motor vehicle population in Beijing (000)

8.3 VEHICLES AS A SOURCE OF EMISSIONS

Throughout Asia, air pollution is a serious problem. Cities as diverse as Hong Kong, Delhi, Bangkok and Seoul, to cite just a few, currently exceed healthy air quality levels, sometimes by a factor of 2 or 3. In Taipei for example, as shown in Figure 8.9, PM_{10} and ozone air quality standards are exceeded several times per year. In Bangkok, it is estimated that roadside emissions of particulate, carbon monoxide and lead must be reduced by 85 per cent, 47 per cent and 13 per cent respectively if acceptable air quality is to be achieved.

While many sources contribute to pollution in these cities, vehicles clearly stand out as major sources. Figures 8.10 and 8.11 show that vehicles emit approximately 80 per cent of the CO in both Beijing and Guangzhou, China, and almost 40 per cent of the NO_x. In Delhi, India, vehicles are the major source of hydrocarbons, nitrogen oxides and carbon dioxide (Figure 8.12).

In high traffic areas of Hong Kong, diesel vehicles have been found to be responsible for more than half the respirable particles (Figure 8.13); at two locations in Bangkok, motorcycles were found to be the major source of particulate air pollution (Figure 8.14).

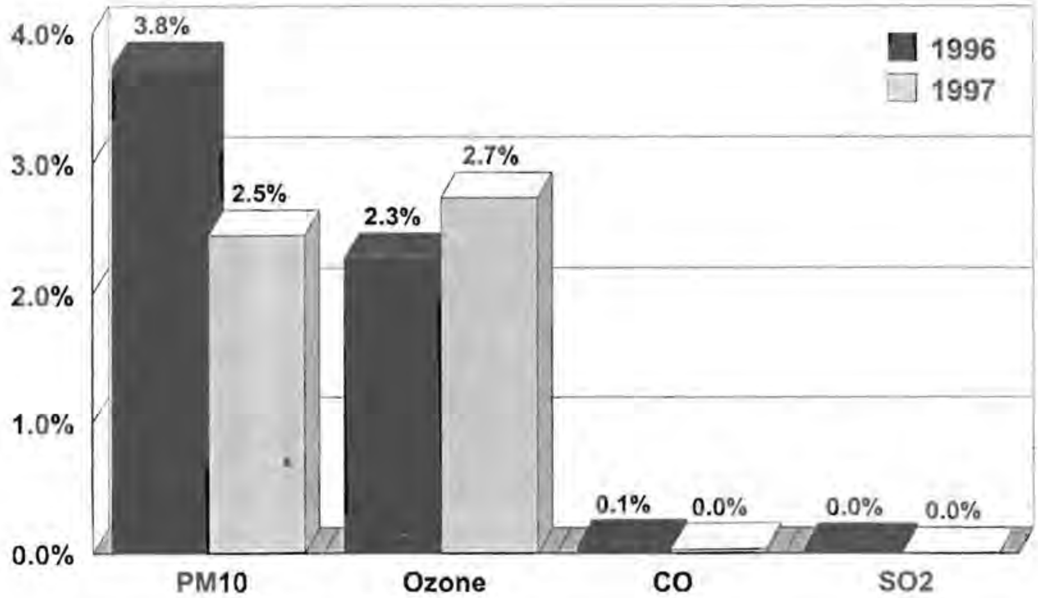


Figure 8.9 Violations of the air quality standards - Taipei

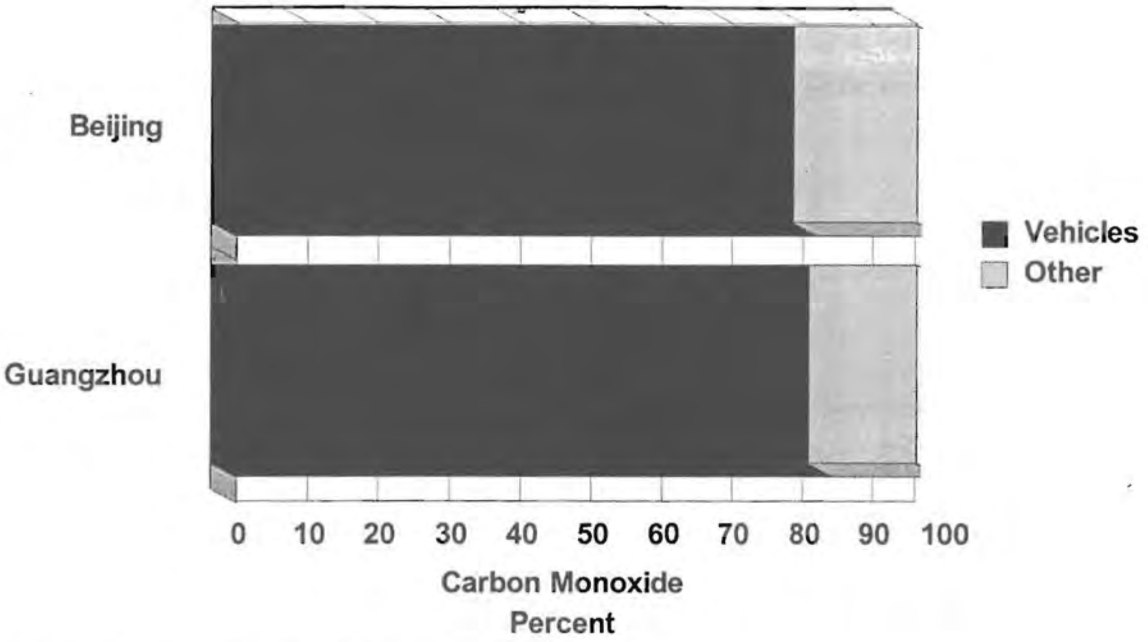


Figure 8.10 Vehicle contribution to emissions

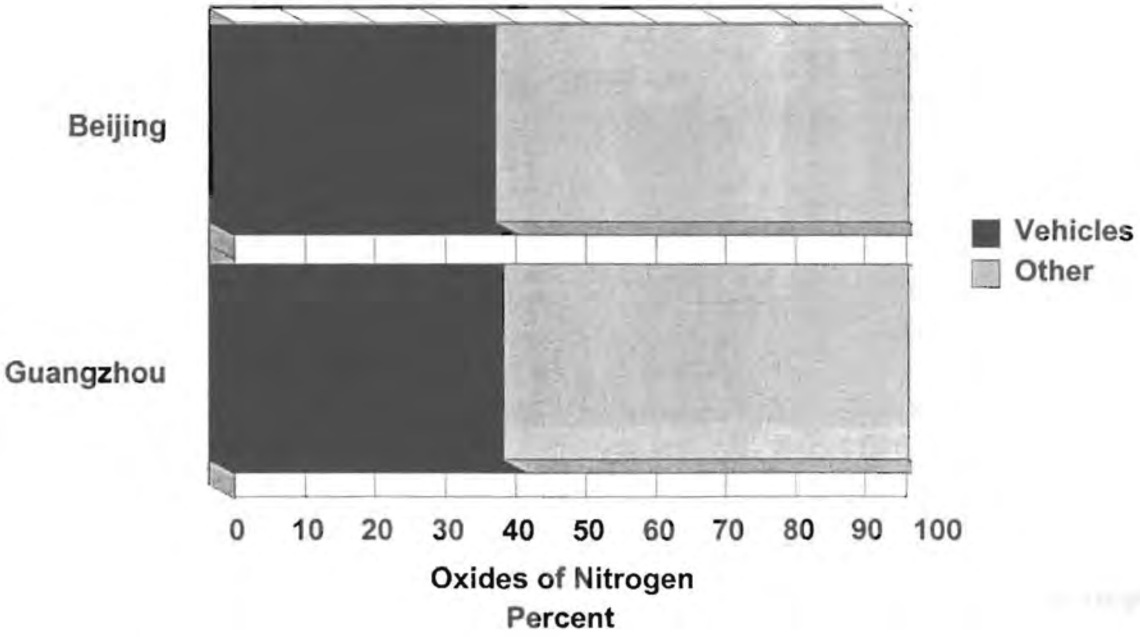


Figure 8.11 Vehicle contribution to emissions

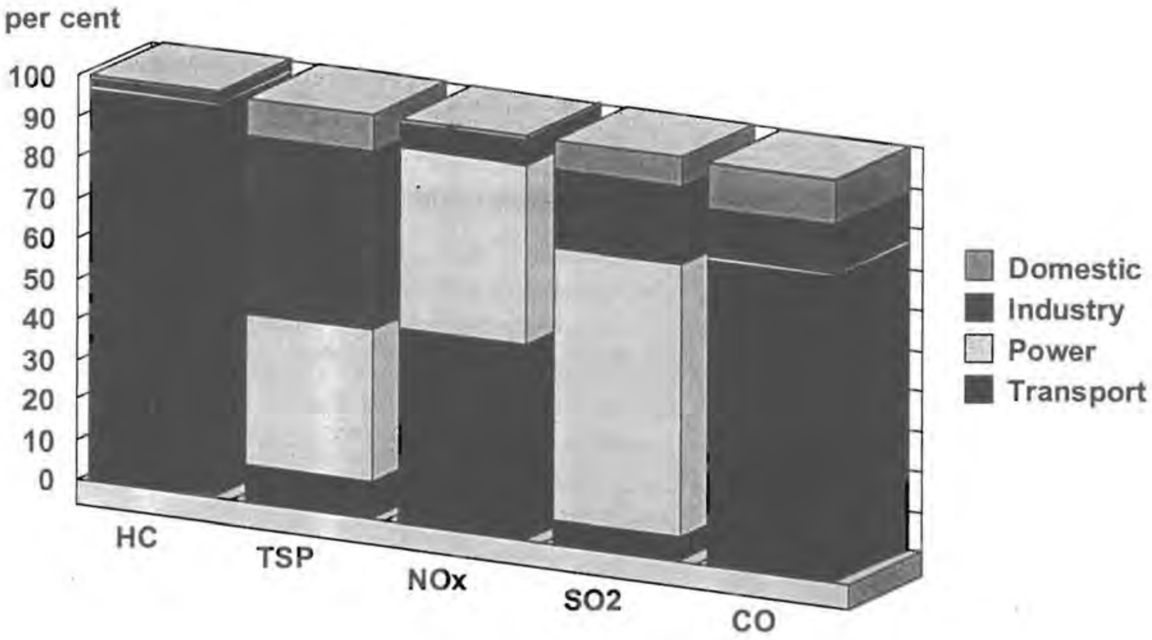


