



# Health risks of particulate matter from long-range transboundary air pollution

## **Health risks of particulate matter from long-range transboundary air pollution**

Address requests about publications of the WHO Regional Office for Europe to:

Publications  
WHO Regional Office for Europe  
Scherfigsvej 8  
DK-2100 Copenhagen Ø, Denmark

Alternatively, complete an online request form for documentation, health information, or for permission to quote or translate, on the WHO/Europe web site at <http://www.euro.who.int/pubrequest>.

**Keywords:**

**AIR POLLUTION**  
**POLLUTANTS, ENVIRONMENTAL – adverse effects**  
**ENVIRONMENTAL EXPOSURE**  
**RISK FACTORS**  
**EUROPE**

**© World Health Organization 2006**

All rights reserved. The Regional Office for Europe of the World Health Organization welcomes requests for permission to reproduce or translate its publications, in part or in full.

The designations employed and the presentation of the material in this publication do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Where the designation “country or area” appears in the headings of tables, it covers countries, territories, cities, or areas. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement.

The mention of specific companies or of certain manufacturers’ products does not imply that they are endorsed or recommended by the World Health Organization in preference to others of a similar nature that are not mentioned. Errors and omissions excepted, the names of proprietary products are distinguished by initial capital letters.

The World Health Organization does not warrant that the information contained in this publication is complete and correct and shall not be liable for any damages incurred as a result of its use. The views expressed by authors or editors do not necessarily represent the decisions or the stated policy of the World Health Organization.

Text editor: Frank Theakston. Cover design and layout: Sven Lund

Printed in Denmark



**EUROPE**

# **Health risks of particulate matter from long-range transboundary air pollution**

**Joint WHO/Convention Task Force  
on the Health Aspects of Air Pollution**

**European Centre for Environment and Health  
Bonn Office**

## **Abstract**

Particulate matter is a type of air pollution that is generated by a variety of human activities, can travel long distances in the atmosphere and causes a wide range of diseases and a significant reduction of life expectancy in most of the population of Europe. This report summarizes the evidence on these effects, as well as knowledge about the sources of particulate matter, its transport in the atmosphere, measured and modelled levels of pollution in ambient air, and population exposure. It shows that long-range transport of particulate matter contributes significantly to exposure and to health effects. The authors conclude that international action must accompany local and national efforts to cut pollution emissions and reduce their effects on human health.

# Contents

Foreword	vii
Executive summary	ix
1. Introduction	1
2. What is PM?	5
3. Hazard assessment of PM	11
4. Sources of PM	25
5. PM levels	33
6. Population exposure	65
7. The approach to estimating risk	73
8. Risk estimates	89
9. Conclusions and recommendations	95
Annex 1.	99

## **Main contributors**

Markus Amann, Richard Derwent,  
Bertil Forsberg, Fintan Hurley,  
Michal Krzyzanowski, Birgit Kuna-Dibbert,  
Steinar Larssen, Frank de Leeuw, Sally Jane Liu,  
Jürgen Schneider, Per E. Schwarze, David Simpson,  
John Stedman, Peter Straehl, Leonor Tarrasón  
and Leendert van Bree.



This report was prepared by the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution according to a Memorandum of Understanding between the United Nations Economic Commission for Europe (UNECE) and the WHO Regional Office for Europe (ECE/ENHS/EOA/2005/001), based on work covered by Memorandum of Understanding ECE/ENHS/EOA/2004/001 between UNECE and the Regional Office.

## Foreword

The scale and seriousness of impacts of air pollution on health that have been detected by scientific investigations over the past decade are the subject of media reports and policy debate throughout Europe. Evidence on those impacts has been gathered through numerous studies conducted by scientists of various disciplines and published mostly by highly specialized scientific journals. Comprehensive evaluation of this evidence is needed in order to formulate effective pollution reduction strategies and national and international policies for reducing health risks due to pollution.

This report focuses on particulate matter, a type of air pollution that causes a wide range of diseases in children and adults, contributing to disability and a significant reduction in life expectancy. Particulate matter is present everywhere where people live and is generated to a great extent by human activities: transport, energy production, domestic heating and a wide range of industries. As presented in this report, this pollution can be transported in the atmosphere for hundreds or even thousands of kilometres and thus affect people living far from the source of the pollution. Particulate matter is therefore not only a serious local problem but also of regional and international concern, and one of the core issues addressed by the Convention on Long-range Transboundary Air Pollution.

The multidisciplinary group of experts who prepared this report, convened by the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution, has summarized the available information on particulate matter – the risk it poses to human health, its sources, transport and distribution in the atmosphere, and population exposure to it. The report also presents estimates of the magnitude of the current

impacts of particulate matter on health in Europe, and concludes that a significant part of these effects is due to particles transported over long distances in the atmosphere.

There is sufficient evidence to indicate that reducing emissions of major pollutants leads to reduced levels of particulate air pollution, of population exposure and of health effects. Current pollution reduction strategies are expected to benefit the health of many Europeans, but even with their full implementation the health impacts will remain significant. A strong commitment from all Member States is needed to implement existing plans and to extend efforts to reduce population exposure and the effects of particulate air pollution.

The Children's Environment and Health Action Plan for Europe, adopted at the Fourth Ministerial Conference on Environment and Health in Budapest in June 2004, sets the reduction of child morbidity caused by air pollution as one of four regional priority goals. Reduction of exposure to particulate matter is essential to the achievement of this goal, and the Convention on Long-range Transboundary Air Pollution can be an important instrument contributing to that achievement.

We are grateful to the experts who prepared this report for summarizing the evidence and for sending a clear message to decision- and policy-makers on the significance for health of particulate matter from long-range transboundary air pollution. The evidence clearly points to the need for health-oriented policies and coordinated local, regional and international action by all polluting economic sectors in all Member States. Action is necessary if we are to reduce the pollution-related burden of disease and improve the health of both children and adults across Europe.



Roberto Bertollini, MD, MPH  
*Director*

Special Programme on Health and Environment  
WHO Regional Office for Europe



Kaj Bärlund  
*Director*

Environment and Human Settlements Division  
United Nations Economic Commission for Europe





## Executive summary

This report summarizes the results of multidisciplinary analysis aiming to assess the effects on health of suspended particulate matter (PM) and especially that part that is emitted by remote sources or generated in the atmosphere from precursor gases. The analysis indicates that air pollution with PM, and especially its fine fraction (PM<sub>2.5</sub>), affects the health of most of the population of Europe, leading to a wide range of acute and chronic health problems and to a reduction in life expectancy of 8.6 months on average in the 25 countries of the European Union (EU). PM from long-range transport of pollutants contributes significantly to these effects.

PM is an air pollutant consisting of a mixture of solid and liquid particles suspended in the air. These particles differ in their physical properties (such as size), chemical composition, etc. PM can either be directly emitted into the air (primary PM) or be formed secondarily in the atmosphere from gaseous precursors (mainly sulfur dioxide, nitrogen oxides, ammonia and non-methane volatile organic compounds). Primary PM (and also the precursor gases) can have anthropogenic and nonanthropogenic sources (for primary PM, both biogenic and geogenic sources may contribute to PM levels).

Several different indicators can be used to describe PM. Particle size (or aerodynamic diameter) is often used to characterize them, since it is associated with the origin of the particles, their transport in the atmosphere and their ability to be inhaled into respiratory system. PM<sub>10</sub> (particles with a diameter <10 µm) and PM<sub>2.5</sub> (those with a diameter <2.5 µm) are nowadays commonly used to describe emissions and ambient concentrations of PM (here, mass concentrations of these indicators are used). Ultrafine particles comprise those with a diameter <0.1 µm. The most important chemical constituents of PM are sulfate, nitrate, ammonium, other inorganic ions (such as Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, Mg<sup>2+</sup> and Cl<sup>-</sup>), organic and elemental carbon, crustal material, particle-bound water and heavy metals. The larger particles (with the diameter between 2.5 and 10 µg/m<sup>3</sup>, i.e. the coarse fraction of PM<sub>10</sub>) usually contain crustal materials and fugitive dust from roads and industry. PM in the size between

0.1 µm and 1 µm can stay in the atmosphere for days or weeks and thus can be transported over long distances in the atmosphere (up to thousands of kilometres). The coarse particles are more easily deposited and typically travel less than 10 km from their place of generation. However, dust storms may transport coarse mineral dust for over 1000 km.

Exposure to PM in ambient air has been linked to a number of different health outcomes, ranging from modest transient changes in the respiratory tract and impaired pulmonary function, through increased risk of symptoms requiring emergency room or hospital treatment, to increased risk of death from cardiovascular and respiratory diseases or lung cancer. This evidence stems from studies of both acute and chronic exposure. Toxicological evidence supports the observations from epidemiological studies. Recent WHO evaluations point to the health significance of PM<sub>2.5</sub>. In particular, the effects of long-term PM exposure on mortality (life expectancy) seem to be attributable to PM<sub>2.5</sub> rather than to coarser particles. The latter, with a diameter of 2.5–10 µm (PM<sub>2.5–10</sub>), may have more visible impacts on respiratory morbidity. The primary, carbon-centred, combustion-derived particles have been found to have considerable inflammatory potency. Nitrates, sulfates and chlorides belong to components of PM showing lower toxic potency. Nevertheless, despite these differences among PM constituents under laboratory conditions, it is currently not possible to precisely quantify the contributions of different components of PM, or PM from different sources, to the health effects caused by exposure to PM. While long- and short-term changes in PM<sub>2.5</sub> (or PM<sub>10</sub>) mass concentration have been shown to be associated with changes in various health parameters, available evidence is still not sufficient to predict the health impacts of changing the composition of the PM mixture.

Health effects are observed at all levels of exposure, indicating that within any large population there is a wide range of susceptibility and that some people are at risk even at the lowest end of the observed concentration range. People with pre-existing heart and lung disease, asthmatics, socially disadvantaged

and poorly educated people and children belong to the more vulnerable groups. Despite the rapid expansion of the evidence, the well documented and generally accepted mechanistic explanation of the observed effects is still missing and requires further study.

There is as yet only incomplete quantitative knowledge available about sources of particle emissions in the various European countries. By 2003, only 19 of the 48 Parties to the Convention had submitted some PM emission data to UNECE. Since these submissions do not allow a consistent and quality-controlled European-wide picture to be drawn, the evaluation of PM emissions summarized in this report relies on the emission inventory developed with the Regional Air Pollution Information and Simulation (RAINS) model.

According to RAINS estimates, mobile sources, industry (including energy production) and domestic combustion contributed 25–34% each to primary PM<sub>2.5</sub> emissions in 2000. These sectors are also major emitters of the precursor gases sulfur dioxide, nitrogen oxides and volatile organic compounds, while agriculture is a dominant contributor to ammonia.

In general, primary emissions of both PM<sub>2.5</sub> and PM<sub>10</sub> from anthropogenic sources fell by around half across Europe between 1990 and 2000. During this period the relative contribution from transport increased compared to industrial emissions, as illustrated by a smaller emission reduction for carbonaceous particles. Future projections by RAINS suggest that further reductions in primary PM emissions of the same magnitude will continue in the EU as a result of existing legislation. In addition to the transport sector, the domestic sector will become an increasingly important source of PM emissions in the future. Furthermore, in contrast to all other sources of primary PM, emissions from international shipping are predicted to increase in the next 20 years.

According to the Convention's Cooperative Programme for Monitoring and Evaluation of the Long-range Transmission of Air Pollutants in Europe (EMEP), significant reductions of between 20% and 80% were also made in emissions of the PM precursors ammonia, nitrogen oxides and sulfur dioxide between 1980 and 2000. RAINS estimates that further reductions of the same magnitude are achievable

owing to legislation currently in place. Nevertheless, as with primary PM emissions, precursor emissions from international shipping are predicted to increase in the next couple of decades.

The expected reduction in primary PM emissions in the non-EU countries of the EMEP area is markedly smaller than those expected in the EU.

The availability of data on PM<sub>10</sub> concentrations has increased rapidly in the last few years, owing mainly to the requirements of EU directives. Data on PM<sub>10</sub> measured at 1100 monitoring stations in 24 countries were available in the EEA's AirBase database for 2002. In some 550 urban areas included in this database, annual mean PM<sub>10</sub> was 26 µg/m<sup>3</sup> in the urban background and 32 µg/m<sup>3</sup> at traffic locations. In rural areas, annual mean PM<sub>10</sub> amounted to 22 µg/m<sup>3</sup>. Limit values set by the EU directive were exceeded in cities in 20 countries. PM<sub>10</sub> levels in Europe are dominated by the rural background component, and the rural concentration is at least 75% of the urban background concentration.

Available data allow European trends in PM concentrations to be assessed only from 1997 onwards. Between 1997 and 1999/2000 there was a downward trend in PM<sub>10</sub>, while PM<sub>10</sub> values increased between 1999/2000 and 2002. This tendency was similar at rural, urban background and traffic locations, but does not follow the trends in emission: reported emissions of precursor gases fell and primary PM<sub>10</sub> emissions did not change significantly during this period in Europe. It is likely that inter-annual meteorological variations affected trends in PM concentrations. Analysis of well validated United Kingdom data indicates that the fall in emissions corresponds well with observed trends in concentrations.

PM<sub>2.5</sub> and smaller size fractions of PM are measured to a much lesser extent in Europe than PM<sub>10</sub>. Data from 119 PM<sub>2.5</sub> stations for 2001 indicate on average a fairly uniform rural background concentration of 11–13 µg/m<sup>3</sup>. Urban levels are considerably higher (15–20 µg/m<sup>3</sup> in urban background and typically 20–30 µg/m<sup>3</sup> at traffic sites). The PM<sub>2.5</sub>/PM<sub>10</sub> ratio was 0.65 for these stations (range 0.42–0.82).

The EMEP model generally underestimates the observed regional background levels of PM<sub>10</sub> and PM<sub>2.5</sub> in Europe, a feature shared by other models. The underestimation is larger for PM<sub>10</sub> (–34%) than

for  $PM_{2.5}$  (-12%). The validation of the models and pollution patterns are affected by the lack of monitoring data in large areas of Europe. Temporal correlations are lower for  $PM_{10}$  (0.4–0.5 on average) than for  $PM_{2.5}$  (0.5–0.6 on average), indicating that the sources and processes presently not described in the model are probably more important for the coarse fraction of PM.

The EMEP model is able to reproduce well the spatial variability and observed levels of secondary inorganic aerosols across Europe, contributing 20–30% of  $PM_{10}$  mass and 30–40% of  $PM_{2.5}$  mass. For the organic aerosols, representing about 25–35% of the background  $PM_{2.5}$  mass, however, the discrepancies between modelled and observed PM concentrations are substantial, with concentrations of elemental carbon underestimated by about 37% and organic carbon represented very poorly in the model.

Calculations from the validated EMEP model show that the regional background concentrations of anthropogenic PM have a considerable transboundary contribution of about 60% on average across Europe for  $PM_{2.5}$ , ranging from about 30% in large European countries to 90% in smaller ones. For primary coarse PM concentrations, the transboundary contribution is calculated to be smaller though still significant, ranging from 20% to 30% in central Europe.

Organic carbon, together with mineral dust, seems to be a major contributor to the differences between traffic site concentrations and regional background. Further analysis of the origins and transport of organic carbon involve efforts to validate anthropogenic emissions and determine the contribution of biogenic and geogenic sources, in particular from condensation of volatile organic compounds, biomass burning and primary biological sources.

Ambient concentrations of PM from long-range transport of pollution, as estimated by secondary sulfate, are representative of population exposure to long-range transported PM. The differences between PM measurements at centrally located monitors and personal exposure measurements are due to proximity to local sources, such as traffic emissions, as well as to personal activities or residential ventilation characteristics, which may be less important when averaging across the population.

Although both primary and secondary PM contribute to long-range transported PM, available modelling results indicate that secondary PM dominates exposure and is more difficult to control, even under the maximum feasible reduction (MFR) scenario. Quantitative knowledge about the sources of particle emission plays an important role in fine tuning these exposure estimates and in finding the best control strategy for reducing risks.

Present knowledge on the sources of population exposure is based on a very limited number of exposure assessment studies on the origins of PM. Large uncertainties were noted in the source apportionment analyses of personal exposure, owing to the limited sample size. Further exposure assessment studies should be conducted to identify contributions from long-range transport to population PM exposure.

The assessment of the risk to health of PM presented in this report follows the conclusions and recommendations of WHO working groups as well as decisions of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution. The impact estimation was prepared and published within the framework of the preparation of the European Commission's Clean Air for Europe (CAFE) programme. The main indicator of health impact chosen for the analysis is mortality. Population exposure is indicated by annual average  $PM_{2.5}$  concentration provided by the EMEP model. Concentration–response function is based on the largest available cohort study, including 0.5 million people followed for 16 years. An increase in risk of all-cause mortality by 6% per 10  $\mu\text{g}/\text{m}^3$  of  $PM_{2.5}$ , resulting from this cohort study, was recommended for use in the health impact assessment conducted for this analysis. Quantification of impacts of PM exposure on morbidity is less precise than that for mortality, since the database concerning concentration–response functions and background rates of health end-points is poorer. Nevertheless, selected estimates of impacts on morbidity are included in the analysis.

The results of analysis indicate that current exposure to PM from anthropogenic sources leads to an average loss of 8.6 months of life expectancy in Europe. The impacts vary from around 3 months in Finland to more than 13 months in Belgium. The total number of premature deaths attributed to exposure

amounts to about 348 000 in the 25 EU countries. Effects other than mortality, including some 100 000 hospital admissions per year, can be also attributed to exposure. Several other impacts on morbidity are expected to occur as well, but the weakness of the existing database affects the precision and reliability of the estimates.

Currently existing legislation on the emission of pollutants is expected to reduce the impacts by about one third. Further reduction of impacts could be achieved by implementation of all currently feasible emission reductions (MFR scenario).

Reduction of the remaining substantial uncertainties regarding the assessment will require further concerted efforts by scientists of various disciplines and improvements in data on pollutants emissions and air quality and a deeper understanding of those components of PM that are crucial to the observed impacts. Nevertheless, the scientific evidence indicating that exposure to ambient PM causes serious health effects and will continue to do so in the coming years is sufficient to encourage policy action for further reduction of PM levels in Europe. Since the long-range transport of pollution contributes a major part of the ambient levels of PM and of population exposure, international, action must accompany local and national efforts to cut pollution emissions and reduce their effects on human health.

# 1. Introduction

In most UNECE countries, ambient air quality has improved considerably in the last few decades. This improvement was achieved by a range of measures to reduce harmful air emissions, including those stipulated by the various protocols under the Convention on Long-range Transboundary Air Pollution (LRTAP). On the other hand, there is convincing evidence that current levels of air pollution still pose a considerable risk to the environment and to human health.

While early agreements on LRTAP were driven by environmental concerns about the transboundary transport of acidifying pollutants, worries about the effect of air pollution from long-range transport on human health have attracted more and more attention in recent years. This led to the creation of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution. The main objective of this Task Force, which is chaired by WHO, is to prepare state-of-the-art reports on the direct and indirect effects of long-range air pollutants on human health.

The first assessment prepared by the Task Force was entitled *Health risk of particulate matter from long-range transboundary air pollution: preliminary assessment (1)*. Its executive summary was presented to the 18th session of the UNECE Working Group on Effects in August 1999, and the full report was made available at the 17th session of the Executive Body for the Convention. The report concluded that “although there is considerable uncertainty with respect to the present information and monitoring methods, preliminary analysis indicates that the particles from long-range transport may lead to tens of thousands of premature deaths in Europe”. The report also recognized that “further intensive work in epidemiology, atmospheric modelling and air quality assessment has been identified as necessary to improve the reliability and precision of the estimates”.

Since this report was prepared and published, enormous progress has been made in the above-mentioned areas. As an example, health effects of particulate matter were assessed within the WHO project entitled “Systematic review of health aspects of air pollution in Europe” (2,3) and considerable progress was made in

model development within the Convention’s Cooperative Programme for Monitoring and Evaluation of the Long-range Transmission of Air Pollutants in Europe (EMEP). Recent analyses have also confirmed that, although the highest concentrations of particulate matter (PM) are obviously found at “hot spot” sites, considerable levels can occur even at rural background sites and transboundary transport of PM is high. This can be explained by the long residence time in the atmosphere (up to several days) of particles in sizes ranging up to a few micrometers, and the fact that they can therefore be transported over long distances (1000 km or more).

There have also been a number of recent activities on PM air pollution outside the Convention, including the preparation of the *Second position paper on particulate matter* by a working group under the European Commission’s Clean Air for Europe (CAFE) programme (4) and the US Environmental Protection Agency’s criteria document on PM (5).

Taking the large increase in knowledge into account, it was considered necessary to prepare an updated report on the risk to human health of PM from LRTAP. This report is also timely, since the review of the Gothenburg Protocol is expected to begin in the next few months. This review will most probably also include an assessment of the health effects of PM. The Joint WHO/Convention Task Force therefore agreed, at its seventh session in Bonn in May 2004, to prepare a report on the risks to health of PM from LRTAP (6). The detailed content of the report was discussed by an editorial group meeting in Vienna in November 2004, and the second draft was evaluated by the 8th meeting of the Task Force in April 2005. A full list of participants in this meeting is presented in Annex 1.

This report provides a concise summary of the current knowledge on the risks to health of PM from LRTAP. It relies strongly on input provided by other processes and groups, most notably:

- the WHO systematic review of health aspects of air pollution in Europe;
- the work under the aegis of EMEP on emission inventories and atmospheric modelling;

- the work of the European Topic Centre on Air and Climate Change of the European Environment Agency (EEA);
- the integrated assessment carried out by the International Institute for Applied Systems Analysis (IIASA) as part of the CAFE programme; and
- the Cost–Benefit Analysis of the CAFE programme (CAFE CBA).

The report aims to bring together and synthesize the most relevant findings of these projects in relation to the effects on health of PM from LRTAP.

This report is targeted at the various groups within the Convention on Long-range Transboundary Air Pollution, including the Working Group on Strategies and Review and the Executive Body. It is also aimed at decision-makers at national level who are concerned with policies on pollution abatement, as well as at those scientists who can contribute further information for all stages of the risk assessment of PM air pollution.

The main objective is to provide a reasonable estimate of the magnitude, spatial distribution and trends in health burden caused by exposure to PM in ambient air in Europe, including the contribution to PM from long-range transport.

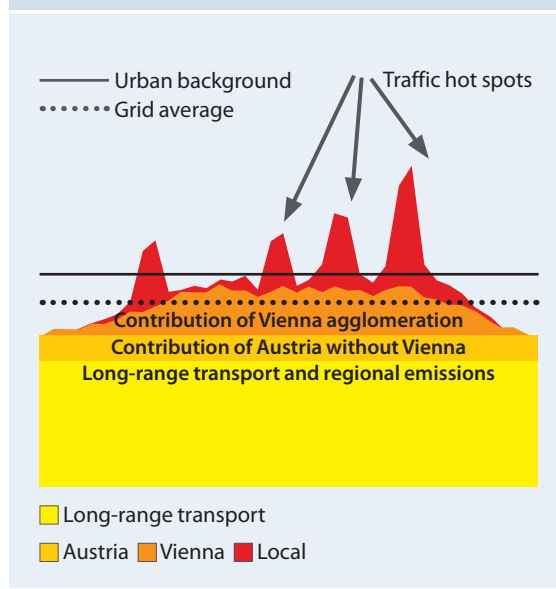
PM has various sources, both anthropogenic and natural. Nevertheless, although both may contribute significantly to PM levels in the atmosphere, this report focuses on PM from anthropogenic sources, since only this fraction may be influenced by human activity.

Fine PM has a long atmospheric residence time and may therefore be subject to long-range transport. In addition, a significant contribution to fine PM mass comes from secondary aerosols (inorganics such as ammonium sulfate and ammonium nitrate but also secondary organic aerosols), which are formed in the atmosphere through chemical/physical processes. As with other secondary air pollutants, the secondary aerosols generally have a rather smooth spatial pattern. Recent analyses have confirmed that in many areas in Europe, long-range transport makes a substantial contribution to PM levels.

This report also contains an assessment of the health effects of exposure to PM, including urban

contributions. The concept of different contributions (regional, urban and local) is illustrated schematically in Fig. 1.1, which shows the different PM levels at monitoring sites in and around Vienna. It should be noted, however, that the regional background is to some extent influenced by emissions from the urban area, since urban hot spots influence the urban background.

**Fig. 1.1. Schematic illustration of different PM<sub>10</sub> levels in different locations for Vienna**



Note: The black line illustrates the city background used to estimate health effects. The dotted line provides the grid average that would be expected from a regional model, and includes all anthropogenic and nonanthropogenic sources of PM.

The beginning of the report provides a short description of “particulate matter” and this is followed by a summary of available data on the hazardous properties of PM. This summary is based on a recent WHO systematic review of epidemiological and toxicological studies (2,3). There then follows a brief overview of sources of PM. The emission data are derived both from national submissions to the UNECE secretariat and from expert estimates. Atmospheric distribution and transformations and current ambient levels are described in Chapter 5. Modelled PM concentrations were calculated with the EMEP unified Eulerian model. Observations on PM complement the description of modelled data. Chapter 5 also contains a discussion on the strengths and weaknesses

of the available models and monitoring data and their robustness as related to policy applications. Data on ambient levels of PM are a prerequisite for Chapter 6 on exposure assessment and Chapters 7 and 8 on risk estimation for human health. Assessment of the effects is made using a classical risk assessment approach, including the following steps:

- hazard identification: review of relevant evidence (epidemiological, toxicological, etc.) to determine whether the agent poses a hazard;
- exposure assessment: determination of the exposure;
- exposure–response function: quantifying the relationship between exposure and adverse health effects; and
- risk characterization: integration of the first three steps above leads to an estimation of the health burden of the hazard.

The methodology of the impact assessment of PM, conducted for the CAFE programme by IIASA and by the CAFE CBA project group, was discussed and agreed on at the sixth and seventh meetings of the Joint WHO/Convention Task Force, using the advice of WHO working groups (6,7). Each step of the risk assessment requires certain assumptions and decisions based on scientific judgements and evaluation of the available, though often limited, scientific evidence. Discussion of the limitations of the existing information is included in each of the chapters.

While the general objective of the review is to evaluate the contribution of LRTAP to the health impact of PM, no direct estimates of this contribution exist. Therefore each of the chapters tries to interpret available data on overall pollution from the perspective of its long-range transport potential. Chapter 9 evaluates the combined evidence, provides conclusions from the analysis and points to key uncertainties in current understanding of the impacts.

## References

1. *Health risk of particulate matter from long-range transboundary air pollution: preliminary assessment*. Copenhagen, WHO Regional Office for Europe, 1999 (document EUR/ICP/EHBI 04 01 02).
2. *Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2003 (document EUR/03/5042688) (<http://www.euro.who.int/document/e79097.pdf>, accessed 1 October 2005).
3. *Health aspects of air pollution – answers to follow-up questions from CAFE. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2004 (document EUR/04/5046026) (<http://www.euro.who.int/document/E82790.pdf>, accessed 1 October 2005).
4. *Second position paper on particulate matter*. Brussels, CAFE Working Group on Particulate Matter, 2004 ([http://europa.eu.int/comm/environment/air/cafe/pdf/working\\_groups/2nd\\_position\\_paper\\_pm.pdf](http://europa.eu.int/comm/environment/air/cafe/pdf/working_groups/2nd_position_paper_pm.pdf), accessed 1 October 2005).
5. *Air quality criteria for particulate matter*. Washington, DC, US Environmental Protection Agency, 2004 (<http://cfpub.epa.gov/ncea/cfm/partmatt.cfm>, accessed 1 October 2005).
6. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2004 (document EB.AIR/WG.1/2004/11) (<http://www.unece.org/env/documents/2004/eb/wg1/eb.air.wg1.2004.11.e.pdf>, accessed 1 October 2005).
7. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2003 (document EB.AIR/WG.1/2003/11) (<http://www.unece.org/env/documents/2003/eb/wg1/eb.air.wg1.2003.11.pdf>, accessed 1 October 2005).





## 2. What is PM?

### KEY MESSAGES

- PM is an air pollutant consisting of a mixture of solid and liquid particles suspended in the air.
- PM can either be directly emitted into the air (primary PM) or be formed in the atmosphere from gaseous precursors (mainly sulfur dioxide, oxides of nitrogen, ammonia and non-methane volatile organic compounds).
- Primary PM and the precursor gases can have anthropogenic and nonanthropogenic sources.
- Commonly used indicators describing PM refer to the mass concentration of PM<sub>10</sub> (particles with a diameter <10 µm) and PM<sub>2.5</sub> (particles with a diameter <2.5 µm). Part of PM<sub>2.5</sub> and PM<sub>10</sub> comprises ultrafine particles having a diameter <0.1 µm.
- PM between 0.1 µm and 1 µm in diameter can remain in the atmosphere for days or weeks and thus be subject to long-range transboundary transport.
- The most important chemical constituents of PM are sulfates, nitrates, ammonium, other inorganic ions such as Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, Mg<sup>2+</sup> and Cl<sup>-</sup>, organic and elemental carbon, crustal material, particle-bound water and heavy metals.

PM is an air pollutant consisting of a mixture of solid and liquid particles suspended in the air. These suspended particles vary in size, composition and origin. Particles are often classified by their aerodynamic properties because (a) these properties govern the transport and removal of particles from the air; (b) they also govern their deposition within the respiratory system; and (c) they are associated with the chemical composition and sources of particles. These properties are conveniently summarized by the aerodynamic diameter, which is the size of a unit-density sphere with the same aerodynamic characteristics. Particles are sampled and described by their mass concentration (µg/m<sup>3</sup>) on the basis of their aerodynamic diameter, usually called simply the particle size. Other important parameters are number concentration and surface area.

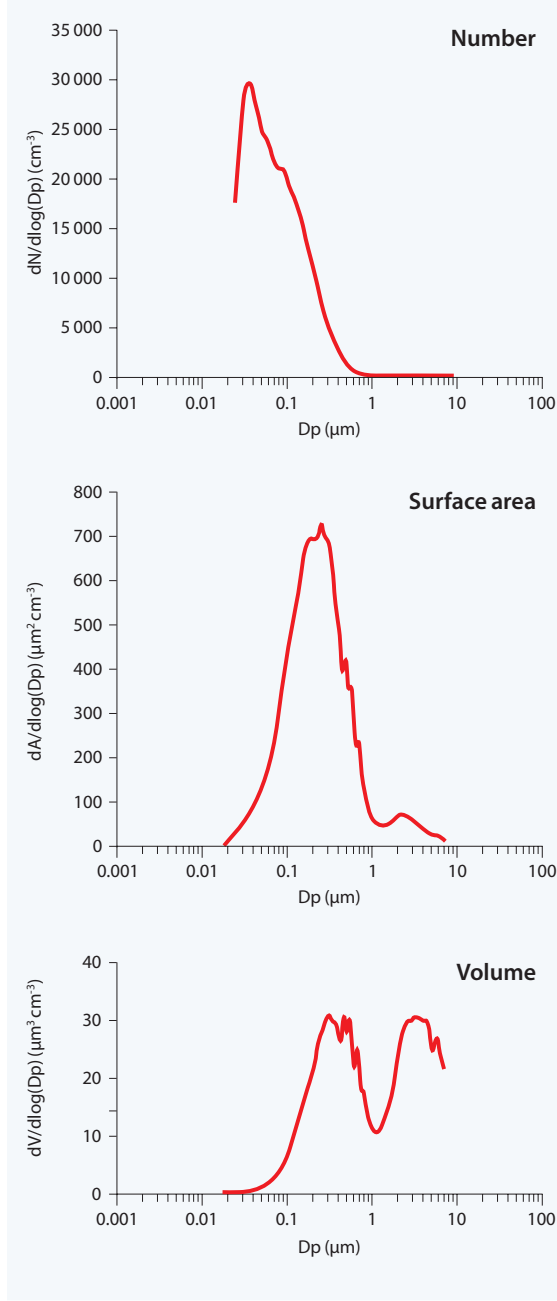
The most commonly used size fractions are the following.

- TSP (total suspended particulates) comprises all airborne particles.
- The term PM<sub>10</sub> is used for particles with an aerodynamic diameter <10 µm.
- The term PM<sub>2.5</sub> is used for particles with an aerodynamic diameter <2.5 µm.
- The coarse fraction comprises particles with an aerodynamic diameter between 2.5 µm and 10 µm.
- The term ultrafine particles is used for particles with an aerodynamic diameter <0.1 µm.
- BS (black smoke) has been widely used as an indicator of the “blackness” of aerosols (and therefore as a surrogate for soot). The definition is linked to a monitoring method used to measure BS. Monitoring is based on an optical method (1). The optical density can be converted by a calibration curve into gravimetric TSP units. However, the conversion depends on the content of black particles within the suspended particulates and thus varies over time and between different types of monitoring site. No validated international standard exists for this method.
- BC (black carbon) is also used as a surrogate for soot. Monitoring is based on an optical method, the aethalometer, which compares the transmission of light through a filter loaded with particulates with transmission through an unloaded part of the filter.

Based on the results of measurements conducted in suburban Birmingham, Fig. 2.1 shows the distri-

butions of the number, surface area and volume of the particles according to their size. These distributions show that most of the particles are quite small,

**Fig. 2.1. Particle size distribution measured in Birmingham, England**



Note: DGV = geometric mean diameter by volume; DGS = geometric mean diameter by surface area; DGN = geometric mean diameter by number; Dp = particle diameter.

Source: Department for Environment, Food and Rural Affairs (2).

<0.1  $\mu\text{m}$ , whereas most of the particle volume (and therefore most of the mass) is found in particles >0.1  $\mu\text{m}$  (2).

Airborne PM represents a complex mixture of organic and inorganic substances. Mass and composition in urban environments tend to be divided into two principal groups: coarse particles and fine particles. The boundary between these two fractions usually lies between 1  $\mu\text{m}$  and 2.5  $\mu\text{m}$ . However, the limit between coarse and fine particles is sometimes fixed by convention at an aerodynamic diameter of 2.5  $\mu\text{m}$  (PM<sub>2.5</sub>) for measurement purposes. Fine and coarse fractions are illustrated in Fig. 2.2.

The heterogenic composition of PM is also illustrated in Fig. 2.3, which shows electron microscopic images of PM sampled at two different Austrian monitoring sites.

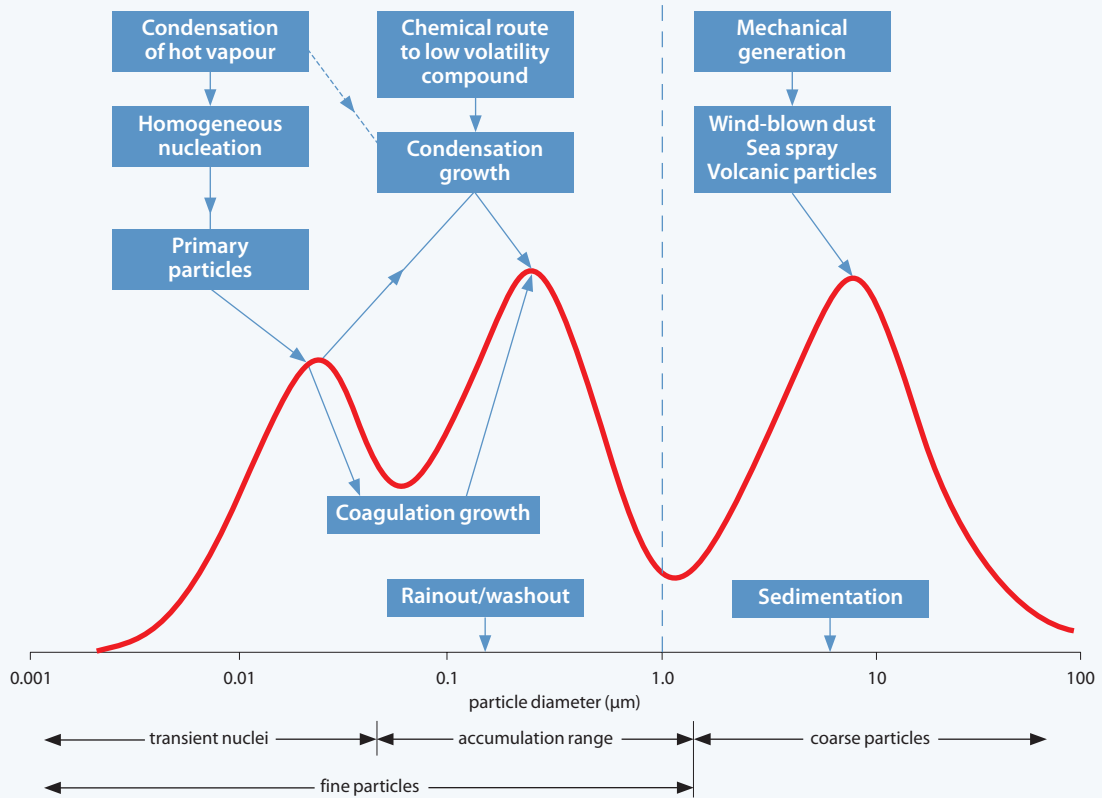
Fine particles contain the secondarily formed aerosols (gas-to-particle conversion), combustion particles (mainly from solid and liquid fuels) and recondensed organic and metal vapours. The fine fraction contains most of the acidity (hydrogen ion) and mutagenic activity of PM, whereas contaminants such as bacterial toxins seem to be most prevalent in the coarse fraction. The most important chemical species contributing to fine PM mass are usually secondary inorganic ions (nitrates, sulfates and ammonia), carbonaceous material (both organic and elemental carbon), water, crustal materials and heavy metals. The size distribution of the main components of PM<sub>10</sub> is shown in Fig. 2.4.

Table 2.1 provides an overview of different characteristics of fine and coarse PM.