Figure 8.12 Sources of emissions in Delhi, India - 1995

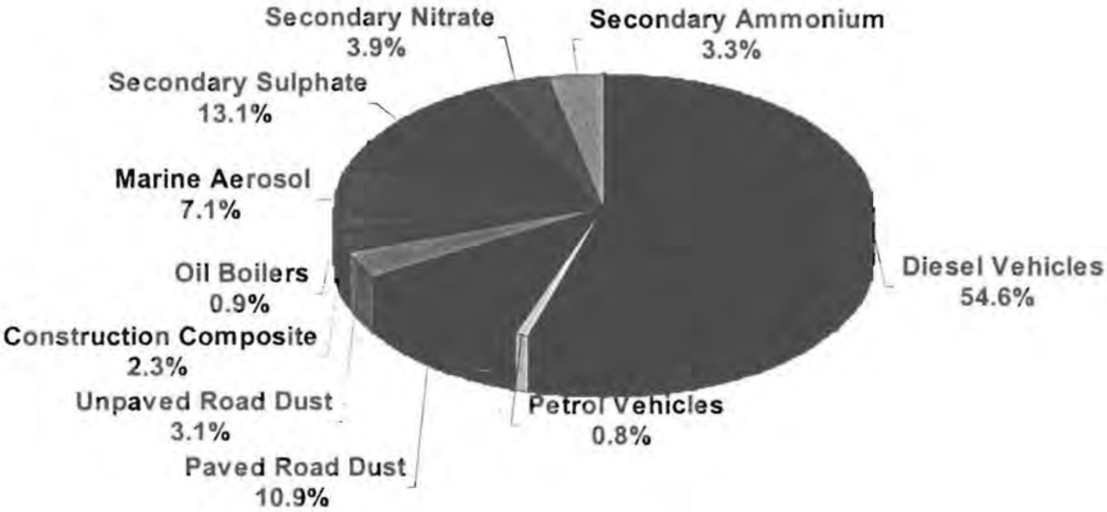
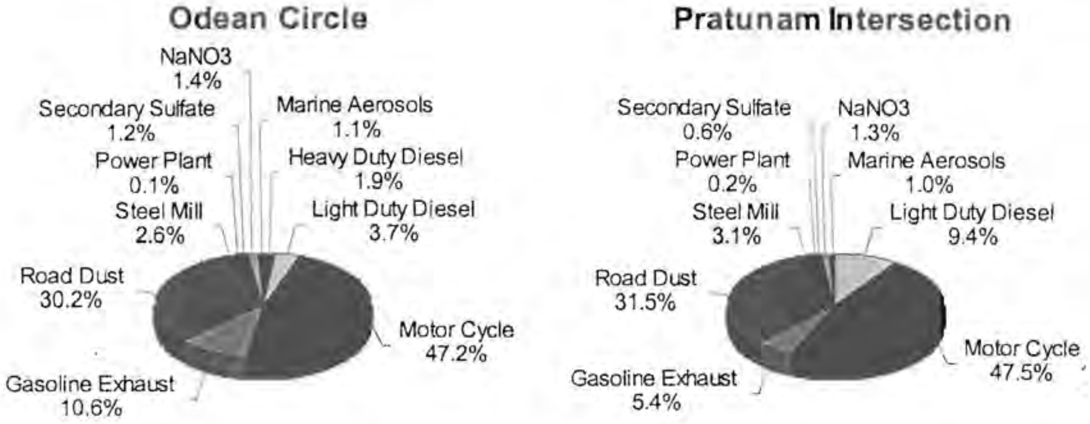


Figure 8.13 Source contribution for RSP Mong Kok annual average



Source: PM Abatement Strategy for the Bangkok Metropolitan Area
Draft Final Report
Radian International

Figure 8.14 Sources of PM emissions in high traffic areas of Bangkok - 1996

Recently, the Taiwan EPA took advantage of routine air raid drills in three major urban areas - Taipei, Taichung and Kaohsiung - to determine the impact of vehicles. During air raids, all traffic is required to stop and vehicle engines are turned off. By comparing air quality readings before and during the drills, the role of motor vehicles can be ascertained. The results summarized in Table 8.1 indicated that CO and NO_x concentrations fell by about 80 per cent after vehicles came to a halt and that levels quickly returned to normal once traffic began moving.

Table 8.1 Comparison of air pollution in urban areas between traffic and non-traffic situations

Pollutant		Taipei	Taichung	Kaohsiung
CO	Avg. concentration (ppm)	3.00	1.50	3.00
	Vehicles stationary (ppm)	0.50	0.50	0.50
	Reduction (%)	83.33	66.67	83.33
NO_x	Avg. concentration (ppb)	300.00	60.00	200.00
	Vehicles stationary (ppb)	50.00	10.00	50.00
	Reduction (%)	83.33	83.33	75.00

The survey results clearly indicate the substantial degree to which motor vehicles contribute to air pollution in urban areas.

8.4 ADVERSE HEALTH EFFECTS RESULTING FROM VEHICLE EMISSIONS

Emissions from vehicles and other sources can result in serious adverse effects on health as summarized below.

8.4.1 Photochemical oxidants (ozone)

Ground-level ozone (O_3) is the prime ingredient of smog, the pollution that blankets many areas during the summer[1]. Short-term exposures (1-3 hours) to high ambient O_3 concentrations have been linked to increased hospital admissions and emergency room visits for respiratory problems. Repeated exposures to O_3 can exacerbate symptoms and the frequency of episodes for people with respiratory diseases such as asthma. Other health effects attributed to short-term exposures include significant decreases in lung function and increased respiratory symptoms such as chest pain and cough. These effects are generally associated with moderate or heavy exercise or exertion. Those most at risk include children who are active outdoors during the summer, outdoor workers, and people with pre-existing respiratory diseases such as asthma. In addition, long-term exposures to O_3 may cause irreversible changes in the lungs which can lead to chronic ageing of the lungs or chronic respiratory disease.

As discussed in the Introduction and Chapter 2, ozone is not emitted directly into the atmosphere, but is formed by a reaction of VOC and NO_x in the presence of heat and sunlight. Ground-level O_3 forms readily in the atmosphere, usually during hot summer weather. Changing weather patterns contribute to yearly differences in O_3 concentrations and differences from city to city. O_3 can also be transported into an area from pollution sources found hundreds of miles upwind.

In the US, EPA recently tightened the air quality standard from 0.12 parts per million of O_3 measured over one hour to 0.08 parts per million measured over eight hours, with the average fourth highest concentration over a three-year period determining whether an area is out of compliance. The updated standard recognizes the current scientific view that exposure to O_3 levels at and below the <u>current</u> standard causes significant adverse health effects in children and in healthy adults engaged in outdoor activities.

8.4.2 Particulate (PM)

Particulate matter has been characterized in the Introduction and Chapter 2. As discussed in previous chapters, scientific studies show a link between particulate matter (alone or in combination with other pollutants in the air) and a series of health effects. Studies of human populations and laboratory studies of animals and humans have established linkages to major human health impacts including breathing and respiratory symptoms; aggravation of existing respiratory and cardiovascular disease; alterations in the body's defence systems against foreign materials; damage to lung tissue; carcinogenesis; and premature mortality.

Motor vehicle particle emissions and the particles formed by the transformation of motor vehicle gaseous emissions tend to be in the fine particle range (Chapter 2). Scientific studies

---

[1] Ozone occurs naturally in the stratosphere and provides a protective layer high above the Earth.

have linked fine particles (alone or in combination with other air pollutants), with a series of significant health problems, including premature death; respiratory related hospital admissions and emergency room visits; aggravated asthma; acute respiratory symptoms, including aggravated coughing and difficult or painful breathing; chronic bronchitis; and decreased lung function that can be experienced as shortness of breath. The World Health Organization (WHO) estimates that approximately 460,000 people die prematurely each year as a result of exposure to PM in the air.

As with ozone, the US EPA recently tightened the air quality standards for particulate. The standard for coarse particles (10 microns or less) remains essentially unchanged, while a new standard for fine particles (2.5 microns or less) was set at an annual limit of 15 micrograms per cubic metre and a 24-hour limit of 65 micrograms per cubic metre.

Some particles have been found to be more hazardous than others. For example, the California Air Resources Board (CARB) recently evaluated diesel exhaust as a candidate toxic air contaminant under the State's air toxics identification programme. To evaluate whether or not diesel exhaust causes cancer, they reviewed all controlled animal and mutagenicity studies as well as studies of worker populations exposed to diesel exhaust. In the last decade, seven studies on rats have demonstrated that exposure to diesel exhaust through inhalation causes cancer. In each of these studies, rats were exposed to concentrations of diesel exhaust greater than 2.5 mg/m^3 (2,500 µg/m^3) and were observed for periods longer than 24 months.

The report also analysed over 30 human studies concerning lung cancer risk and workplace exposure to diesel exhaust. Workers who were exposed to diesel exhaust were more likely than others to develop lung cancer. The consistent results are unlikely to be due to chance, confounding, or bias, according to CARB.

The report concludes that a reasonable and likely explanation for the increased rates of lung cancer observed in the epidemiological studies is a causal association between diesel exhaust exposure and lung cancer.

8.4.3 Lead

Over the past century, a range of clinical, epidemiological and toxicological studies have continued to define the nature of lead toxicity, to identify young children as a critically susceptible population, and to investigate mechanisms of action of lead toxicity. Lead toxicity has been briefly summarized in Chapter 3. In summary, lead affects many organs and organ systems in the human body with subcellular changes and neurodevelopmental effects appearing to be the most sensitive. The most substantial evidence from cross sectional and prospective studies of populations with lead levels generally below 25 µg/deciliter of blood relates to decrements in intelligence quotient (IQ).

Existing epidemiological studies do not provide definitive evidence of a threshold. Below the range of about 10-15 µg/deciliter of blood, the effects of confounding variables and limits in the precision in analytical and psychometric measurements increase the uncertainty attached to any estimate of effect. However, there is some evidence of an association below this range. Animal studies provide support for a causal relationship between lead and nervous system effects, reporting deficits in cognitive functions at lead levels as low as 11-15 µg/deciliter of blood which can persist well beyond the termination of lead exposure. Other effects which may occur include:

- impaired sensory motor function;
- impaired renal function;
- a small increase in blood pressure has been associated with lead exposure;
- some but not all epidemiological studies show a dose dependent association of pre-term delivery and some indices of fetal growth and maturation at blood lead levels of 15 μg/deciliter or more.

In countries where leaded gasoline is still used, the major air emission is from mobile and stationary combustion of gasoline. Because of the concerns highlighted above, a global consensus has emerged to phase out the use of lead in gasoline (CDC, 1991; Howson and Hernandez Avila, 1996; IPCS, 1995; NRC, 1993; US EPA, 1986).

As recently noted by the US EPA (US EPA, 1998):

- in December 1994, at the Summit of the Americas, heads of state from a number of countries pledged to develop national action plans for the phase out of leaded gasoline in the Western Hemisphere;
- in May 1996, the World Bank called for a global phase out of leaded gasoline and offered to help countries design feasible phase out schedules and incentive frameworks;
- a key recommendation of the Third "Environment for Europe" Ministerial Conference held in Sofia, Bulgaria in October 1995 called for the reduction and ultimate phase out of lead in gasoline;
- in June 1996, the second United Nations Conference on Human Settlements, called Habitat II, included the elimination of lead from gasoline as a goal in its agenda;
- in May 1997, environmental ministers from the Group of Seven plus Russia endorsed the phase out of leaded gasoline in the 1997 Declaration of Environmental Leaders of the Eight on Children's Environmental Health.

8.4.4 Lead scavengers

When lead additives were first discovered to improve gasoline octane quality, they were also found to cause many problems with vehicles. Notable among these was a very significant build up of deposits in the combustion chamber and on spark plugs, which caused durability problems. To relieve these problems, lead scavengers were added to gasoline at the same time as the lead to encourage greater volatility in the lead combustion by-products so they would be exhausted from the vehicle. These scavengers continue to be used today with leaded gasoline.

Ultimately, a significant portion of these additives is emitted from vehicles. This is important because, unfortunately, these lead scavengers, most notably ethylene dibromide, have been found to be carcinogenic in animals and have been identified as potential human carcinogens by the National Cancer Institute (Sigsby *et al.*, 1982). Therefore, their removal along with the removal of lead may result in significant benefits to health.

8.4.5 Carbon monoxide (CO)

CO is a major vehicle emission. It is a tasteless, odourless and colourless gas produced though the incomplete combustion of carbon-based fuels. The health threat from CO has been discussed in Chapter 3, and it is most serious for those who suffer from cardiovascular disease, particularly those with angina or peripheral vascular disease. Healthy individuals also are affected, but only at higher levels. Exposure to elevated CO levels is associated with impairment of visual perception, work capacity, manual dexterity, learning ability and performance of complex tasks.

8.4.6 Nitrogen oxides (NO_x)

Nitrogen oxides are emitted in substantial amounts into the air of cities by combustion of fuels by vehicles. NO_x emissions produce a wide variety of health effects, as discussed in Chapter 3. Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infection (such as influenza). NO_x emissions are an important precursor to acid rain and may affect both terrestrial and aquatic ecosystems. Nitrogen dioxide and airborne nitrate also contribute to pollutant haze, which impairs visibility and can reduce residential property values and revenues from tourism.