The fine particles are sometimes divided into separate modes:

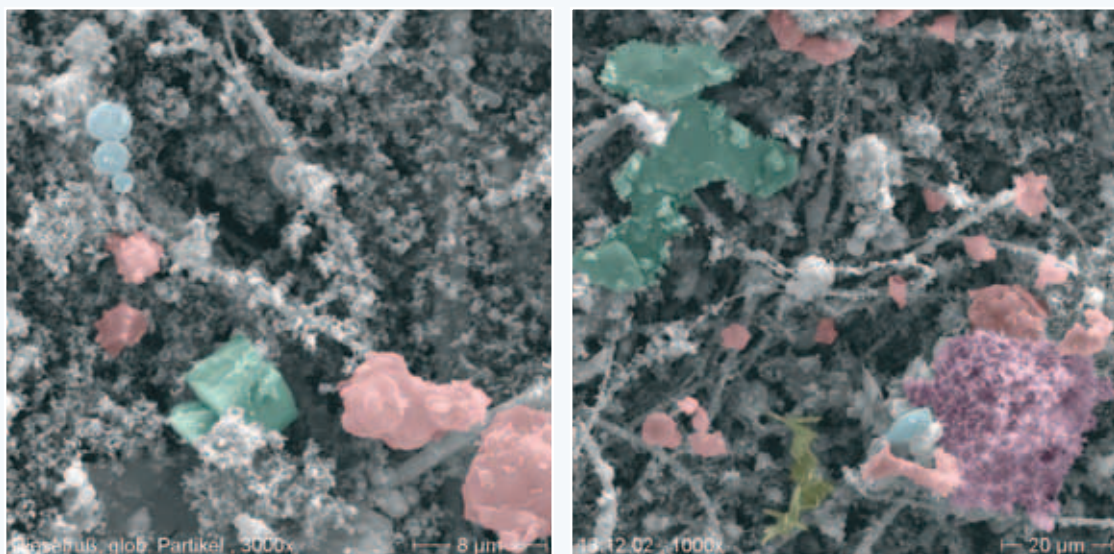
- Ultrafine particles, a term used in various studies, comprise particles of the nucleation and Aitkin modes. Nucleation- and Aitkin-mode particles grow by coagulation (two particles combining to form one) or by condensation (low-equilibrium vapour pressure gas molecules condensing on a particle) and “accumulate” in this size range.
- The accumulation mode covers the range between 0.1  $\mu\text{m}$  and up to 1  $\mu\text{m}$ . These particles do not normally grow into the coarse mode.

**Fig. 2.2. Schematic representation of the size distribution of PM in ambient air**



Source: Department for Environment, Food and Rural Affairs (2).

**Fig. 2.3. Electron microscopic images of PM<sub>10</sub> sampled at two traffic monitoring sites in Austria**



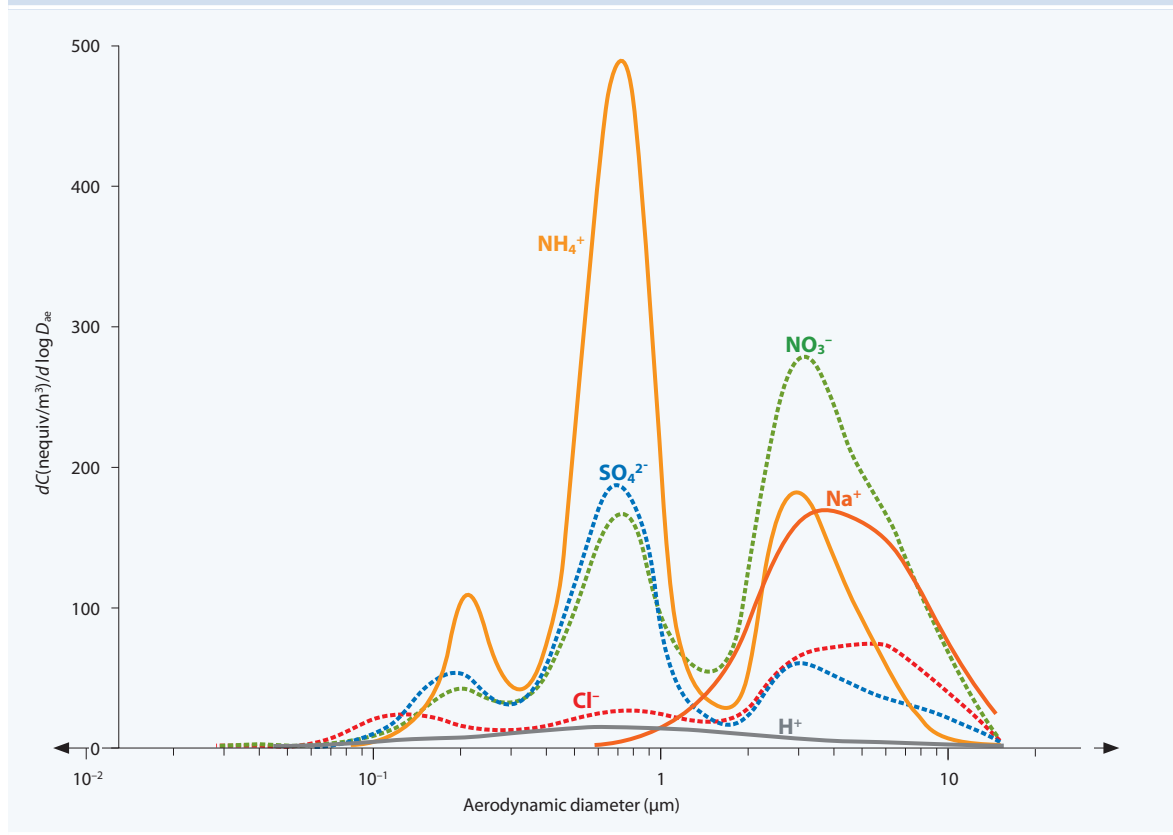
**Table 2.1. Comparison of fine- and coarse-mode particles**

	Fine (< 2.5 µm)		Coarse (2.5–10 µm)
	Ultrafine (< 0.1 µm)	Accumulation (0.1–1 µm)	
Formation processes	Combustion, high-temperature processes and atmospheric reactions		Break-up of large solids/droplets
Formation	Nucleation Condensation Coagulation	Condensation Coagulation Reaction of gases in or on particles Evaporation of fog and cloud droplets in which gases have dissolved and reacted	Mechanical disruption (crushing, grinding, abrasion of surfaces) Evaporation of sprays Suspension of dusts Reactions of gases in or on particles
Composition	Sulfate Elemental carbon Metal compounds Organic compounds with very low saturation vapour pressure at ambient temperature	Sulfate, nitrate, ammonium and hydrogen ions Elemental carbon Large variety of organic compounds Metals: compounds of lead, cadmium, vanadium, nickel copper, zinc, manganese, iron, etc. Particle-bound water	Suspended soil or street dust Fly ash from uncontrolled combustion of coal, oil and wood Nitrates/chlorides from nitric acid/hydrochloric acid Oxides of crustal elements (silicon, aluminium, titanium, iron) Calcium carbonate, sodium chloride, sea salt Pollen, moulds, fungal spores Plant and animal fragments Tyre, brake pad and road wear debris
Solubility	Probably less soluble than accumulation mode	Often soluble, hygroscopic and deliquescent	Largely insoluble and nonhygroscopic
Sources	Combustion Atmospheric transformation of sulfur dioxide and some organic compounds High-temperature processes	Combustion of coal, oil, gasoline, diesel fuel, wood Atmospheric transformation products of nitrogen oxides, sulfur dioxide and organic carbon, including biogenic organic species such as terpenes High-temperature processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads and streets Suspension from disturbed soil (e.g. farming, mining, unpaved roads) Construction and demolition Uncontrolled coal and oil combustion Ocean spray Biological sources
Atmospheric half-life	Minutes to hours	Days to weeks	Minutes to days
Removal processes	Grows into accumulation mode Diffuses to raindrops	Forms cloud droplets and is deposited in rain Dry deposition	Dry deposition by fallout Scavenging by falling rain drops
Travel distance	<1 to tens of km	Hundreds to thousands of km	<1 to hundreds of km

Source: US Environmental Protection Agency (4).

Table 2.1 shows that PM, and especially the fine fraction, remains airborne for a long time in the atmosphere and can travel for hundreds or even thousands of kilometres, crossing borders of regions and coun-

tries. Owing to chemical reactions, condensation and accumulation, the particles change their chemical composition, mass and size. The primary particles emitted in Europe grow 10-fold in mass in a few days,

Fig. 2.4. Aerodynamic parameter of the main chemical components of PM<sub>10</sub>

Source: Wall et al. (3).

forming particles dominated by inorganic salts such as sulfates, nitrates and biogenic organics carrying soot and anthropogenic organics (5). They are able to deposit themselves and affect receptors remote from the source of emission of the primary PM or of the precursor gases.

## References

1. *Methods of measuring air pollution. Report of the working group on methods of measuring air pollution and survey techniques.* Paris, Organisation for Economic Co-operation and Development, 1964.
2. *Air Quality Expert Group report on particulate matter in the United Kingdom.* London, Department for Environment, Food and Rural Affairs, 2005 (<http://www.defra.gov.uk/environment/airquality/aqeg/particulate-matter/index.htm>, accessed 22 December 2005).
3. Wall SM et al. Measurement of aerosol size distributions for nitrate and major ionic species. *Atmospheric Environment*, 1988, 22:1649–1656.
4. *Air quality criteria for particulate matter.* Washington, DC, US Environmental Protection Agency, 2004 (<http://cfpub.epa.gov/ncea/cfm/partmatt.cfm>, accessed 1 October 2005).
5. Forsberg B et al. Comparative health impact assessment of local and regional particulate air pollutants in Scandinavia. *Ambio*, 2005, 34:11–19.



### 3. Hazard assessment of PM

#### KEY MESSAGES

##### Main results

- Exposure to PM in ambient air has been linked to a number of different health outcomes, starting from modest transient changes in the respiratory tract and impaired pulmonary function and continuing to restricted activity/reduced performance, visits to the hospital emergency department, admission to hospital and death. There is also increasing evidence for adverse effects of air pollution on the cardiovascular system as well as the respiratory system. This evidence stems from studies on both acute and chronic exposure. The most severe effects in terms of overall health burden include a significant reduction in life expectancy of the average population by a year or more, which is linked to long-term exposure to PM. A selection of important health effects linked to specific pollutants is summarized in Table 3.1. Most epidemiological studies on large populations have been unable to identify a threshold concentration below which ambient PM has no effect on mortality and morbidity.

##### Main uncertainties

- Despite differences in toxic properties found among PM constituents studied under laboratory conditions, it is currently not possible to quantify precisely the contributions from different sources and different PM components to the effects on health caused by exposure to ambient PM. Thus there remain some uncertainties as to the precise contribution of pollution from regional versus local sources in causing the effects observed in both short- and long-term epidemiological studies.

##### Conclusions

- The body of evidence on health effects of PM at levels currently common in Europe has strengthened considerably over the past few years. Both epidemiological and toxicological evidence has contributed to this strengthening; the latter provides new insights into possible mechanisms for the hazardous effects of air pollutants on human health and complements the large body of epidemiological evidence. The evidence is sufficient to strongly recommend further policy action to reduce levels of PM. It is reasonable to assume that a reduction in air pollution will lead to considerable health benefits (1).

**Table 3.1. Important health effects associated with exposure to PM**

##### Effects related to short-term exposure

- Lung inflammatory reactions
- Respiratory symptoms
- Adverse effects on the cardiovascular system
- Increase in medication usage
- Increase in hospital admissions
- Increase in mortality

##### Effects related to long-term exposure

- Increase in lower respiratory symptoms
- Reduction in lung function in children
- Increase in chronic obstructive pulmonary disease
- Reduction in lung function in adults
- Reduction in life expectancy, owing mainly to cardiopulmonary mortality and probably to lung cancer



### 3.1 Approaches to assessing the health effects of PM

Information on the health effects of PM comes from different disciplines. A review and assessment of the health risks of PM is a major challenge, since a remarkably large body of evidence has to be taken into account. In the last decade, there have been hundreds of new scientific publications addressing exposure, and providing new toxicological and epidemiological findings on adverse health effects. By necessity, any review will have to be selective, focusing on the most significant and relevant studies and on meta-analyses when available.

The literature represented a variety of papers with different sources of information, including observational epidemiology, controlled human exposures to pollutants, animal toxicology and *in vitro* mechanistic studies. Each of these approaches has its strengths and weaknesses. Epidemiology is valuable because it generally deals with the full spectrum of susceptibility in human populations. Children, the elderly and people with pre-existing disease are usually included. In fact, the effects in such susceptible groups may dominate the health outcomes reported. In addition, exposure occurs under real life conditions. Extrapolation across species and to different levels of exposure is not required. Sensitive methodologies, such as time series analysis, allow the identification of even small increases in overall mortality. Nevertheless, exposures are complex in epidemiological studies; observational epidemiology, for example, unless it is a study in the workplace, inevitably includes mixtures of gases and particles. By contrast, in controlled human exposures, exposure can be to a single agent that can be carefully generated and characterized, and the nature of the subjects can be rigorously selected and defined. Yet such studies are limited because they generally deal with short-term, mild, reversible alterations and a small number of individuals exposed to single pollutants, and do not include those with severe disease who may be at most risk of adverse effects.

Animal studies have the same strengths of well-characterized exposures and more uniform subjects. Invasive mechanistic studies can be carried out. More profound toxic effects can be produced in animals than in experimental human studies. Other limitations occur, however, such as possible interspecies

differences and the frequent need to extrapolate from the higher levels used in animal studies to lower (and more relevant) ambient concentrations.

For these reasons, the best synthesis incorporates different sources of information. Thus the WHO review did not rely solely on (new) epidemiological evidence but included also new findings from toxicological and clinical studies.

### 3.2 Epidemiological studies on effects of exposure to PM

Most of the currently available epidemiological studies on the health effects of PM use mortality as the indicator of health effect. The main reason for this obvious limitation is the relatively easy access to information on population mortality necessary for time series studies. In most cases, the quality of routinely collected mortality data is good and permits cause-specific analysis. Information on daily admissions to hospital are also used by time series studies, but their intercountry comparison and use for health impact assessment are limited by differences in national or local practices in hospital admissions and in the use of other forms of medical care in the case of acute symptoms. Also, for long-term studies, information on case mortality is easier to obtain than on less severe health problems, which can also indicate adverse effects of air pollution. Consequently, the risk estimates for mortality can be compared between populations, and a common estimate can be generated either in multicentre studies or in meta-analysis. Such estimates provide strong support for health impact assessment. Unfortunately, comparison between populations of morbidity risk coefficients is less reliable owing to less certainty about the definition and ascertainment of the health outcome under study.

#### Studies on the effects of long-term exposure to PM on mortality

Results from studies on the effects of long-term exposure to PM on mortality are specifically relevant for this report, since they provide essential information for assessing the health impact of PM exposure (Table 3.2). Recently, the available knowledge has been expanded by three new cohort studies (2–4), an extension of the American Cancer Society (ACS) cohort study (5) and a thorough re-analysis of origi-

nal study papers by the Health Effects Institute (HEI) (6). In view of the extensive scrutiny that was applied in the HEI re-analysis to the Harvard Six Cities Study and the ACS study, it is reasonable to attach most weight to these two. The HEI re-analysis largely corroborated the findings of the original two American cohort studies, both of which showed an increase in mortality with an increase in fine PM and sulfate. The increase in mortality was mostly related to increased cardiovascular mortality. A major concern remaining was that spatial clustering of air pollution and health data in the ACS study made it difficult to disentangle air pollution effects from those of spatial auto-correlation of health data per se. The extension of the ACS study found statistically significant increases in relative risk for PM<sub>2.5</sub> in the case of cardiopulmonary and lung cancer deaths and deaths from all causes. TSP and coarse particles (PM<sub>15</sub>-PM<sub>2.5</sub>) were not significantly associated with mortality (5,6). The effect estimates remained largely unchanged even after taking spatial auto-correlation into account.

Another concern was about the role of sulfur dioxide. Inclusion of sulfur dioxide in multi-pollutant models decreased PM effect estimates consider-

ably in the re-analysis, suggesting that there was an additional role for sulfur dioxide or for pollutants spatially co-varying with it. This issue was not further addressed in the extension of the ACS study, although a statistically significant effect of sulfur dioxide was found in a single-pollutant model. The HEI re-analysis report concluded that the spatial adjustment might have over-adjusted the estimated effect for regional pollutants such as fine particles and sulfate compared to effect estimates for more local pollutants such as sulfur dioxide. The discussion of available evidence by the WHO systematic review of epidemiological and toxicological studies points to an unlikely role of sulfur dioxide as the cause of health effects attributed to PM (7).

More recent publications from the extended follow-up of the ACS study indicate that the long-term exposures to PM<sub>2.5</sub> were most strongly associated with mortality attributable to ischemic heart disease, dysrhythmias, heart failure and cardiac arrest (8). For these cardiovascular causes of death, a 10- $\mu\text{g}/\text{m}^3$  elevation of PM<sub>2.5</sub> was associated with an 8–18% increase in risk of death. Mortality attributable to respiratory disease had relatively weak associations. Analysis of

**Table 3.2. Comparison of excess relative risk for mortality from American cohort studies**

Study	PM <sup>a</sup> metric <sup>c</sup>	Total mortality		Cardiopulmonary mortality		Lung cancer mortality	
		Excess RR <sup>b</sup>	95% CI (%)	Excess RR	95% CI (%)	Excess RR	95% CI (%)
Six City (6,11)	PM <sub>2.5</sub>	13%	4.2–23	18%	6.0–32	18%	-11–57
Six City new (6)	PM <sub>2.5</sub>	14%	5.4–23	19%	6.5–33	21%	-8.4–60
ACS (6)	PM <sub>2.5</sub>	6.6%	3.5–9.8	12%	6.7–17	1.2%	-8.7–12
ACS new (6)	PM <sub>2.5</sub>	7.0%	3.9–10	12%	7.4–17	0.8%	-8.7–11
ACS new (6)	PM <sub>15–2.5</sub>	0.4%	-1.4–2.2	0.4%	-2.2–3.1	-1.2%	-7.3–5.1
ACS new (6)	PM <sub>10/15</sub>	4.1%	0.9–7.4	7.3%	3.0–12	0.8%	-8.1–11
ACS new (6)	PM <sub>10/15</sub> SSI <sup>c</sup>	1.6%	-0.8–4.1	5.7%	2.5–9.0	-1.6%	-9.1–6.4
ACS extended (5)	PM <sub>2.5</sub> 1979–1983	4.1%	0.8–7.5	5.9%	1.5–10	8.2%	1.1–16
ACS extended (5)	PM <sub>2.5</sub> 1999–2000	5.9%	2.0–9.9	7.9%	2.3–14	12.7%	4.1–22
ACS extended (5)	PM <sub>2.5</sub> average	6.2%	1.6–11	9.3%	3.3–16	13.5%	4.4–23
AHSMOG (2) <sup>d</sup>	PM <sub>10/15</sub>	2.1%	-4.5–9.2	0.6%	-7.8–10	81%	14–186
AHSMOG (12) <sup>e</sup>	PM <sub>2.5</sub>	8.5%	-2.3–21	23%	-3.0–55	39%	-21–150
Veterans Administration <sup>f</sup>	PM <sub>2.5</sub>	-10.0%	-15–-4.6				

<sup>a</sup> Increments are 10  $\mu\text{g}/\text{m}^3$  for PM<sub>2.5</sub> and 20  $\mu\text{g}/\text{m}^3$  for PM<sub>10/15</sub>.

<sup>b</sup> Excess RR (percentage excess relative risk) =  $100 \times (\text{RR}-1)$ , where the RR has been converted from the highest-to-lowest range to the standard increment (10 or 20) by the equation  $\text{RR} = \exp(\log(\text{RR for range}) \times \text{range})$ .

<sup>c</sup> PM measured with size-selective inlet (SSI) technology. The other PM measurements in ACS new (6) were based on dichotomous sampler with 15- $\mu\text{m}$  and 2.5- $\mu\text{m}$  cut-off points.

<sup>d</sup> Pooled estimate for males and females.

<sup>e</sup> Using two-pollutant (fine- and coarse-particle) models; males only.

<sup>f</sup> Males only, exposure period 1979–1981, mortality 1982–1988 (from Table 7 in Lipfert et al. (13)).

Source: US Environmental Protection Agency (10).

the Los Angeles part of the ACS cohort suggests that the chronic health effects associated with within-city gradients in exposure to PM<sub>2.5</sub> may be even larger than those reported across metropolitan areas (9).

The Adventist Health and Smog (AHSMOG) study (2) found significant effects of PM<sub>10</sub> on nonmalignant respiratory deaths in men and women and on lung cancer deaths in men in a relatively small sample of non-smoking Seventh-Day Adventists. Results for PM<sub>10</sub> were insensitive to adjustment for co-pollutants. In contrast to the Six Cities and ACS studies, no association with cardiovascular deaths was found. For the first 10 years of the 15-year follow-up period PM<sub>10</sub> was estimated from TSP measurements, which were also much less related to mortality in the other two cohorts. A later analysis of the AHSMOG study suggested that effects became stronger when analysed in relation to PM<sub>2.5</sub> estimated from airport visibility data, which further reduces the degree of discrepancy with the other two cohort studies.

The EPRI-Washington University Veterans' Cohort Mortality Study used a prospective cohort of up to 70 000 middle-aged men (51 ± 12 years) assembled by the Veterans Administration (13). No consistent effects of PM on mortality were found. However, statistical models included up to 230 terms and the effects of active smoking on mortality in this cohort were clearly smaller than in other studies, calling into question the modelling approach that was used. Also, only data on total mortality were reported, precluding conclusions with respect to cause-specific deaths.

The first European cohort study reported was from the Netherlands (4) and suggested that exposure to traffic-related air pollution, including PM, was associated with increased cardiopulmonary mortality in people living close to main roads.

The relationship between air pollution and lung cancer has also been addressed in several case-control studies. A study from Sweden found a relationship with motor vehicle emissions, estimated as the nitrogen dioxide contribution from road traffic, using retrospective dispersion modelling (14). Diesel exhaust may be involved in this but, so far, diesel exhaust has not been classified by the International Agency for Research on Cancer (IARC) as a proven human carcinogen. Nevertheless, new evaluations are under way, both in the United States and at IARC,

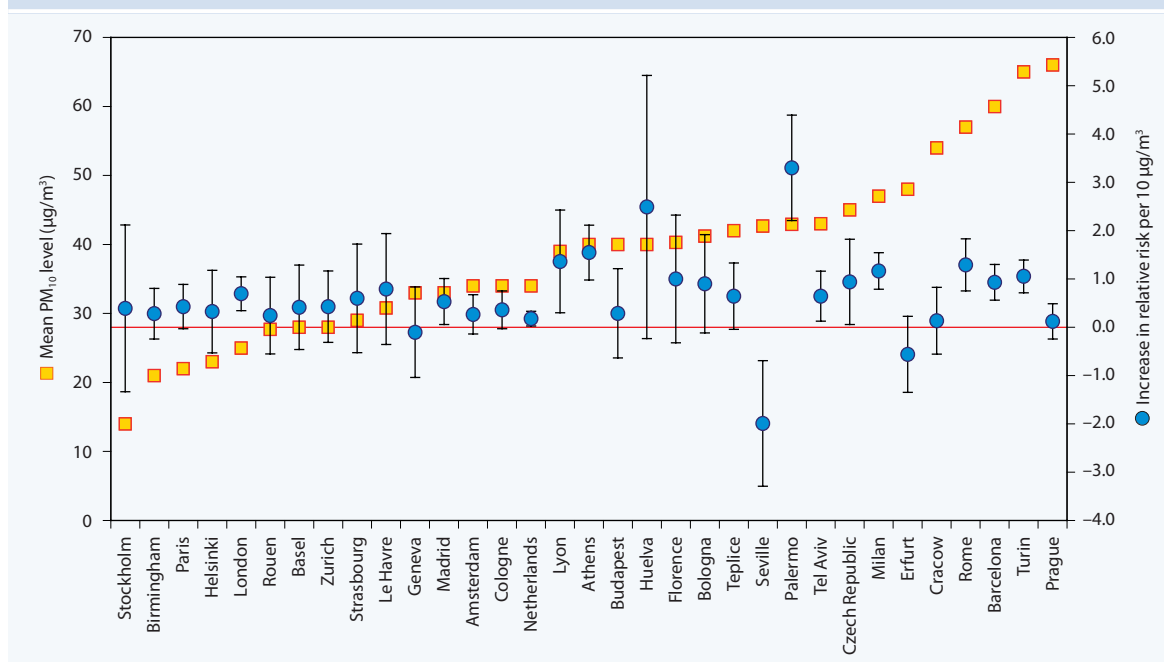
since new studies and reviews have appeared since IARC last evaluated diesel exhaust in 1989.

The effects of long-term PM exposure on a number of other health parameters were also evaluated in a number of studies. Notably, work from Southern California has shown that lung function growth in children is reduced in areas with high PM concentrations (15,16) and that the lung function growth rate changes in step with relocation of children to areas with higher or lower PM concentrations than before (17). Impacts of pollution on the prevalence of respiratory symptoms in children and adults were also found, though high correlation of various pollutants in those studies precludes attribution of the results of these studies to PM alone.

### Studies on the effects of short-term exposure to PM

Since the early 1990s, more than 100 studies on the effects of short-term exposure to air pollution, including PM, have been published in Europe and other parts of the world. Most of them are "time series" studies, analysing the association between daily variations in the ambient concentrations of the pollutants measured by the air quality monitoring networks and daily changes in health status of the population indicated by counts of deaths or admissions to hospital. As part of the WHO project "Systematic review of health aspects of air pollution in Europe", WHO commissioned a meta-analysis of peer-reviewed European studies to obtain summary estimates for certain health effects linked to exposure to PM and ozone. The data for these analyses came from a database of time series studies (ecological and individual) developed at St George's Hospital Medical School at the University of London. Also, the meta-analysis was performed at St George's Hospital according to a protocol agreed on by a WHO Task Group in advance of the work. This analysis confirmed statistically significant relationships between levels of PM and ozone in ambient air and mortality, using data from several European cities (18).

Estimates of the effect of PM<sub>10</sub> on all-cause mortality were taken from 33 separate European cities or regions. The random-effects summary of relative risk for these 33 results was 1.006 (95% CI 1.004–1.008) for a 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> (Fig. 3.1). Of these

Fig. 3.1. Relative risk for all-cause mortality and a 10- $\mu\text{g}/\text{m}^3$  increase in daily  $\text{PM}_{10}$ 

Source: WHO (18).

estimates, 21 were taken from the APHEA 2 study (19) and hence the summary estimate derived from this review is dominated by this multi-city study. Cause-specific results for mortality from the APHEA 2 project have yet to be published. Thus, the numbers of estimates for cardiovascular and respiratory mortality are smaller than for all-cause mortality – 17 and 18, respectively. The corresponding summary estimates were 1.009 (1.005–1.013) and 1.013 (1.005–1.020) for a 10- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$  (Fig. 3.2). The majority of the estimates in these two categories come from multi-city studies conducted in France, Italy and Spain.

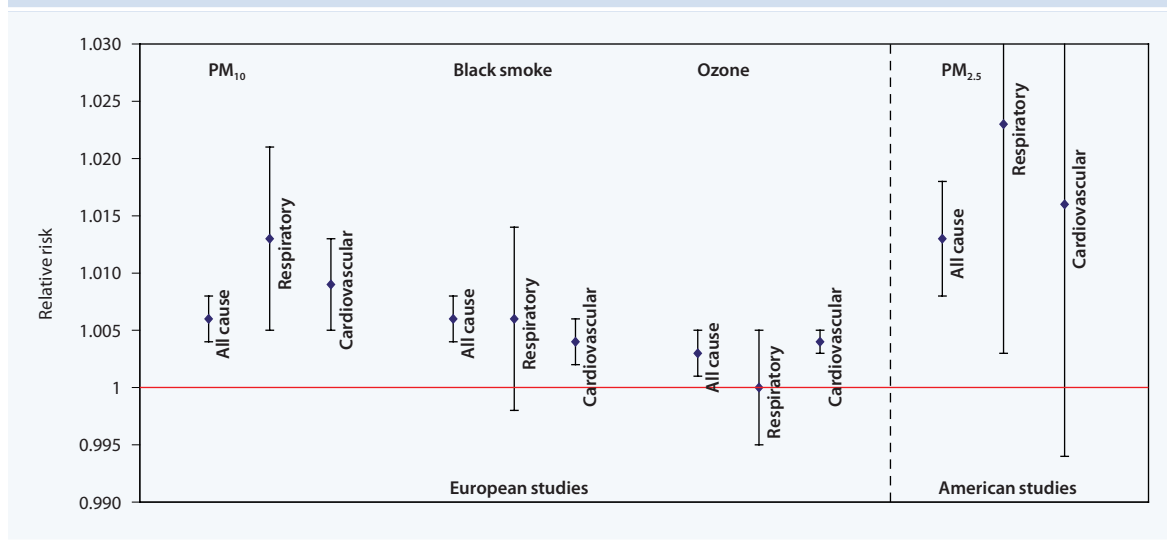
The estimates for all-cause and cause-specific mortality are comparable to those originally reported from the National Mortality, Morbidity and Air Pollution Study (NMMAPS), based on the 20 largest cities in the United States (20). For a 10- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$  they reported a 0.51% (0.07–0.93) increase in daily mortality from all causes, and for cardiorespiratory mortality the corresponding percentage change was slightly larger at 0.68% (0.2–1.16). A recent re-analysis of the NMMAPS data, organized by HEI because of concern over the statistical procedures used in the original analyses, revised the NMMAPS

summary estimates downwards to 0.21% for all-cause mortality and 0.31% for cardiorespiratory mortality (21). A similar re-analysis of the APHEA 2 mortality data revealed that the European results were more robust to the method of analysis. It is at present uncertain why the European estimates are markedly higher than those from North America, and whether this difference is also valid for the risk associated with long-term exposure.

There are few European epidemiological studies on the health effects of  $\text{PM}_{2.5}$ . The WHO meta-analysis of non-European studies indicates significant effects of  $\text{PM}_{2.5}$  on total mortality as well on mortality due to cardiovascular and respiratory diseases (18).

In their recent analysis of the available evidence on the effects of coarse airborne particles on health, Brunekreef & Forsberg (22) conclude that increases in mortality are mainly related to an increase in  $\text{PM}_{2.5}$  and not to that of the coarse fraction. However, in studies of chronic obstructive pulmonary disease, asthma and admissions to hospital caused by respiratory diseases, coarse PM has at least as strong a short-term effect as fine PM, suggesting that coarse PM may trigger adverse responses in the lungs requiring hospital treatment.

Fig. 3.2. Summary estimates for relative risks for mortality and different air pollutants



Note: There were not enough European results for a meta-analysis of effects of PM<sub>2.5</sub>. The relative risk for this pollutant is from North American studies and is shown for illustrative purposes only.

Source: WHO (18).

Only for hospital admissions due to respiratory diseases in those aged 65+ was there a sufficient number of estimates for the WHO meta-analysis of European short-term studies on effects of PM on morbidity (18). The relative risk for a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> was 1.007 (95% CI 1.002–1.013).

### 3.3 Intervention studies and evidence for a causal relationship between particulate air pollution and health effects

Positive effects of reductions in ambient PM concentrations on public health have been shown following the introduction of clean air legislation. Such positive effects have also been reported more recently in a limited number of studies.

Some studies have addressed directly the question of whether public health benefits can be shown as a result of planned or unplanned reductions in air pollution concentrations. A recent study from Dublin documented the health benefits of the ban on the use of coal for domestic heating enforced in 1990 (23). In the Utah Valley, PM concentrations fell markedly during a 14-month strike at a local steel factory in the 1980s, and mortality as well as respiratory morbidity was found to be reduced during this period (24,25). It is worth mentioning that toxicological studies have been performed to examine whether a change in the

concentration of inert vs active components in the PM fraction could reduce the inflammatory/toxic potential of ambient PM. Both controlled human exposures (26) and animal studies (27) using Utah Valley PM<sub>10</sub> sampled before, during and after closing of the steel factory showed considerable coherence of inflammatory outcomes in the lung and changes in airway hyperresponsiveness compared to the epidemiological findings. The change of toxicity potential was attributed to a change in metal (or metal cation) concentrations in the PM (28).

Studies from the area of the former German Democratic Republic reveal a reduction in childhood bronchitis and improved lung function along with sharp reductions in SO<sub>2</sub> and PM concentrations after German reunification (29–31). The effect of reduced air pollution is, however, confounded with other socioeconomic and cultural changes that happened at the same time (“westernization”) and so is difficult to identify reliably.

On balance, these studies suggest that reductions in ambient PM concentrations bring about benefits to public health that can be observed in the months and years immediately following the reduction. (There may also be further, delayed, benefits.) However, the available epidemiological intervention studies do not give direct, quantitative evidence as to the rela-

tive health benefits that would result from selective reduction of specific PM size fractions.

Ambient PM per se is also considered responsible for the health effects seen in the large multi-city epidemiological studies relating ambient PM to mortality and morbidity such as NMMAPS (32) and APHEA (19). In the Six Cities (6) and ACS cohort studies (5), PM but not gaseous pollutants (with the exception of sulfur dioxide) was associated with mortality. That ambient PM is responsible per se for effects on health is substantiated by controlled human exposure studies, and to some extent by experimental findings in animals. Overall, the body of evidence strongly suggests causality and so implies that reductions in mixed ambient PM will be followed by benefits to health.

### 3.4 Thresholds

The WHO systematic review analysed in depth the question of whether there is a threshold below which no effects of the pollutant on health are expected to occur in all people. After thorough examination of all the available evidence, the review concluded (33) that:

Most epidemiological studies on large populations have been unable to identify a threshold concentration below which ambient PM has no effect on mortality and morbidity. It is likely that within any large human population, there is a wide range in susceptibility so that some subjects are at risk even at the low end of current concentrations.

There are only few studies available on the effects of long-term exposure of PM on mortality, and even fewer of these examined the shape of the exposure-response relationship. The most powerful study (5) used non-parametric smoothing to address this issue and found no indication of a threshold for PM<sub>2.5</sub>, either for cardiopulmonary or for lung cancer mortality, within the range of observed PM<sub>2.5</sub> concentrations of about 8–30 µg/m<sup>3</sup>. Further modelling of these data suggested that the exposure-response relationship for PM<sub>2.5</sub> was actually steeper in the low-exposure range up to about 16 µg/m<sup>3</sup>. In contrast, analyses for sulfates suggested that a threshold might exist at about 12 µg/m<sup>3</sup> (34).

### 3.5 Susceptible groups

A number of groups within the population have potentially increased vulnerability to the effects of exposure to particulate air pollutants. These groups comprise those who are innately more susceptible to the effects of air pollutants than others, those who become more susceptible (for example as a result of environmental or social factors or personal behaviour) and those who are simply exposed to unusually large amounts of air pollutants. Members of the last group are vulnerable by virtue of exposure rather than as a result of personal susceptibility. Groups with innate susceptibility include those with genetic predisposition that renders them unusually sensitive (35).

Very young children and probably unborn babies seem also particularly sensitive to some pollutants, as concluded by a WHO Working Group (36). The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the post-neonatal period. Evidence is also sufficient to infer a causal relationship between exposure to ambient air pollutants and adverse effects on lung function development. Both reversible deficits of lung function as well as chronically reduced lung growth rates and lower lung function levels are associated with exposure to particulates. The available evidence is also sufficient to assume a causal relationship between exposure to PM and aggravation of asthma, as well as a causal link between increased prevalence and incidence of cough and bronchitis due to particulate exposure.

Groups that develop increased sensitivity include the elderly, those with cardiorespiratory disease or diabetes (37), those who are exposed to other toxic materials that add to or interact with air pollutants, and those who are socioeconomically deprived. When compared with healthy people, those with respiratory disorders (such as asthma or chronic bronchitis) may react more strongly to a given exposure, either as a result of increased responsiveness to a specific dose or as a result of a larger internal dose of some pollutants. In short-term studies, elderly people (38) and those with pre-existing heart and lung disease (39,40) were found to be more susceptible to effects of ambient PM on mortality and morbidity. In panel studies, asthmatics have also been shown to

respond to ambient PM with more symptoms, larger lung function changes and increased medication use than non-asthmatics (5,41). In long-term studies, it has been suggested that socially disadvantaged and poorly educated populations respond more strongly in terms of mortality (4–6).

Increased particle deposition and retention has been demonstrated in the airways of subjects suffering from obstructive lung disease (42). Lastly, those exposed to unusually large amounts of air pollutants, including PM, perhaps as a result of living near a main road or spending long hours outdoors, may be vulnerable as result of their high level of exposure.

### 3.6 Critical components and critical sources

As stated above, PM in ambient air has various sources. In targeting control measures, it would be important to know if PM from certain sources or of a certain composition gave rise to special concern from the point of view of health, for example owing to high toxicity. The few epidemiological studies that have addressed this important question specifically suggest that combustion sources are particularly important for health (43,44). Toxicological studies have also pointed to primary combustion-derived particles as having a higher toxic potential (45). These particles are often rich in transition metals and organic compounds, and also have a relatively high surface area (46). By contrast, several other single components of the PM mixture (e.g. ammonium salts, chlorides, sulfates, nitrates and wind-blown dust such as silicate clays) have been shown to have a lower toxicity in laboratory studies (47). Despite these differences found among constituents studied under laboratory conditions, it is currently not possible to precisely quantify the contributions from different sources and different PM components to the effects on health caused by exposure to ambient PM. It seems also premature to rule out any of the anthropogenic components in contributing to adverse health effects. It is, however, prudent to check that proposed control measures do indeed target those components of PM, which studies to date have suggested are relatively more toxic (or, equivalently, to check that reductions in PM are not achieved principally by reductions in the less toxic fractions).

It is worth noting that some of the components identified as hazardous in toxicological studies can also be found in rural sites in considerable concentrations. These include organic material and transition metals, even though the latter are clearly enriched near sources. However, some of the components with less toxicological activity are also present at considerable levels in aerosols subject to long-range transboundary air pollution, including secondary inorganic aerosols and sea salt.

### 3.7 Regional differences

The heterogeneity of effect estimates between cities or areas has been identified and investigated in several studies on the health effects of air pollution (31,48–50). Thus, in the APHEA project it was first noted that the short-term effects of particles on mortality were lower in cities in central-eastern Europe (48). Similarly, in the NMMAPS project the highest effects of particles were estimated for the northeast United States (32). This issue was investigated further in the APHEA 2 project, where a number of variables (city characteristics) hypothesized to be potential effect modifiers were recorded and tested in a hierarchical modelling approach (19). This led to the identification of several factors that can explain part of the observed heterogeneity. Nevertheless, much of the variation between studies and regions remains unexplained. The following were the most important effect modifiers identified.

- Larger estimates of the effects of particles on mortality are found in warmer cities (e.g. 0.8% versus 0.3% increase in mortality per 10- $\mu\text{g}/\text{m}^3$  change in  $\text{PM}_{10}$ ).
- In cities with higher  $\text{NO}_2$  levels the estimated effects were higher (e.g. 0.8% versus 0.2% increase in mortality per 10- $\mu\text{g}/\text{m}^3$  change in  $\text{PM}_{10}$ ). This may reflect a real interaction between  $\text{NO}_2$  and PM or it may indicate that high  $\text{NO}_2$  levels imply larger proportions of particles originating from traffic.
- It is generally accepted that air pollution causes larger effects in members of sensitive population subgroups. There is evidence that the effects are larger among the elderly (51,52). In the APHEA 2 analyses it was found that in cities with higher age-

standardized mortality and those with a smaller proportion of elderly people (>65 years) the estimated effects were lower (19).

In the re-analyses of the Six City and ACS cohort studies on long-term effects of air pollution on mortality, several socioeconomic variables were tested as potential effect modifiers (49).

### **3.8. Relevance of exposure at urban background versus hot spot**

There are locations at which short-term and/or long-term exposure to air pollution is significantly increased in comparison to the rural or urban background. These include locations in the vicinity of traffic and industrial and domestic sources. Much new evidence has been produced in recent years on traffic hot spots. PM can be significantly elevated near such sources, especially PM components such as elemental carbon and ultrafine particles, while PM mass (such as PM<sub>10</sub> and PM<sub>2.5</sub>) has a much more even spatial distribution. Levels of secondary PM components such as sulfates and nitrates are hardly elevated near traffic sources (see also Chapter 5).

Recent evidence has shown that people living near busy roads (the best investigated type of hot spot) are insufficiently characterized by air pollution measurements obtained from urban background locations, and that they are also at increased risk of adverse health effects (4,53–57). It is worth noting that a significant part of the urban population may be affected. Roemer & van Wijnen (53) estimated that 10% of the population of Amsterdam were living on roads carrying more than 10 000 vehicles a day.

Thus there remain some uncertainties on the precise contribution of pollution from regional vs local sources in causing the effects observed in both short- and long-term epidemiological studies. As a first approximation, the contribution of regional sources to urban background concentrations can be used as a proxy to estimate the effects of regional air pollution on health.

### **3.9 How PM seems to exert its effects – conclusions from mechanistic studies**

Human experimental studies and animal and cellular experiments all indicate that PM initiates or exacer-

bates disease or its markers through several mechanisms, as indicated below.

Central with respect to lung disorders is the induction of an inflammatory response that the lung tissue cannot cope with. This response involves the influx of inflammatory cells following the formation of reactive oxygen/nitrogen compounds, cascades of intracellular signals in response to the PM-associated stress factors, changes in gene expression and a network of signalling substances between cells (58,59). The tissue defence, such as different types of antioxidant (vitamins), anti-inflammatory lung proteins (SP-A, CCSP) and cytokines (IL-10), may be overwhelmed. PM and the inflammatory response may induce tissue damage and repair, which may lead to remodelling of the lung structure and loss of function (60), although an additional effect of PM on allergen-induced inflammation could not be demonstrated in two recent reports (61,62). PM seems to be able to exert an effect as adjuvant in the induction of an allergic reaction with specific IgE production and eosinophilic inflammation (63) and also an exacerbation of the allergic response (64).

PM, and in particular fine PM, has been also been found to elicit DNA damage, mutations and carcinogenesis (65–66). These effects are related to oxidative DNA damage, metabolism of organic compounds and formation of adducts. The effects may be exacerbated by insufficient DNA repair (67) and by low capability of detoxification of activated, carcinogenic metabolites (68).

The effects of PM on the cardiovascular system seem to involve the activation of clotting factors, leading to the formation of thrombosis, but the destabilization of atherosclerotic plaques also cannot be excluded. In addition, there may be effects on the heart, mediated through effects on the nervous system or directly on the heart itself. The latter mechanism may include the release/leakage of stress mediators from the lung and/or the direct effect of soluble compounds or of ultrafine particles on the heart cells.

Which PM components might be most important for health effects is still a matter of intense investigation. Different studies point to different components. Many PM components have been shown to be able to induce oxygen radical formation. Surface and composition, however, seem to be more important



determinants of particle effects than mass. Different components may be involved in eliciting the diverse effects. Certain PAH are especially potent in causing DNA damage and cancer. Some metals, but also metal-free ultrafine particles, are strong inducers of inflammation. Ultrafine particles are also suspected to initiate cardiovascular responses, whereas coarse particles may not affect the cardiovascular system. However, these issues have not yet been resolved.

## References

1. *Health aspects of air pollution. Results from the WHO project "Systematic review of health aspects of air pollution in Europe"*. Copenhagen, WHO Regional Office for Europe, 2004 (<http://www.euro.who.int/document/E83080.pdf>, accessed 17 November 2005).
2. Abbey DE et al. Long-term inhalable particles and other pollutants related to mortality of non-smokers. *American Journal of Respiratory and Critical Care Medicine*, 1999, 159:373–382.
3. Lipfert FW et al. The Washington University-EPRI veterans' cohort mortality study: preliminary results. *Inhalation Toxicology*, 2000, 12:41–73.
4. Hoek G et al. The association between mortality and indicators of traffic-related air pollution in a Dutch cohort study. *Lancet*, 2002, 360:1203–1209.
5. Pope CA et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 2002, 287:1132–1141.
6. Krewski D et al. *Re-analysis of the Harvard Six-Cities Study and the American Cancer Society study of air pollution and mortality*. Cambridge, MA, Health Effects Institute, 2000.
7. *Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2003 (document EUR/03/5042688) (<http://www.euro.who.int/document/e79097.pdf>, accessed 1 October 2005).
8. Pope CA et al. Cardiovascular mortality and long-term exposure to particulate air pollution. *Circulation*, 2004, 109:71–77.
9. Jarett M et al. Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiology*, 2005, 16:727–736.
10. *Air quality criteria for particulate matter*. Washington, DC, US Environmental Protection Agency, 2004 (<http://cfpub.epa.gov/ncea/cfm/partmatt.cfm>, accessed 1 October 2005).
11. Dockery DW, Pope CA, Xu X. An association between air pollution and mortality in six US cities. *New England Journal of Medicine*, 1993, 329:1753–1759.
12. McDonnell WF et al. Relationships of mortality with the fine and coarse fractions of long-term ambient PM<sub>10</sub> concentrations in nonsmokers. *Journal of Exposure Analysis and Environmental Epidemiology*, 2000, 10:427–436.
13. Lipfert FW et al. The Washington University-EPRI veterans' cohort mortality study: preliminary results. *Inhalation Toxicology*, 2000, 12(Suppl. 4):41–73.
14. Nyberg F et al. Urban air pollution and lung cancer in Stockholm. *Epidemiology*, 2000, 11:487–495.
15. Gauderman WJ et al. Association between air pollution and lung function growth in southern California children. *American Journal of Respiratory & Critical Care Medicine*, 2000, 162:1383–1390.
16. Gauderman WJ et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. *American Journal of Respiratory & Critical Care Medicine*, 2002, 166:76–84.
17. Avol EL et al. Respiratory effects of relocating to areas of differing air pollution levels. *American Journal of Respiratory & Critical Care Medicine*, 2001, 164:2067–2072.
18. *Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O<sub>3</sub>)*. Copenhagen, WHO Regional Office for Europe, 2004 (<http://www.euro.who.int/document/E82792.pdf>, accessed 6 November 2005).

19. Katsouyanni K et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: Results from 29 European cities within the APHEA 2 project. *Epidemiology*, 2001, 12:521–531.
20. Samet JM et al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *New England Journal of Medicine*, 2000, 343:1742–1749.
21. Revised analyses of time-series studies of air pollution and health. Boston, MA, Health Effects Institute, 2003.
22. Brunekreef B, Forsberg B. Epidemiological evidence of effects of coarse airborne particles on health. *European Respiratory Journal*, 2005, 26:309–318.
23. Clancy L et al. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet*, 2002, 360:1210–1214.
24. Pope CA et al. Daily mortality and PM<sub>10</sub> pollution in Utah Valley. *Archives of Environmental Health*, 1992, 47:211–217.
25. Pope CA. Particulate pollution and health: a review of the Utah valley experience. *Journal of Exposure Analysis and Environmental Epidemiology*, 1996, 6:23–34.
26. Ghio AJ et al. Diffuse alveolar damage after exposure to an oil fly ash. *American Journal of Respiratory and Critical Care Medicine*, 2001, 164:1514–1518.
27. Dye JA et al. Acute pulmonary toxicity of particulate matter filter extracts in rats: coherence with epidemiologic studies in Utah Valley residents. *Environmental Health Perspectives*, 2001, 109:395–403.
28. Molinelli AR et al. Effect of metal removal on the toxicity of airborne particulate matter from the Utah Valley. *Inhalation Toxicology*, 2002, 14:1069–1086.
29. Heinrich J et al. Decline of ambient air pollution and respiratory symptoms in children. *American Journal of Respiratory and Critical Care Medicine*, 2000, 161:1930–1936.
30. Heinrich J et al. Improved air quality in reunified Germany and decreases in respiratory symptoms. *Epidemiology*, 2002, 13:394–401.
31. Frye C et al. Association of lung function with declining ambient air pollution. *Environmental Health Perspectives*, 2003, 111:383–387.
32. Samet JM et al. *National morbidity, mortality and air pollution study*. Cambridge, MA, Health Effects Institute, 2000 (HEI Report 94, Part 2).
33. *Health aspects of air pollution – answers to follow-up questions from CAFE. Report on a WHO working group meeting, Bonn, Germany, 15–16 January 2004*. Copenhagen, WHO Regional Office for Europe, 2004 (document EUR/04/5046026) (<http://www.euro.who.int/document/E82790.pdf>, accessed 3 November 2005).
34. Abrahamowicz M et al. Flexible modelling of exposure–response relationship between long-term average levels of particulate air pollution and mortality in the American Cancer Society study. *Journal of Toxicology and Environmental Health*, 2003, 66:1625–1654.
35. Gilliland FD et al. Effect of glutathione-S-transferase M1 and P1 genotypes on xenobiotic enhancement of allergic responses: randomised, placebo-controlled crossover study. *Lancet*, 2004, 363:119–125.
36. *Effects of air pollution on children's health and development: a review of the evidence*. Copenhagen, WHO Regional Office for Europe, 2005 (<http://www.euro.who.int/document/E86575.pdf>, accessed 3 November 2005).
37. Zanobetti A, Schwartz J. Are diabetics more susceptible to the health effects of airborne particles? *American Journal of Respiratory and Critical Care Medicine*, 2001, 164:831–833.
38. Schwartz, J. What are people dying of on high air pollution days? *Environmental Research*, 1994, 64:26–35.
39. Dockery DW et al. Epidemiologic evidence of cardiovascular effects of particulate air pollution. *Environmental Health Perspectives*, 2001, 109:483–486.

40. Goldberg MS et al. Identification of persons with cardiorespiratory conditions who are at risk of dying from the acute effects of ambient air particles. *Environmental Health Perspectives*, 2001, 109:487–494.
41. Boezen HM et al. Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. *Lancet*, 1999, 353:874–878.
42. Brown JS et al. Ultrafine particle deposition and clearance in the healthy and obstructed lung. *American Journal of Respiratory and Critical Care Medicine*, 2002, 166:1240–1247.
43. Laden F et al. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environmental Health Perspectives*, 2000, 108:941–947.
44. Janssen NA et al. Air conditioning and source-specific particles as modifiers of the effect of PM(10) on hospital admissions for heart and lung disease. *Environmental Health Perspectives*, 2002, 110:43–49.
45. Cassee FR et al. Effects of diesel exhaust enriched concentrated PM<sub>2.5</sub> in ozone pre-exposed or monocrotaline-treated rats. *Inhalation Toxicology*, 2002, 14:721–743.
46. Donaldson K et al. The pulmonary toxicology of ultrafine particles. *Journal of Aerosol Medicine*, 2002, 15:213–220.
47. Schlesinger RB, Cassee F. Atmospheric secondary inorganic particulate matter: the toxicological perspective as a basis for health effects risk assessment. *Inhalation Toxicology*, 2003, 15:197–235.
48. Katsouyanni K et al. Short-term effects of ambient sulfur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. *British Medical Journal*, 1997, 314:1658–1663.
49. Krewski D et al. Overview of the reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of particulate air pollution and mortality. *Journal of Toxicology and Environmental Health*, 2003, 66:1507–1551.
50. Levy JJ et al. Estimating the mortality impacts of particulate matter: what can be learned from between study variability? *Environmental Health Perspectives*, 2000, 108:109–117.
51. Viegi G, Sandstrom T, eds. Air pollution effects in the elderly. *European Respiratory Journal*, 2003, 21(Suppl. 40).
52. Gouveia N, Fletcher T. Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. *Journal of Epidemiology and Community Health*, 2000, 54:750–755.
53. Roemer WH, van Wijnen JH. Daily mortality and air pollution along busy streets in Amsterdam, 1987–1998. *Epidemiology*, 2001, 12:649–653.
54. Venn A et al. Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study. *Occupational and Environmental Medicine*, 2000, 57:152–158.
55. Garshick E et al. Residence near a major road and respiratory symptoms in U.S. *Veterans Epidemiology*, 2003, 14:728–736.
56. Janssen N et al. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. *Environmental Health Perspectives*, 2003, 111:1512–1518.
57. Nicolai T et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *European Respiratory Journal*, 2003, 21:956–963.
58. Tao F, Gonzalez-Flecha B, Kobzik L. Reactive oxygen species in pulmonary inflammation by ambient particulates. *Free Radical Biology & Medicine*, 2003, 35:327–40.
59. Donaldson K et al. Oxidative stress and calcium signaling in the adverse effects of environmental particles (PM10). *Free Radical Biology & Medicine*, 2003, 34:1369–1382.
60. Holgate ST et al. Epithelial-mesenchymal interactions in the pathogenesis of asthma. *Journal of Allergy and Clinical Immunology*, 2000, 105:193–204.

61. Harkema JR et al. Effects of concentrated ambient particles on normal and hypersecretory airways in rats. *Research Report (Health Effects Institute)*, 2004, 120:1–68.
62. Last JA et al. Ovalbumin-induced airway inflammation and fibrosis in mice also exposed to ultrafine particles. *Inhalation Toxicology*, 2004, 16:93–102.
63. Steerenberg PA et al. Adjuvant activity of ambient particulate matter of different sites, sizes, and seasons in a respiratory allergy mouse model. *Toxicology and Applied Pharmacology*, 2004, 200:186–200.
64. Svartengren M et al. Short term exposure to air pollution in a road tunnel enhances the asthmatic response to allergen. *European Respiratory Journal*, 2000, 15:716–724.
65. Sorensen M et al. Linking exposure to environmental pollutants with biological effects. *Mutation Research*, 2003, 544:255–271.
66. Gabelova A et al. Genotoxicity of environmental air pollution in three European cities: Prague, Kosice and Sofia. *Mutation Research*, 2004, 563:49–59.
67. Hartwig A. Role of DNA repair in particle- and fiber-induced lung injury. *Inhalation Toxicology*, 2002, 14:91–100.
68. Pavanello S, Clonfero E. Biological indicators of genotoxic risk and metabolic polymorphisms. *Mutation Research*, 2000, 463:285–308.



## 4. Sources of PM

### KEY MESSAGES

- Mobile sources, industry (including energy production) and domestic combustion contributed 25–34% each to primary PM<sub>2.5</sub> emissions in 2000. These sectors are also important emitters of the precursor gases sulfur dioxide, nitrogen oxides and volatile organic compounds (VOC), while agriculture is a dominant contributor of ammonia.
- Anthropogenic emissions of PM<sub>2.5</sub> and PM<sub>10</sub> across Europe generally fell by about a half between 1990 and 2000. During this period, the relative contribution from transport increased compared to industrial emissions. The emission of precursor gases fell by 20–80% between 1980 and 2000.
- Projections by the Regional Air Pollution Information and Simulation (RAINS) model suggest that, owing to the existing legislation, further reductions in emissions of primary PM and precursor gases of the same magnitude will continue in the European Union (EU). In addition to the transport sector, the domestic sector will become an increasingly important source of primary PM emissions in the future. Furthermore, in contrast to all other sources of primary PM and precursor gases, international shipping emissions are predicted to increase in the next 20 years.
- The expected reduction of primary PM emissions in non-EU countries covered by EMEP is markedly smaller than the reduction expected in the EU.

Small particles in ambient air originate from a wide range of sources. It is useful to distinguish particles that are directly emitted (primary particles) and those (secondary particles) that are formed in the atmosphere from gaseous precursors. Both primary and secondary particles originate from natural sources and from human activities. Natural sources are either biogenic (such as pollen and parts of plants and animals) or geogenic (such as soil dust and sea salt).

**Table 4.1. Precursors of secondary aerosols and their PM component**

Precursor	PM component
Sulfur dioxide	Sulfate
Nitrogen oxides	Nitrate
Ammonia	Ammonium
Anthropogenic VOC	Various compounds of organic carbon
Biogenic VOC	Various compounds of organic carbon

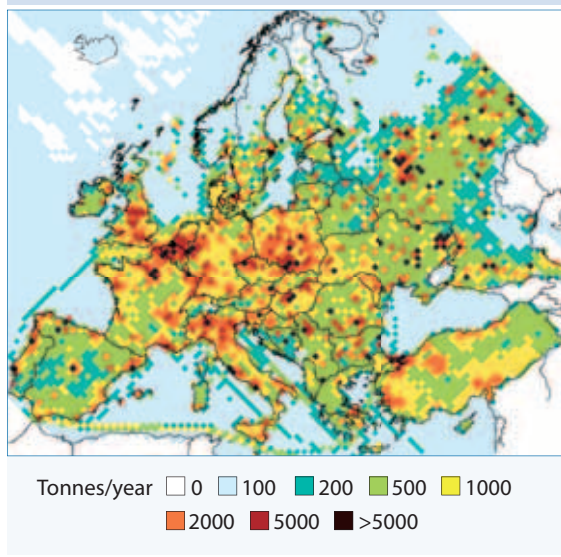
Anthropogenic sources of primary particles include fuel combustion, handling of different materials during industrial production, mechanical abrasion of various surfaces (e.g. road, tyre and brake wear) and agricultural activities. Most of the traditional air pollutant gases such as sulfur dioxide, nitrogen oxides, ammonia and VOC act as precursors to the formation of secondary particles (aerosols) in the atmosphere (Table 4.1).

The following section summarizes our current understanding of the emission of primary particles and precursor gases from anthropogenic sources. These are caused by human action and can be influenced by targeted measures.

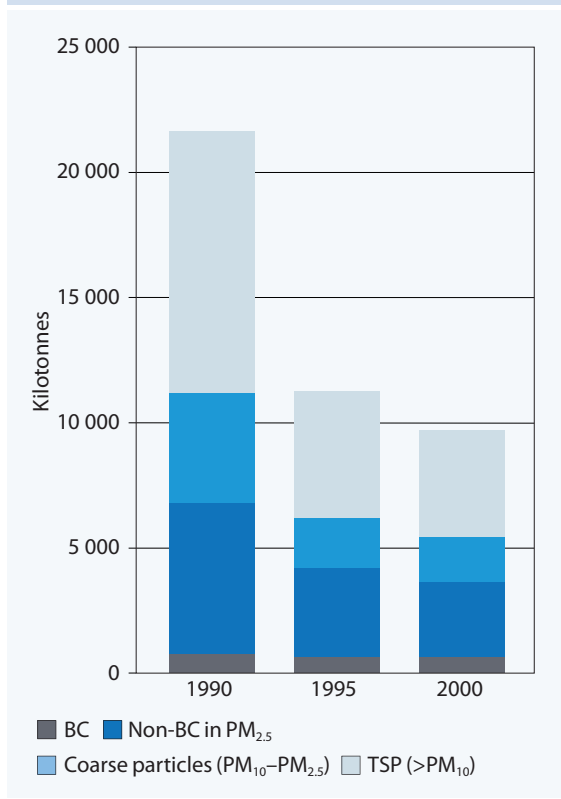
### 4.1 Primary emissions

There is as yet only incomplete quantitative information available about the sources of particle emission in the various European countries. By 2003, only 19 of the 48 Parties to the Convention had submitted some PM emission data to UNECE. Since these submissions do not allow one to draw a consistent and qual-

**Fig. 4.1. Identified anthropogenic contribution of the grid average  $PM_{2.5}$  emissions in Europe for 2000, including international shipping emissions**



**Fig. 4.2. Emissions of PM in the EMEP domain (all European countries up to the Ural Mountains)**



ity controlled European-wide picture, this chapter relies on the emission inventory developed with the RAINS model for all European countries under the CAFE programme (1).

The CAFE programme aims at a comprehensive assessment of the available measures for further improving European air quality, beyond the achievements expected from the full implementation of all current air quality legislation. The EU has established a comprehensive legislative framework that allows for economic development while moving towards sustainable air quality. A large number of directives specify minimum requirements for emission controls from specific sources. The CAFE baseline assessment quantifies for each Member State the impact of the legislation on future emissions. In this chapter we consider the penetration of emission control legislation in the various Member States that is of maximum technical feasibility in the coming years, thereby outlining information on future emissions of primary PM emissions up to 2020 compared to the current legislation baseline for the year 2000.

The RAINS model estimates emissions based on national data on sectoral economic activities and reviewed emission factors from the literature and from national sources. These estimates thus provide an internationally consistent overview of emissions of PM, although for individual countries they may deviate from national inventories to the extent they are available. RAINS distinguishes a range of different size classes and chemical fractions of PM, such as TSP,  $PM_{10}$ ,  $PM_{2.5}$  and BC. For example, Fig. 4.1 illustrates the emission density of  $PM_{2.5}$  across Europe for 2000 where similar distributions exist for the other air pollutants (i.e. nitrogen oxides and sulfur dioxide).

According to the RAINS estimates, the total volume of emission of primary  $PM_{10}$ ,  $PM_{2.5}$  and BS between 1990 and 2000 decreased by 51%, 46% and 16%, respectively (Table 4.2). Overall, the relative contribution of industry to  $PM_{10}$  and  $PM_{2.5}$  emissions decreased slightly and the contribution of transport increased (in particular for BS emissions). The decline in primary PM emissions is a result of the decrease in the consumption of solid fuels, especially following economic restructuring in central and eastern Europe, and of tightened emission control requirements for stationary and mobile sources

**Table 4.2. RAINS estimates of PM emissions from all land-based sources in the EMEP domain in 1990 and 2000: percentage contribution by various economic sectors**

Economic sector	PM <sub>10</sub>		PM <sub>2.5</sub>		BS	
	1990	2000	1990	2000	1990	2000
Energy industries	16%	13%	11%	9%	1%	1%
Manufacturing industries and construction	15%	9%	13%	10%	3%	4%
Transport	6%	11%	9%	13%	31%	37%
Other sectors	33%	34%	36%	38%	46%	43%
Fugitive emissions from fuels	5%	5%	4%	3%	12%	7%
Mineral products	1%	3%	0%	0%	0%	0%
Chemical industry	4%	2%	4%	2%	0%	0%
Metal production	14%	11%	17%	14%	0%	0%
Other production	1%	3%	1%	1%	0%	0%
Manure management	2%	5%	1%	1%	0%	0%
Agricultural soils	0%	1%	0%	0%	0%	0%
Agriculture	0%	3%	2%	4%	5%	5%
Waste incineration	2%	1%	0%	1%	1%	1%
Other	0%	2%	1%	3%	1%	1%
Total	100%	100%	100%	100%	100%	100%
Total kilotonnes	11 195	5 442	6 814	3 651	759	639

(Fig. 4.2). This decline is especially large for TSP and the coarse fraction of particles (larger than 2.5 µm), owing to the decline in coal consumption by homes and small industry in central and eastern Europe. The change in PM<sub>2.5</sub> and, most notably, in BC emissions is significantly smaller.

To put shipping emissions of PM into perspective, the RAINS baseline emissions for the EU (25 countries) in the year 2000 for land-based shipping sources (included in the more general transport sector within RAINS, see Table 4.2) accounted for only 16.9 and

16.0 kilotonnes of primary PM<sub>10</sub> and PM<sub>2.5</sub>, respectively. On the other hand, the primary emissions due to international shipping in Europe are about one order of magnitude higher (Table 4.3). Nevertheless, the primary emissions of nitrogen oxides and sulfur dioxide associated with shipping, and the potential formation of secondary inorganic aerosols from this source, make this an important transboundary issue (see section 4.3).

For the most recent inventory for the year 2000 in Europe, the major share of TSP emissions is estimat-

**Table 4.3. RAINS estimates (by sea regions within the EMEP area) of emissions of PM, nitrogen oxides and sulfur dioxide from international shipping (not including land-based shipping sources) in 1990 and 2000, in kilotonnes**

Region	PM <sub>10</sub>		PM <sub>2.5</sub>		Nitrogen oxides		Sulfur dioxide	
	1990	2000	1990	2000	1990	2000	1990	2000
Atlantic Ocean	28	36	27	34	444	566	307	396
Baltic Sea	17	22	16	21	273	249	188	242
Black Sea	6	8	6	7	93	118	65	83
Mediterranean Sea	88	114	83	108	1 415	1 808	958	1 237
North Sea	33	42	31	40	518	659	357	460
Total	171	222	162	210	2 743	3 501	1 874	2 418



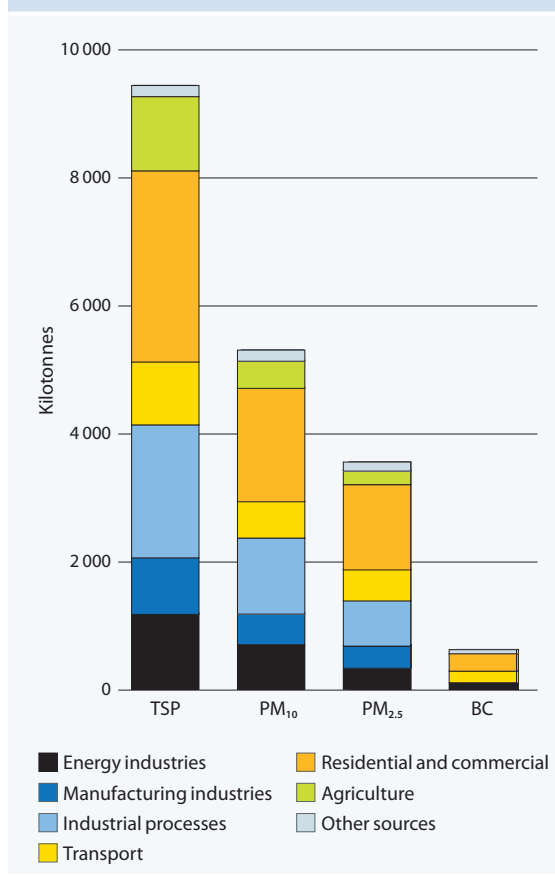
ed to originate from the combustion of solid fuels in small stoves in the residential and commercial sectors, followed by industrial emissions from energy combustion and manufacturing processes and from agricultural activities (Fig. 4.3). Since a significant share of primary particles from industrial and agricultural sources is typically larger than 10  $\mu\text{m}$ , these sources make a smaller contribution to  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ . The same applies to non-exhaust emissions from traffic sources (road, tyre and brake wear), which are a minor source for particles smaller than 2.5  $\mu\text{m}$ . BC emissions originate predominantly from combustion processes in the transport sector (diesel vehicles) and small domestic stoves.

For the year 2000, the RAINS estimates (excluding international shipping) suggest that in the EU (15 countries) about one third of the primary  $\text{PM}_{10}$  emissions (637 kilotonnes) originated from industrial processes and other non-combustion sources (e.g. in agriculture). The transport sector contributes another 521 kilotonnes (including non-exhaust emissions), while combustion in the domestic sector (mainly wood fuel use in small stoves) is calculated to emit 360 kilotonnes. In the new EU Member States, the largest share of primary  $\text{PM}_{10}$  emissions was caused by the combustion of coal, mainly in the domestic sector.

## 4.2 Projections of primary PM emissions

The change in emissions of PM will continue in the future. Economic activities are undergoing constant change, and a wealth of emission control legislation has been adopted in Europe that will further contribute to a decline in particle emissions. In the EU with its new Member States, full implementation of the large combustion plants directive (2001/80/EC), of the Euro IV and Euro V emission control standards for mobile sources, of legislation on non-road mobile machinery and of the provisions of the integrated pollution prevention and control directive (96/61/EC) will affect emissions of fine PM. With all these measures, primary  $\text{PM}_{10}$  emissions from stationary combustion of fossil fuels are expected to decline significantly in the coming years. Emissions from mobile sources (including non-exhaust emissions) show a declining trend too, but less steep than that for stationary sources.

**Fig. 4.3. Contribution to primary PM emissions from different anthropogenic source categories in 2000 (EMEP domain)**



Emissions by sector for  $\text{PM}_{2.5}$  and  $\text{PM}_{10}$  in the EU are presented in Fig. 4.4 and 4.5, respectively. Sector contributions to primary  $\text{PM}_{2.5}$  emissions in the EU (15 countries) for 2000 and projections for the year 2020 are illustrated in Fig. 4.6 and 4.7, respectively. Overall, the CAFE baseline scenario estimated that  $\text{PM}_{10}$  emissions will decrease between 2000 and 2010 by approximately 24% in the EU (15 countries) and by more than 40% in the new Member States. For 2020, total primary  $\text{PM}_{10}$  emissions would be 34% and 55% lower, respectively.

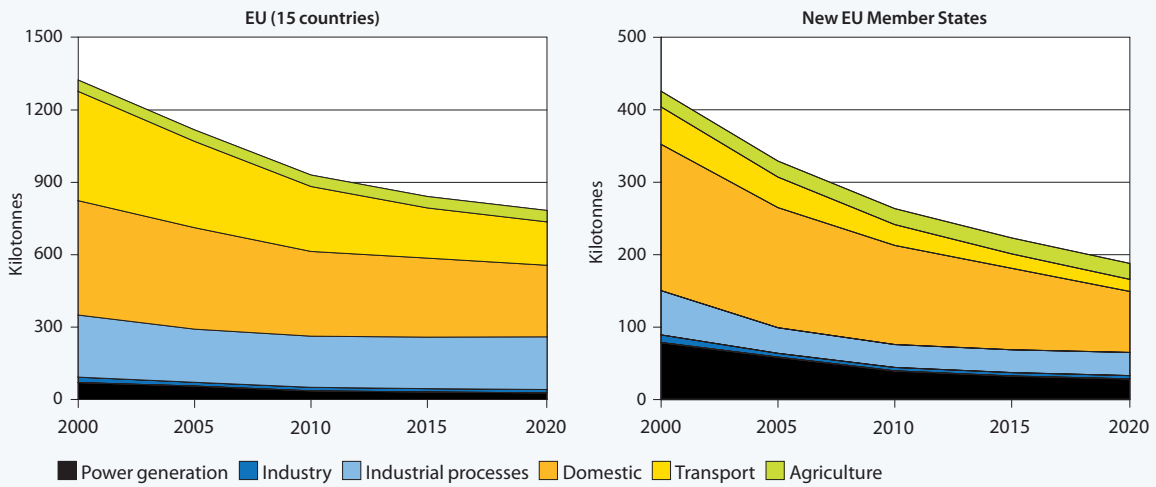
Progress in the implementation of emission control technologies and continuing changes in the composition of emission source categories will alter the contributions of the various emission source sectors to total  $\text{PM}_{2.5}$  emissions (Fig. 4.6 and 4.7). Overall, the share of mobile sources will decline from a third

to slightly more than a fifth. Implementation of the Euro V standard for heavy-duty diesel vehicles will reduce the contribution of exhaust emissions from this category from 7% in 2000 to 1% in 2020. The share of exhaust emissions from diesel passenger cars is calculated to decline from 12% to 6% in 2020, while the contribution from off-road mobile sources will increase to 9%. Overall, the largest sources of

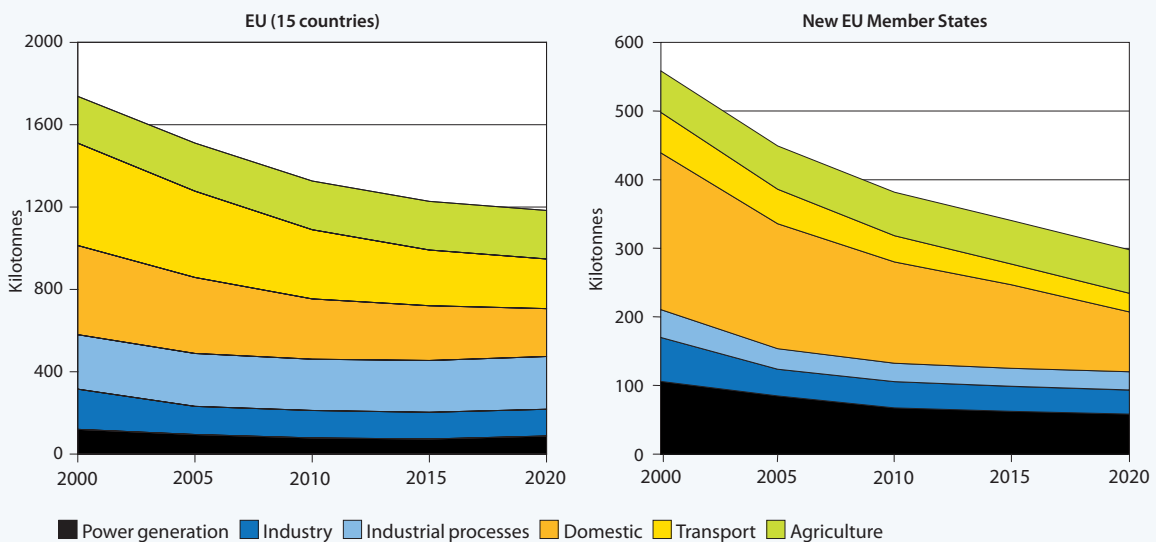
primary PM<sub>2.5</sub> emissions will be combustion of wood in domestic stoves (38%) and industrial processes (28%).

According to IIASA estimates, the expected changes in primary PM emissions due to currently adopted legislation will be much smaller in non-EU than in EU countries in the coming decades (2). On the other hand, the maximum technically feasible

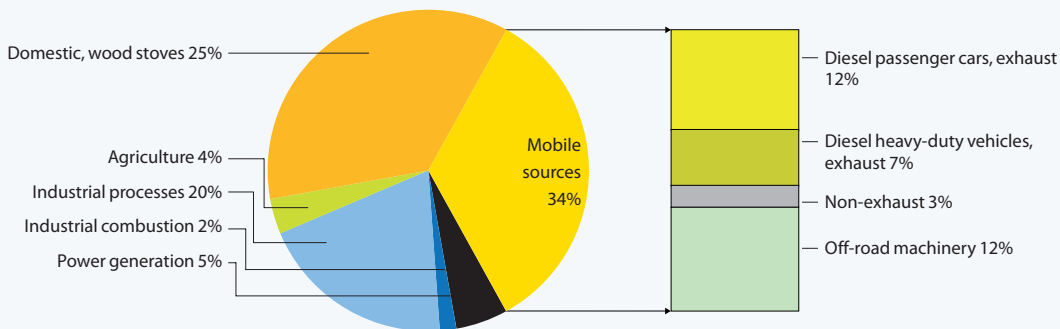
**Fig. 4.4. PM<sub>2.5</sub> emissions by sector for the CAFE baseline scenario**



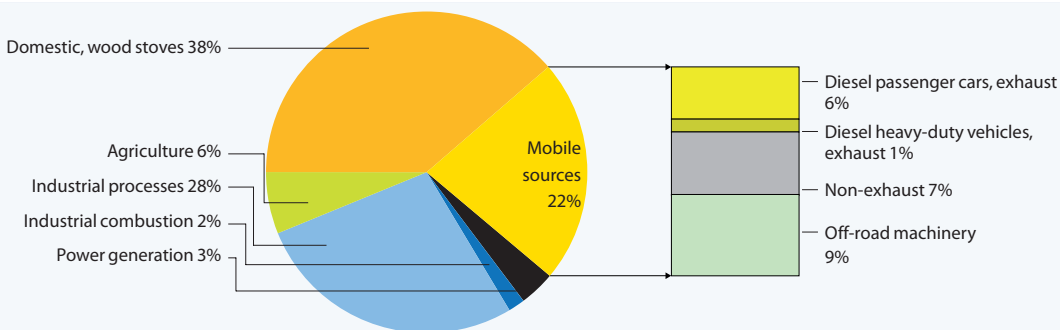
**Fig. 4.5. PM<sub>10</sub> emissions by sector for the CAFE baseline scenario**



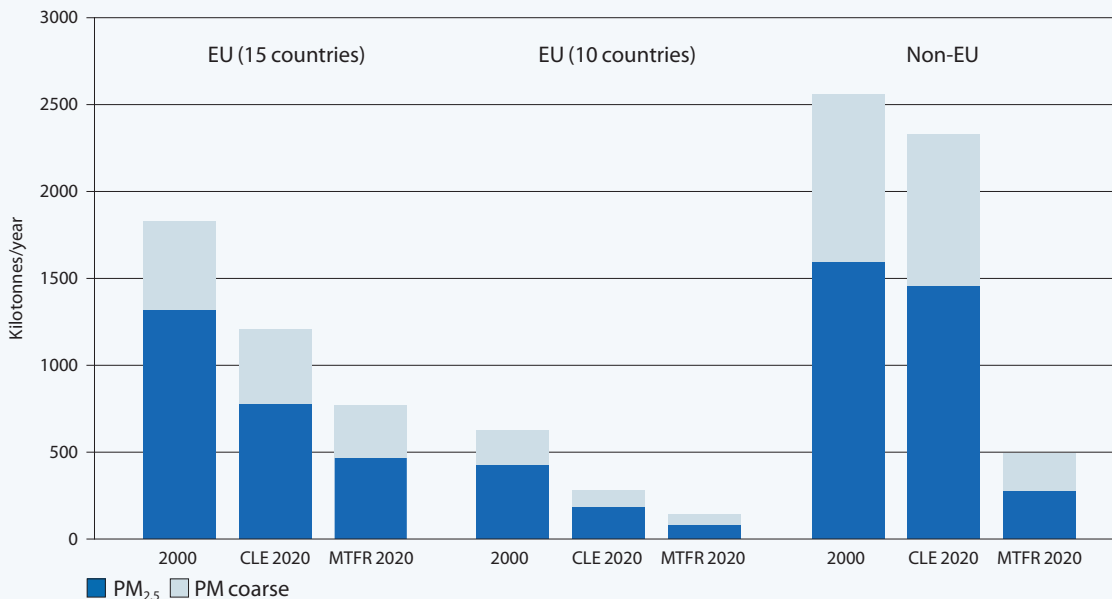
**Fig. 4.6. Sector contribution to primary PM<sub>2.5</sub> emissions in the EU (15 countries) in 2000**



**Fig. 4.7. Sector contribution to primary PM<sub>2.5</sub> emissions in the EU (15 countries) in 2020**



**Fig. 4.8. RAINS estimates of primary PM emissions from anthropogenic sources in various parts of the EMEP domain in 2000 and 2020 under current legislation (CLE) and maximum technically feasible reductions (MTR) scenarios**



Source: Amann et al. (2).

reductions in emissions in non-EU countries may cut them to about a fifth of the current levels (Fig. 4.8). The largest potential for feasible reductions exists in industrial and nonindustrial combustion facilities, in other production processes, in agriculture and in road transport.