8.4.7 Other toxics

The 1990 Clean Air Act (CAA) directed the US EPA to complete a study of emissions of toxic air pollutants associated with motor vehicles and motor vehicle fuels. The study found that in 1990 the aggregate risk is 720 cancer cases in the US. For all years, 1,3-butadiene is responsible for the majority of the cancer incidence, ranging from 58 to 72 per cent of the total motor vehicle toxics risk. This is due to the high unit risk of 1,3-butadiene. Gasoline and diesel particulate matter, which are considered to represent motor vehicle polycyclic organic matter (POM), are roughly equal contributors to the risk. The combined risk from gasoline and diesel particulate matter ranges from 16 to 28 per cent of the total, depending on the year examined. Benzene is responsible for roughly 10 per cent of the total for all years. The aldehydes, predominately formaldehyde, are responsible for roughly 4 per cent of the total for all years.

A variety of studies have found that in individual metropolitan areas, mobile sources are one of the most important and possibly the most important source category in terms of contributions to health risks associated with air toxics. For example, according to the US EPA, mobile sources are responsible for almost 60% of the air pollution related cancer cases in the US per year.

8.5 VEHICLE POLLUTION CONTROL PROGRAMMES

Serious pollution problems are not inevitable and several countries in the region have adopted significant efforts to reduce vehicle emissions.

8.5.1 Motorcycle pollution controls in Taiwan

Because almost 75 per cent of road vehicles in Taiwan are motorcycles, the most distinctive feature of its vehicle pollution control programme is its motorcycle control component which is summarized below.

- The emissions standards for new motorcycles were reduced to 3.5 grams per kilometre for CO, and 2.0 for HC and NO_x combined in 1998. As a result of these requirements, most of the engines for four stroke motorcycles have been redesigned to use secondary air injection and most of the two stroke motorcycles are fitted with catalytic converters.

- Since 1991, all new motorcycles must also be equipped with evaporative controls.

- In order to reduce the pollution from in-use motorcycles, the EPA is actively promoting a motorcycle Inspection and Maintenance (I/M) system. As of January 1998, there were a total of 1,061 stations, which include 999 stationary stations and 62 mobile stations spreading over 15 cities and/or counties. Approximately 1.4 million motorcycles were inspected in 1996; 22.5 per cent failed. Approximately 2.8 million motorcycles were inspected in 1997 with 19.4 per cent failing. Of the failing vehicles, 70 per cemt were repaired and re-inspected and, of these, 77 per cent passed. On average, CO improved by 48 per cent and HC by 35 per cent after repair. Three-quarters of the failed motorcycles spent less than NTD $500 for repair.

- Since 1992, electric motorcycles have been available in the market but sales have been modest. The market share for electric powered motorcycles will be mandated at 2 per cent by 2000. Currently, there are approximately 10 million motorcycles in Taiwan. Pollution emission can be greatly reduced if a significant number of them can switch from the use of gasoline fuel to electricity. Thirty-five colleges and/or universities have been entrusted to promote the use of electric vehicles. Meanwhile, pertinent studies are being carried out by the Industrial Technology Research Institute. $5,000 is given as a supplement to each electric vehicle purchased. Currently, there are more than 600 electric motorcycles in operation. In the future, charging stations in public areas including parking lots and battery exchange stations will also be established.

- The EPA announced on 5 August that the *Fourth Stage Motorcycle Emission Standards* are to come into effect on 31 December, 2003. The main features are:
  - Sets different emission standards for two- and four-stroke motorcycles. First, second and third stage standards used the same standards for both two- and four-stroke motorcycles. According to investigation results, however, the average emissions value of a cold engine tested two-stroke motorcycle was roughly triple that of a four-stroke motorcycle and the results were even worse when the motorcycle was in poor condition. In an effort to force the dirtier engines out of the market, the standards for two-stroke motorcycles in the fourth stage standards are twice as strict as that for four-stroke motorcycles.
  - Changes tests from warm to cold engine. First, second and third stage standards testing procedures all used the warm engine method whereby tests were conducted after the motorcycle was driven for 10 kilometres until the engine was warm. According to the EPA, investigations indicated that about 70 per cent of trips averaged less than 10 kilometres round trip with a one-way journey of no more than five kilometres. Moreover, the actual quantity of emissions detected in a cold engine test was 2.5 times that for a warm engine test.
  - Tightens emission standards for in-use motorcycles. For the sake of convenience, standards for CO and HC used to audit in-use motorcycles remained for many years at an average of 4.5 per cent and 9,000 ppm respectively. Given the

increased performance of motorcycles and to ensure that catalytic converters continue to be used, the standards for CO and HC are to be tightened to 3.5 per cent and 2,000 ppm respectively. In the future, in-use motorcycles that are not properly maintained may have trouble passing inspection.

- Two-stroke models currently account for about half of all motorcycles. Under current conditions, two-stroke models will likely have trouble adjusting to the fourth stage standards when they come into effect and thus two-stroke motorcycles are likely to be eliminated.

8.5.2 Singapore's land transport policy

Singapore's land transport policy strives to provide free-flowing traffic within the constraint of limited land. A four-pronged approach has been adopted to achieve this. Firstly, the need to travel is minimized through systematic town planning. Secondly, an extensive and comprehensive network of roads and expressways, augmented by traffic management measures, has been built to provide quick accessibility to all parts of Singapore. Thirdly, a viable and efficient public transport system that integrates both the Mass Rapid Transit (MRT) and bus services is promoted. Finally, the growth and use of vehicles are managed to prevent congestion on the road.

8.5.3 Lead-free petrol in China

8.5.3.1 China moving ahead quickly

From 1 January, 2000, all petroleum enterprises in China are to produce only lead-free petrol, according to an announcement made in mid-September by the State Council. All cars must use unleaded petrol from 1 July that year. The circular was released at a State Environmental Protection Administration (SEPA) press conference in Beijing on 18 September.

From 1 July, 1999, petrol stations in major cities, including provincial capitals and special economic zones, should only sell lead-free petrol. One year later, all petrol stations nationwide will follow suit, the circular specified. In addition, from 1 January, 2000 car-manufacturing enterprises must make their models suitable for use with lead-free petrol. Catalytic converters and electronic fuel injection systems should be installed on all new cars. The notice also required that public transport buses should gradually begin switching to cleaner fuels such as natural gas and electricity.

The consumption tax rates on leaded petrol for vehicle use will be raised, making prices for leaded petrol higher than for lead-free petrol. To date, 18 provinces or cities including Beijing, Shanghai and Guangzhou, have adopted lead-free petrol in all or some of the areas under their jurisdiction.

Beijing has taken the lead regarding new car standards, having decided to implement ECE R83.01 (so-called Euro 1) regulations in early 1999; these are the same standards which were first required in the European Union in 1992.

8.5.4 Recent progress in India

A series of measures has been initiated by the Government of the National Capital Territory of Delhi to mitigate the problem of vehicular pollution. As a part of a public interest litigation, the Supreme Court of India has issued several directions to the Delhi Government and fixed deadlines for various actions. While these actions pertain to Delhi, the measures are likely to be emulated by other Indian states.

8.5.4.1 Leaded petrol banned in Delhi

The National Capital Territory of Delhi has switched over fully to unleaded petrol. No leaded petrol has been permitted to be sold in the region since 1 September, 1998.

8.5.4.2 Old vehicle phase out proceeds

Under sustained pressure from the Supreme Court, the Delhi Government is now implementing a plan by which it disallows the use of old commercial vehicles in the National Capital Territory of Delhi. Diesel buses and trucks, three wheeler autorikshaws and taxis, which are more than 15 years old, were allowed to be used after 1 January, 1999. Holders of permits for these vehicles will be allowed to buy new vehicles complying with the current emission standards, provided they either scrap the old vehicle or show proof of its sale outside the Delhi territory. In case of autorikshaws and taxis, the replacement vehicles will be expected to comply with the Indian 2000 emission standards from 1 April, 1999. Issue of new permits for autorikshaws had been stopped more than two years ago. It is estimated that the total number of such vehicles is around 18,000. The Government is drawing up a package of incentives such as low interest finance, subsidy on interest, etc. It is proposed to follow this with a similar ban on 12 years and older vehicles from 1 April, 1999. An option to allow replacement of the engines on such vehicles with those which comply with the current emission standards is likely to be made available. The move is already meeting with some resistance from the associations and unions of transporters, as they consider the measure to be too severe, especially for the individual and small fleet operators.