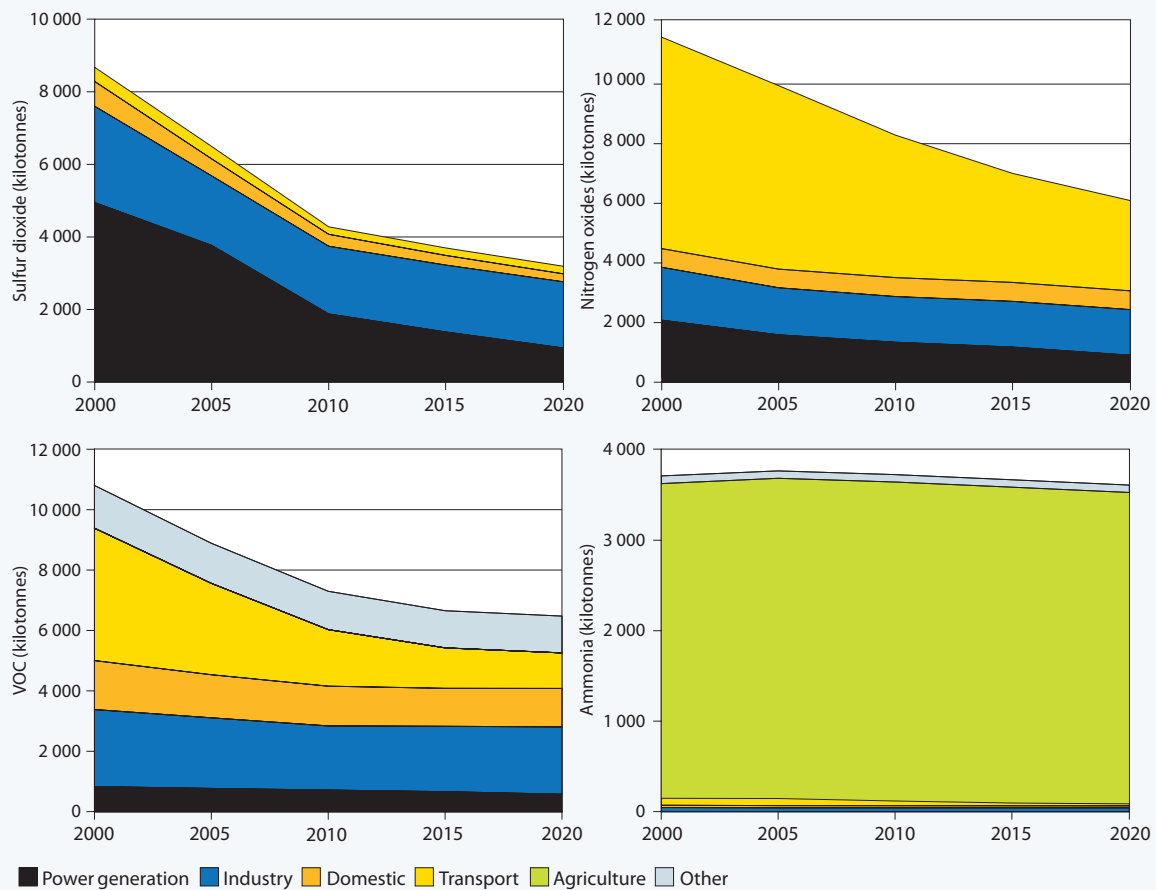
### 4.3 Emissions of PM precursors

All relevant precursor gases for atmospheric PM formation have been included in the emissions reporting of the Convention for many years. The decline in primary PM emissions will be accompanied by shrinking emissions of the precursors that contribute to the formation of secondary particles. For the EU (25 countries) it is estimated that, with full implementation of present emission control legislation, sulfur

dioxide emissions will decline by 70% between 2000 and 2020 and that emissions of nitrogen oxides and VOC will fall by approximately 55% over the same period. For ammonia emissions, however, only little change is anticipated (see Fig. 4.9). A clear exception is for international shipping, where precursor emissions of nitrogen oxides and sulfur dioxide are predicted to increase by 28% and 52%, respectively.

Historical trends for precursor emissions have been outlined in the most recent EMEP assessment report (3). The reduction in sulfur dioxide emissions has been significant in most countries in Europe, often more than 50% compared to 1980. Some countries have managed to reduce their emissions even further, by 80–90% between 1980 and 2000. In total, the reduction in sulfur dioxide emissions is 67%

Fig. 4.9. Projections of the PM precursor emissions for the CAFE baseline scenario of the EU (25 countries)



(shipping emissions excluded). Emissions of nitrogen oxides show a less pronounced downward trend over the same period; overall, the officially reported decrease was around 25% between 1990 and 2000. The reduction in ammonia emissions was similar to that of nitrogen oxides. The largest relative decrease for ammonia took place after 1990 in the eastern European countries, where emissions fell by nearly 50%, although in most areas the reduction has only been around 10%. VOC were not assessed as part of this report.

## References

1. Amann M et al. *Baseline scenarios for the Clean Air for Europe (CAFE) programme*. Laxenburg, International Institute for Applied Systems Analysis, 2005 ([http://europa.eu.int/comm/environment/air/cale/activities/pdf/cale\\_scenario\\_report\\_1.pdf](http://europa.eu.int/comm/environment/air/cale/activities/pdf/cale_scenario_report_1.pdf), accessed 17 November 2005).
2. Amann M et al. *Baseline emission projections and scope for further reductions in Europe up to 2020*. Laxenburg, International Institute for Applied Systems Analysis, 2005 (<http://www.iiasa.ac.at/rains/presentations/amann-TFIAM30-baseline.ppt>, accessed 17 November 2005).
3. Tarrasón L et al. *Transboundary acidification, eutrophication and ground level ozone in Europe in 2003*. Oslo, Norwegian Meteorological Institute, 2005 (EMEP Report 1/2005) ([http://www.emep.int/publ/common\\_publications.html](http://www.emep.int/publ/common_publications.html), accessed 17 November 2005).

## 5. PM levels

### KEY MESSAGES

- Data on PM<sub>10</sub> measured at 1 100 monitoring stations in 24 countries were available in the EEA's AirBase database for 2002. In some 550 urban areas included in this database, annual mean PM<sub>10</sub> was 26 µg/m<sup>3</sup> in urban background and 32 µg/m<sup>3</sup> in traffic. In rural areas, annual mean PM<sub>10</sub> amounted to 22 µg/m<sup>3</sup>.
- In more than 130 cities in 20 countries, the daily limit value set by Council Directive 1999/30/EC was exceeded. The annual limit value was exceeded in 37 cities from 8 countries in background locations and in 48 cities from 12 countries at traffic stations.
- PM<sub>10</sub> levels in Europe are dominated by the rural background component. In most areas, the rural concentration is at least 75% of the urban background concentration, and in some very densely populated areas, such as in the Netherlands, the rural concentration is more than 90% of the urban background.
- The available data allow assessment of European trends in PM concentrations only from 1997 onwards. From 1997 to 1999/2000 there was a downward trend in PM<sub>10</sub>, while values increased between 1999/2000 and 2002. This tendency was similar at the three types of monitoring station: rural, urban background and hot spot.
- The tendencies reported above do not follow the trends in emission: the reported emissions of precursor gases decreased and primary PM<sub>10</sub> emissions did not change significantly during this period in Europe. It is likely that inter-annual meteorological variations affected trends in PM concentrations.
- Analysis of data from the United Kingdom since 1990 shows a downward trend in PM<sub>10</sub> concentration, especially at the beginning of the 1990s. In this case, where quality controlled emission data are available and meteorological variability is taken into account, the decrease in emissions corresponds well with the observed concentration tendencies.
- PM<sub>2.5</sub> and smaller size fractions of PM are measured to a much smaller extent than PM<sub>10</sub> in Europe. Data from 119 PM<sub>2.5</sub> stations for 2001 indicate on average fairly uniform rural background concentrations of 11–13 µg/m<sup>3</sup>. The urban levels are considerably higher (15–20 µg/m<sup>3</sup> in urban background and typically 20–30 µg/m<sup>3</sup> at traffic sites). The PM<sub>2.5</sub> : PM<sub>10</sub> ratio for these stations was 0.65 (range 0.42–0.82).
- The unified EMEP model generally underestimates the observed regional background levels of PM<sub>10</sub> and PM<sub>2.5</sub> in Europe, a feature shared by other models. The underestimation is larger for PM<sub>10</sub> (–34%) than for PM<sub>2.5</sub> (–12%). Spatial correlations between model estimates and observed levels are currently high: 0.70 for PM<sub>10</sub> and 0.78 for PM<sub>2.5</sub>. However, these results are affected by the lack of monitoring data in large areas of Europe and mostly show the ability of the model to reproduce the north–south distribution gradients of rural background PM across Europe. Temporal correlations are lower for PM<sub>10</sub> (0.4–0.5 on average) than for PM<sub>2.5</sub> (0.5–0.6 on average), indicating that the sources and processes currently not described in the model are probably more important for the coarse fraction of PM.
- The contribution of *secondary inorganic aerosols* to PM mass varies considerably across sites but it represents between 20–30% of PM<sub>10</sub> mass and 30–40% of the PM<sub>2.5</sub> mass.

The EMEP model is able to reproduce well the spatial variability and the observed levels of secondary inorganic aerosols across Europe, with an average positive bias of 18% and spatial correlations of 0.87.

- The *carbonaceous (OC+EC) contribution* represents about 25–35% of the background  $PM_{2.5}$  mass. The largest discrepancies between modelled and observed PM components are found for organic aerosols.
  - The EMEP model underestimates measured background concentrations of EC by –37%. Spatial correlations are high (0.88) as are the temporal correlations (0.5–0.6). Therefore, the generalized model underestimation may indicate a systematic underestimation in the primary PM emissions.
  - The underestimation for OC is more than a factor of 3 (–84%) and the model has difficulties representing the temporal variability and seasonal variation of the measurements, especially during the summer. The underestimation seems also to be related to the fact that relevant sources of organic carbon are missing in the model

simulations. Except from the formation of secondary organic aerosols from anthropogenic VOC, these missing sources are of biogenic/natural origin.

- The *mineral contribution* to  $PM_{10}$  at regional sites is estimated to be around  $2 \mu\text{g}/\text{m}^3$  for all countries with the exception of Spain, where the mineral contribution is usually 2–3 times higher owing to Saharan dust intrusions. This represents about 10% of observed  $PM_{10}$  levels averaged over Europe. The introduction of wind-blown dust processes in the model calculations improves the performance of the model for both  $PM_{10}$  and  $PM_{2.5}$ .
- Calculations from the validated EMEP model show that the regional background concentrations of anthropogenic PM have a considerable transboundary contribution: about 60% on average over Europe for  $PM_{2.5}$  concentrations. For primary coarse PM concentrations, the transboundary contribution is calculated to be smaller though still significant, ranging from 20% to 30% in central Europe.

## 5.1 Introductory remarks

In the framework of an integrated assessment, ambient concentrations of a pollutant have to be calculated from emissions of this pollutant and its precursor substances. Thus pollution levels have to be calculated, starting from information on the main emission sources and taking account of dispersion and chemical reactions in the atmosphere. This is not an easy task, since PM in ambient air stems from a number of different sources; in addition, PM is not only emitted directly into the atmosphere by anthropogenic and nonanthropogenic sources (such as geogenic and biogenic sources) but can also be formed by atmospheric reactions from precursor substances. The most important precursors are sulfur dioxide, oxides of nitrogen, ammonia and non-methane VOC (again from anthropogenic and nonanthropogenic sources).

The section on modelled PM levels is complemented by sections on PM monitoring data, on

source apportionment and on the results of the CITY-DELTA project (1), which investigated the differences between rural and urban pollution concentrations.

The EMEP monitoring network and the EMEP model provide estimates of the regional contribution to ambient PM concentrations. In urban environments and other source regions, higher PM concentrations are usually observed than in rural regions. However, there are large differences in the spatial variability for different PM components and therefore in the contribution of LRTAP to those components.

The results that follow are primarily concerned with concentrations of two specific PM size fractions such as  $PM_{10}$  and  $PM_{2.5}$ , because this is the most common way of quantifying ambient air PM for regulatory purposes and has been also used for evaluating health effects.

## 5.2 PM monitoring and assessment of concentrations and trends

### PM monitoring programmes and methods

This section provides an overview of monitored PM concentrations in Europe, within the EMEP programme as well as data reported by countries to the European Commission and to the AirBase database of the EEA via the Exchange of Information (EoI) Decision structure (Council Decision 97/101/EC). Currently, within these two networks, PM<sub>10</sub> is routinely reported from more than 1000 sites in Europe. In addition, there are probably a number of national and local PM networks with more stations. The high number of sites reporting data is primarily a consequence of the requirements stipulated in the EU air quality directives, primarily Directive 1999/30/EC relating to limit values for sulfur dioxide, nitrogen dioxide and oxides of nitrogen, PM and lead in ambient air. The network for PM<sub>2.5</sub>, the main parameter to be used for risk assessment, is much less dense. Although the extent of PM<sub>2.5</sub> monitoring is increasing rapidly in Europe, PM<sub>2.5</sub> data are still reported to AirBase only from a few stations. As part of a position paper produced by the CAFE Working Group on Particulate Matter (3), PM<sub>2.5</sub> data were collected via a questionnaire from 119 stations with annual time series of data, some with data from both 2000 and 2001 and some with data from only one of these years. There is only a very limited number of sites concerned with other PM parameters such as number concentration and chemical composition. The data collection by the Working Group produced data from 15 stations with annual PM<sub>1</sub> time series and 21 stations with ultrafine particle annual time series (number of particles). Of these, 15 stations also had size distribution data for ultrafine particles.

PM concentrations at the various main types of station (rural, urban background and hot spot (mainly traffic)) are compared. It should be kept in mind that there are uncertainties related to the classification of stations. Stations may be classified differently in different countries, and it has also been found that some stations in AirBase may be misclassified. Siting criteria differ somewhat between EMEP and EoI-related stations. "Rural" EoI stations may in some cases not be as strictly rural as the EMEP stations.

The PM data reported to AirBase are, for almost all of the stations, restricted to mass concentration. Within EMEP, a number of different PM parameters are part of the monitoring strategy. The strategy differentiates three levels: Levels 1 and 2 are mandatory while Level 3 is voluntary. The parameters to be measured in the three levels are shown in Table 5.1. However, the monitoring strategy is far from fully implemented. Currently, there are about 20 sites fulfilling the requirements of Level 1 while Level 2 is fully implemented at only a few sites.

The measurement of PM concentrations in ambient air is not straightforward. There are a variety of techniques available to measure mass concentrations but, owing to the complex nature of PM, the method that is selected can significantly influence the result. There are problems of loss of PM matter and artefact formation for both of the main types of method used: (a) collection of matter on filters with subsequent weighing in the laboratory and (b) online measurement of mass on filters, using instruments with sensors based on various physical principles. The reference method of the European Committee for Standardization for PM<sub>10</sub> (4) is based on filtering with subsequent weighing of filters. Many automatic instruments incorporate a heated manifold, which causes inevitable losses of semi-volatile species and makes direct comparison with the European reference sampler difficult. Thus correction factors need to be applied; these vary between instruments and area, and range from close to 1.0 up to almost 1.5. The reference method has not yet been defined for PM<sub>2.5</sub>. The indication is that a correction fraction for PM<sub>2.5</sub>, using automatic monitors, might be even larger than those for PM<sub>10</sub>. The implication of this is that dedicated procedures of measurements and quality assurance are necessary to produce reliable data for PM mass.

About half of the time series reported to AirBase are measured with non-reference automatic monitors, requiring that correction factors be determined and applied to the data.

### PM limit and target values

As a reference for the assessment of PM<sub>10</sub> levels in Europe, the following sections of this chapter use the limit values for PM concentrations introduced by



**Table 5.1. EMEP monitoring programme for PM**

Programme	Parameter	Resolution	Method	Remarks
<b>Level 1</b>				
Inorganic compounds in air	SO <sub>2</sub> , SO <sub>4</sub> <sup>-</sup> , NO <sub>3</sub> <sup>-</sup> , HNO <sub>3</sub> , NH <sub>4</sub> <sup>+</sup> , NH <sub>3</sub> , (sNO <sub>3</sub> , sNH <sub>4</sub> ), HCl, Na <sup>+</sup> , K <sup>+</sup> , Ca <sup>++</sup> , Mg <sup>++</sup>	Daily	Filter pack	Needs to be complemented with low-cost denuders
PM mass in air	PM <sub>2.5</sub> , PM <sub>10</sub>	Hourly/daily	Low-volume sampler, high-volume sampler or equivalent	Monitors can be used where equivalence can be demonstrated
Gas-particle ratios	NH <sub>3</sub> , NH <sub>4</sub> , HCl, HNO <sub>3</sub> , NO <sub>3</sub> (in combination with filter pack sampling)	Monthly	Low-cost denuder	Low-cost alternative to basic PM speciation that provides necessary gas-particle ratios for Level 1 sites
<b>Level 2</b>				
Major inorganics in both PM <sub>2.5</sub> and PM <sub>10</sub>	SO <sub>4</sub> <sup>-</sup> , NO <sub>3</sub> <sup>-</sup> , NH <sub>4</sub> <sup>+</sup> , Na <sup>+</sup> , K <sup>+</sup> , Ca <sup>++</sup> , Mg <sup>+</sup> (Cl <sup>-</sup> )	Hourly/daily	Manual denuders or continuous monitors	Continuous denuder/steam-jet and other instruments may also be used
Mineral dust in PM <sub>10</sub>	Si	Daily/weekly	Chemical (XRF, INAA, PIXE)	Reference methodology is under development
Elemental carbon (EC) Organic carbon (OC)	EC, OC	Daily/weekly	Thermo-optical	
<b>Level 3</b>				
OC speciation	Both water-soluble and water-insoluble OC	Hourly/daily	—	Contributes to EMEP programme for PM evaluation of effects on health and analysis of synergies with global change
Black carbon	BC	Hourly/daily	—	
Size/number distribution	Particle number distribution (dN/dlogDp)	Hourly/daily	—	
Light scattering	Aerosol optical depth	Hourly/daily	—	

Council Directive 1999/30/EC, which entered into force on 1 January 2005 (Table 5.2).

### PM measurements at rural background stations

In 2002, measurements of PM<sub>10</sub> were performed at 36 EMEP sites; at 19 of these PM<sub>2.5</sub> was also measured, mostly as daily samples. The location of sites, as well as detailed statistics and metadata, can be found at the EMEP web site ([www.emep.int](http://www.emep.int)). The regional distribution of PM<sub>10</sub> and PM<sub>2.5</sub> in Europe, as assessed by the EMEP network, is shown in Fig. 5.1. The EMEP PM<sub>10</sub> reference method follows the European Committee for Standardization standard (4) and most of the measurements are in accordance with this.

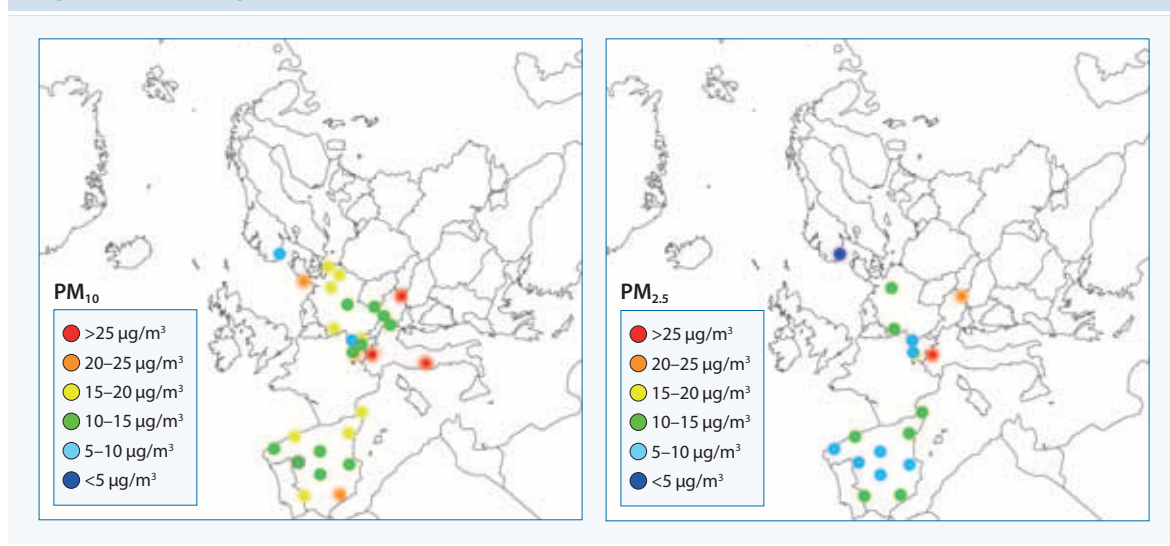
A comparatively large concentration of fine particles in the PM<sub>10</sub> mass occurred at several sites. None of

the EMEP sites exceeded the annual limit value set by the EU in Council Directive 1999/30/EC. The highest concentration is seen in Italy (two sites) with an annual average of around 35 µg/m<sup>3</sup>. The corresponding 24-hour limit was slightly exceeded in northern Italy at the Ispra IT04 site (37 higher values in 2002). For PM<sub>2.5</sub> the annual arithmetic average from the Illmitz AT02 site, at 23.3 µg/m<sup>3</sup>, was higher than the annual limit value in the United States standard (15 µg/m<sup>3</sup> for annual arithmetic mean). The 98th percentile of the 24-hour PM<sub>2.5</sub> concentration from this site in 2002 was also slightly higher than the United States standard.

AirBase contains for 2002 data from 154 PM<sub>10</sub> monitoring stations classified as rural stations. (For metadata and statistics, see <http://air-climate.eionet.eu.int/databases/airbase/index.html>.) Fig. 5.2 gives

**Table 5.2. EU air pollution limit values for PM<sub>10</sub> for health protection**

Compound	Limit/target value	Value (µg/m <sup>3</sup> )		
PM <sub>10</sub> stage 1	Annual average	40	—	2005
	Daily average	50	May be exceeded up to 35 days a year	2005
PM <sub>10</sub> stage 2	Annual average	20	Indicative; may be exceeded up to 7 days a year	2010
	Daily average	50	Indicative; may be exceeded up to 7 days a year	2010

**Fig. 5.1. Annual average concentration of PM<sub>10</sub> and PM<sub>2.5</sub> in 2002 at EMEP sites**

an overview of the measured annual average concentrations. Many of these stations are EMEP stations, and thus also found in Fig. 5.1. Note that the spatial coverage of these rural background stations is at present limited mostly to Spain and central European countries. There are few data in Scandinavia, eastern Europe and most Mediterranean countries.

The AirBase data show larger areas with an annual average PM<sub>10</sub> concentration above the stage 2 indicative 2010 limit value of 20 µg/m<sup>3</sup>. An area in Silesia even has concentrations above the 2005 limit value.

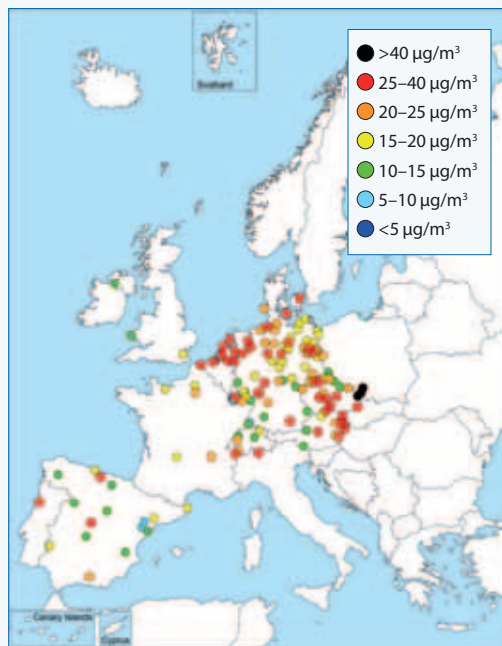
### Urban and rural PM<sub>10</sub> measurements reported to AirBase

AirBase contained, for 2002, 1306 PM<sub>10</sub> monitoring stations in 24 countries, and included stations in some 550 cities and towns as well as 154 rural stations. The amount of PM<sub>10</sub> data in AirBase is substantial only from 1997 onwards. The new EU Member States are less well represented than the original 15 (111 of the

stations are in the new Member States and of these 53 are in the Czech Republic).

As a basis for evaluating the population exposure situation for PM<sub>10</sub>, it is useful to look at both urban background and rural areas, as well as hot spot areas (traffic and industrial stations). The data for 2002 presented in Fig. 5.3 show that the annual average PM<sub>10</sub> level in urban areas varies across Europe. It is usually above 20 µg/m<sup>3</sup> and sometimes even above 30 µg/m<sup>3</sup> in German cities. In France and the United Kingdom it seems to be somewhat lower, while it is higher in Belgium Greece, the Netherlands, Spain and eastern Europe. Traffic and industrial stations have higher PM<sub>10</sub> levels. The annual average limit PM<sub>10</sub> value for the EU (40 µg/m<sup>3</sup>) was exceeded (red and black dots) at some urban background stations in Belgium, Italy, Spain and eastern Europe and at traffic hot spot stations in several more countries. Regarding high daily concentrations, the indicator of the EU limit value (the 36th highest daily value in a

**Fig. 5.2. Annual average concentration of PM<sub>10</sub> in 2002 at rural stations in AirBase**

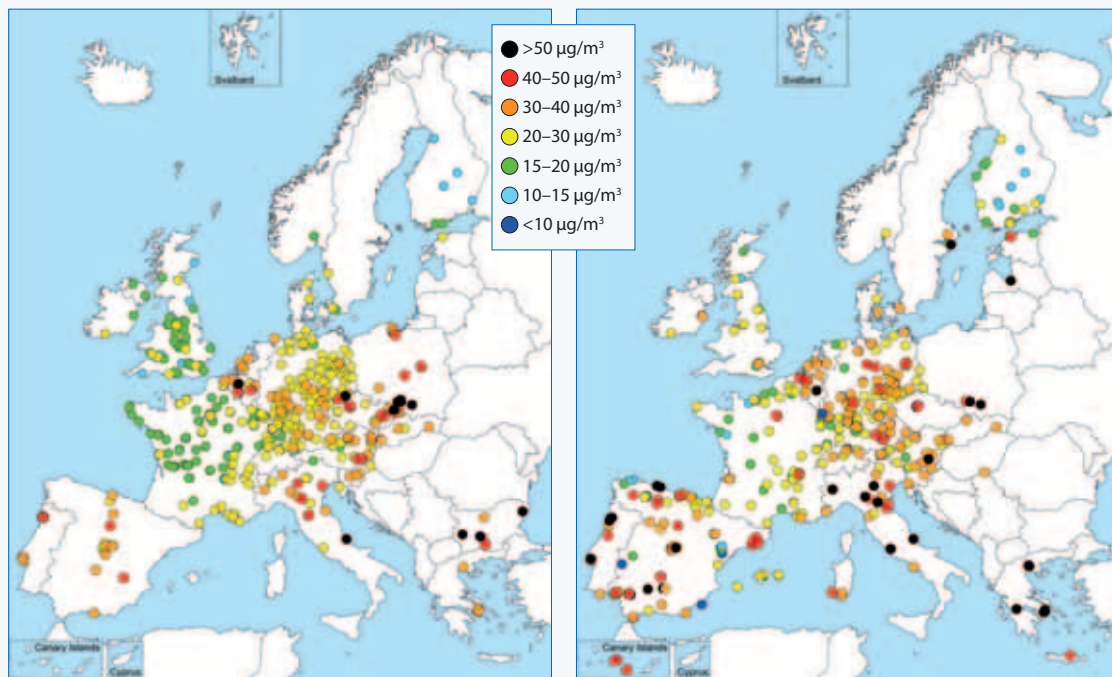


year) was above 50 µg/m<sup>3</sup> at a large number of urban stations and also at some rural background stations. As a further basis for estimating population exposure in Europe, Fig. 5.4 gives average PM<sub>10</sub> concentrations (annual average and the 36th highest daily concentration) at urban background and traffic stations. The average concentrations at the sites where the concentration in 2002 exceeded the limit values are up to 27% higher than their respective limit value. Maximum concentrations can reach over three times the limit value.

Fig. 5.5 shows annual average PM<sub>10</sub> concentrations over the period 1997–2002, averaged over station types. The figure represents only the relatively few stations that have data for all the years in the period, i.e. 137 stations in 9 countries (Belgium, the Czech Republic, Finland, the Netherlands, Poland, Portugal, Spain, Switzerland and the United Kingdom). For these stations, monitoring method correction factors have been applied consistently for all years.

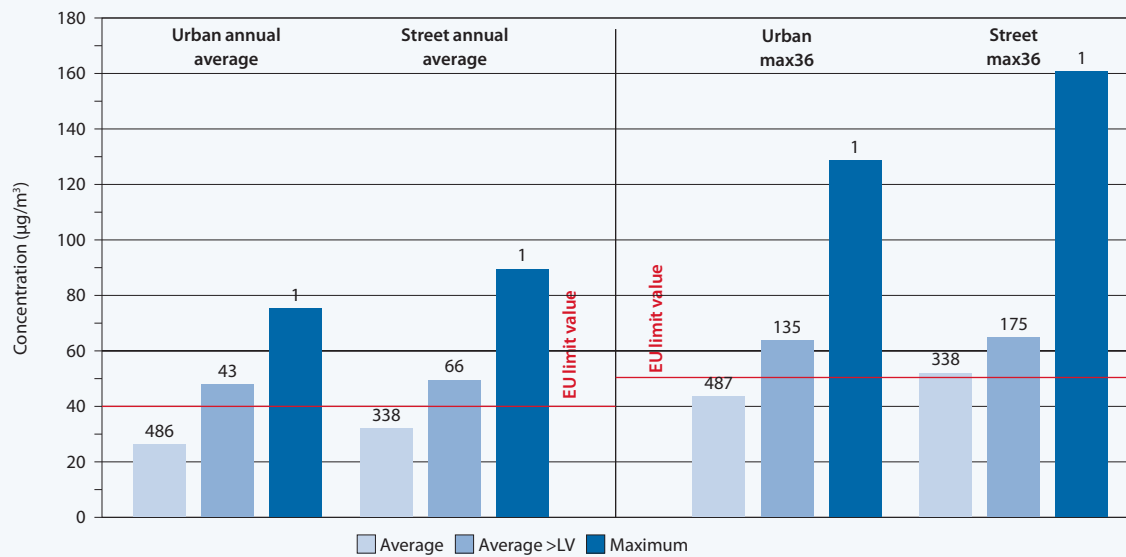
These data show a downward trend in PM<sub>10</sub> concentrations from 1997 to 1999, followed by an upward

**Fig. 5.3. Annual average PM<sub>10</sub> in urban background (left) and urban hot spot (right) stations in 2002**



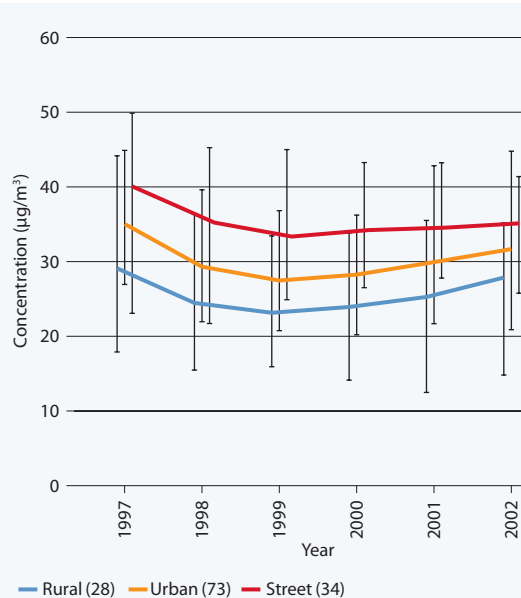
Note: The coding corresponds to the highest average obtained in all relevant stations in the city.

**Fig. 5.4. Average concentrations of PM<sub>10</sub> at European monitoring stations, 2002**



Note: The Average bar represents all stations in a class; the Average >LV bar represents the stations that are above the EU limit value; Maximum represents the station with the highest concentration (numbers of stations are shown on top of bars).

**Fig. 5.5. Variation of annual mean PM<sub>10</sub> from 1997 to 2002 in stations with data from all six years**



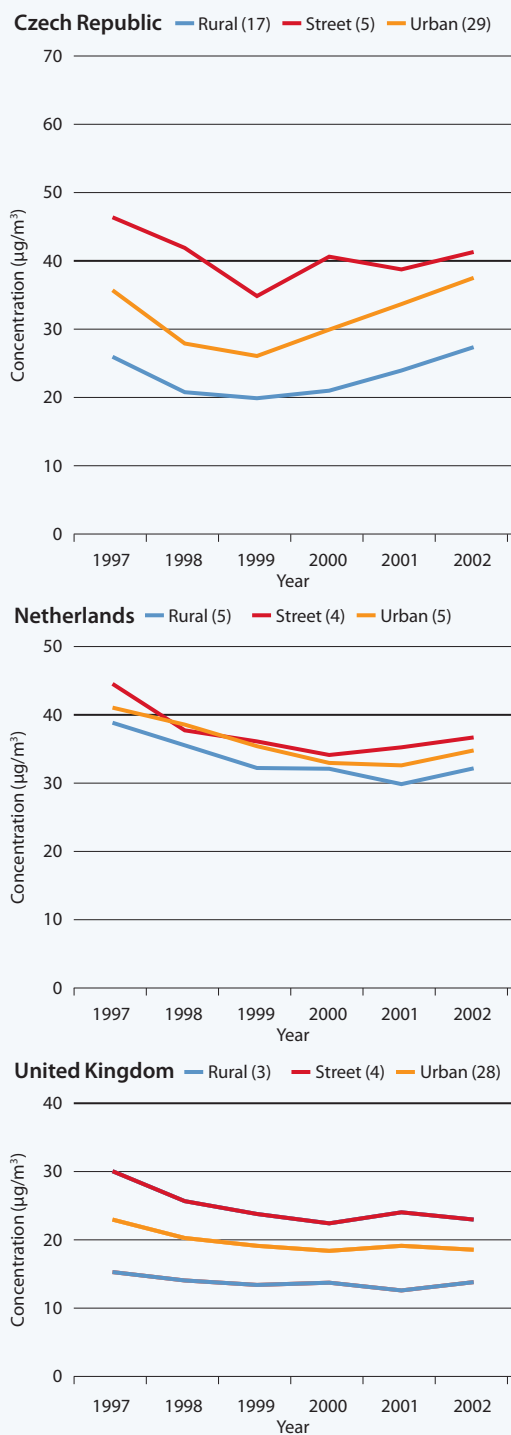
Note: Rural: 28 stations in the Czech Republic (17), the Netherlands (7), Switzerland (1) and the United Kingdom (3); Urban: 73 stations in Belgium (3), the Czech Republic (29), the Netherlands (5), Poland (3), Spain (1), Switzerland (4) and the United Kingdom (28); Street: 34 stations in Belgium (4), the Czech Republic (5), Finland (2), the Netherlands (5), Poland (1), Portugal (1), Spain (10), Switzerland (2) and the United Kingdom (4). Vertical bars show the 10th and 90th percentiles. Many of the rural background stations represented are EMEP stations.

trend towards 2002<sup>1</sup> (Fig. 5.5). For 2003 for the same set of stations, the concentrations were higher than for 2002, at about the same level as for 1997. Preliminary data for 2004 indicate a rather significant decrease from 2003. This development needs to be analysed together with data on emissions and meteorological conditions. Longer time series, such as that from the United Kingdom described below (page 43), show that PM<sub>10</sub> concentrations there have been falling since the early 1990s, and probably also before that time.

An attempt has been made to study whether there are regional differences in PM<sub>10</sub> trends in Europe (5). Owing to the low number of stations with long time series of various classes in each country, definitive conclusions cannot be drawn. The results indicate, however, that the decreasing trend in PM<sub>10</sub> (annual average) from 1997 to 1999 and the increase after 1999 or 2000 showed up in most countries. Notable exceptions are Spain and the United Kingdom, where reported concentrations have stayed almost constant since 2000.

<sup>1</sup> The trend is based only on data from stations where the use of correction factors is known for all years with data, and thus the trend shown is not influenced by inconsistent use of correction factors.

**Fig. 5.6. PM<sub>10</sub> trends and difference between station classes for the Czech Republic, the Netherlands and the United Kingdom, 1997–2002**



Note: The number of stations is given in parentheses.

Fig. 5.5 shows clearly that the PM<sub>10</sub> concentrations in Europe are dominated by the rural background. The averages for the three types of station are not directly comparable, since they do not necessarily represent stations from the same areas. Nevertheless, on average, the additional contribution in urban areas is relatively small compared to the rural level. The traffic in the streets gives an additional contribution, which is also limited. The vertical bars show the large variability within each station class. Some rural stations have higher PM<sub>10</sub> concentrations than many urban and even traffic stations.

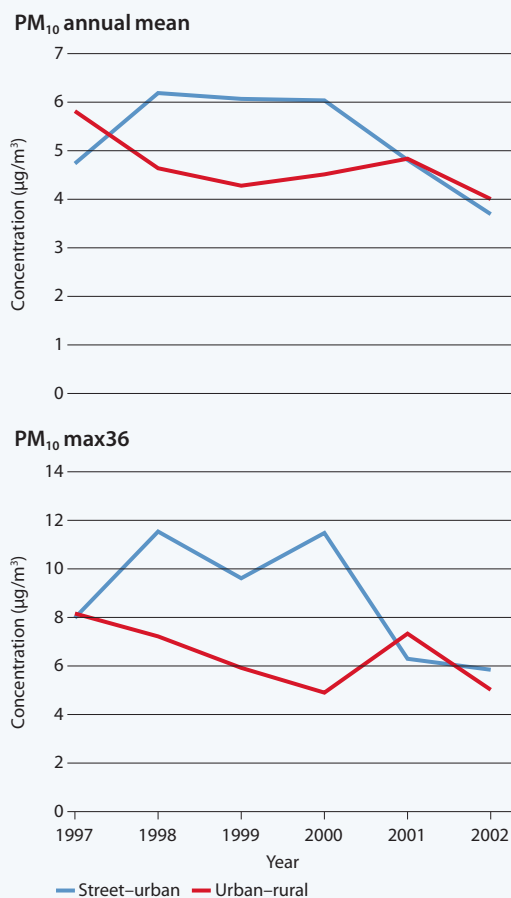
An analysis was carried out of PM<sub>10</sub> data in Air-Base for 2002, from 16 station pairs (pairs of traffic and urban background stations in individual cities), where the distance between the stations was less than 1 km. It showed that the PM<sub>10</sub> concentration (annual average) at the traffic stations was on average 6.9 µg/m<sup>3</sup> (25%) higher than at the urban stations of these pairs, with a standard deviation of ±20%. The corresponding increase for the 36th highest day concentration was 11.7 µg/m<sup>3</sup> (26%). This indicates that the added concentrations in streets, compared to the urban background, for well defined station pairs are in general larger than indicated by the lines in Fig. 5.5, which do not in general represent station pairs.

The absolute and relative additions to PM<sub>10</sub> from rural-to-urban and urban-to-street locations vary greatly, however, between countries and regions. Fig. 5.6 shows three examples, for countries with several stations in each class: the Czech Republic, the Netherlands and the United Kingdom. The very small additions from rural-to-urban and urban-to-street in the densely populated Netherlands are apparent, while they are larger (more typical?) for the Czech Republic and the United Kingdom.

Part of the street contribution to PM comes from the suspension of road dust due to turbulence from the traffic, as well as from tyre and brake wear. Attempts have been made to quantify this contribution, for example as summarized in CAFE's second position paper on particulate matter (3). It was concluded here that the available studies leave a large uncertainty in the estimate of the strength of this source. Relative to the strength of the exhaust particle source, this source is somewhere between 0.5 and 10 for PM<sub>10</sub> on an annual basis. It is much larger in the

North, where studded tyres are used, than in other areas. A recent basis for a closer estimate has been developed in the Street Emissions Ceiling project of the EEA's European Topic Centre on Air and Climate Change (6). Analysis of measurements at four good station pairs (urban-street) in cities in Europe indicate that the strength of the road/tyre/brake source of  $PM_{10}$  is closer to the lower part of the ratio above, around 1–2 times the exhaust particle source, on an annual basis. In areas with studded tyres it is estimated to be 3–4 times higher. There is of course still a considerable uncertainty in these estimates. Thus, the road source of  $PM_{10}$ , which is predominantly in the coarse fraction, has a strength that is probably larger than the exhaust particle source, which is mainly in the fine fraction.

**Fig. 5.7. Differences in  $PM_{10}$  between station classes, 1997–2002**



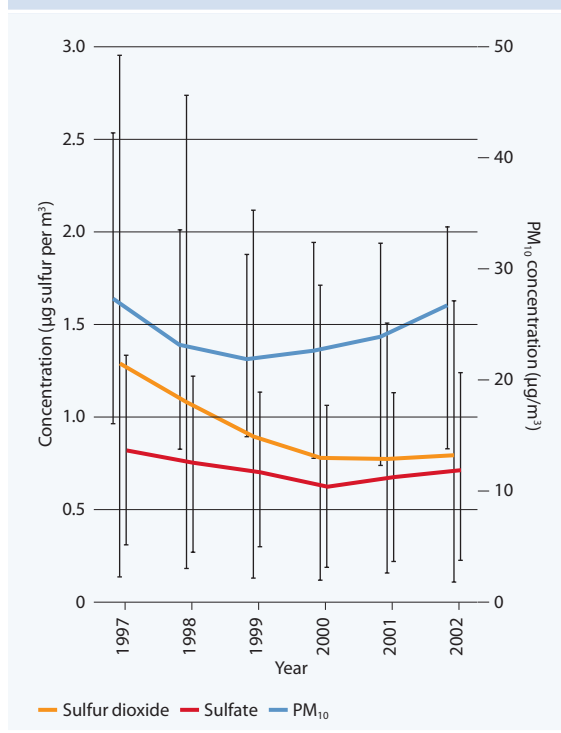
The road/tyre/brake source also contributes to  $PM_{2.5}$ . In Stockholm, where there is extensive use of studded tyres, the road dust contribution to  $PM_{2.5}$  in winter was estimated at 10% of the contribution to  $PM_{10}$  (7). The *Compilation of air pollutant emission factors* of the US Environmental Protection Agency gives a factor of 0.25 between these (8).

The rural background is the result of natural and primary PM sources as well as formation of secondary particles (mainly inorganic particles formed from sulfur dioxide and nitrogen compounds, while secondary organic particles also contribute). The absolute changes between years in  $PM_{10}$  concentrations are, for the 137 stations in 9 countries, rather similar for rural and urban stations.  $PM_{10}$  trends in urban areas compared to rural areas are better shown in Fig. 5.7. It points to little difference in the urban and rural trends, although there is a slight downward tendency at urban background stations compared to rural. For traffic hot spot stations, however, there has been a more pronounced reduction compared to rural (and urban) stations since 2000. This could reflect reduced traffic volumes or changed vehicle composition in the represented streets, but could also reflect reduced average PM emissions from vehicles as a result of stricter emissions regulations.

The year-to-year changes in rural  $PM_{10}$  concentrations can be partly explained by changes in the rural sulfate concentrations, which show a similar variation (Fig. 5.8). Although the  $PM_{10}$  and sulfate lines do not represent the same set of stations, the trends are similar. The sulfate variations are, however, too small to account for all the  $PM_{10}$  variation.

In conclusion, exceedances of  $PM_{10}$  limit values are widespread in urban areas in Europe, and they are also exceeded in rural areas in some countries. The concentrations, averaged over a limited set of stations in nine countries, decreased between 1997 and 1999 and then increased towards 2003. The inter-annual meteorological conditions can explain part of this variation, as can variations in the emission of primary PM. The increase since 1999 seems, however, to be controlled by an increase in the rural  $PM_{10}$  concentration. This increase occurs simultaneous with an increase in secondary sulfate concentrations, which again can be related to meteorological inter-annual variability.

**Fig. 5.8. Annual average concentrations of sulfur dioxide, sulfate and PM<sub>10</sub> for the period 1997–2002 for rural stations (72, 68 and 26 stations, respectively)**



Note: The vertical bars indicate the range between the 10th and 90th percentiles.

### Measurements of PM<sub>2.5</sub>, PM<sub>1.0</sub> and ultrafine particles

As mentioned above, data from stations measuring PM<sub>2.5</sub>, PM<sub>1.0</sub> and ultrafine particles were collected by the CAFE Working Group in connection with the development of the second position paper on particulate matter (3). The number of stations with annual time series data for 2000 and 2001 (many stations had data from only one of these years) was 119 for PM<sub>2.5</sub>, 15 for PM<sub>1.0</sub> and 21 for ultrafine particles. Summaries of the analysis of those data are as follows, cited from the second position paper (3).

Rural background concentrations [of PM<sub>2.5</sub>] seem to be on average quite uniform in Europe (between 11 and 13 µg/m<sup>3</sup>) and considerably lower than urban background levels (around 15–20 µg/m<sup>3</sup>), which in turn are lower than PM<sub>2.5</sub> annual averages at traffic exposed sites (typical range from 20 to 30 µg/m<sup>3</sup>). However,

in interpreting these figures the limited data set has to be borne in mind. The PM<sub>2.5</sub>/PM<sub>10</sub> ratio for 2001 centres around 0.65 (range from 0.42 to 0.82). ... The PM<sub>2.5</sub>/PM<sub>10</sub> ratios are quite uniform at the majority of European stations, with only slight tendencies towards somewhat higher ratios at rural background sites compared with urban traffic sites. Putaud et al. observed somewhat lower ratios for kerbside sites, suggesting large contributions of suspended road dust to the coarse fraction.

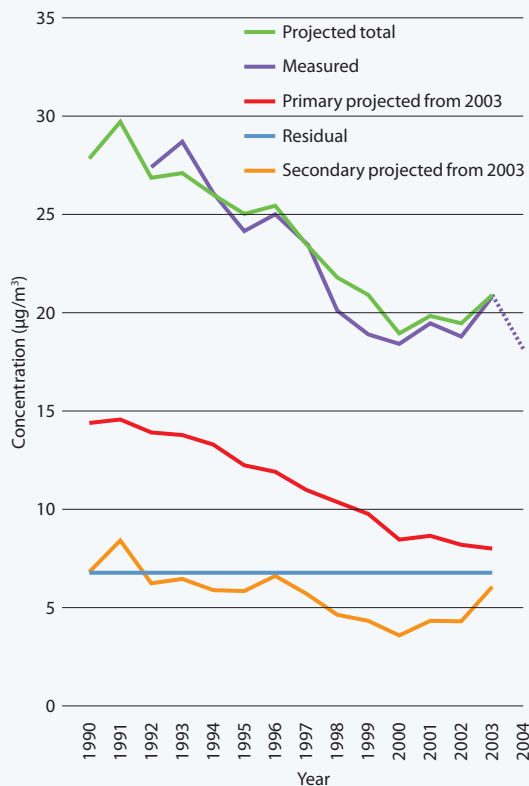
The PM<sub>1.0</sub> data set ... was too small (15 station years) to detect statistically significant differences between station types, although urban traffic sites tended to show somewhat higher PM<sub>1.0</sub> levels than urban background sites. ... The annual means [for 2001] ranged between 6 (Helsinki) and 21 µg/m<sup>3</sup> (Llodio in Spain), but it should be noted that there are no rural background data. The ratio PM<sub>1.0</sub>/PM<sub>2.5</sub> varied around an average of 0.73 ... [while] the ratio PM<sub>1.0</sub>/PM<sub>10</sub> centred around 0.49. The maximum of daily means exceeded 50 µg/m<sup>3</sup> at the majority of stations (highest value: 147 µg/m<sup>3</sup>).

Most of the UFP [ultrafine particle] data received ... were from urban traffic stations (7, with 17 station years in total) ... The levels behaved as expected: lowest at rural background (annual mean 4000–10 000 particles per m<sup>3</sup>), highest at traffic stations (annual mean 10 000–80 000 particles per m<sup>3</sup>). The range was over an order of magnitude, so considerably larger than for PM mass concentrations, which is consistent with the picture that there are near sources relatively many very small particles with a limited lifetime. As a consequence, the ratio of particle number and mass concentration tended to decrease with distance to sources: at rural sites this ratio was considerably lower than at traffic stations both for PM<sub>10</sub> and PM<sub>2.5</sub>.

### Comparison between concentration and emissions trends

Primary PM and precursor gas emissions in Europe are reported to have fallen significantly and steadily

**Fig. 5.9. The components of annual mean PM<sub>10</sub> concentrations at 10 background monitoring sites in the United Kingdom**



since 1990 (9). Precursor gas emissions are about 10 times (in mass) primary PM emissions. Precursor gas emissions (nitrogen oxides, sulfur dioxide, ammonia) fell by about 16% between 1997 and 2002, and reported primary PM emissions fell by about 6% over the same period. The tendency in measured PM<sub>10</sub> concentrations across Europe does not, since 1999–2000, reflect this significant reported decrease in Europe-wide emissions. However, as mentioned in Chapter 4, there are significant gaps in countries' reporting of primary PM emissions; it is thus uncertain whether this reduction is representative of the entire EEA area, or even of the EU (15 countries). Therefore, more complete data from the United Kingdom, covering longer period, were chosen to study the relationships of trends in PM concentrations and emissions.

### Trends in the United Kingdom

Fig. 5.9 illustrates the results of an analysis of the downward trend in annual mean PM<sub>10</sub> concentrations in the United Kingdom since measurements began in 1992. The United Kingdom was chosen for this analysis because of the availability of a detailed time series of primary PM<sub>10</sub> emissions. A simple receptor model was used to assign the annual mean concentrations at measurement sites in 2003 to primary, secondary and residual components. A site-specific projection model (10) was used to predict the concentrations of these components in earlier years. Primary particle concentrations were predicted using published emission inventory trends, secondary particle trends were derived from measurements of sulfate and nitrate at rural sites in the United Kingdom, and the residual contribution was assumed to be constant. Fig. 5.9 shows the predicted concentrations of each component, the predicted total concentrations and the measured concentrations averaged over nine urban background and one rural background monitoring sites.

The predicted trend in PM<sub>10</sub> concentrations reproduced the measured trend quite well. This analysis suggests that the decline in measured concentrations from the early 1990s to 2000 was due to the combined control of primary PM emissions and of the precursors of secondary PM. The concentrations of secondary PM, however, showed considerable year-to-year variation owing to the meteorological conditions. The rate of decline in primary PM concentrations became less steep from 2000, and this coincided with an increase in the secondary PM concentration. A partial annual mean for 2004 is also included in Fig. 5.9 as a broken line. Measured concentrations in 2004 were lower than in 2003, suggesting that the recent upward trend in PM<sub>10</sub> concentrations is unlikely to continue.

Measured PM<sub>10</sub> trends are affected by inter-annual variations in meteorological conditions as well as by changes in emissions. An analysis of the United Kingdom PM<sub>10</sub> concentration and emissions data, with data for annual average meteorological conditions (wind speed, precipitation) showed that a lack of proportionality between inter-annual changes in concentrations and emissions could be explained by the parallel variations in meteorology (5).

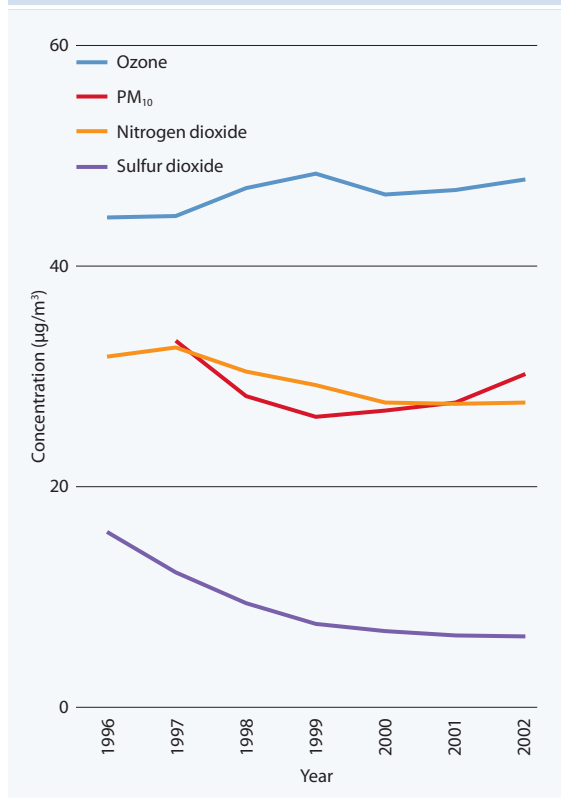


Thus, the analysis of the United Kingdom data indicates that where emissions, concentration and meteorological data of controlled quality are available, the concentration trends can be explained to a large extent by the parallel variations in emission, meteorological conditions and secondary inorganics. The apparent mismatch between European-wide average concentration data and emission trends may indicate a relative stability of primary PM emissions in the (short) period with the data, as well as the need to further study the impact of inter-annual changes in meteorological conditions on PM levels.

### Recent trends for sulfur dioxide, nitrogen dioxide, PM<sub>10</sub> and ozone

In Fig. 5.10, the trends in PM<sub>10</sub> levels during the last few years can be compared to those for other compounds. The graphs show trends in concentrations,

**Fig. 5.10. Summary of measured concentrations of sulfur dioxide, nitrogen dioxide, PM<sub>10</sub> and ozone in Europe, averaged over all stations (all types) contained in AirBase, annual averages, 1996–2002**



as an average for all types of stations reported to AirBase. For each year, the plotted value represents the average of all stations in all countries (only stations with data for all years are shown).

Sulfur dioxide levels show a definite downward trend, and the same tendency applies to nitrogen dioxide since 1996. For ground-level ozone, the tendency is towards an increasing annual average concentration. For sulfur dioxide, nitrogen dioxide and PM<sub>10</sub> the majority of stations show a downward trend, though this change is significant over the period at fewer than half of the nitrogen dioxide and PM<sub>10</sub> stations. The stations showing upward trends are mainly some of the hot spot stations dominated by local traffic or industry, which have presumably been increasing in strength over the entire period (e.g. streets with an increasing amount of traffic). For ozone, the majority of stations show a (nonsignificant) upward tendency; on the other hand the data indicate a decreasing trend in maximum 1-hour concentration (11).

### 5.3 Modelling PM

The use of models allows us to test the validity of our understanding of PM, its origins and transport patterns. Models need to be evaluated against observations in a large variety of places and at different times in order to establish their ability. At present, several models are available in Europe that are able to reproduce long-term observed PM levels in regional areas with documented accuracy (e.g. CHIMERE, EMEP, MATCH, LOTOS and REM3). Results from the EMEP model are presented here because this is the model that has been internationally reviewed (12) and is presently used to support integrated assessment modelling for policy applications within the Convention on LRTAP and the European Commission.

#### The EMEP model

The EMEP model has been developed to calculate long-term source–receptor relationships of air pollutants at the European regional scale (13, 14). It describes the emission, transport, chemical transformation and removal by dry and wet deposition of approximately 75 species (7 of them in particulate form) and about 140 chemical reactions. The model has an aerosol version especially dedicated to studying the transport and transformation of aerosols in air. The aerosol ver-

sion is kept as a research tool and is used to test the validity of the unified EMEP model in describing PM mass. Aerosol dynamic processes are included in the model through a monodisperse multicomponent approach (MULTIMONO) developed in cooperation with the University of Helsinki (15). MULTIMONO includes seven different chemical components and distinguishes four different size classes. Dry and wet deposition processes are considered differently according to size classes. The unified EMEP model was originally developed for regional-scale applications on a  $50 \times 50$ -km grid covering Europe, but it has recently been extended to link hemispheric-regional and local air pollutions, allowing it to describe a chosen subdomain in a finer grid embedded in a hemispheric coarser grid (16,17). The EMEP model is suitable for use in intensive computer simulations such as transfer matrixes between different European countries and over long periods of time (years).

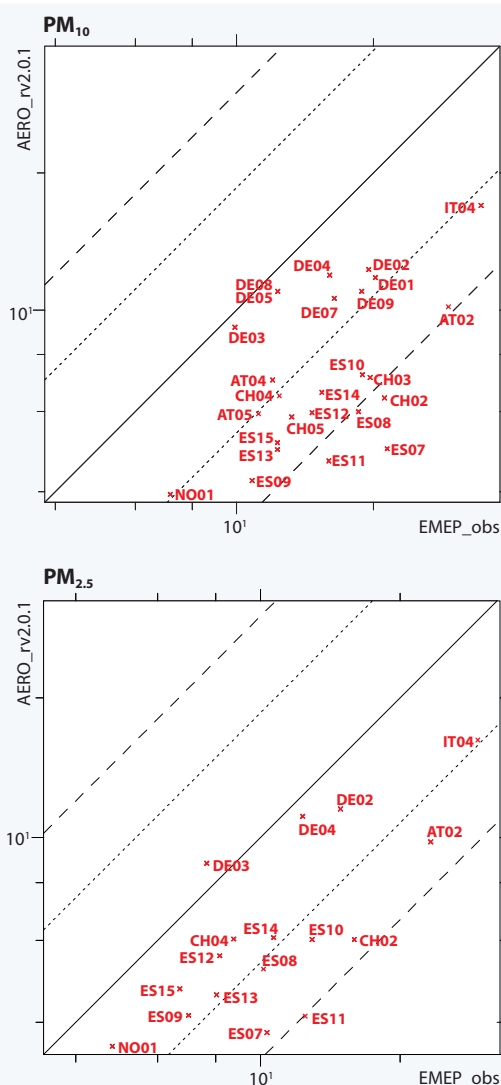
The sources of PM presently included in the unified EMEP model are both anthropogenic and natural. The anthropogenic sources include gaseous precursors of secondary inorganic aerosols, namely emissions of sulfur dioxide, nitrogen oxides and ammonia and primary PM emissions from industrial, residential and agricultural sources classified as fine and coarse primary particle emissions. The anthropogenic emissions are based on officially reported data from countries to the Convention on LRTAP under the EMEP programme. National emission totals can be found in Vestreng et al. (18) and the spatial distribution of these sources is documented in Tarrasón et al. (19). The only natural source of PM currently included in the unified EMEP model is sea salt, and the latest version of the aerosol model also includes wind-blown sources from agricultural soils and from Saharan dust.

Thus there are identified sources of PM that are not yet included in the unified EMEP model, and all these contribute to the carbon content of the aerosol. These are (a) secondary organic aerosols formed in the atmosphere through condensation of volatile organic compounds of both anthropogenic and natural origin; (b) PM emissions from wildfires and biomass burning; and (c) primary biological aerosol particles such as pollen and fungal spores. The consequences of the omission of these sources are further discussed below.

### General underestimation of annual mean aerosol concentrations (regional gradients)

The performance of the unified EMEP model is evaluated annually by comparison with observations from the EMEP and AirBase monitoring networks (2,20). The general conclusion from the comparison is that the model underestimates annual mean  $PM_{10}$  and  $PM_{2.5}$  levels in Europe. The underestimation is well illustrated in Fig. 5.11, where model-calculated annual mean  $PM_{10}$  and  $PM_{2.5}$  concentrations are presented.

**Fig. 5.11. Scatter-plots for model-calculated vs EMEP-measured  $PM_{10}$  and  $PM_{2.5}$  in 2002**



Source: Tsyro (2).

In 2002, PM measurements from six countries were available to EMEP: at 26 sites for  $PM_{10}$  and 17 sites for  $PM_{2.5}$ . The model underestimates observed  $PM_{10}$  concentrations by a factor of 1.5 at German sites and by a factor of 2–2.5 at all other sites. The underestimation of measured  $PM_{2.5}$  concentrations is smaller, within a factor of 2 for all sites. In general, the model reproduces the regional distribution of  $PM_{10}$  and  $PM_{2.5}$  with spatial correlation coefficients of 0.56 and 0.78, respectively. However, calculated  $PM_{10}$  and  $PM_{2.5}$  gradients in Germany and Switzerland are smaller than observed.

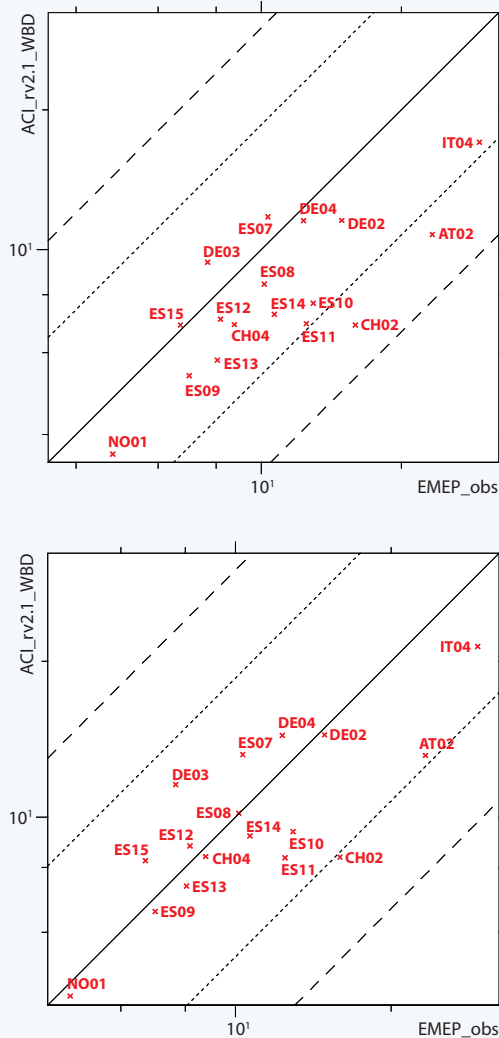
The general underestimation of the model is probably related to the contribution of sources currently not included in the model calculations. Another possible reason can be related to water bound to particles: while model calculations usually provide dry PM mass, the equilibration of filters used to compile measurement data does not always remove all particle-bound water, so that a significant part of the unaccounted observed PM mass could be associated with water. As illustrated in Fig. 5.12, the performance of the unified EMEP model improves considerably when wind-blown dust sources and particle-bound water are taken into account in the model calculations. For  $PM_{2.5}$ , the original general underestimation of –42% is reduced to –28% when wind-blown dust emissions are taken into account, and further to –12% when particle-bound water is taken into account in comparing modelled results with observations.

Although the spatial correlations are currently high at 0.70 for  $PM_{10}$  and 0.78 for  $PM_{2.5}$ , these results are affected by the lack of monitoring data in large areas of Europe and mostly show the ability of the model to reproduce the north–south distribution gradients of rural background PM across Europe. Further understanding of the reasons for the general underestimation of modelled results would require analysis of individual sources and processes through direct comparison with daily observations.

### Temporal correlations

Temporal correlations of modelled  $PM_{10}$  and  $PM_{2.5}$  mass concentrations with daily observations also improve comparison when emissions from wind-blown dust sources are included in the model calculations and when particle-bound water is considered

**Fig. 5.12. Scatter plots in 2002 for  $PM_{2.5}$  calculations when including natural wind-blown dust sources (top) and, in addition, particle-bound water (bottom)**



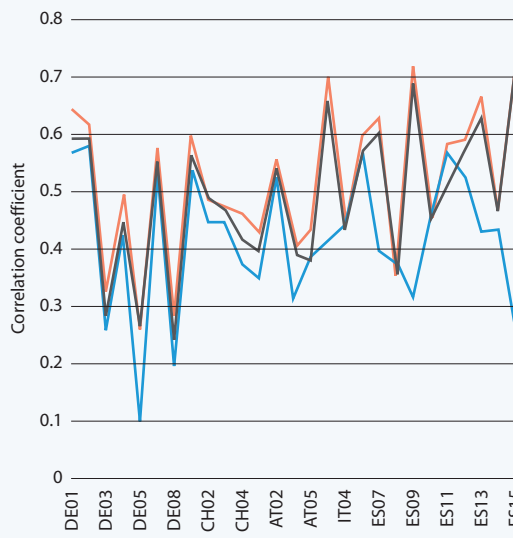
Source: Tsyro (2).

as part of the modelled output (Fig. 5.13 and 5.14). The increase in temporal correlations is an indication that both processes are relevant to PM mass calculations. Since mineral emissions from wind-blown dust sources affect mainly the coarse mode of PM, the effect of introducing wind-blown dust emission in the model is consequently larger for  $PM_{10}$  than for  $PM_{2.5}$ . It is also interesting to note that the increase in temporal correlations is largest for southern Europe–

an (especially Spanish) stations, where the influence of Saharan dust is well established.

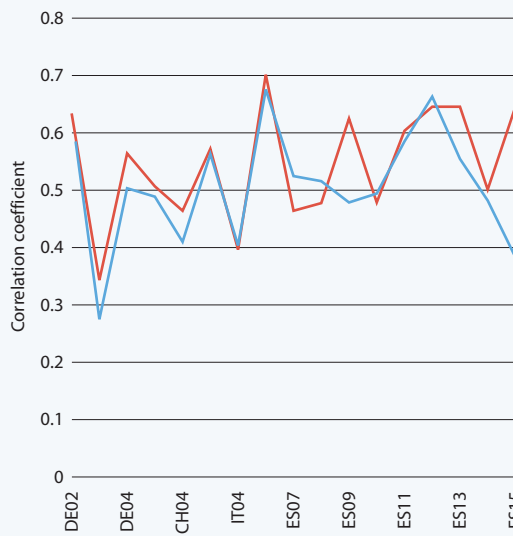
The temporal correlations are lower for  $PM_{10}$  (0.4–0.5 on average) than for  $PM_{2.5}$  (0.5–0.6 on average).

**Fig. 5.13. Temporal correlation of modelled  $PM_{10}$  mass with daily observations in the EMEP network in 2002**



Note: The blue curve represents dry  $PM_{10}$  mass, the green curve dry  $PM_{10}$  mass with wind-blown dust, and the red curve wet  $PM_{10}$  mass with wind-blown dust.

**Fig. 5.14. Temporal correlation of modelled  $PM_{2.5}$  mass with daily observations in the EMEP network in 2002**



Note: The blue curve represents dry  $PM_{2.5}$  mass and the red curve shows results for  $PM_{2.5}$  mass when both wind-blown dust sources and particle-bound water are included in the calculations.

This indicates that the sources and processes currently not described in the model are probably more important for the coarse fraction of PM.

### Seasonal variations in particle concentrations

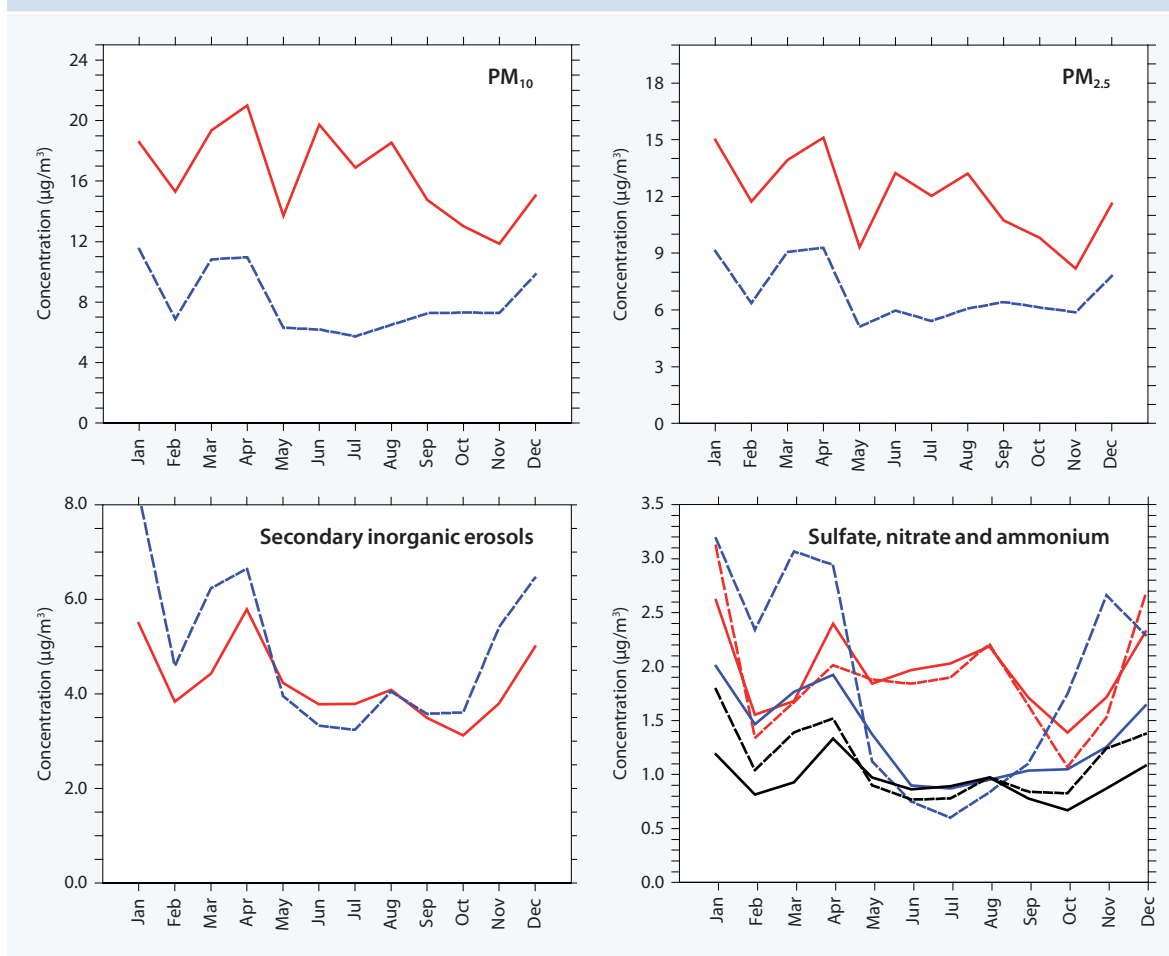
The general underestimation of PM mass concentrations by the model is not uniform over the year. The situation for 2002 is representative of what has been observed in other years. As indicated in Fig. 5.15, the model underestimation of observed concentrations of  $PM_{2.5}$ , and especially  $PM_{10}$ , is largest in summer.

In the summer months, the underestimation of  $PM_{2.5}$  and  $PM_{10}$  seems to be related to the model not yet accounting for secondary organic aerosols of biogenic origin or for primary biogenic particles. Both source types are expected to have a summer maximum. Also, wind-blown sources are not yet included in the unified EMEP model and, as indicated above from the results of the test aerosol model, wind-blown sources represent a significant contribution to PM mass concentrations. This lack of relevant sources with a summer maximum in the model description is also the reason that the negative bias for both  $PM_{2.5}$  and  $PM_{10}$  is largest in the summer and the spatial and temporal correlations are relatively low.

In the winter, underestimation of  $PM_{2.5}$  and  $PM_{10}$  is smaller than in the summer, primarily because the model overestimates secondary inorganic aerosols from November through to March. Thus, the model's overestimation of secondary inorganic aerosols (mostly nitrate) in cold situations compensates to some extent for its underestimation of PM due to other causes. Also, as presented in Table 5.3, the spatial correlation between calculated and measured  $PM_{10}$  concentrations is lowest in winter. This is probably because the model has difficulties representing free tropospheric intrusions at high mountain stations. Since there were five such mountain stations reporting to the EMEP network in 2002, these biased the results of the model comparison with observations in 2002 and also affected the results on spatial gradients and variability, as pointed out above. The effect of mountain stations on the model performance statistics in winter is less pronounced for  $PM_{2.5}$  since only two such sites measured  $PM_{2.5}$ .

On average, the model performance with respect to both  $PM_{2.5}$  and  $PM_{10}$  is better in spring and autumn

**Fig. 5.15. Monthly time series of calculated (dashed line) and measured (solid line)  $PM_{10}$ ,  $PM_{2.5}$ , secondary inorganic aerosols (sulfate + nitrate + ammonium) and individual concentrations of sulfate, nitrate and ammonium averaged over all EMEP sites where measurements were available, 2002**



Note: In the bottom right panel, sulfate is represented as a red, nitrate as a blue and ammonium as a black line.

Source: Tsyro (2).

**Table 5.3. Comparison of the modelled and measured  $PM_{10}$  and  $PM_{2.5}$  for 2002 averaged over all EMEP sites with measurements**

Particle size	Season	Observed mean	Modelled mean	Bias (%)	Root square mean error	Correlation
$PM_{10}$	winter	16.62	9.17	-44	13.39	0.29
	spring	18.00	9.35	-48	9.42	0.71
	summer	18.32	6.19	-66	12.81	0.46
	autumn	13.20	7.29	-44	7.57	0.58
$PM_{2.5}$	winter	13.17	7.77	-40	11.05	0.60
	spring	12.74	7.81	-38	5.63	0.87
	summer	13.04	5.92	-54	7.85	0.41
	autumn	9.57	6.17	-35	5.19	0.79

Note: Bias is calculated as (modelled mean - observed mean)/observed mean; root square mean error is calculated as  $1/N \sum (\text{modelled} - \text{observed})^2$ .

when the spatial correlation coefficients are quite high (0.58–0.87) and root square mean error is smallest (Table 5.3).

## 5.4 PM speciation

PM is a complex mixture of different pollutants from a variety of sources. In order to determine its origin, an analysis of the individual chemical components is necessary. Chemically speciated PM data from measurements at rural, urban and traffic sites are available from Austria, Germany (Berlin), the Netherlands, Spain, Sweden, Switzerland and the United Kingdom and have been analysed in the second position paper on particulate matter (3) and recent EMEP reports.

The following is a summary of the findings of source contributions to PM based on the speciation data and modelled values, identifying the main areas of uncertainty and consequences for further conclusions on source allocation of PM.

### Secondary inorganic aerosols

The contribution of secondary inorganic aerosols, mainly from industrial, agricultural and traffic emissions, range from 4 to 13  $\mu\text{g}/\text{m}^3$  and from 3 to 11  $\mu\text{g}/\text{m}^3$  for  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ , respectively. The contribution of secondary inorganic aerosols to PM mass varies considerably across sites, but it represents some 20–30% of  $\text{PM}_{10}$  mass and 30–40% of  $\text{PM}_{2.5}$  mass.

In general, the unified EMEP model is able to reproduce well the spatial variability and observed levels of secondary inorganic aerosols across Europe, with an average positive bias of 18% and spatial correlations of 0.87. The good performance of the EMEP model for secondary inorganic aerosols underpins the confidence in the modelled contributions of precursor gas emissions (sulfur dioxide, nitrogen oxides and ammonia) to total PM mass.

Although these results on model performance are satisfactory, the model tends to overestimate measured concentrations of secondary inorganic aerosols close to major emission areas and to underestimate long-range transported secondary inorganic aerosols, as indicated in Fig. 5.16. The underestimation of secondary inorganic aerosols in Nordic countries is associated with the underestimation of nitrate and ammonium in these areas. In other regions, the general underestimation of particulate sulfate concen-

trations is compensated for by overestimated nitrate values, as a consequence of equilibrium chemistry. Further improvements would require the study of nitrate chemistry and transport, using additional denuder measurements, as recommended in the new EMEP monitoring strategy (21).

### Sea salt

The marine contribution to  $\text{PM}_{10}$  is estimated to be 2–4  $\mu\text{g}/\text{m}^3$  for most European coastal areas. For  $\text{PM}_{2.5}$  this contribution is lower, ranging from 0.2 to 0.8  $\mu\text{g}/\text{m}^3$ , except in Scandinavia where it can reach up to 2  $\mu\text{g}/\text{m}^3$  in southern Sweden.