8.5.4.3 Ban on sale of loose two stroke oil

A majority of the 1.7 million two and three wheelers in Delhi are powered by two stroke engines which use a blend of two-stroke lubricant (2T oil) and petrol. Most of the consumers leave it to the pump attendant to manually mix the 2T oil from open drums with petrol. This leads to the use of incorrect, mostly excessive, proportions and inferior grades of 2T oils, leading to high smoke emissions. With a view to curb the emission of visible smoke, under directions from the Government, around half the petrol retail outlets in Delhi have made arrangements to dispense pre-mixed 2T oil for two and three wheelers. It is now proposed to make a complete change over to pre-mixed dispensing only from 1 January, 1999 which will also be accompanied by a regulation that will ban the sale of loose 2T oils.

8.5.4.4 Promotion of CNG

Proposals to promote the progressive change over of autorikshaws and taxis in Delhi to CNG are under active consideration. The Gas Authority of India Ltd. (GAIL) has been asked to provide 80 CNG filling stations throughout the Delhi region by April 2000 in order to achieve this objective. It is also proposed to convert the entire state-owned public transport buses to CNG by April 2001.

8.5.4.5 Other items

The Supreme Court has also directed the Delhi Government to take up the installation of a centralized Inspection and Maintenance Centre with private sector participation. It is also planned to set up an independent laboratory to undertake regular monitoring of the quality of petrol and diesel sold in Delhi mainly with a view to check adulteration of petrol with kerosene or diesel fuel, both of which are sold at significantly lower (roughly one-third) prices than petrol under an administered pricing mechanism.

At first the Indian Government had notified that all new cars for sale in 42 Indian cities should be fitted with catalytic converters from 1 June onwards. The date was subsequently postponed to 1 August and now has been implemented.

8.6 CONCLUSIONS

While developing countries currently have very few motorized vehicles per capita compared to the OECD countries, the vehicle population is growing very rapidly. Further, the mix of vehicles, especially the predominance of motorcycles and scooters with highly polluting two stroke engines, is unique and emits substantially more pollution per kilometre driven than is typical in the OECD countries. Most of the current vehicle population is concentrated in the major cities with the result that these cities are already highly polluted.

As a result of these pollutants, serious health problems result, especially with the very old, the very young and those least able to cope.

Significant progress is being made, however. Taiwan has been implementing an aggressive motorcycle control programme which is expected to eliminate new two stroke motorcycles by about 2003. Further, they are rapidly expanding the conversion to electric motorcycles. A number of other Asian countries, including Thailand and China, are expected to follow Taiwan's lead in cleaning up motorcycles.

Singapore has put in place one of the pre-eminent land transport planning programmes in the World serving as a model to its neighbours.

Most other countries in the region have also made progress or will in the near future. Thailand banned all sales of leaded gasoline in 1996 and China will do so by 2000. India and the Philippines are expected to follow suit. All new cars sold in Hong Kong, Singapore, Taiwan, Thailand and South Korea have been equipped with catalysts for several years and China, India and the Philippines will require these controls within the next few years.

While there is some reason for optimism as noted above, there is also great cause for concern as vehicle populations continue to grow rapidly and to concentrate in the megacities which are increasingly spread across the region. Fundamental shifts to inherently clean transport technologies such as fuel cells and electric vehicles are gaining increased attention.

8.7 REFERENCES

Howson, C. and Hernandez Avila, M. 1996. *Lead in the Americas*. Washington: NAS Press.

International Programme on Chemical Safety (IPCS). 1995. *Environmental Health Criteria Document: Lead*. IPCS, WHO, Geneva.

National Research Council (NRC). 1993. *Measuring Lead Exposures in Infants, Children and Other Sensitive Populations*. NAS Press, Washington.

Sigsby, J.E., Dropkin, D.L., Bradow, R.L. and Lang, J.M. 1982. *Emissions of Ethylene Dibromide*. Society of Automotive Engineers. No. 820786. US EPA and Northrop Services. p. 17.

US Centers for Disease Control (CDC). 1991. *Preventing Lead Poisoning in Young Children*. US DHHS, Atlanta. October 1991.

US Environmental Protection Agency (US EPA). 1986. *Ambient Air Quality Criteria Document for Lead*. Research Triangle Park, NC. EPA ORD.

US Environmental Protection Agency (US EPA). 1988. *Reducing Health Risks Worldwide*. EPA's International Lead Risk Reduction Program. March 1988.

Chapter 9

AIR QUALITY IN HONG KONG AND THE IMPACT OF POLLUTION ON HEALTH 1988-1997

Anthony Johnson Hedley, Chit-Ming Wong, Tai-Hing Lam, Sarah Morag McGhee and Stefan Ma

Department of Community Medicine, The University of Hong Kong

Abstract

Hong Kong's air quality is similar in many ways to that of other capitals and major urban centres around the World; therefore, the epidemiological studies in Hong Kong may contribute to the global evidence on the relationship between air pollution and health. This Chapter summarizes some studies of air pollution and impacts on health in Hong Kong, as they may provide an indication of impacts in other similar cities. The studies found that overall the relative risks for health effects of pollution, including admissions for all respiratory disease, asthma and lung disease, showed higher excess risks for all age groups than results for western European cities using similar methodologies. Similar findings were made for hospital deaths.

Pollution is now responsible for eroding the previously gained economic advantages of Asia, which have resulted in improved population health. However, policy-makers can be confident that controls on pollution sources will, in addition to arresting degradation of the environment, bring about measurable improvements in health, particularly in children and the elderly. Pollution controls will reduce premature deaths in the chronic sick and elderly *and thereby reduce avoidable morbidity and mortality and associated hospital costs.*

9.1 BACKGROUND

There has been increasing awareness in the Hong Kong Special Administrative Region (HKSAR) over the past ten years that air pollution is degrading the quality of the environment and affecting the health of its citizens of all ages.

In 1988 exceedances of Air Quality Objectives (AQOs) were common (Environmental Protection Department, 1989) and the Environmental Protection Department received over 2000 complaints about air quality in the late 1980s and early 1990s (Figure 9.1).

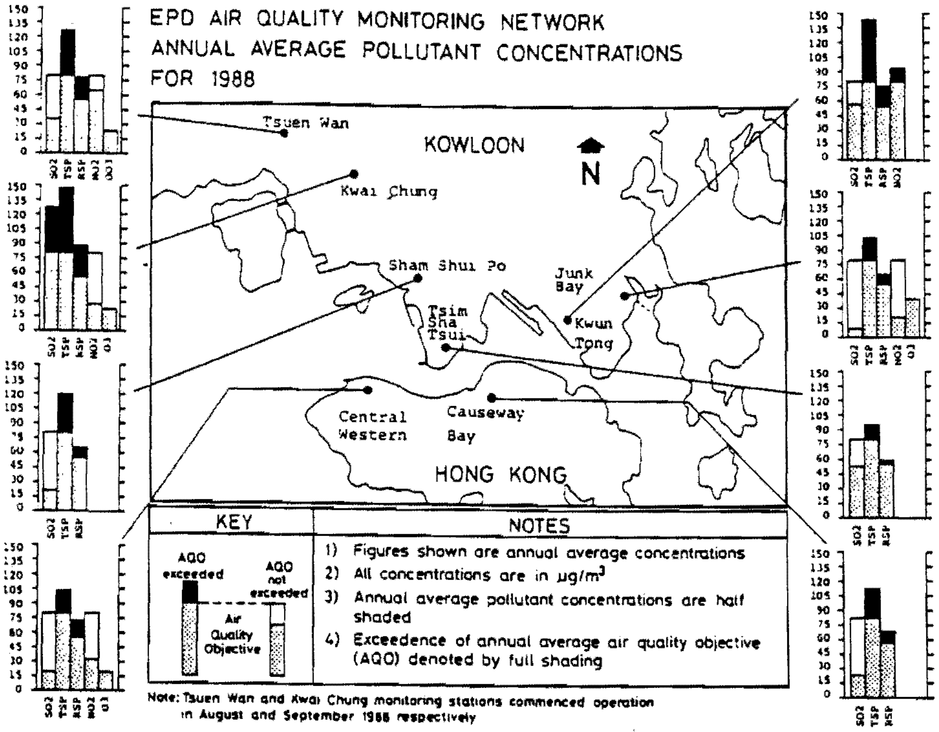


Figure 9.1 Hong Kong's average pollutant concentrations recorded at air quality monitoring stations during 1988

In July 1990 the Air Pollution Control (Fuel Restriction) Regulation became law. This prohibited the use of fuel oil containing more than 0.5 per cent by weight of sulphur (Hong Kong Government, 1990). There was an immediate decrease in ambient concentrations of sulphur dioxide (SO_2) by over 80 per cent in the most polluted districts (Figure 9.2) (Environmental Protection Department, 1991; Environmental Protection Department, 1992; Environmental Protection Department, 1993). Sulphate concentrations in respirable suspended particulates (RSP) also fell by up to 35 per cent.

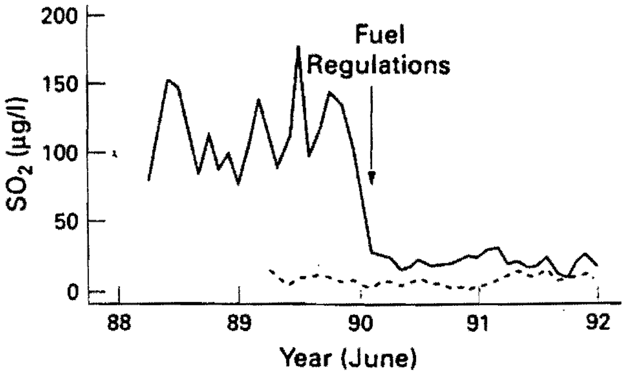


Figure 9.2 Changes in SO_2 following the 1990 fuel regulations

Levels of other pollutants such as nitrogen oxides (NO_x), particulates and ozone (O_3) were unchanged following this intervention. Lower levels of SO_2 were maintained but overall air quality has deteriorated and, in 1997, exceedances of nitrogen dioxide (NO_2) and RSP occurred at six out of nine air quality monitoring stations located in different districts of the HKSAR (Table 9.1) (The Government of the Hong Kong Special Administrative Region, 1998).

Table 9.1 Compliance with air quality objectives in 1997 in nine districts of Hong Kong, for O_3, NO_2 and RSP (modified from The Government of the Hong Kong Special Administrative Region (1998))

	Ozone	Nitrogen dioxide			Respirable suspended	
Station	**1-hour**	**1-hour**	**24-hour**	**1-year**	**24-hour**	**1-year**
1	√	√	x	√	√	x
2	√	√	√	√	√	√
3	√	√	√	√	√	√
4	√	√	√	√	√	x
5	-	√	x	√	√	x
6	√	√	√	√	√	x
7	√	√	√	√	√	x
8	√	√	√	√	√	√
9	-	x	x	x	√	x

√: Complied X: Not complied -: Not available

Hong Kong's air quality is similar in many ways to that of other capitals and major urban centres around the World; therefore, the epidemiological studies in the HKSAR may contribute to the global evidence on the relationship between air pollution and health. It is clear that the status of a city may be categorized in different ways by different pollutants, assuming that the measurements are comparable. In comparison with other Asian cities, Hong Kong ranks middling to low for ambient concentrations of SO_2 and carbon monoxide (CO) and, for particulates, it has lower ambient concentrations than Taipei, Shanghai and Guangzhou but higher than Seoul, Tokyo and Singapore. Hong Kong has higher ambient ozone concentrations than Kuala Lumpur, Tokyo and Singapore but lower concentrations than the Chinese cities of Guangzhou and Taipei (The Government of the Hong Kong Special Administrative Region, 1998). In 1999 Hong Kong's pollution made international headlines as tourists observed visibility dropping to 3000 metres and once famous views disappearing. In Hong Kong, in a series of different studies, variations in air pollutants by both geographical zone and time have been used to measure the impact of pollution on health and, where possible, the effects of intervention.

This Chapter reviews some of the more pertinent studies, but a consideration of methods is needed to properly appreciate the results.

9.2 METHODS

9.2.1 Measurements of health in children

Respiratory symptoms: Children are the most sensitive members of the general population to health effects of air pollution. Studies of the impact of pollution on health have been based on comparisons of the health of primary schoolchildren, aged mainly 8 to 10 years, in two districts with higher and lower levels of pollution respectively. Children were sampled from schools considered to be located in constituencies which were representative of the air quality in the two districts (Ong *et al.*, 1991). Data were collected by standardized questionnaires from children and parents who were asked about respiratory symptoms including cough, phlegm, wheeze, sore throat, nasal problems and doctor-diagnosed asthma. Questions were also asked about smoking experience and how many categories of people (father, mother, siblings, other relatives or lodgers) smoked in the family home, parental educational attainment, occupational status, use of domestic fuels, incense and mosquito coils. The response rate ranged from 96 per cent (parents) up to 99 per cent (children). In the first year of the survey, before the introduction of new fuel regulations in the territory, 3521 children took part; 1504 in the low pollution district and 2017 in the high pollution district.

Bronchial reactivity: An abnormal tendency for the pulmonary airways to narrow when challenged with an aerosol of the compound histamine is a characteristic of individuals with asthma. This reaction is followed by a fall in lung function which can be measured with a meter. This response, called bronchial hyper-responsiveness, can be observed in well children and may reflect sensitization by exposure to environmental factors such as chemical pollutants, biological allergens, pollen or house dust mite. This test was used to examine whether there was any variation in bronchial reactivity between children with higher or lower levels of exposure to air pollution

The children were examined and tested on two occasions, in 1989 and 1990, before the introduction of the fuel regulations and again on two occasions, in 1991 and 1992, after the intervention.

9.2.2 Measures of health in adults

A well-population of workers: A comprehensive health enquiry was conducted in a young adult public sector workforce (mean age ~32y) with responsibilities assigned either outdoors at street level, indoors in offices, or in a marine environment. Information obtained included current respiratory symptoms and a detailed history of both active and passive smoking (Department of Community Medicine, 1997).

Measurement of lung function: Representative samples of individuals in the workforce who were engaged in street level outdoor activities took part in lung function testing sessions before and after eight-hour shifts. In this survey the test was used to determine whether lung function was impaired by a normal working shift, usually of eight hours, at street level.

Variation in population health by ambient air pollution: In order to take a whole population approach to the estimation of possible variations in health caused by air pollution, statistical modelling techniques were used to analyse patterns of daily hospital admissions to public sector hospitals and hospital deaths, in relation to daily variations in pollutants.

The sources of information included the public sector hospital service Integrated Patient Administration System and Medical Records Abstracting System (Hospital Authority Hong Kong) and the Environmental Protection Department database of daily ambient pollutant concentrations (Environmental Protection Department). The pollutants included in the analyses were nitrogen dioxide (NO_2), respirable particulates (RSP) measured as PM_{10}, ozone (O_3) and sulphur dioxide (SO_2).

The statistical modelling techniques were based on the protocols used by the European collaborating group known as APHEA (Air Pollution on Health: a European Approach) (Katsouyanni *et al.*, 1996).

9.3 FINDINGS

9.3.1 Respiratory health of children

Inferences about the effects of air pollution on children's health were drawn from two sources: the first a cross sectional survey and, second, a cohort follow-up study which spanned the introduction of the new fuel regulations.