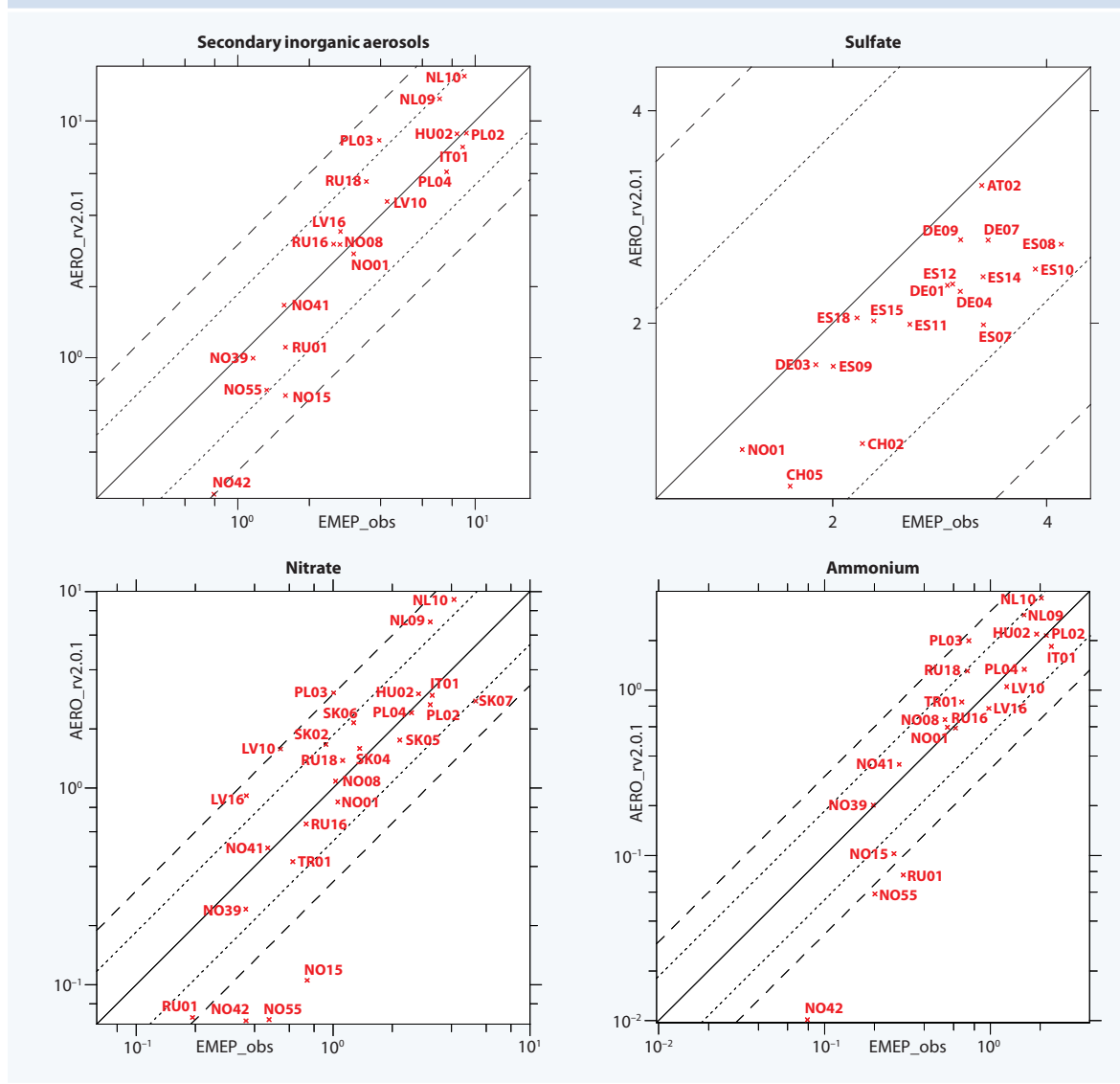
Sea salt particles are a natural component of ambient aerosols and have been accounted for in the unified EMEP model in order to better characterize the PM mass closure. Model-calculated concentrations of sodium originating from sea salt aerosols have been compared with sodium measurements collected at three Danish and seven Norwegian sites during 2001 and 2002. The scatter plots in Fig. 5.17 show that the model tends to underestimate measured sodium concentrations at all of the sites except for Spitsbergen (NO42). The spatial correlation between modelled and observed sodium concentrations in these years is quite high (0.84 and 0.72, respectively).

The underestimation of sodium at some stations can be related to the rather large gradients of sea salt in coastal areas that impose large sub-grid concentration variability, which cannot be resolved accurately enough with the EMEP model. Nevertheless, the temporal correlation coefficient between calculated and measured daily sodium is quite high, between 0.4 and 0.7, which indicates that the model manages to capture sea salt episodes. The best agreement between model and observation results was found at Skreådalen (NO08), Tustervatn (NO15) and Anholt (DK08) in 2001 and 2002, and the worst at Spitsbergen (NO42) and Tange (DK03) in 2002, as shown in Fig. 5.18.

### Mineral dust from anthropogenic and natural sources

The mineral contribution to  $\text{PM}_{10}$  at regional sites is estimated to be around 2  $\mu\text{g}/\text{m}^3$  for all countries except Spain, where the mineral contribution is usually 2–3 times higher. At urban sites, the mineral contribution ranges from 3–5  $\mu\text{g}/\text{m}^3$  in central Europe to 7–9  $\mu\text{g}/\text{m}^3$

Fig. 5.16. Scatter plots for model-calculated vs measured total secondary inorganic aerosols, sulfate, nitrate and ammonium at EMEP sites in 2002



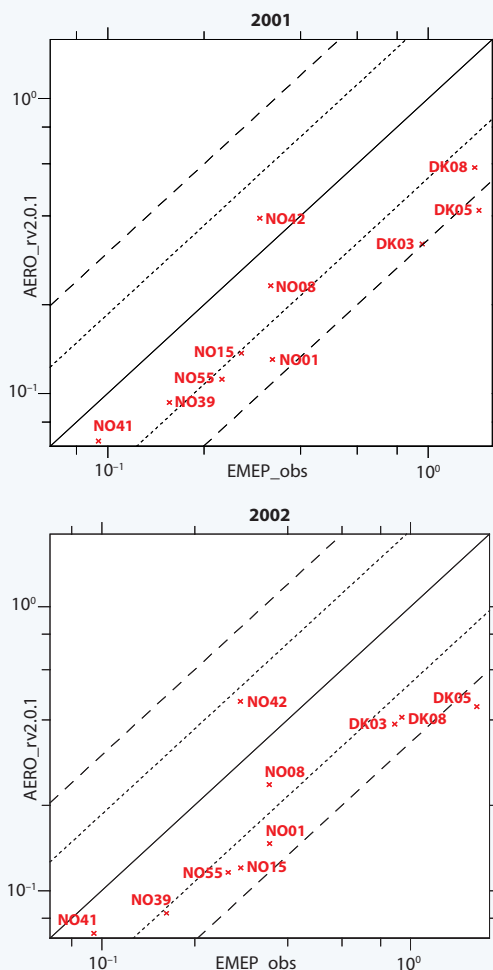
Source: Tsyro (2).

in Sweden and up to  $10 \mu\text{g}/\text{m}^3$  in Spain. At roadside sites, the mineral contribution again increases from  $4\text{--}7 \mu\text{g}/\text{m}^3$  in central Europe to  $16 \mu\text{g}/\text{m}^3$  in Spain and  $17\text{--}36 \mu\text{g}/\text{m}^3$  in Sweden. A clear differentiation is also evident for the mineral contribution to  $\text{PM}_{2.5}$ , the lowest levels being recorded at regional background sites and the highest at roadside stations.

The higher concentrations of mineral dust at Spanish stations are due to the influence of Saharan dust intrusions. This natural contribution comes in

addition to natural wind-blown dust from agricultural soils and the anthropogenic dust contributions from cement and construction activities. On the other hand, the higher concentrations of  $\text{PM}_{10}$  mineral contribution over Sweden (and Scandinavia as a whole) are mostly associated with urban and roadside sites and not Saharan dust intrusions. In this case, the origin of mineral  $\text{PM}_{10}$  is most probably resuspension of road dust. Although road dust affects urban and roadside sites all over Europe, its contribution

**Fig. 5.17. Scatter plots of calculated vs measured sodium concentrations in 2001 and 2002**



Source: Tsyro (2)

to Scandinavian  $PM_{10}$  levels is expected to be larger, specially during winter owing to the common use of studded tyres and the salting of roads.

On a regional scale, anthropogenic mineral dust emissions and the natural contribution from wind-blown dust from the Sahara and from European agricultural soils are considered in EMEP model calculations. The effect of resuspension of road dust is considered to be more important at urban level and has not yet been introduced into the model calculations.

As mentioned above, the introduction of wind-blown dust emissions improves the performance of

the EMEP unified model. For  $PM_{10}$  concentrations, the model bias is reduced from  $-48\%$  to  $-34\%$  when introducing wind-blown dust sources, and spatial correlations increase from 0.56 to 0.65. Considering water content in  $PM_{10}$  results in further reductions of the model bias down to  $-22\%$  and increased spatial correlations (0.70). Such increases in both spatial and temporal correlations is an indication of the significance of wind-blown dust sources for an appropriate representation of processes involved with the emission, transport and transformation of PM in surface air (Fig. 5.19).

### Particle-bound water in $PM_{2.5}$ and $PM_{10}$

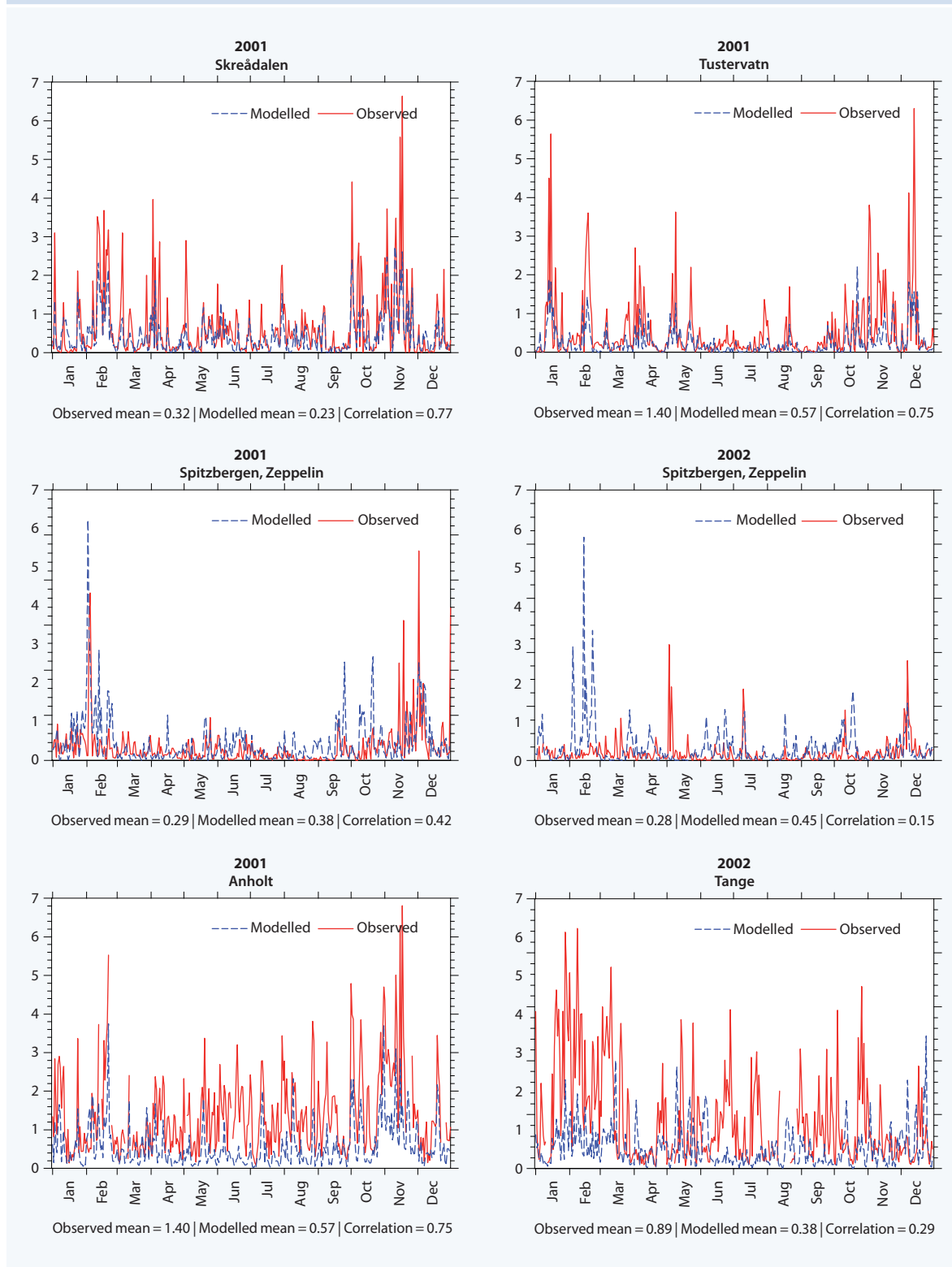
Available measurements on the chemical characterization of  $PM_{10}$  and  $PM_{2.5}$  (22–25) reveal that full chemical PM mass closure is rarely achieved. In many cases, a fraction as large as 30–40% of the gravimetric  $PM_{10}$  or  $PM_{2.5}$  mass can remain unidentified in the chemical analysis. This unaccounted PM mass is believed to be partly due to non-carbon atoms in organic aerosols and/or to sampling and measurement artefacts. Moreover, a considerable part of the unaccounted PM mass is likely to consist of water associated with particles. Thus, the gravimetrically measured mass of filter-collected particles does not necessarily represent dry  $PM_{10}$  and  $PM_{2.5}$  mass.

Filter-based gravimetric methods are recommended by the EMEP measurement manual and employed for determining  $PM_{10}$  mass concentrations at all EMEP sites. It is required that the dust-loaded filters should be equilibrated at  $20^\circ\text{C}$  ( $\pm 1^\circ\text{C}$ ) and 50% relative humidity ( $\pm 5\%$ ) for 48 hours before they are weighed, both prior to and after sample collection. However, equilibration of filters does not remove all particle-bound water. A number of experimental studies revealed that particles can contain 10–30% water by mass at a relative humidity of 50% (26–27).

This is thought to be one of the reasons for models under-predicting gravimetrically measured PM if calculated dry  $PM_{10}$  and  $PM_{2.5}$  concentrations are compared with observations. To account for this effect, the water content of  $PM_{10}$  and  $PM_{2.5}$  has been calculated with the EMEP model for the conditions required for sample equilibration ( $20^\circ\text{C}$  and 50% relative humidity). According to these simulations, gravimetrically measured annual mean  $PM_{10}$  concentrations can con-



**Fig. 5.18. Time series of calculated vs measured sodium concentrations in 2001 and 2002 (selected stations)**



tain between 0.5 and 6.5  $\mu\text{g}/\text{m}^3$  of water, in Scandinavia and in Belgium and the Netherlands, respectively. For  $\text{PM}_{2.5}$ , residual water content varies between 0.3 and 5  $\mu\text{g}/\text{m}^3$ . Particle water content is determined by the mass fraction and the type of mixture of soluble PM constituents. The model calculated particle water constitutes 20–35% of the annual mean  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  concentrations, which is effectively consistent with existing experimental data.

The calculated aerosol water in  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  can explain about 30–80 % of the unaccounted for PM mass compared with data on PM chemical composition available at six stations (28). An example of these results is presented in Fig. 5.20 for two Austrian stations.

In general, accounting for particle water in  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  improves the general agreement between calculated and measured PM concentrations. However, the model-calculated aerosol water needs to be verified against measurements. Measurements of particle water in  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  are presently not included in monitoring framework activities. The general lack of measurement data on particle-bound water hampers verification of model calculations of particle water content.

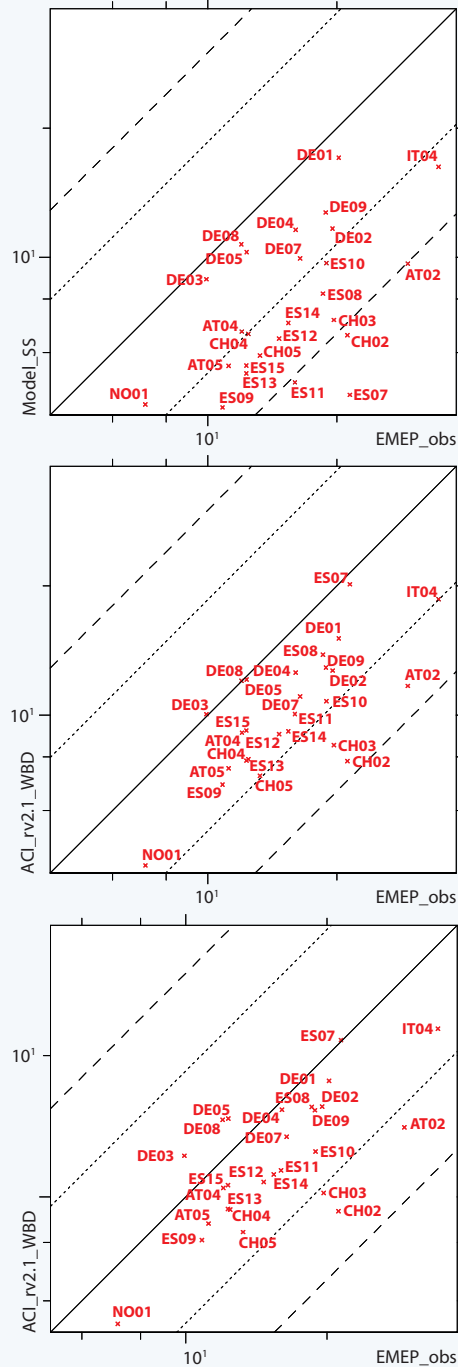
These results, as well as experimental evidence, suggest that particle water should be accounted for in model-calculated  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  when evaluating them against gravimetrically measured PM mass.

### Organic and elemental carbon (OC+EC)

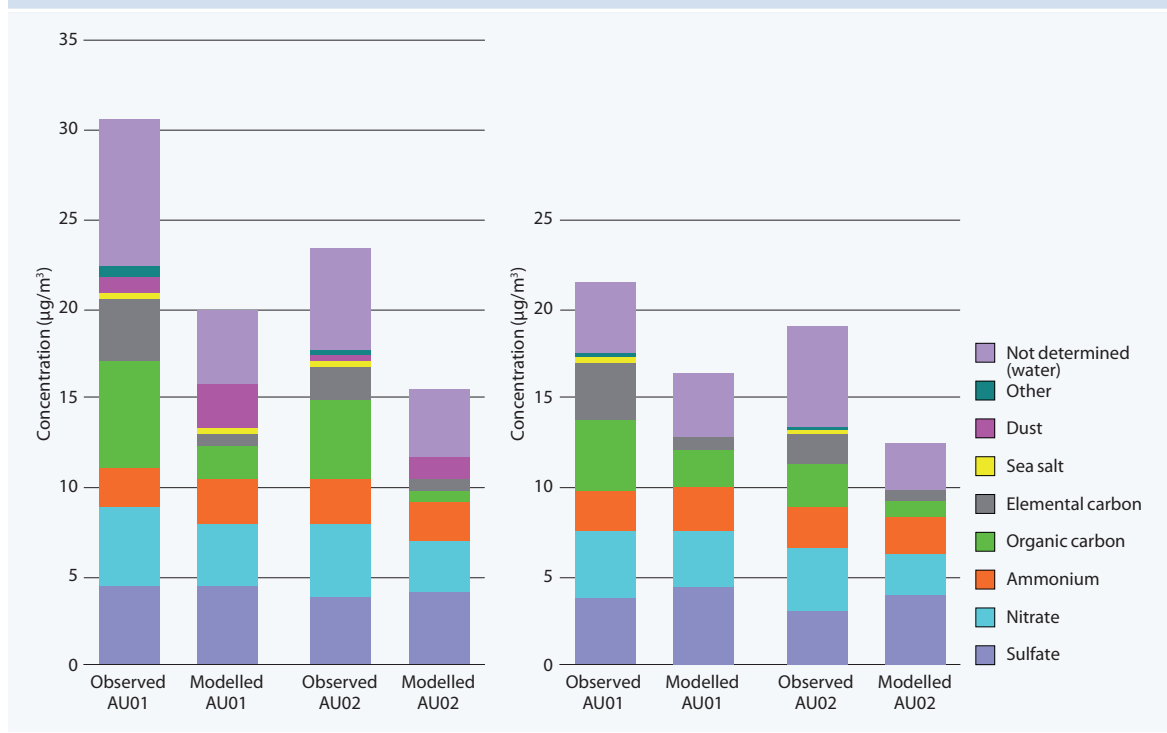
The contribution of OC+EC to  $\text{PM}_{10}$  at regional background sites ranges from 1  $\mu\text{g}/\text{m}^3$  in Sweden to 3  $\mu\text{g}/\text{m}^3$  in Spain to 7  $\mu\text{g}/\text{m}^3$  in Austria. The carbonaceous contribution is larger at urban background than at regional background sites. The OC+EC contribution is largest at roadside sites and usually ranges from 13 to 21  $\mu\text{g}/\text{m}^3$ . The same gradient in concentrations is observed for the OC+EC contributions in  $\text{PM}_{2.5}$ , although the actual concentrations of OC+EC in  $\text{PM}_{2.5}$  are about 10–20% below those in  $\text{PM}_{10}$ .

During 2002 and 2003, EMEP carried out an EC/OC measurement campaign to address the level of carbonaceous material present in ambient aerosols at 14 representative rural background sites in Europe for one year. Results from four of these stations are depicted in Fig. 5.21. The lowest concentrations of EC

**Fig. 5.19. Improvement in model performance for annual averaged  $\text{PM}_{10}$  concentrations from PM dry concentration (upper panel), addition of wind-blown dust (middle) and taking into account both natural wind-blown dust and water content in aerosols (lower panel)**



**Fig. 5.20. Measured and modelled chemical composition of PM<sub>10</sub> (left) and PM<sub>2.5</sub> (right) at Austrian sites in Vienna (AU01) and Streithofen (AU02) over the period 1 January–31 May 2000**



Note: The purple colour denotes the unaccounted for (not determined) PM fraction in measurements and particle water in model calculations.  
Source: Tsyro (2).

were in general observed in Scandinavia and the British Isles, whereas the highest ones were reported for the central, eastern and southern parts of Europe. EC was found to account for 1–5 % of PM<sub>10</sub> on an annual basis while organic matter was found to account for 12–45% of PM<sub>10</sub>.

Wintertime (October–March) concentrations of EC were found to be higher than those recorded during summer (April–September), except at the Norwegian site. The increased levels of EC found during winter may be explained by increased emissions from residential heating (coal, oil and wood) and traffic (cold starts) during winter and possibly more frequent inversions. The highest annual average concentration of organic matter is seen in Italy and the lowest concentration in Norway. At the three Scandinavian sites and at the site in Slovakia, the summertime concentrations of OC were found to be a factor of 1.4–1.6 higher than those recorded during winter. One possible explanation may be that this is due to biogenic OC and primary biological aerosol particles contrib-

uting to the OC fraction at these sites during summer, together with a low effect of anthropogenic OC. For the other sites, the concentration of OC increases by a factor 1.2–2.7 from summer to winter. This is most likely explained, as for EC, by increased emissions from residential heating and traffic during winter.

Model-calculated EC and OC concentrations were evaluated against measurements from the above-mentioned EMEP OC/EC campaign. The comparison of modelled and measured concentrations of OC and EC aimed at characterizing the model's performance against observations and also the possibility of validating primary PM emissions through this study.

Different tracers for sources of primary aerosols are currently under evaluation, including levoglucosan for wood burning emissions, hopane for vehicle emissions, benzothiazole for tyre wear emissions and sugars and sugar alcohols for primary biological aerosol particles. However, the use of such tracers is still under discussion and conclusions are masked by artefacts in measurements and differences in the

chemical composition of OC in different European regions. At present, EC seems to be the most useful component for evaluating the appropriateness of PM emission inventories.

In the current EMEP model calculations, primary PM emissions are considered to be chemically divided into three large categories: OC, EC and mineral dust. For fine primary particles, the fraction of EC and OC components are defined for each emission activity sector according to Kupiainen & Klimont (29). The remaining fraction of fine primary particle emissions is considered to be mineral dust. Coarse particles are considered to be emitted as mineral dust and EC, where the relative contribution of the EC component varies depending on the activity sector of the emission source. (Z. Klimont, personal communication, 2005).

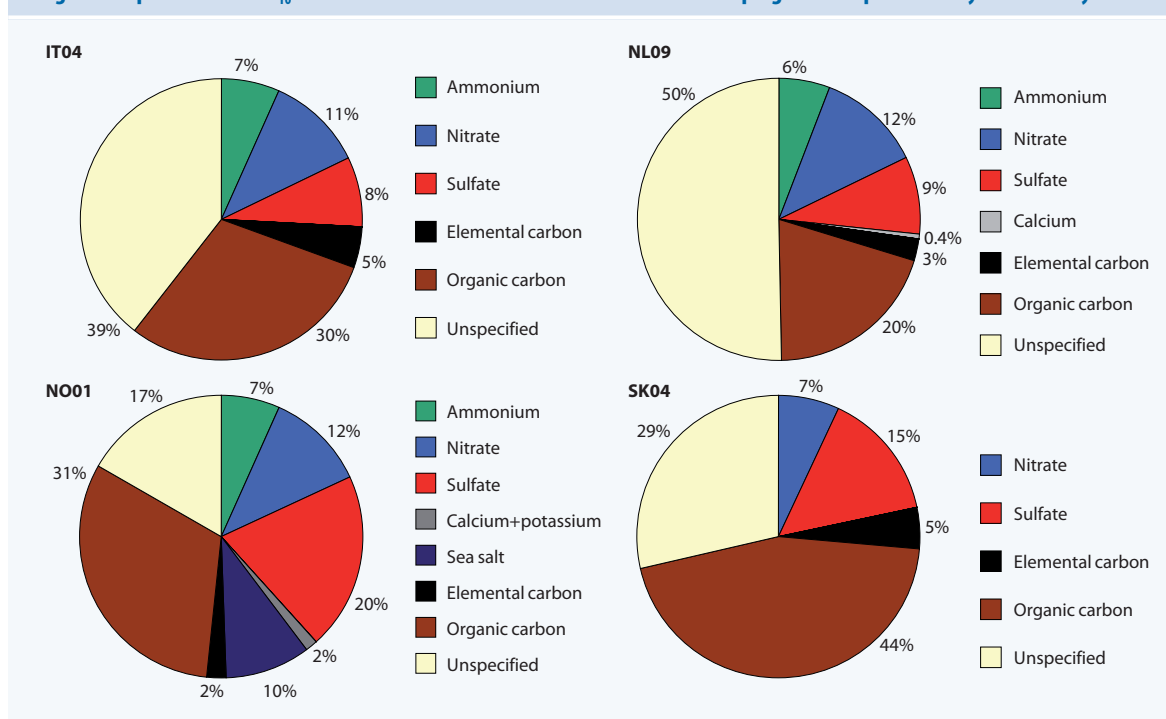
Fig. 5.22 presents the modelled and observed geographical distribution of EC and OC in Europe. Measured concentrations are shown as the averages over the whole measurement campaign period, while calculated concentrations are annual means in 2002. The model manages to reproduce the main features of the observed distribution pattern of EC and OC, with

the highest values in Belgium and northern Italy and the lowest values in northern Europe. Nevertheless, the model underestimates measured concentrations of EC and specially OC. The scatter plots for the campaign period in Fig. 5.23 show a general underestimation of  $-37\%$  for EC and of over a factor of 3 ( $-84\%$ ) for OC. This is the largest discrepancy between model and observation of any of the validated aerosol components.

The spatial correlations are high for EC (0.88), as are the temporal correlations (0.5–0.6). Thus the generalized model underestimation may indicate a systematic underestimation in the primary PM emissions.

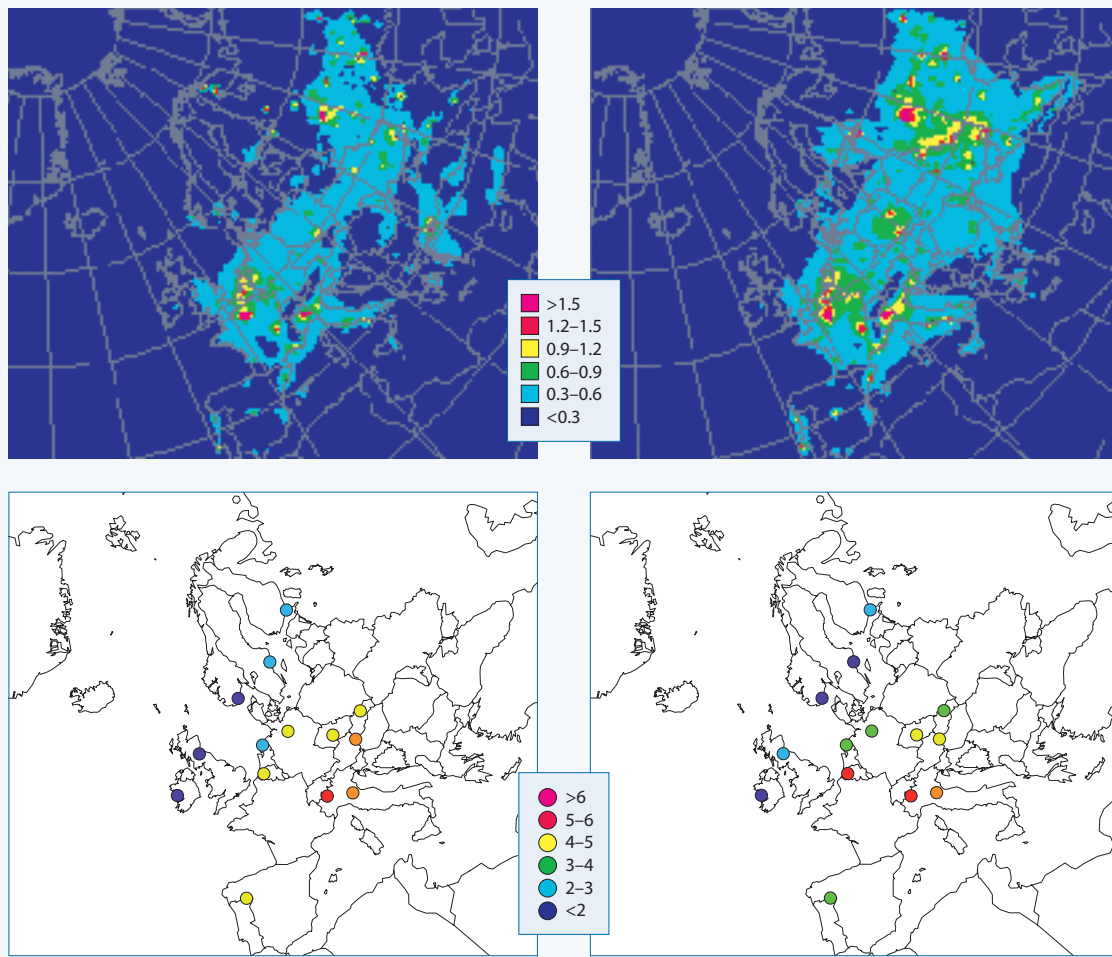
For OC, however, the model has difficulties representing the temporal variability and seasonal variation of the measurements, especially during the summer. In this case, the underestimation seems also to be related to the fact that relevant sources of OC are missing in the model simulations. The model-calculated OC does not represent all atmospheric organic aerosols but only their anthropogenic primary fraction. Secondary organic aerosols, both biogenic and

**Fig. 5.21. Speciation of PM<sub>10</sub> mass concentrations from the measurement campaign for the period 1 July 2002–1 July 2003**



Note: Organic matter is OC multiplied by a factor of 1.6. (IT04) or 2.0 (SK04, NO01, NL09). The concentrations of inorganic ions are from 2002.

Fig. 5.22. Maps of EC (left) and OC (right) concentrations: model-calculated 2002 annual mean (upper panel) and measured, averaged over the campaign period (lower panel)



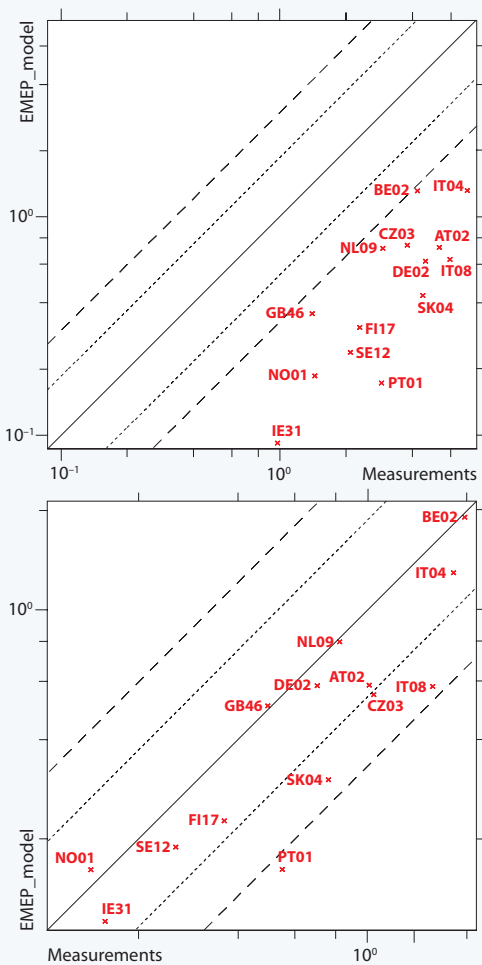
Note. There is a different colour scale in the legends for OC model results and observations.  
Source: Tsyro (2).

anthropogenic, are not included in the model. Neither is organic matter from primary biological aerosol particles or from biomass burning. Therefore, OC concentrations are considerably under-predicted by the model.

The largest uncertainties in the anthropogenic contribution of the observed PM are related to the OC component. While there are indications that primary anthropogenic PM emissions may be somewhat underestimated, the main uncertainties still remain associated with the primary biogenic sources of OC and to the formation of secondary organic aerosols (30). Models that aim to define various heterogene-

ous mechanisms to condense volatile substances onto an aerosol can give rise to differences of up a factor of 10 in calculated concentrations of secondary organic aerosols (31). This level of uncertainty advises against introducing such calculations into models to be used as a basis for policy calculations. In this context it would be useful to determine the extent of the anthropogenic contribution to secondary organic aerosols. Recent estimates at Swiss stations indicate that 60–80% of the observed daytime secondary organic aerosols can be of biological origin (32), but conclusions vary across Europe. Further analysis of the origin and transport of organic carbon involve efforts to:

**Fig. 5.23. Scatter plots of model-calculated vs measured EC (upper) and OC (lower) averaged over the period July–December 2002**



Note: Measurements were taken one day a week over the campaign period.  
Source: Tsyro (2).

- validate the anthropogenic emission component and analysis of biogenic sources through the use of speciated emissions and speciated OC monitoring data; and
- determine the contribution of biogenic and geogenic sources of OC, in particular from biological VOC condensation, biomass burning and primary biological sources.

Such studies are of further relevance in studying the differences between regional background concentrations and urban and road site concentrations. OC

seems to be a major contributor to the differences between road site concentrations and regional background.

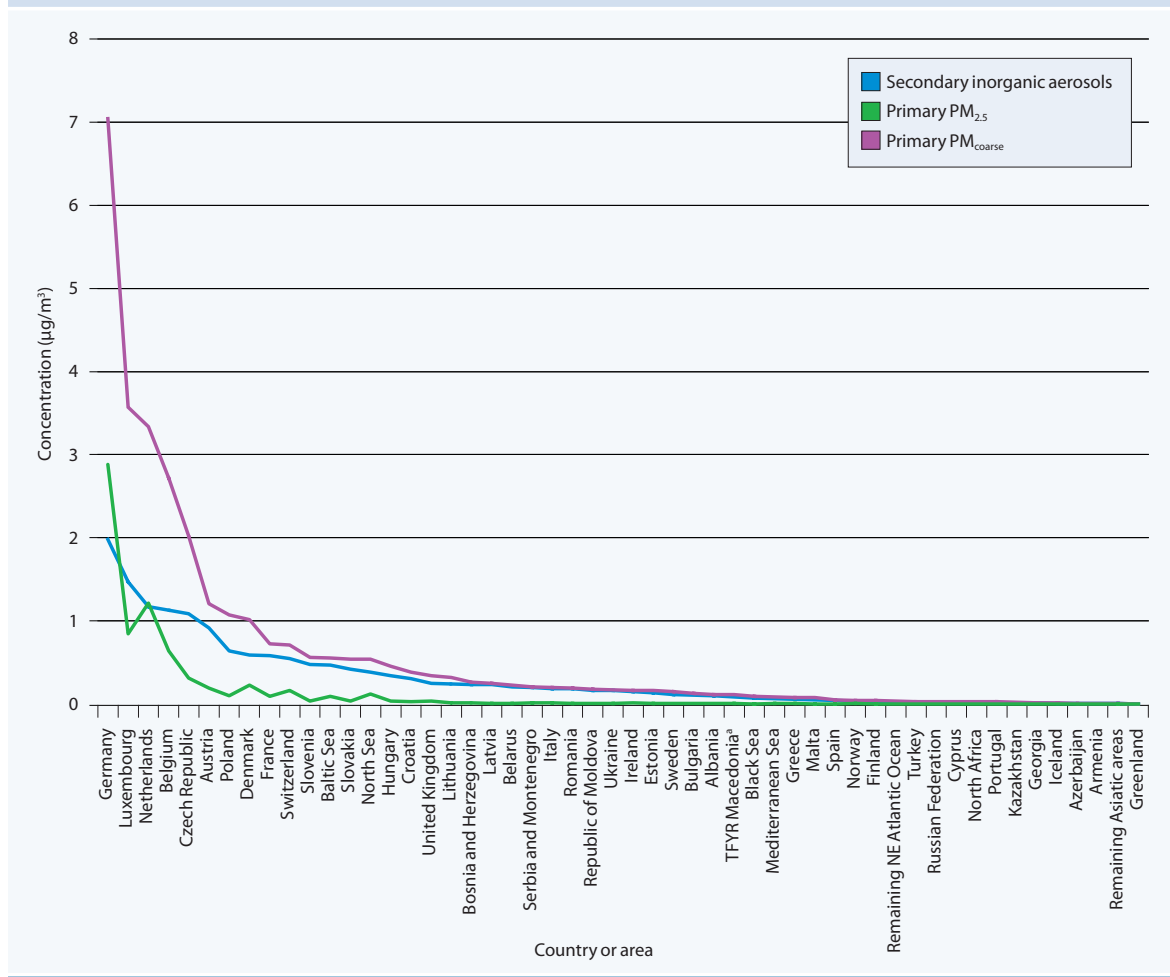
### 5.5 Source apportionment: is PM a transboundary problem?

The travel distance of pollutants in air is determined by their residence time in the atmosphere. For PM, residence times depend mainly on their dry Lagrangian path, that is, the time particles can be transported in the atmosphere without being removed by precipitation. The residence time of PM in the atmosphere ranges from 1–2 days to 4–6 days, depending mostly on the size of the particles and their chemical composition. For instance, coarse particles have shorter residence times than fine particles because they are more effectively removed by dry deposition.

Typical travel distances are about 3000–4000 km for secondary inorganic aerosols, about 2000–3000 km for primary fine particles and 500–1000 km for primary coarse particles. The fact that PM can travel over such long distances implies that pollutants emitted in one European country can affect PM concentrations in neighbouring countries and even countries far distant from the source. An example is provided in Fig. 5.24, showing the area of influence of German anthropogenic emissions of primary PM and precursor gases. Emissions of fine primary particles in Germany can be traced to the Czech Republic, France and the United Kingdom, affecting PM levels in these countries. Note here that PM of biogenic and geogenic origin is also transported over long distances but it is not included in the analysis of transboundary contributions as this is addressed to the anthropogenic contribution to PM.

Over Europe in general, regional background  $PM_{2.5}$  levels have a considerable transboundary contribution of 40–80% of the concentrations in air. For primary fine particle concentrations the transboundary contribution is slightly lower, and for primary coarse particles it varies from 20% to 60%. The size of the country, its geographical position and the size of emissions in neighbouring countries compared to its own also determine the extent of the contribution from transboundary PM levels in a particular European country.

**Fig. 5.24. Average concentrations ( $\text{ng}/\text{m}^3$ ) of secondary inorganic aerosols, primary fine particles and primary coarse particles over different countries following a 1-kilotonne emission of precursor gases and primary PM in Germany**



Note: The model assumes the chemical situation projected for the year 2010 under current legislation (CLE scenario) and using 1997 meteorological conditions for the pollution transport.

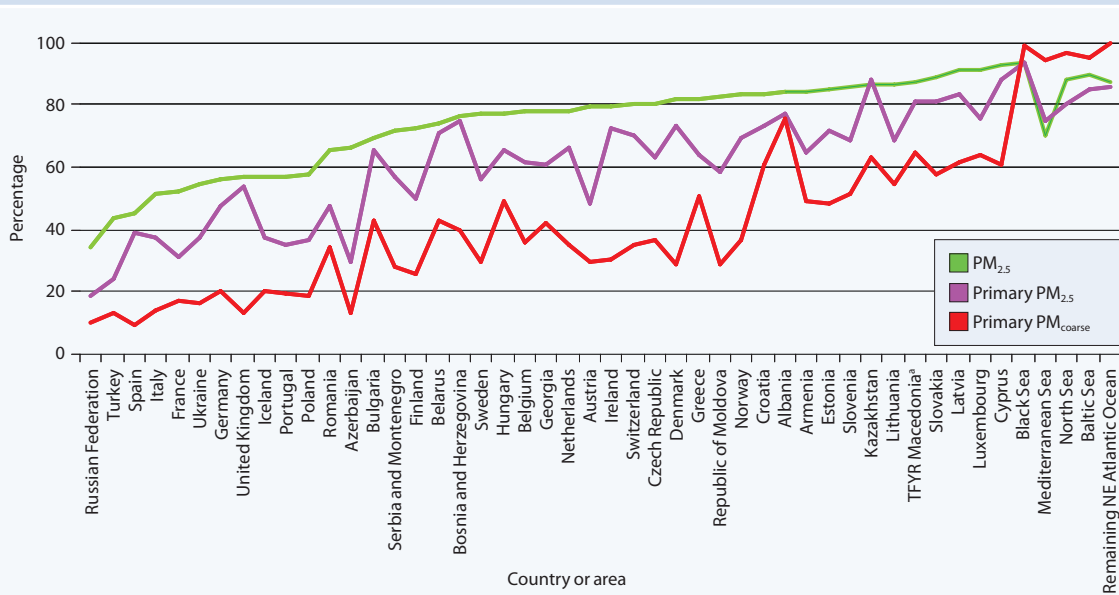
\*The former Yugoslav Republic of Macedonia.

Fig. 5.25 presents an overview of the contribution of transboundary PM in different European countries and regions. The calculations were carried out with the unified EMEP model for the meteorological conditions of 1997 and for emission levels in Europe expected in 2010 under the scenario assuming implementation of current legislation (CLE scenario). Emissions from each country were studied separately to identify the contribution of each of them to total PM levels. The transboundary contribution is largest over sea areas, since there are few indigenous pollution sources over the seas. The transboundary contribution is also large for most eastern European countries, especially those situated downwind from

the main emission pollution source areas in central Europe. The smallest contribution from transboundary  $\text{PM}_{2.5}$  (about 40%) is found for large countries with significant indigenous emissions and affected by particular atmospheric transport conditions, such as Italy, the Russian Federation, Spain and Turkey.

The transboundary contribution to PM in the different countries is made up of a large number of small contributions. Typically, the contribution from one country to immediate neighbouring areas ranges from 3% to 15%. For longer transport distances, contributions from individual countries are usually below 3%. Nevertheless, as indicated in Fig. 5.24, the sum of small individual country contributions can add up to

**Fig. 5.25. Percentage contribution of anthropogenic transboundary pollution to PM concentrations in different European countries and regions**



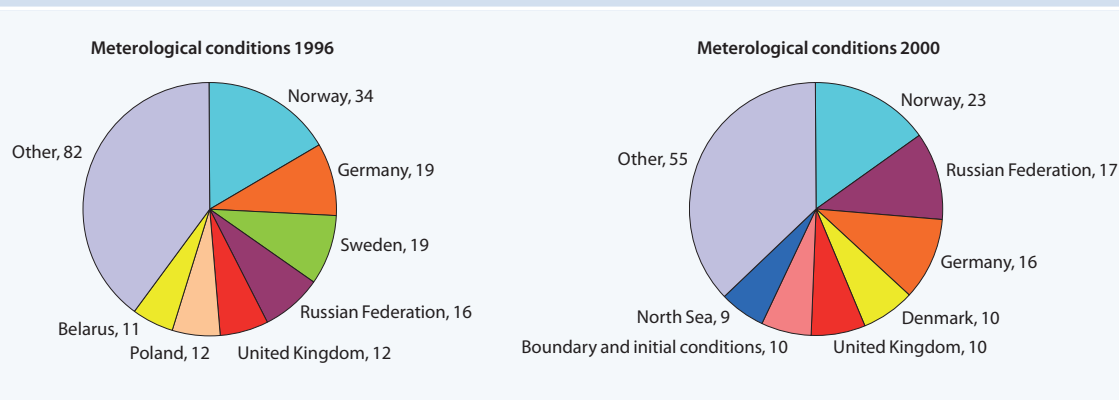
\*The former Yugoslav Republic of Macedonia.

30% of the calculated PM<sub>2.5</sub> levels, as in this example from Norway. Fig. 5.26 also shows that individual country contributions vary from year to year according to meteorological conditions. Meteorological variability can imply changes in PM levels of 15–25% (33) and it affects also the transboundary exchange between countries. For this reason, it is recommended to consider averages over different meteorological years when considering the contribution of regional transboundary pollution. Table 5.4 provides such an

average calculated for five different years (1996, 1997, 1998, 2000 and 2003) and projections for 2010.

In conclusion, the calculations from the validated unified EMEP model show that regional background concentrations of anthropogenic PM have a considerable transboundary contribution – about 60% on average over Europe for PM<sub>2.5</sub>. For primary coarse PM, the transboundary contribution is calculated to be smaller but still significant, ranging from 20% to 30% in central Europe.

**Fig. 5.26. Contributions from individual countries to predicted PM<sub>2.5</sub> levels in Norway by 2010**



Note: Individual contributions below 3% of the calculated PM<sub>2.5</sub> levels are placed together under "Other".



Table 5.4. Percentage contribution to PM<sub>2.5</sub> concentration levels in receptor countries and areas due to emissions of precursor gases and

		Emitter country or area																								
		AL	AM	AT	AZ	BA	BE	BG	BY	CH	CS	CY	CZ	DE	DK	EE	ES	FI	FR	GB	GE	GR	HR	HU	IE	IS
Albania	AL	15	0	1	0	6	0	9	0	0	11	0	1	2	0	0	1	0	1	0	0	7	1	2	0	0
Armenia	AM	0	13	0	7	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0
Austria	AT	0	0	19	0	1	1	1	0	2	1	0	5	20	0	0	1	0	5	1	0	0	2	4	0	0
Azerbaijan	AZ	0	3	0	33	0	0	1	1	0	0	0	0	0	0	0	0	0	0	4	0	0	0	0	0	0
Bosnia and Herzegovina	BA	1	0	2	0	23	0	3	0	0	10	0	2	5	0	0	1	0	2	0	0	1	7	6	0	0
Belgium	BE	0	0	1	0	0	21	0	0	1	0	0	1	15	0	0	1	0	21	8	0	0	0	0	0	0
Bulgaria	BG	1	0	1	0	2	0	32	1	0	6	0	1	2	0	0	0	0	1	0	0	3	1	3	0	0
Belarus	BY	0	0	0	0	0	0	1	25	0	1	0	1	4	1	0	0	1	1	1	0	0	0	1	0	0
Switzerland	CH	0	0	2	0	0	2	0	0	17	0	0	1	15	0	0	2	0	17	2	0	0	0	1	0	0
Serbia and Montenegro	CS	1	0	1	0	7	0	7	1	0	27	0	2	4	0	0	0	0	1	0	0	1	2	7	0	0
Cyprus	CY	0	0	0	0	1	0	5	0	0	1	6	0	0	0	0	0	0	0	0	2	0	0	0	0	0
Czech Republic	CZ	0	0	6	0	1	1	1	1	1	0	18	22	1	0	1	0	5	2	0	0	1	4	0	0	0
Germany	DE	0	0	2	0	0	3	0	1	2	0	0	3	41	1	0	1	0	10	4	0	0	0	1	0	0
Denmark	DK	0	0	1	0	0	2	0	1	0	0	0	1	18	17	0	1	0	6	7	0	0	0	1	0	0
Estonia	EE	0	0	0	0	0	1	1	8	0	0	0	1	5	1	13	0	5	2	2	0	0	0	1	0	0
Spain	ES	0	0	0	0	1	1	0	0	0	0	0	2	0	0	53	0	10	2	0	0	0	0	0	0	0
Finland	FI	0	0	0	0	0	0	0	4	0	0	0	0	3	1	3	0	25	1	1	0	0	0	0	0	0
France	FR	0	0	1	0	0	3	0	0	2	0	0	1	10	0	0	5	0	45	6	0	0	0	0	0	0
United Kingdom	GB	0	0	0	0	0	3	0	0	0	0	0	1	9	1	0	1	0	12	40	0	0	0	0	0	2
Georgia	GE	0	3	0	7	0	0	2	1	0	0	0	0	0	0	0	0	0	0	19	0	0	0	0	0	0
Greenland	GL	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Greece	GR	2	0	0	0	3	0	20	1	0	4	0	1	1	0	0	0	0	1	0	0	18	1	2	0	0
Croatia	HR	0	0	4	0	7	0	2	0	0	7	0	2	6	0	0	1	0	2	1	0	0	15	7	0	0
Hungary	HU	0	0	4	0	2	0	3	1	0	6	0	3	7	0	0	0	0	2	1	0	0	4	21	0	0
Ireland	IE	0	0	0	0	0	2	0	0	0	0	0	1	7	1	0	1	0	10	28	0	0	0	0	19	0
Iceland	IS	0	0	0	0	0	1	0	1	0	0	0	1	5	0	0	1	0	3	8	0	0	0	0	2	23
Italy	IT	0	0	2	0	3	0	2	0	1	2	0	1	4	0	0	1	0	5	1	0	1	2	2	0	0
Kazakhstan	KZ	0	0	0	0	0	0	1	2	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
Lithuania	LT	0	0	1	0	0	1	1	12	0	0	0	1	6	1	1	0	1	2	2	0	0	0	1	0	0
Luxembourg	LU	0	0	1	0	0	10	0	0	1	0	0	2	24	0	0	1	0	25	5	0	0	0	0	0	0
Latvia	LV	0	0	0	0	0	1	1	12	0	0	0	1	6	1	2	0	2	2	2	0	0	0	1	0	0
Republic of Moldova	MD	0	0	1	0	1	0	4	2	0	2	0	1	3	0	0	0	1	0	0	0	0	0	2	0	0
TFYR Macedonia*	MK	4	0	1	0	4	0	14	0	0	12	0	1	2	0	0	0	0	1	0	0	9	1	3	0	0
Malta	MT	0	0	0	0	4	0	3	0	0	2	0	0	1	0	0	2	0	3	0	0	1	1	1	0	0
Netherlands	NL	0	0	1	0	0	8	0	0	0	0	0	1	19	1	0	1	0	12	9	0	0	0	0	1	0
Norway	NO	0	0	0	0	0	1	0	2	0	0	0	1	9	5	1	1	2	4	7	0	0	0	0	1	0
Poland	PL	0	0	2	0	1	1	1	3	0	1	0	4	11	1	0	0	0	3	2	0	0	1	3	0	0
Portugal	PT	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	31	0	4	1	0	0	0	0	0	0
Romania	RO	0	0	1	0	2	0	8	1	0	5	0	1	3	0	0	0	0	1	0	0	1	1	5	0	0
Russian Federation	RU	0	0	0	0	0	0	1	3	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0
Sweden	SE	0	0	0	0	0	1	0	3	0	0	0	1	10	5	1	0	3	3	4	0	0	0	1	0	0
Slovenia	SI	0	0	8	0	2	0	1	0	1	2	0	2	9	0	0	1	0	3	1	0	0	7	4	0	0
Slovakia	SK	0	0	3	0	1	1	2	1	1	3	0	6	8	0	0	0	0	2	1	0	0	2	13	0	0
Turkey	TR	0	0	0	0	1	0	6	1	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0
Ukraine	UA	0	0	0	0	1	0	2	4	0	1	0	1	2	0	0	0	0	1	0	0	0	0	2	0	0
Remaining NE Atlantic Ocean	ATL	0	0	0	0	0	2	0	1	0	0	0	0	5	0	0	5	0	12	11	0	0	0	0	2	1
Baltic Sea	BAS	0	0	1	0	0	1	0	4	0	0	0	1	13	5	2	0	3	3	3	0	0	0	1	0	0
Black Sea	BLS	0	0	0	0	1	0	7	2	0	1	0	0	1	0	0	0	0	0	0	1	1	0	1	0	0
Mediterranean Sea	MED	1	0	1	0	3	0	6	0	0	2	0	0	2	0	0	3	0	4	0	0	3	1	1	0	0
North Sea	NOS	0	0	0	0	0	4	0	1	0	0	0	1	15	4	0	1	0	14	18	0	0	0	0	1	0
Remaining Asiatic areas	ASI	0	0	0	3	0	0	2	1	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0
North Africa	NOA	0	0	0	0	3	0	8	0	0	2	0	0	1	0	0	3	0	2	0	0	3	1	1	0	0

\*The former Yugoslav Republic of Macedonia.

## primary PM in emitter countries and areas

## Emitter country or area

Emitter country or area																													
IT	KZ	LT	LU	LV	MD	MK	MT	NL	NO	PL	PT	RO	RU	SE	SI	SK	TR	UA	ATL	BAS	BLS	MED	NOS	ASI	NOA	BIC	VOL	NAT	
7	0	0	0	0	1	5	0	0	0	3	0	5	1	0	0	1	1	3	0	0	0	10	0	0	0	1	1	0	AL
0	1	0	0	0	0	0	0	0	0	1	0	1	4	0	0	0	46	3	0	0	0	1	0	10	0	3	3	0	AM
14	0	0	0	0	0	0	0	1	0	6	0	2	1	0	2	1	0	2	0	1	0	2	1	0	0	2	0	0	AT
0	2	0	0	0	0	0	0	0	0	1	0	1	12	0	0	0	19	5	0	0	0	1	0	9	0	4	1	0	AZ
9	0	0	0	0	0	1	0	0	0	5	0	5	1	0	1	1	0	3	0	0	0	5	0	0	0	1	1	0	BA
2	0	0	1	0	0	0	0	9	0	2	0	0	1	0	0	0	0	1	1	0	0	7	0	0	3	0	1	BE	
2	0	0	0	0	2	1	0	0	0	4	0	17	3	0	0	1	2	10	0	0	1	2	0	0	1	0	0	BG	
1	0	2	0	1	1	0	0	0	0	14	0	3	15	1	0	1	0	18	0	2	0	0	1	0	0	1	0	0	BY
27	0	0	0	0	0	0	0	1	0	2	0	1	0	0	1	0	0	1	0	0	2	2	0	0	2	0	0	0	CH
5	0	0	0	0	1	1	0	0	0	6	0	10	2	0	0	1	1	5	0	0	0	3	0	0	0	1	1	0	CS
1	0	0	0	0	0	0	0	0	0	1	0	2	2	0	0	0	48	4	0	0	1	14	0	2	1	1	2	1	CY
4	0	0	0	0	0	0	0	1	0	14	0	2	1	0	1	2	0	3	0	1	0	1	1	0	0	2	0	0	CZ
3	0	0	0	0	0	0	0	5	0	7	0	1	1	1	0	0	0	2	0	2	0	0	4	0	0	2	0	0	DE
1	0	0	0	0	0	0	0	4	1	8	0	1	2	3	0	0	0	2	1	8	0	0	9	0	0	2	0	1	DK
1	0	3	0	3	0	0	0	1	1	8	0	1	19	4	0	0	0	7	0	6	0	0	1	0	0	2	0	1	EE
3	0	0	0	0	0	0	0	0	0	1	5	0	0	0	0	0	0	0	3	0	0	10	1	0	1	4	1	1	ES
0	0	1	0	1	0	0	0	1	1	5	0	1	29	5	0	0	0	4	0	5	0	0	1	0	0	3	0	1	FI
6	0	0	0	0	0	0	0	2	0	2	0	0	0	0	0	0	0	1	1	0	0	3	5	0	0	2	0	1	FR
1	0	0	0	0	0	0	0	4	0	2	0	0	1	0	0	0	0	1	3	1	0	0	9	0	0	4	0	2	GB
0	1	0	0	0	0	0	0	0	1	0	1	12	0	0	0	31	7	0	0	2	1	0	4	0	2	2	0	0	GE
0	0	0	0	0	0	0	0	0	0	1	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	94	0	2	GL
4	0	0	0	0	1	3	0	0	0	3	0	8	2	0	0	0	4	6	0	0	1	10	0	0	0	1	1	0	GR
13	0	0	0	0	0	0	0	0	0	6	0	4	1	0	3	1	0	3	0	0	0	5	0	0	0	1	0	0	HR
5	0	0	0	0	0	0	0	1	0	11	0	8	2	0	1	4	0	7	0	0	0	2	1	0	0	1	0	0	HU
1	0	0	0	0	0	0	0	3	0	2	0	0	0	0	0	0	0	0	5	1	0	0	7	0	0	5	0	4	IE
1	0	0	0	0	0	0	0	1	1	3	0	0	3	1	0	0	0	1	3	1	0	0	3	0	0	22	0	16	IS
48	0	0	0	0	0	0	0	0	0	2	0	2	1	0	1	0	0	1	0	0	0	12	0	0	0	2	1	0	IT
0	15	0	0	0	0	0	0	0	0	2	0	1	47	0	0	0	1	13	0	0	0	0	1	0	10	0	0	KZ	
1	0	13	0	1	1	0	0	1	0	18	0	2	12	2	0	1	0	10	0	4	0	0	1	0	0	1	0	0	LT
2	0	0	8	0	0	0	0	5	0	2	0	0	0	0	0	0	0	1	1	0	0	1	4	0	0	2	0	0	LU
1	0	7	0	8	0	0	0	1	0	13	0	2	15	3	0	1	0	10	0	4	0	0	1	0	0	1	0	0	LV
1	0	0	0	0	17	0	0	0	0	7	0	15	6	0	0	1	1	27	0	1	1	1	0	0	0	1	0	0	MD
4	0	0	0	0	1	13	0	0	0	4	0	8	2	0	0	1	1	4	0	0	0	5	0	0	0	1	1	0	MK
15	0	0	0	0	0	1	11	0	0	2	0	2	1	0	0	0	1	1	0	0	0	38	0	0	1	2	3	2	MT
1	0	0	0	0	0	0	0	21	0	4	0	0	1	0	0	0	0	1	1	1	0	0	10	0	0	3	0	1	NL
1	0	1	0	0	0	0	0	3	15	6	0	1	11	6	0	0	0	2	1	3	0	0	5	0	0	7	0	3	NO
2	0	1	0	0	0	0	0	1	0	40	0	2	4	1	0	2	0	7	0	2	0	0	1	0	0	1	0	0	PL
1	0	0	0	0	0	0	0	0	0	39	0	0	0	0	0	0	0	0	7	0	0	4	1	0	0	5	0	2	PT
2	0	0	0	0	3	0	0	0	0	7	0	34	3	0	0	1	1	13	0	0	1	1	0	0	0	1	0	0	RO
0	2	0	0	0	0	0	0	0	0	2	0	1	67	0	0	0	1	12	0	0	0	0	0	0	0	3	0	0	RU
1	0	1	0	0	0	0	0	2	3	9	0	1	9	21	0	0	0	3	1	7	0	0	3	0	0	3	0	1	SE
23	0	0	0	0	0	0	0	1	0	5	0	3	1	0	14	1	0	2	0	0	0	5	1	0	0	1	0	0	SI
4	0	0	0	0	0	0	0	1	0	20	0	6	2	0	1	11	0	7	0	1	0	1	1	0	0	1	0	0	SK
1	0	0	0	0	1	0	0	0	0	2	0	4	4	0	0	0	58	6	0	0	2	4	0	2	0	2	2	0	TR
1	0	0	0	0	2	0	0	0	0	8	0	5	13	0	0	1	1	46	0	1	1	0	0	0	0	1	0	0	UA
1	0	0	0	0	0	0	0	2	1	2	2	0	6	1	0	0	0	1	8	1	0	0	4	0	0	19	0	12	ATL
1	0	2	0	1	0	0	0	2	1	14	0	1	8	8	0	0	0	5	0	10	0	0	3	0	0	2	0	1	BAS
1	1	0	0	3	0	0	0	4	0	8	15	0	0	0	0	11	27	0	0	7	1	0	1	0	1	1	0	1	BLS
11	0	0	0	0	0	1	0	0	0	2	0	3	1	0	0	0	8	3	0	0	1	29	0	0	1	2	2	2	MED
1	0	0	0	0	0	0	0	7	1	4	0	0	1	1	0	0	0	1	2	2	0	0	12	0	0	3	0	3	NOS
0	3	0	0	0	0	0	0	0	0	1	0	1	10	0	0	0	32	5	0	0	1	5	0	20	1	7	3	0	ASI
7	0	0	0	0	0	1	0	0	0	2	0	3	1	0	0	0	6	3	0	0	0	24	0	0	9	6	6	1	NOA

BIC: Boundary and initial conditions; VOL: Volcanic emissions; NAT: Natural marine sources.

## 5.6 CITY-DELTA

The available atmospheric long-range transport models address European-scale air pollution with a limited spatial resolution, typically of  $50 \times 50$  km. For European-scale analysis, such a resolution is considered adequate for capturing the features of long-range transported pollutants and, with additional information, for conducting impact assessment with reasonable accuracy.

Nevertheless, it is clear that ambient concentrations of some air pollutants show strong variability at a much finer scale (e.g. in urban areas, in street canyons and at hot spots close to industrial point sources of emission) and that at least some of these differences result in small-scale variations in the effect of pollution on humans and the environment. Thus there is a need for a health impact assessment to address air quality problems that occur on a finer scale than the  $50 \times 50$ -km grid that is considered adequate for regional-scale pollution.

The CITY-DELTA model intercomparison – initiated by IIASA, the Institute for Environment and Sustainability of the Joint Research Centre at Ispra, Italy, the Norwegian Meteorological Institute and EUROTRAC-2 – conducted a systematic comparison of regional- and local-scale dispersion models. The aim of the exercise was to identify and quantify the factors that lead to systematic differences between air pollution in urban background air and rural background concentrations.

CITY-DELTA explored:

- systematic differences (deltas) between rural and urban background air quality;
- how these deltas depend on urban emissions and other factors;
- how these deltas vary across cities; and
- how these deltas vary across models.

Based on the findings of CITY-DELTA, functional relationships have been developed that allow the estimation of urban levels of pollution ( $PM_{2.5}$ ) as a function of rural background concentrations and local factors, such as local emission densities of low-level sources and meteorological factors that represent the ventilation conditions in a city. The analysis addresses the response of health-relevant parameters

of pollution (i.e. mostly long-term concentrations of  $PM_{2.5}$ ) to changes in local and regional precursor emissions, including the formation of secondary aerosols. This enables the generic analysis of urban air quality for a large number of European cities, based on information available in the RAINS model framework. For the time being, the methodology has been implemented in the RAINS model for 150 European cities.

## References

1. CITY-DELTA European Modelling Exercise. An inter-comparison of long-term model responses to urban-scale emission-reduction scenarios (<http://rea.ei.jrc.it/netshare/thunis/citydelta>, accessed 22 October 2005).
2. Tsyro S. Model assessment of particulate matter in Europe in 2002. In: Torseth K, ed. *Transboundary particulate matter in Europe*. Kjeller, Norwegian Institute for Air Research, 2004 (EMEP Status Report 4/2004).
3. *Second position paper on particulate matter. CAFE Working Group on Particulate Matter*. Brussels, European Commission, 2004.
4. *Air quality. Determination of the  $PM_{10}$  fraction of suspended particulate matter. Reference method and field test procedure to demonstrate reference equivalence of measurement methods*. Brussels, European Committee for Standardization, 1998 (document EN 12341).
5. Snel SC, Larssen S. *Trends in  $PM_{10}$  concentrations in European Cities 1997–2002*. Kjeller, Norwegian Institute for Air Research, 2004.
6. Larssen S et al. *Report on data analysis and comparison with emission estimates*. Bilthoven, RIVM, 2005.
7. Johansson, C. (2002) Source contributions of PM in Sweden – implications for abatement strategies. Institute of Applied Environmental Research (ITM). Stockholm. Presentation at ‘Particles in the size of 2.5 to 10  $\mu m$  in urban areas’ workshop, in support of the Clean Air for Europe (CAFE) programme of DG ENV in Berlin, Germany, November 4–6, 2002.

8. Gustavsson M. [Emission of wear and resuspension particles in the road environment]. Linköping, Swedish National Road Administration, 2003 (Report No. VTI 944) (in Swedish).
9. Emissions of primary particles and secondary particulate precursors (CSI 003) – May 2005 Assessment. Copenhagen, European Environment Agency, 2005 ([http://themes.eea.eu.int/IMS/ISpecs/ISpecification20041001123025/IAssessment1116511151442/view\\_content](http://themes.eea.eu.int/IMS/ISpecs/ISpecification20041001123025/IAssessment1116511151442/view_content), accessed 25 November 2005).
10. Stedman JR. The use of receptor modelling and emission inventory data to explain the downward trend in UK PM<sub>10</sub> concentrations. *Atmospheric Environment*, 2002, 36:4089–4101.
11. Eerens H et al. *European environmental outlook 2005: background document air quality 1990–2030*. Copenhagen, European Environment Agency, 2005 (EEA/ETC Technical Paper 2005/2).
12. *Review of the unified EMEP model*. Geneva, United Nations Economic Commission for Europe, 2004 (document EB.AIR/GE.1/2004/6) ([http://www.unece.org/env/emep/emep28\\_docs.htm](http://www.unece.org/env/emep/emep28_docs.htm), accessed 25 October 2005).
13. Simpson D et al. *Transboundary acidification, eutrophication and ground level ozone in Europe. Part II. Unified EMEP model performance*. Oslo, Norwegian Meteorological Institute, 2003 (EMEP Report 1/2003).
14. Tarrasón L, ed. *Transboundary acidification, eutrophication and ground level ozone in Europe. Part III. Source–receptor calculations*. Oslo, Norwegian Meteorological Institute, 2003 (EMEP Report 1/2003).
15. Pirjola L et al. A monodisperse aerosol dynamics module – a promising candidate for use in long-range transport models: box-model tests. *Journal of Geophysical Research*, 2003, 108(D9):4258.
16. Wind P et al. *Development of a modelling system able to link hemispheric-regional and local air pollution*. Oslo, Norwegian Meteorological Institute, 2002 (Joint MSC-W & NILU Note 5/2002).
17. Wind P et al. *Further development of a modelling system able to link hemispheric-regional and local air pollution*. Oslo, Norwegian Meteorological Institute, 2003 (MSC-W Technical Report 2/2003).
18. Vestreng V, Adams M, Goodwin J. *EMEP/EEA Joint Review Report, Inventory Review 2004 Emission Data reported to CLRTAP and under the NEC Directive*. Oslo, Norwegian Meteorological Institute, 2004 (MSC-W Technical Report 1/2004).
19. Tarrasón L et al. *Transboundary acidification, eutrophication and ground level ozone in Europe*. Oslo, Norwegian Meteorological Institute, 2004 (EMEP Status Report 1/2004).
20. Tsyro S, Kahnert M, Tarrasón L. Physical characterisation of particulate matter across Europe in 2000 and 2001. In: Kahnert M, Tarrasón L, eds. *Transboundary particulate matter in Europe*. Kjeller, Norwegian Institute for Air Research, 2003 (EMEP Report 4/2003).
21. *Draft EMEP monitoring strategy and measurement programme 2004–2009*. Geneva, United Nations Economic Commission for Europe, 2004 (document EB.AIR/GE.1/2004/5) ([http://www.unece.org/env/emep/emep28\\_docs.htm](http://www.unece.org/env/emep/emep28_docs.htm), accessed 27 October 2005).
22. Putaud JP et al. A European aerosol phenomenology – 1: physical characteristics of particulate matter at kerbside, urban, rural and background sites in Europe. *Atmospheric Environment*, 2004, 38:2561–2577.
23. Putaud JP et al. A European aerosol phenomenology – 2: chemical characteristics of particulate matter at kerbside, urban, rural and background sites in Europe. *Atmospheric Environment*, 2004, 38:2579–2595.
24. Yttri KE, Tørseth K. Chemical characterisation and mass concentration of ambient aerosol at the EMEP site in Birkenes (southern Norway) – a one-year study. In: Kahnert M ed. *Measurements of particulate matter. Status report 2002*. Kjeller, Norwegian Institute for Air Research, 2002:20–25 (EMEP Report 4/2002).

25. Zappoli S et al. Inorganic, organic and macromolecular components of fine aerosol in different areas of Europe in relation to their water solubility. *Atmospheric Environment*, 1999, 33:2733–2743.
26. Neusüß C et al. Characterization and parameterization of atmospheric aerosol number-, mass-, and chemical size distributions in central Europe during LACE 98 and MINT. *Journal of Geophysical Research*, 2002, 107(D21):8127 (doi: 10.29/2000JD000514).
27. Schwela D, Morawska L, Kotzias D, eds. *Guidelines for concentration and exposure-response measurement of fine and ultra fine particulate matter for use in epidemiological studies*. Geneva, World health Organization, 2002 (document EUR 20238 EN) (<http://whqlibdoc.who.int/hq/2002/a76621.pdf>, accessed 26 November 2005).
28. Tsyro S. To what extent can aerosol water explain the discrepancy between model calculated and gravimetric PM<sub>10</sub> and PM<sub>2.5</sub>? *Atmospheric Chemistry and Physics*, 2005, 5:515–532.
29. Kupiainen K, Klimont Z. *Primary emissions of submicron and carbonaceous particles in Europe and the potential for their control*. Laxenburg, International Institute for Applied Systems Analysis, 2004 (IIASA Interim Report IR-04-079).
30. Simpson D, Makar P. Modelling SOA and OC in Europe. In: Torseth K, ed. *Transboundary particulate matter in Europe*. Kjeller, Norwegian Institute for Air Research, 2004 (EMEP Status Report 4/2004).
31. Pun BK et al. Uncertainties in modeling secondary organic aerosols. Three dimensional modeling studies in Nashville/Western Tennessee. *Environmental Science and Technology*, 2003, 37:3647–3661.
32. Szidat S et al. Radiocarbon (C-14)-deduced biogenic and anthropogenic contributions to organic carbon (OC) of urban aerosols from Zurich, Switzerland. *Atmospheric Environment*, 2004, 38:4035–4044.
33. Tarrasón L et al. Meteorological years available for use under the review of the National Emission Ceilings Directive (NECD) and the Gothenburg Protocol. In: *Transboundary acidification, eutrophication and ground level ozone in Europe in 2003*. Kjeller, Norwegian Institute for Air Research, 2005 (EMEP Report 1/2005).

## 6. Population exposure

### KEY MESSAGES

- Ambient concentrations of PM from long-range transport, as estimated by secondary sulfate, are representative of population exposure. The differences between the PM measurements at centrally located monitors and personal exposure measurements are due to proximity to local sources, such as traffic emissions, as well as personal activities or residential ventilation characteristics, which may be less important when averaging across the population.
- Although both primary and secondary PM contribute to long-range transport, available modelling results indicate that secondary PM dominates the exposure and is more difficult to control, even under the maximum feasible reduction scenario.
- Quantitative knowledge about sources of particle emissions plays an important role in fine tuning these exposure estimates and in finding the best control strategy for reducing risks.
- Current knowledge on sources of population exposure is based on a very limited number of exposure assessment studies. Large uncertainties were noted in the source apportionment analyses of personal exposure, owing to the limited sample size. Further exposure assessment studies should be conducted to identify contributions from long-range transport to population PM exposure.

### 6.1 Introductory remarks

As noted in Chapter 5, the contribution of long-range transport to PM levels (including urban environments) in ambient air is estimated either by measuring outdoor concentrations of PM through monitoring networks or by using appropriate ambient air quality models with available emission estimates. Since people spend the majority of their time indoors, and total exposure often exceeds indoor and outdoor measurements, the adequacy of using outdoor PM measurements or ambient modelling results to represent personal exposure to PM<sub>2.5</sub> has often been questioned.

Conventionally, personal exposure of individuals is calculated using air pollution levels in the different micro-environments, weighted with the time-activity pattern. More recently, personal exposure to PM<sub>2.5</sub> has been separated into two components for regulatory purposes: exposure to ambient-generated particles ( $E_{ag}$ ), such as regional air pollution or secondary aerosols, vehicle exhaust, wood smoke and road dust and non-ambient-generated particles, including particles

produced by indoor and personal activities (cooking, vacuum cleaning, etc.). (1–3).  $E_{ag}$  is dominated by home ventilation and can be estimated from ambient concentrations ( $C_a$ ) multiplied by the fraction of time spent outdoors ( $F_o$ ) and the fraction of time spent indoors ( $F_i$ ) modified by the particle infiltration efficiency ( $F_{inf}$ ), i.e.  $E_{ag} = C_a \times (F_o + F_i \times F_{inf})$ . The overall modification for  $C_a$  is usually referred to as the ambient exposure attenuation factor.

Recent exposure panel studies have shown correlations between personal exposure and ambient measurements within individuals; these correlations are similar in groups of people with various health conditions (e.g. normal vs susceptible populations) (4,5).  $E_{ag}$  accounts for more than 50% of total personal PM<sub>2.5</sub> exposure, explaining the observed correlations between personal exposure and ambient measurements within individuals (3). Several papers published recently further explore sources of  $E_{ag}$  in both general and susceptible populations (see section 6.2).

The concept of different levels of contributions can also be applied to  $E_{ag}$ , which includes a contribution

from regional air pollution in addition to that from urban background. Even higher levels may occur at hot spots such as busy roads. Special environments or activities, such as commuting, may further contribute to exposure.

The differences in pollution levels between various microenvironments depend on the pollutant in question. For secondary pollutants such as sulfate, gradients are rather low and it seems reasonable to assume that regional background levels are important determinants of (personal) exposure. For primary PM components with a limited atmospheric lifetime, such as number concentration of ultrafine particles, however, it is expected that regional contributions will contribute only marginally to overall exposure.

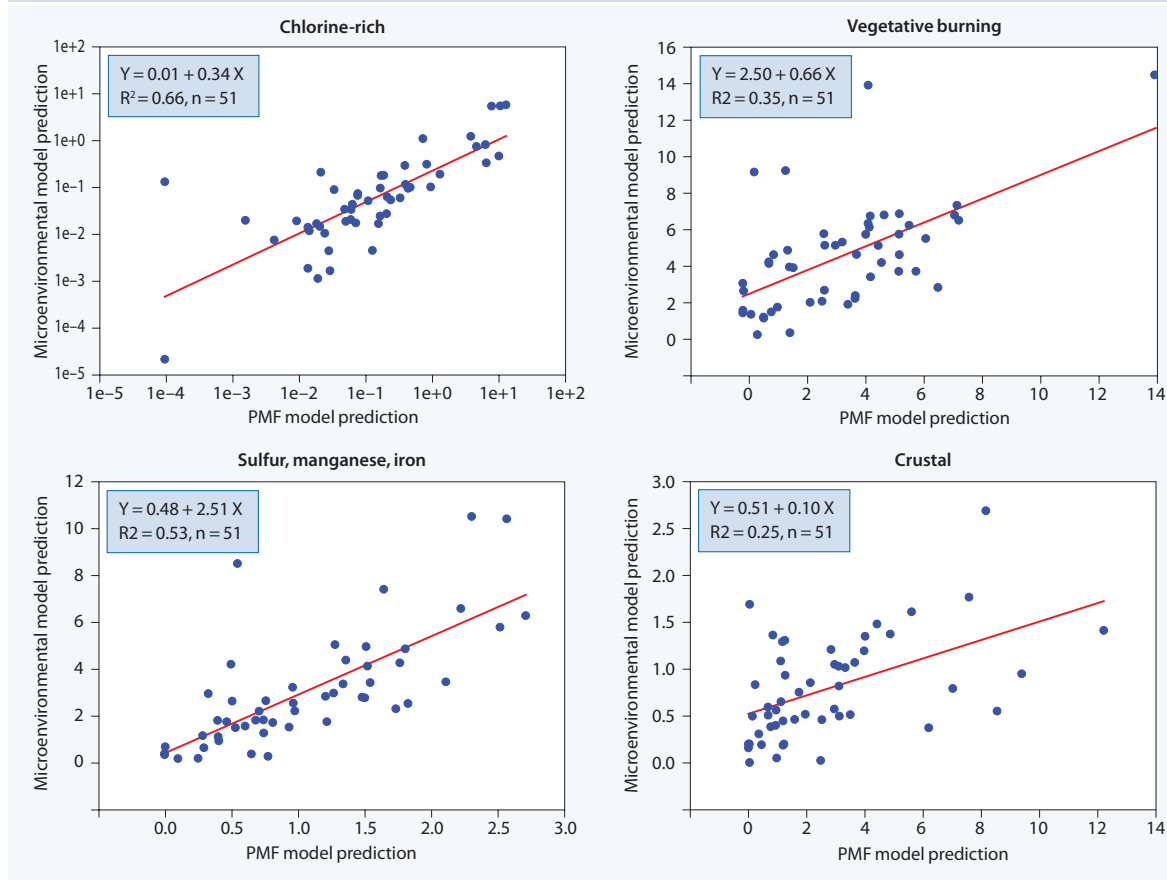
As discussed in section 3.6, there are still uncertainties as to the importance of various PM constitu-

ents in causing the health effects observed in (epidemiological) studies (6,7). Based on the findings of the WHO Systematic Review project and the recommendations of the Task Force on Health (8,9), the effects of PM on mortality will be assessed using PM mass ( $PM_{2.5}$  or  $PM_{10}$ ) as indicator. This chapter explores the relevance of available estimates of ambient PM levels as indicators of population exposure to PM, and in particular the links of exposure with PM from long-range transport of pollution.