Respiratory symptoms: In 1989 and 1990, after adjustment for potential confounding factors (such as smoking in the home), significant excess risks for reported "cough" or "sore throat", "any wheeze or asthmatic symptoms" or "wheezing" were found in the most polluted district (Peters *et al.*, 1996). The frequencies of any respiratory symptoms were highest in the youngest children (aged 8 years), higher in boys than girls and higher in the children living in the most polluted district. Children who lived in a smoking home also had a higher prevalence of symptoms.

Over a three-year period the prevalence ratios declined in both districts (Table 9.2). Apart from the prevalence of nasal symptoms including allergic rhinitis and sinusitis, there was an apparently greater drop in the district with the higher level of pollution. After adjustment for potential confounding factors the finding of significant excess risks in the most polluted district had disappeared by 1991 (Figure 9.3a, b), although there were some apparent residual effects (Department of Community Medicine, 1993).

Table 9.2 Crude prevalence ratios (%) for respiratory symptoms before and after the introduction of restrictions on fuel sulphur content in districts with lower and higher pollution

Respiratory symptoms		1989	1990	1991
Cough/sore throat	S:	16.1	13.9	11.7
	K:	19.8	16.8	11.1
Phlegm	S:	16.8	13.2	9.4
	K:	19.2	14.2	8.1
Wheezing	S:	11.3	10.5	9.2
	K:	12.7	11.2	9.3
Nasal symptoms	S:	35.6	31.0	32.6
	K:	38.1	31.4	32.4

S: Southern district (lower pollution)
K: Kwai Tsing (higher pollution)

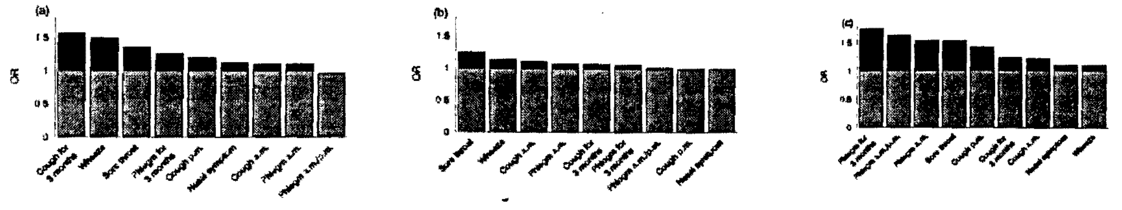


Figure 9.3 Odds ratios and excess risks (black) for nine respiratory symptoms in primary school children associated with exposure to ambient air pollution before (a) and after (b) the introduction of restrictions on the sulphur content of fuel. The excess risks associated with living in a smoking home (c) were unchanged by the intervention

In contrast the observed effects of exposure to environmental tobacco smoke from living in a smoking home, with excess risks for "cough or sore throat" and "phlegm", were unchanged after the intervention of the fuel regulations (Figure 9.3c).

Bronchial responsiveness: The between district differences in the prevalence of bronchial hyper-responsiveness were studied to assess the effectiveness of the intervention. In the pre-intervention period children in the higher pollution district had a higher prevalence of hyper-responsiveness (Tam *et al.*, 1994). In the cohort of children, who were followed up after the introduction of the fuel regulations, a decline in bronchial hyper-responsiveness over a two year period was observed; with the rate of decline in hyper-responsiveness greater in the most polluted district (Wong *et al.*, 1998) (Table 9.3).

Table 9.3 Prevalence of bronchial hyper-responsiveness before (1990) and after (1991-1992) the introduction of restrictions on fuel sulphur content in districts with lower and higher pollution levels

	Prevalence (%) of bronchial hyper-responsiveness before and after air quality intervention		
	Year		
District	**1990**	**1991**	**1992**
Lower pollution	30.6	17.8	10.9
Higher pollution	22.7	11.1	12.0

Costs and benefits of air quality improvement: The information gained from the children's respiratory health survey was used to carry out an economic appraisal of the costs and benefits of the Government intervention based on the fuel regulations. The analysis, which was principally based on the valuation of avoided doctor consultations, concluded that Hong Kong (its citizens and policy-makers) implicitly valued the non-monetized benefits higher than the net costs. In addition, a portion of the intervention costs were offset by the avoidance of acute respiratory symptoms and health care utilization (Barron *et al.*, 1995).

9.3.2 Health effects in adults

A healthy workforce: The types of duties in the workforce studied were used as proxy indicators for exposure to ambient pollution. The frequencies of cough, phlegm and wheeze were used as the health outcome measure. Workers who were engaged in duties at street level experienced significantly increased excess risks for cough and production of phlegm compared to workers in a marine environment. Similarly, when outdoor workers were compared with those with indoor administrative duties, they had significant excess risks for cough, phlegm and wheeze (Table 9.4).

Table 9.4 Excess risks for cough and production of phlegm for workers who were engaged in duties at street level

	Health outcome	
	Cough	Phlegm
Work environment	Excess risk	Excess risk
Street level versus marine workers	49% $p<0.0001$	17% $p=0.0235$
Outdoors versus indoors	57% $p=0.0002$	32% $p=0.0082$

The results, which were adjusted for all factors that might influence health (such as gender, marital status, education level, smoking and exposure to passive smoking), suggest that outdoor shift work is associated with several bronchitic symptoms in this urban environment.

These findings are supported by the results of lung function tests which showed that in the non-smoking subjects who spent the longest periods on the streets, at the kerbside, during shift work, the pre- and post-shift lung function tests indicated a significant drop after an eight-hour shift (Department of Community Medicine, 1997).

Pollution effects on hospital admissions: Reports from other centres based on analyses of daily hospital admission data have shown significant variations in admissions for cardiovascular and respiratory disease by variations in ambient daily pollutant concentrations. In Hong Kong, a sub-tropical environment, time trends, seasonality and weather conditions explained 31-79 per cent of the variations in all the health outcomes under study. In Hong Kong, as in temperate zones, cardiovascular mortality is strongly associated with lower temperatures in the cool season (Wong *et al.*, 1999). In addition, all of the four pollutants which were studied showed a significant association with daily hospital admissions for cardiovascular and respiratory diseases both combined and separately. The excess risks were in the range of 3-10 per cent (Wong *et al.*, 1998). The effects of pollutants on circulatory and respiratory diseases were stronger for older age groups with significant excess risks of 5-10 per cent in those aged 65 or older. Both NO_2 and O_3 were strongly associated with hospital deaths from cardiovascular and respiratory diseases.

Concentrations of ozone in Hong Kong have increased more than 80 per cent in the past seven years, the hourly air quality objective of 24 $\mu g/m^3$ being frequently violated. In an analysis of approximately 87,000 daily hospital admissions through the Accident and Emergency Departments for circulatory diseases in the over 65 year age group between January 1995 and June 1997, there was a significant excess risk of admission for all circulatory diseases, dysrhythmias and heart failure. A significant interaction with season was observed, with excess risks much increased in the cool season (Wong *et al.*, 1999). Overall, the relative risks for health effects of pollution, including admissions for all respiratory disease, asthma and chronic obstructive pulmonary disease, showed higher excess risks for all age groups when compared with the pooled results of western European cities from the APHEA studies. Similar findings were made for hospital deaths.

9.4 DISCUSSION

The evidence available from both well-population epidemiological surveys and analyses of databases for air pollutants and records of hospitalized patients indicate consistent and strong associations between variations in ambient air pollutants and bad health outcomes.

The evidence suggests that both past and current air quality objectives have not protected several large sections of the Hong Kong community from air pollution-related health problems, including particularly the very young, the sick and the elderly.

The findings in a wide spectrum of different types of enquiries show that, whereas specific benefits can be gained from the reduction of one particular pollutant, in this case SO_2, they also demonstrate that all the pollutants measured are associated with adverse health effects. It follows from this that policy-makers must focus on the *sources* of pollution rather than individual pollutant species.

In Hong Kong, Government proposals to reduce ambient air pollution include the conversion of all light commercial diesel road vehicles to run on liquified petroleum gas (LPG). Exhaust emissions from LPG vehicles would be lower for carbon monoxide and hydrocarbons and markedly lower for NO_x and particulates compared with diesel. The choice of LPG has been made because legislators rejected an earlier proposal for a diesel to petrol (gasoline) switch. However, the time scale for the conversion extends up to the year 2005 and this is now the subject of a vigorous public debate in Hong Kong. The short-term conversion of all taxis to LPG power units would cost about US $770m but would eliminate a large source of pollutants very rapidly.

Other Government interventions include routine road side measurement of smoke emissions, the calling in of 30,000 vehicles per year which are reported by "spotters" and the phasing out of leaded petrol by 2000. This is a generally inefficient approach because fines do not remove these vehicles from the road. Given the evidence of detriment to the health of the population the licences of these vehicles should be removed.

The adverse health effects of pollution have been measurable throughout the past ten years. Epidemiological evidence of harm to health, derived from local studies, has been used to evaluate and support action by the Environmental Protection Department and strengthen arguments for legislative action. However, public expressions of concern and growing intolerance of the present situation have been closely related to the visible and other sensory effects of ambient pollution. Hong Kong has lost its horizon, and the aesthetic impact on the environment and quality of life has been an important catalyst in the community calling for action.

All of the analyses which relate morbidity and mortality patterns to ambient levels of NO_2, SO_2, RSP and O_3 show excess risks for health problems, hospital admissions and hospital mortality. On the basis of these findings, it can be predicted that progressive reductions in current levels of pollution would benefit large numbers in the population. Caution must be exercised in the interpretation of the outputs of models. Environmentalists, the media and possibly policy-makers want quotes of "body counts". However, it is not possible to estimate precisely the excess numbers of admissions or deaths by the methods used. It can only be concluded that variations in pollution probably cause or aggravate health problems. This is at least consistent with the findings in well-population subjects that marked variations in the frequency of symptoms are associated with pollution which is a good indicator that people are being adversely exposed. In the case of hospital admissions and deaths it was found that pollutant concentrations are related to acute events (admissions and deaths) for conditions such as coronary heart disease and chronic lung disease, which show an excess over predicted levels whereas outcomes related to cancer do not.

Any strategy to reduce the risk of cardiovascular and respiratory disease and premature death requires an intersectoral approach. The risks from tobacco *for those exposed*, including passive smoking in children, are much greater than from air pollution. Air pollution, on the other hand, now affects everyone in this community and its effects tend to negate the benefits of rapid social and economic development which ironically have in turn created the pollution.

Hong Kong could have acted on more stringent pollution controls at least a decade ago but a general non-interventionist Government philosophy prevailed. A lack of good quality data on the health effects of pollution contributed to this.

The situation clearly signals that the dominance of diesel vehicles in the commercial sector and other aspects of current transportation policies are unsustainable. Nevertheless, the trend in infrastructural developments is to build highways rather than other mass transit systems. The Hong Kong experience indicates that this will be associated with further degradation of air quality with adverse effects on health, tourism and general perceptions of quality of life.

9.5 CONCLUSIONS

- Pollution is now responsible for eroding the previously gained economic advantages of Asia, for example in terms of improved population health and tourism.
- Policy-makers can be confident that controls on pollution sources will, in addition to arresting degradation of the environment, bring about measurable improvements in health, particularly in children and the elderly.
- Pollution controls will reduce premature deaths in the chronic sick and elderly and thereby reduce avoidable morbidity and mortality and associated hospital costs.
- The response time for measurable health benefits to be realized is short, measured in weeks and months rather than years. However, the lead time for policy decisions to take effect is likely to be much longer.
- The solution to air pollution in Hong Kong and China must include a new and radical approach to transportation policy but the prospects for reduction in the rate of increase in personal forms of transport and a shift to mass transport are not promising.

9.6 REFERENCES

Barron, W.F., Liu, J., Lam, T.H., Wong, C.M., Peters, J. and Hedley, A.J. 1995. Costs and benefits of air quality improvement in Hong Kong. *Contemporary Economic Policy* **9**: 105-116.

Department of Community Medicine, The University of Hong Kong. 1997. *Health of the Hong Kong Police*. Department of Community Medicine, 7 Sassoon Road, Hong Kong. ISBN 962-7751-02-2.

Department of Community Medicine, The University of Hong Kong. 1993. *Air Pollution and Respiratory Health in Hong Kong, 1989-1992*. Report to the Environmental Protection Department, Hong Kong Government.

Environmental Protection Department, Hong Kong. CD-ROM: Air Quality in Hong Kong 1986-1996.

Environmental Protection Department. 1989. *Environment Hong Kong 1989*. Hong Kong: Government Printer, pp. 105.

Environmental Protection Department. 1991. *Environment Hong Kong 1991. A Review of 1990*. Hong Kong: Government Printer.

Environmental Protection Department. 1992. *Environment Hong Kong 1992. A Review of 1991*. Hong Kong: Government Printer.

Environmental Protection Department. 1993. *Environment Hong Kong 1993. A Review of 1992*. Hong Kong: Government Printer.

Hong Kong Government. 1990 Air Pollution Control (Fuel Restriction) Regulations 1990. *Air Pollution Control Ordinance (Chapter 311) Gazette* **4/1990**. Hong Kong: Government Printer.

Hospital Authority Hong Kong. *Overview of the HA IT/IS Strategy (1992-2000)*. http://www.ha.org.hk/it/pcp5.htm.

Katsouyanni, K., Schwartz, J., Spix, C., Touloumi, G., Zmirou, D., Zanobetti, A., Wojtyniak, B., Vonk, J.M., Tobias, A., Ponka, A., Medina, S., Bacharova, L. and Anderson, H.R. 1996. Short-term effects of air pollution on health: a European approach using epidemiologic time series data. The APHEA protocol. J Epidemiol Community Health **50**: S12-18.

Ong, S.G., Liu J., Wong, C.M., Lam, T.H., Tam, A.Y.C., Daniel, L. and Hedley, A.J. 1991. Studies on the respiratory health of primary school children in urban communities of Hong Kong. *Sci. Total Environ.* **106**: 121-135.

Peters, J., Hedley, A.J., Wong, C.M., Lam, T.H., Ong, S.G., Liu, J. and Spiegelhalter, D.J. 1996. Effects of ambient air pollution intervention and environmental tobacco smoke on children's respiratory health in Hong Kong. *International Journal of Epidemiology* **25**: 821-828.

Tam, A.Y.C., Wong, C.M., Lam, T.H., Ong, S.G., Peters, J. and Hedley, A.J. 1994. Bronchial responsiveness in children exposed to atmospheric pollution in Hong Kong. *Chest* **104**: 1056-1060.

The Government of the Hong Kong Special Administrative Region. 1998. *A Proposal to Introduce LPG Taxis*. A consultation paper.

Wong, C.M., Lam, T.H., Peters, J., Hedley, A.J., Ong, S.G., Tam, A.Y.C., Liu, J. and Spiegelhalter, D.J. 1998. Comparison between two districts of the effects of an air pollution intervention on bronchial responsiveness in primary school children in Hong Kong. *Journal of Epidemiology and Community Health* **52**: 571-578.

Wong, C.M., Ma, S., Hedley A.J. and Lam, T.H. 1998. *Short-term Effects of Ambient Air Pollution on Public Health in Hong Kong. A Follow-up Study*. Report to Environmental Protection Department, Hong Kong Government. Department of Community Medicine, The University of Hong Kong.

Wong, C.M., Ma, S., Hedley, A.J. and Lam, T.H. Effect of ozone on daily hospital admissions in the elderly in a sub-tropical city. *Journal of Epidemiology and Community Health* (in press).

Wong, C.M., Ma, S., Lam, T.H. and Hedley, A.J. Seasonal variation in coronary heart disease in Hawaii and Hong Kong. *British Medical Journal* (in press).

Woolcock, A.J. 1987. Epidemiologic methods for measuring prevalence of asthma. *Chest* **91**: 895-925.

Yan, K., Salome, C. and Woolcock, A.J. 1983. Rapid method for measurement of bronchial hyper-responsiveness. *Thorax* **38**: 760-5.