## 6.2 Contribution of long-range transport of pollution to personal PM exposure

Few studies have investigated the contribution of different sources to personal PM exposure. In the limited number of studies carried out, exposure to long-range transport of PM was not specifically identified,

**Fig. 6.1. Personal exposures to specific sources vs exposure estimates (in  $\mu\text{g}/\text{m}^3$ ) using a microenvironmental model that includes indoor, outdoor and central-site source estimates and time-activity information**



Source: Larson et al. (12).

although personal exposure to secondary sulfate was estimated. As secondary sulfate is an important component of the long-range transport, it is used here as a rough indicator of the long-range transport of PM.

Yakovleva et al. (10) investigated sources of microenvironmental and personal exposures in the Particle Total Exposure Assessment Methodology (PTEAM) study at Riverside, California. Using a three-way positive matrix factorization method, they found that major sources of personal PM<sub>10</sub> included resuspended indoor soil (31%), emission of nonferrous metals and motor vehicle exhaust (10%), secondary sulfate (17%) and personal activities (15%). The ambient exposure attenuation factor for secondary sulfate was 0.77, i.e. 77% of the outdoor sulfate penetrated indoors and was encountered by the subjects. In contrast to other PM sources, exposure to secondary sulfate did not differ by time of day (day or night), having a job outside the house, or the occurrence of indoor cooking or vacuum cleaning.

Hopke et al. (11) examined sources of personal PM<sub>2.5</sub> exposure in the 1998 Baltimore PM epidemiology exposure study among elderly subjects. They classified the sources to three “ambient” categories: sulfate (46%), unknown (14%) and soil (3%), and to three “internal” sources including gypsum (1%), personal activities (36%) and personal care products (0.4%). Their estimates indicated that the ambient exposure attenuation factor for secondary sulfate was 72%.

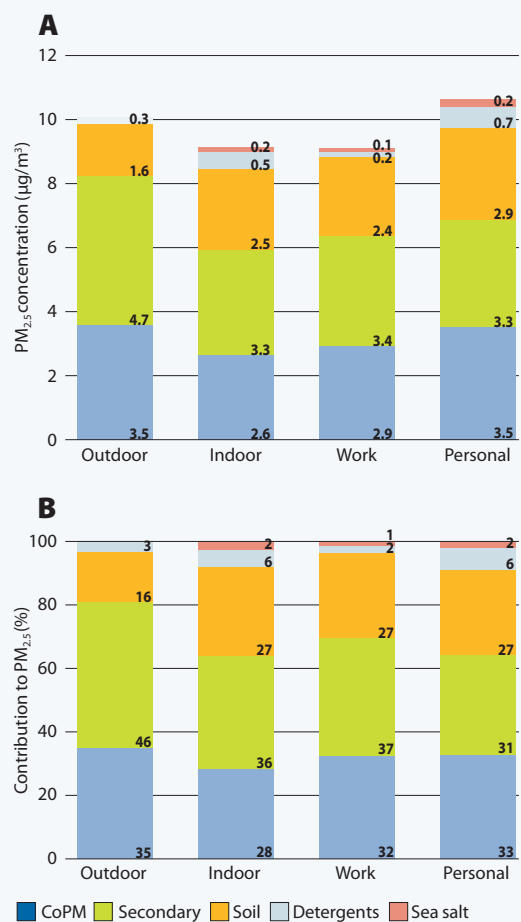
Larson and co-workers (4,12) examined sources of microenvironmental and personal exposures among susceptible subpopulations in the Seattle exposure panel study. Using a three-way positive matrix factorization method, they identified four major personal PM<sub>2.5</sub> sources including burning of vegetable matter (41%), mobile (7%), secondary sulfate (19%) and crustal materials (32.5%). The ambient exposure attenuation factor for secondary sulfate is 0.7. In addition, personal exposure to secondary sulfate can be relatively easily predicted (Fig. 6.1b) using the indoor and outdoor sulfate measurements and time-activity information, as compared with exposures to other sources (Fig. 6.1a,c,d).

Koistinen et al. (13) investigated the contribution of different PM<sub>2.5</sub> sources to microenvironmental and personal exposure as part of the EXPOLIS study in

Helsinki, Finland. Their combined principal component and mass reconstruction method identified five major sources contributing to personal PM<sub>2.5</sub> exposure, including traffic and other combustion sources (33%), long-range transported inorganic secondary PM (31%), soil (27%), detergents (6%) and sea salt (2%) (Fig. 6.2). The ambient exposure attenuation factor for LRT secondary sulfate is 0.67.

All exposure source apportionment studies described above have identified secondary sulfate as one of the major contributing sources to personal

**Fig. 6.2. Absolute (A) and percentage (B) contributions of sources to PM<sub>2.5</sub> in residential outdoor, residential indoor and indoor workplace microenvironments and in personal 48-hour exposures of participants not exposed to environmental tobacco smoke**



Note: CoPM = traffic and other combustion-related PM.  
Source: Koistinen et al. (13).



exposure, ranging between 17% in the western United States and 46% in the sulfate-rich eastern United States, with a middle range contribution (31%) for Helsinki. All studies reported a high ambient exposure attenuation factor for sulfate exposure, ranging between 67% and 77%. The high correlations between personal and central-site measurements of ambient originated  $PM_{2.5}$ , especially sulfate, indicate that central-site measurements are a good surrogate for exposure to particles generated by ambient sources ( $E_{ag}$ ), especially long-range transported PM. Although secondary aerosols tend to be spatially homogeneous,  $PM_{2.5}$  and ultrafine PM from local combustion sources have been shown to be distributed unequally throughout the city. Thus, central-site measurements may not predict equally well personal exposure to other PM sources, such as primary combustion sources and crustal materials.

Findings from the aforementioned exposure assessment studies indicate that most epidemiological study results concerning concentration–response relationships are good estimates for association of health with personal exposure to ambient originated PM. Thus the health impact assessment based on estimates of ambient concentrations of PM is both consistent with the original evidentiary studies and relates to personal exposure to ambient originated PM (14).

### 6.3 Population data

As a part of the EXPOLIS study, Kruize et al. (15) developed a stochastic exposure-modelling framework using the @Risk add-on software in Microsoft Excel to compare exposure distributions of different subpopulations and different scenarios. Their model was based on the conventional microenvironmental model, with input parameters described by probability distributions. Simulations were performed for population exposures to  $PM_{2.5}$  in Athens, Basel, Helsinki and Prague. Additional modelling was conducted for rural (59.2%) vs urban (40.8%) populations and for four age groups (children, working adults, non-working adults and the elderly) to estimate the Dutch population exposure to  $PM_{10}$ .

Table 6.1 presents comparison of the population  $PM_{2.5}$  exposure simulated and measured in the four EXPOLIS cities. Although the order of mean exposure levels between cities was correctly reflected by the simulation, the estimates exaggerated the differences between the extreme cities (Athens vs Helsinki). The order of simulated standard deviations in the cities was different than the order of observed values, with the differences between the observed and simulated means varying by city. For simulated exposure of the Dutch subpopulations, this study showed small differences (about  $3 \mu\text{g}/\text{m}^3$ ) between rural and urban environments, caused solely by the differences in ambient concentrations. The sensitivity analysis results also reinforced a conclusion that the

**Table 6.1. Summary statistics of simulation results and corresponding observed exposures for  $PM_{2.5}$  exposures in 4 EXPOLIS cities**

	Helsinki		Basel		Prague		Athens	
	Simulated 2000	Observed 193	Simulated 2000	Observed 193	Simulated 2000	Observed 193	Simulated 2000	Observed 193
<i><math>PM_{2.5}</math> exposures (<math>\mu\text{g}/\text{m}^3</math>)</i>								
Mean	13	16	25	31	37	35	43	37
SD	30	19	20	43	30	26	30	25
25th centile	6	6	14	15	22	19	28	20
50th centile	9	10	20	20	30	25	37	29
75th centile	15	18	30	30	43	42	52	41
90th centile	24	33	43	50	62	57	69	70
95th centile	34	43	54	74	75	82	82	74

Source: Kruize et al. (15).

**Table 6.2. Average population exposure to PM<sub>10</sub> in Switzerland by source group**

Scenario	Source group	Average PM <sub>10</sub> population exposure					
		Primary		Secondary		Total	
		µg/m <sup>3</sup>	%	µg/m <sup>3</sup>	%	µg/m <sup>3</sup>	%
2000	Anthropogenic, Swiss	6.57	33.6	2.88	14.7	9.45	48.3
	Anthropogenic, foreign	3.14	16.0	5.54	28.3	8.68	44.4
	Biogenic and geogenic	1.05	5.3	0.39	2.0	1.44	7.4
	Total	10.75	54.9	8.81	45.1	19.56	100.0
2010 “business as usual”	Anthropogenic, Swiss	6.09	34.5	2.80	15.8	8.89	50.3
	Anthropogenic, foreign	2.56	14.5	4.78	27.1	7.34	41.6
	Biogenic and geogenic	1.05	5.9	0.39	2.2	1.44	8.1
	Total	9.70	54.9	7.98	45.1	17.68	100.0
2010 “maximum feasible reduction”	Anthropogenic, Swiss	3.89	27.1	2.63	18.3	6.52	45.4
	Anthropogenic, foreign	2.16	15.0	4.25	29.6	6.40	44.6
	Biogenic and geogenic	1.05	7.3	0.39	2.7	1.44	10.0
	Total	7.10	49.4	7.26	50.6	14.36	100.0

Source: Swiss Agency for the Environment, Forests and Landscape (16).

ambient concentrations appeared to have the largest influence on the population exposure distribution. Furthermore, similar to the findings in the exposure panel studies described earlier, the Dutch results also demonstrated small variations in the exposure distributions of the subpopulations (max. difference of 1 µg/m<sup>3</sup>), in spite of the differences in time–activity patterns.

The Swiss Agency for the Environment, Forests and Landscape (16) performed surveys, measurements and source–receptor dispersion modelling to evaluate the level and evolution of primary and secondary PM<sub>10</sub> in Switzerland on a rectangular grid with 200-metre mesh size. Modelling was also performed for PM<sub>2.5</sub> by applying PM<sub>2.5</sub> : PM<sub>10</sub> ratios to the PM<sub>10</sub> emission loads and a reduced particle deposition velocity. For transported PM<sub>2.5</sub>, the PM<sub>2.5</sub> : PM<sub>10</sub> ratio was assumed to be 0.90. Area statistics and population exposure were determined using the annual average ambient concentration per grid cell and population density.

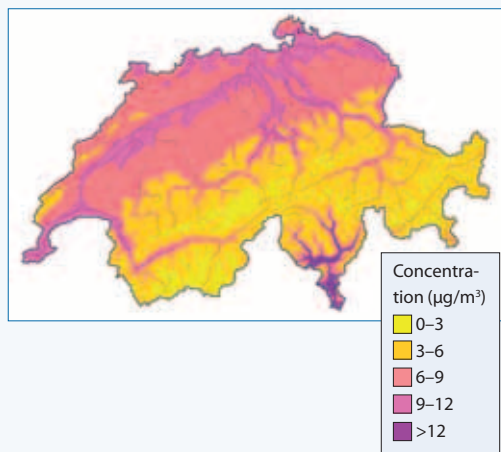
The average Swiss population exposure, based on the 2000 census and the estimated change of population size through 2010, was computed for total PM<sub>10</sub> for different source groups (Table 6.2). The average exposure decreased from 19.6 µg/m<sup>3</sup> in 2000 to 17.7 µg/m<sup>3</sup> and 14.4 µg/m<sup>3</sup> in 2010 for the “business

as usual” and “maximum feasible reduction” scenarios, respectively. Foreign (long-range transported) anthropogenic particles accounted for approximately 8.7 µg/m<sup>3</sup> (44%) of the total population exposure in 2000 and remained at a similar percentage contribution in 2010 under both scenarios. A higher reduction in the foreign anthropogenic PM was expected from the primary than from the secondary sources. In addition, contribution of long-range transported PM<sub>10</sub> varied by location, with the highest contribution (>12 µg/m<sup>3</sup>) occurring in southern Switzerland from sources in northern Italy (Fig. 6.3).

The predicted annually averaged PM<sub>10</sub> and PM<sub>2.5</sub> concentrations were compared to measurements collected throughout Switzerland. For PM<sub>10</sub>, the overall mean predicted and measured values agreed well (22.6 µg/m<sup>3</sup> measured vs 21.4 µg/m<sup>3</sup> predicted). The overall Pearson’s correlation coefficient (*r*) was 0.74 (*n* = 44 monitors), ranging between 0.46 in Ticino (*n* = 5) and 0.69 in the alpine area (*n* = 9). The lower correlations were due to the local effects that were not accounted for in the model. For PM<sub>2.5</sub>, only six data points were available for the comparison (mean difference 0.9 µg/m<sup>3</sup>, *r* = 0.90).

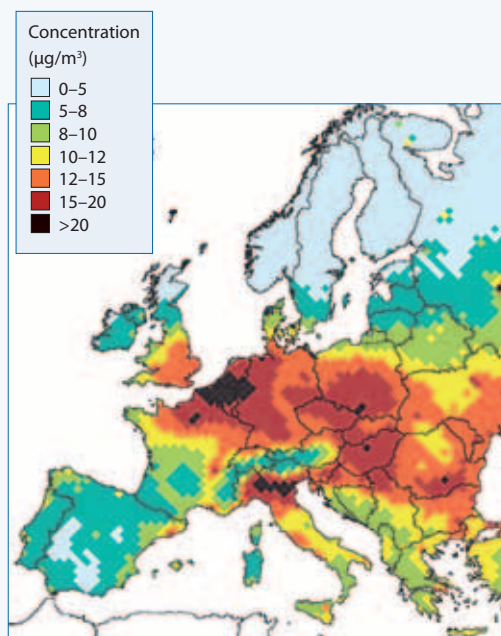
Within the CAFE programme, the EMEP model has been used to calculate mean PM<sub>2.5</sub> concentration from anthropogenic sources in each of 50 × 50-km

**Fig. 6.3. Long-range transported  $PM_{10}$  in Switzerland in 2000 due to imported primary and secondary particles**



Source: Swiss Agency for the Environment, Forests and Landscape (16).

**Fig. 6.4. Identified anthropogenic contribution to modelled grid-average annual mean  $PM_{2.5}$  concentrations in Europe for emissions in 2000**



Note: Includes contributions of primary and secondary inorganic aerosols to  $PM_{2.5}$ . Calculations are based on 1997 meteorology.

grid and its changes resulting from the changes in the precursor emissions (primary  $PM_{2.5}$ , sulfur dioxide, nitrogen oxides and ammonia). Fig. 6.4 presents the model estimates of the identified anthropogenic fraction of  $PM_{2.5}$  for the year 2000. These estimates, together with the data on grid-specific population density, were used to describe distribution of population exposure to regional  $PM_{2.5}$  from anthropogenic sources.

Current knowledge on the relationships of personal and population exposure to PM from specific sources is based on a very limited number of exposure assessment studies. Large uncertainties were noted in the source apportionment analyses for personal exposure owing to the limited sample size. Further exposure assessment studies should continue to identify contributions from long-range transported PM to population PM exposure. Population-based source-specific exposure modelling has not been fully developed and validated, especially for the European populations. The available population exposure model, from the EXPOLIS study, is not source-specific, while the Swiss source-receptor dispersion model is not exposure-specific. The Swiss model is based on ambient concentration estimates and does not take into account PM infiltration and the population time-activity pattern. Furthermore, long-term exposure to specific sources has not been investigated. The adequacy of such a modelling approach is unknown owing to a lack of long-term population exposure data.

## References

1. Wilson WE et al. Estimating separately personal exposure to ambient and nonambient particulate matter for epidemiology and risk assessment: why and how. *Journal of the Air & Waste Management Association*, 2000, 50:1167–1183.
2. Allen R et al. Use of real-time scattering data to estimate the contribution of infiltrated and indoor-generated particles to indoor air. *Environmental Science & Technology*, 2003, 37:3484–3492.
3. Allen RW et al. Estimated hourly personal exposures to ambient and non-ambient

- particulate matter among sensitive populations in Seattle, Washington. *Journal of the Air & Waste Management Association*, 2004, 54:1197–1211.
4. Liu L-J et al. Exposure assessment of particulate matter for susceptible populations in Seattle, WA. *Environmental Health Perspectives*, 2003, 111:909–918.
  5. Williams R et al. The Research Triangle Park particulate matter panel study: PM mass concentration relationships. *Atmospheric Environment*, 2003, 37:5349–5363.
  6. *Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2003 (document EUR/03/5042688) (<http://www.euro.who.int/document/e79097.pdf>, accessed 13 May 2004).
  7. *Health aspects of air pollution – answers to follow-up questions from CAFE. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2004 (<http://www.euro.who.int/document/E82790.pdf>, accessed 13 May 2004).
  8. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2003 (document EB.AIR/WG.1/2003/11) (<http://www.unece.org/env/documents/2003/eb/wg1/eb.air.wg1.2003.11.pdf>, accessed 1 October 2005).
  9. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2004 (document EB.AIR/WG.1/2004/11) (<http://www.unece.org/env/documents/2004/eb/wg1/eb.air.wg1.2004.11.e.pdf>, accessed 1 October 2005).
  10. Yakovleva EP et al. Positive matrix factorization in determining sources of particles measured in EPA's Particle TEAM Study. *Environmental Science & Technology*, 1999, 33:3645–3652.
  11. Hopke PZ et al. Receptor modeling of ambient and personal exposure samples: 1998 Baltimore Particulate Matter Epidemiology-Exposure Study. *Atmospheric Environment*, 2003, 37:3289–3302.
  12. Larson TT et al. Source apportionment of indoor, outdoor and personal PM<sub>2.5</sub> in Seattle, WA using positive matrix factorization. *Journal of the Air & Waste Management Association*, 2004, 54:1175–1187.
  13. Koistinen KJ et al. Sources of fine particulate matter in personal exposures and residential indoor, residential outdoor and workplace microenvironments in the Helsinki phase of the EXPOLIS study. *Scandinavian Journal of Work, Environment & Health*, 2004, 30(Suppl. 2):32–46.
  14. *Quantification of the health effects of exposure to air pollution. Report of a WHO Working Group, Bilthoven, Netherlands 20–22 November 2000*. Copenhagen, WHO Regional Office for Europe, 2001 (document EUR/01/5026342) (<http://www.euro.who.int/document/e74256.pdf>, accessed 2 November 2005).
  15. Kruize H et al. Description and demonstration of the EXPOLIS simulation model: two examples of modeling population exposure to particulate matter. *Journal of Exposure Analysis and Environmental Epidemiology*, 2003, 13:87–99.
  16. *Modelling of PM<sub>10</sub> and PM<sub>2.5</sub> ambient concentrations in Switzerland, 2000 and 2010*. Berne, Swiss Agency for the Environment, Forests and Landscape, 2003 (Environmental Documentation No. 169 (Air)).



## 7. The approach to estimating risk

### KEY MESSAGES

- Following the conclusions of WHO working groups and decisions of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution, the health impact assessment of PM is based on annual average PM<sub>2.5</sub> concentrations provided by the EMEP model.
- The main indicator of health impact chosen for the analysis is mortality. Concentration–response function is based on the largest available cohort study, including 0.5 million people followed for 16 years. The increase in risk of all-cause mortality by 6% per 10 µg/m<sup>3</sup> PM<sub>2.5</sub> resulting from this study was recommended for use in health impact assessment conducted for this analysis.
- Quantification of impacts of PM exposure on morbidity is less precise than that for mortality, since the database concerning concentration–response functions and background rates of health end-points is poorer. However, selected estimates of impacts on morbidity are included in the analysis.

This chapter provides an overview of the approach selected to quantify the health impact from exposure to PM in Europe. The main steps in conducting a health impact assessment are lined out in Fig. 7.1. The sources of information for deriving air pollution data and exposure estimates are described in previous chapters. The selection of appropriate health end-points, air pollution indicators and concentration–response functions are outlined in this chapter. The approach used in the present analysis follows those used and reviewed previously (1–6). The methodology was also reviewed, discussed and approved by experts and stakeholders at two meetings of the Joint WHO/Convention Task Force (7,8). The International Institute for Applied Systems Analysis (IIASA) elaborated the details of estimation of impacts on life expectancy and included it in the RAINS model.

### 7.1 Choice of PM indicator

The WHO systematic review agreed that PM<sub>2.5</sub> is strongly associated with mortality and other effects (9). As summarized in Chapter 3, this recommendation was based on the evidence from the American Cancer Society (ACS) study, was consistent with results from one available, smaller European study, and was corroborated by toxicological evidence. Based on this conclusion, the Task Force recommend-

ed the use of PM<sub>2.5</sub> as the main indicator of exposure in impact assessment of PM within the RAINS model (7).

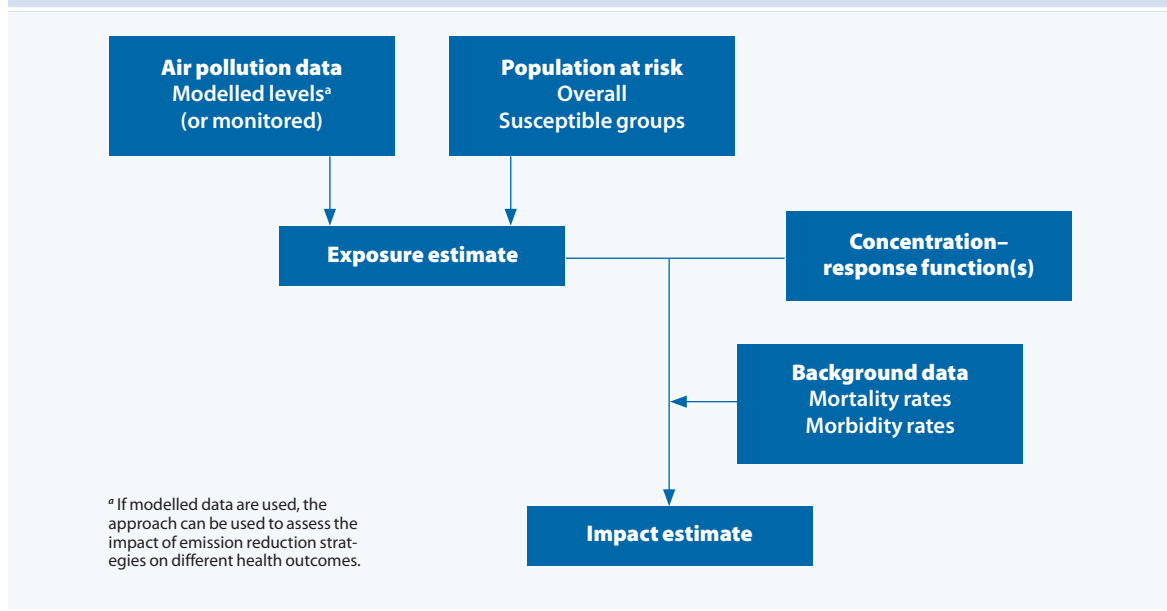
PM-related health effects have been observed in both short-term and long-term epidemiological studies. For this assessment it was decided to choose the annual average of PM<sub>2.5</sub> as the exposure metric, since it indicates long-term level of exposure and is consistent with exposure indicators used in the ACS study, which provided the concentration–response function for assessment of impacts of PM on mortality and life expectancy.

### 7.2 Mortality as the most relevant health end-point

Exposure to outdoor air pollution is associated with a broad spectrum of acute and chronic health effects, ranging from irritant effects to death (9,10). While all these outcomes are potentially relevant for health impact assessment, all-cause mortality was chosen as the essential health effect for this assessment, for the following reasons.

1. *Strength of evidence.* There is a large a number of (European and non-European) time series studies showing an association between mortality and exposure to PM. In addition, there are a few

Fig. 7.1. Schematic presentation of the main steps of a health impact assessment



long-term studies showing significant associations between exposure to PM and a reduction in life expectancy.

2. *Consistent definition of the end-point.* Mortality per se is a well defined event that is registered in all European countries. For this reason, epidemiologists have frequently assessed the effect of air pollution on mortality (either all-cause or cause-specific). Other outcomes (such as bronchitis) are subject to very large variations in severity, and without such qualification their health impact is difficult to assess. The definitions of other possible health outcomes, such as restricted activity days, use of primary care services, etc., are likely to vary with national culture and among health care systems.
3. *Availability of baseline occurrence rates.* In all European countries, mortality data are collected or estimated using consistent methods. This is not necessarily the case for some important morbidity outcomes such as number of asthma attacks.
4. *Importance of the end-point in terms of health impact.* Air pollution has an impact on other health end-points; nevertheless, there are studies showing that mortality, quantified as numbers of

deaths or reduction in life expectancy, is the most important health impact in terms both of disability-adjusted life years (DALYs) (11) and the monetary value of the impact (12).

It should be stressed, however, that the focus on mortality does not imply that there are no other relevant health end-points, some of which impose a considerable health burden.

### 7.3 Use of cohort and time series study results in risk estimation

Associations between air pollution exposure and mortality have been assessed mainly through two types of epidemiological study:

- time series studies, which investigate the association between daily mortality and variations in recent PM concentrations; and
- cohort studies, which follow large populations for years and relate mortality to exposure to air pollution over extended periods.

Both designs provide estimates of relative risk of mortality that can be associated with exposure to air pollution. It is important to point out that the relative risks derived from time series and cohort studies have

different meanings, but refer to similar effects of air pollution.

The WHO Working Group (6) concluded that both designs could contribute useful, albeit different, information for health impact assessment. Through their design, time series studies yield estimates of deaths due to recent exposure, in all likelihood among those who are frail due either to chronic disease or to some transient condition. Because such studies cannot quantify chronic effects of long-term exposure, some deaths attributable to air pollution will be missed and the extent to which air pollution advances the time of death cannot be quantified (4,13). For this reason, the use of risk estimates from time series studies of daily mortality will underestimate the impact of pollution exposure on both the attributable numbers of deaths and average lifespan in a given population. Therefore, the WHO Working Group (6) concluded that the most complete estimates of both attributable numbers of deaths and average reduction in lifespan associated with exposure to air pollution are those based on cohort studies. Such studies include not only those whose deaths were advanced by very recent exposure to air pollution, but also those who died from chronic disease caused by long-term exposure.

#### 7.4 Concentration–response function

Based on the findings of the WHO systematic review, presented in Chapter 3, the Joint WHO/Convention Task Force (7) recommended using the risk coefficients generated by the ACS study (14) for quantification of impacts of PM in Europe.

The ACS study is by far the largest cohort study of air pollution and long-term mortality. It is based on the ACS Cancer Prevention II Study, an ongoing prospective cohort of approximately 1.2 million adults from all 50 American states. Participants were enrolled in 1982 when they were at least 30 years of age, and deaths were monitored up to 1998. Data on a wide range of risk factors for cancer and other chronic diseases were obtained from each participant. The ACS study links the data on approximately 500 000 cohort members with air pollution data for metropolitan areas throughout the United States. The first study of air pollution and mortality in this cohort (15) was based on follow-up through to 1990. The findings of the first study were subsequently corroborat-

ed in an independent reanalysis (16). A more recent analysis of this cohort extended follow-up through to 1998, and ascertained 40 706 deaths from cardiopulmonary disease and 10 749 from lung cancer. Data were analysed using Cox proportional hazards regression models that incorporated both random effects and non-parametric spatial smoothing to adjust for unmeasured factors correlated spatially with air pollution and mortality across the United States. The models also adjusted for age, sex, race, education, marital status, body mass, diet, alcohol consumption, occupational exposures, and the duration and intensity of cigarette smoking, all measured via questionnaire at enrolment.

In general, ambient air pollution concentrations declined across the United States between 1982 and 1998. Measurements of ambient PM<sub>2.5</sub> concentrations in the cities where subjects resided at enrolment were available for periods both briefly preceding enrolment (1979–1983) and immediately after follow-up (1999–2000). Mean PM<sub>2.5</sub> levels in the two periods were highly correlated ( $r = 0.78$ ) and the rank ordering of cities by relative pollution levels remained nearly the same. In separate regression analyses, cohort members were assigned exposure estimates specific to their city of residence value for each of those periods, as well as for the average value across the two periods. For a 10- $\mu\text{g}/\text{m}^3$  change in the ambient PM<sub>2.5</sub> concentration, the smallest relative increases were observed for the mean concentration of the period 1979–1983. The relative risk for a 10- $\mu\text{g}/\text{m}^3$  change in the ambient PM<sub>2.5</sub> concentration was larger when exposure was specified as the average of the ambient concentrations of the two periods (Table 7.1). This difference may be explained by the fact that the estimates from the earliest periods are more subject to random (and non-differential) error. However, it also suggests that more recent exposures may be exerting the strongest effects on mortality, an interpretation also offered in the recent reanalysis of the earlier follow-up of the ACS cohort (16). It was therefore decided to use the risk estimate derived for the average of the two periods. This decision was endorsed by the Task Force (7), which felt that this risk coefficient was a more appropriate choice than the estimates specific to the PM levels in the initial or final period of the follow-up in the ACS study. The Task Force felt there were



**Table 7.1. Relative risk estimates associated with a 10- $\mu\text{g}/\text{m}^3$  change in long-term exposure to  $\text{PM}_{2.5}$** 

Cause of death	Adjusted relative risk (95% confidence interval)		
	1979–1983	1999–2000	Average
All-cause	1.04 (1.01–1.08)	1.06 (1.02–1.10)	1.06 (1.02–1.11)

Source: Pope et al. (14).

indications that for some health end-points (such as cardiopulmonary mortality) recent exposure was relevant while for others (such as lung cancer) it could be assumed that exposure dating from earlier periods of exposure was important.

Using the risk coefficient from the ACS study might, possibly, lead to an underestimation of the effects, since the population followed in the ACS cohort had an above-average educational status for the United States while the risk was higher for those with a lower level of education.

Preliminary reports from the analysis of mortality in the Los Angeles subcohort of the ACS study indicate that a more precise exposure assessment results in significantly (up to three times) higher risk estimates for the association between cardiovascular mortality and  $\text{PM}_{2.5}$  level (17) than those reported by Pope et al. (14). This is consistent with previous observations in the Harvard Six Cities Study (also with a more precise exposure assessment and higher risk coefficients), and with the role of the random error of exposure indicator in estimating exposure–response relationships.

The Pope et al. (14) study population was exposed to  $\text{PM}_{2.5}$  at concentrations of about 8–30  $\mu\text{g}/\text{m}^3$  as an annual mean, which is roughly similar to the  $\text{PM}_{2.5}$  levels observed in populated areas of Europe (see Chapter 5). The impact assessment presented below addresses  $\text{PM}_{2.5}$  from anthropogenic sources, estimated by the EMEP model. This model does not cover well secondary organic aerosols, underestimating real  $\text{PM}_{2.5}$  levels even from anthropogenic sources. Thus one can assume that, even if one subtracted all of the  $\text{PM}_{2.5}$  estimated by the model, the remaining  $\text{PM}_{2.5}$  concentrations would exceed the lower end of the  $\text{PM}_{2.5}$  concentration range from the ACS study. On the other hand, long-term average  $\text{PM}_{2.5}$  levels do not exceed 30  $\mu\text{g}/\text{m}^3$  in large areas of Europe. Thus the shape of the concentration–response function estimated by the ACS study should be adequate for impact assessment of  $\text{PM}_{2.5}$  in Europe.

Consequently, the use of a linear concentration–response function seems the most appropriate choice and was recommended by the Task Force. This choice was also supported by the experience of the WHO comparative quantification of health risks, which tested the sensitivity of impact estimates in selecting various options for the concentration–response function (2).

Estimation of the burden of disease attributed to a certain exposure requires specification of a theoretical minimum level of exposure. For the purpose of estimating health effects of PM in Europe, it is assumed that any variations in  $\text{PM}_{2.5}$  concentration that are caused by changes in anthropogenic emissions – on top of the natural background – contribute to the burden of disease. As a consequence, health effects are calculated only for the anthropogenic fraction without a threshold in anthropogenic  $\text{PM}_{2.5}$ .

## 7.5 Transferability

A health impact assessment applies air pollution effect estimates derived from one (evidentiary) population to estimate impacts in another (target) population, based on the assumption that these estimates can be transferred. Care must be taken if one cannot assume that the contribution of various causes of death is similar, if the mixture of pollutants differs, if the baseline health statuses of the populations are not the same, or if exposure ranges do not overlap.

Currently, only a limited number of cohort studies on the effect of PM on mortality are available to be used as a basis for impact assessments. Since all but one of these cohort studies was conducted in the United States, the generalization of their results to populations in Europe and elsewhere is critical in quantifying effects in Europe. Recent studies have begun to explore effect modifiers that may explain the variation in air pollution effect estimates observed among locations in Europe and the United States (18,19). However, results for  $\text{PM}_{2.5}$  are not yet

available and present knowledge is quite limited, so that it is difficult to include other factors in a practical impact assessment at the moment.

The WHO project “Systematic review on health aspects of air pollution in Europe” also assessed the question of possible regional characteristics modifying the effects of air pollution (20):

Potentially this could be a very influential issue since the characteristics of populations, environments and pollution (including particle concentration, size distribution and composition) vary throughout Europe. However, at this stage there is not sufficient evidence to advocate different guidelines for particles or other priority pollutants in different parts of Europe.

To get some idea about the consistency of PM-related health effects between North America and Europe, it might also be worth comparing the effect estimates derived from the corresponding multi-city time series studies presented in Chapter 3. The results of American studies tend to yield lower risk coefficients. If this tendency is also reflected in long-term effects, the use of ACS study coefficients for impact assessment in Europe may lead to an underestimation of the impacts.

## 7.6 Further considerations in estimating impacts of PM on mortality

Mortality in adults from long-term exposure, captured via cohort studies, implemented via life tables and expressed as changes in life expectancy, is the principal indicator of the adverse effects of ambient PM. There are, however, other important aspects of quantifying the effects on mortality of ambient PM.

### Estimating attributable deaths from long-term exposure to PM

Many health impact assessments of the effects on mortality of long-term exposure to PM seek to estimate numbers of deaths attributable to the exposure rather than changes in life expectancy. There are three main reasons. First, the method is easy to implement and as such was the method used initially in quantifying effects on mortality of cohort studies (21) and is still used widely (see, for example, 12,22). Second, it is

widely thought that these results can be simply interpreted as the changes in number of annual deaths attributable to changes in ambient PM<sub>2.5</sub>. This simple interpretation may be misleading because the method ignores the more complex fact, captured by life tables, that changes in mortality in one year also affect the population at risk in subsequent years. This cumulative effect becomes important in the longer term. Finally, many economists consider that monetary valuation of attributable deaths is more reliable than corresponding valuation of life expectancy, although empirical studies have now begun to address directly the issue of valuing life expectancy.

For these reasons, it was considered helpful to the development of air pollution policy in Europe that the effects of mortality in the Cost-Benefit Analysis of the CAFE programme (CAFE CBA) should be expressed in terms of number of deaths as well as life expectancy; this was done, as follows. The relative risk coefficient discussed earlier (0.6% per µg/m<sup>3</sup> PM<sub>2.5</sub>, as estimated from the ACS study) was linked to (changes in) anthropogenic PM<sub>2.5</sub>, without threshold, and applied to the annual number of deaths at age 30 years or more in the target population (23). This gives results in terms of numbers of “extra” deaths (or lives saved) annually.

The advantages and disadvantages of using attributable deaths as an alternative to life expectancy in assessing the mortality effects implied by the cohort studies remain under active discussion. The arguments are summarized in various documents of CAFE CBA, including Volume 2 (23), the peer review of an earlier draft methodology (24), UNICE’s concerns about the CAFE methodology, and the CAFE CBA team’s response to those concerns (25). It is an area of continuing methodological development and clarification.

### Should we include attributable deaths from studies of short-term exposure to PM?

Another issue is whether the cohort studies capture all the effects on mortality of ambient PM and, if not, whether deaths as estimated from time series studies of short-term exposures (daily variations in PM) should in some way be included also. A suitable coefficient of 0.6% (95% CI 0.4–0.8) per 10 µg/m<sup>3</sup> PM<sub>10</sub> is available from the WHO meta-analysis of studies

in Europe (26). The time series studies capture effects at all ages, whereas the ACS cohort study included only adults at age 30 years or more. Also, by using the metric of  $PM_{10}$ , they capture some direct effects of the coarse fraction of PM. Consequently, deaths as estimated from time series studies are sometimes estimated, for example APHEIS (22) and CAFE CBA (23). The associated effects are small, however, compared with mortality in adults associated with longer-term exposure to  $PM_{2.5}$  and, to avoid double-counting, it is usual *not* to add time series mortality effects to those from cohort studies. This was also the approach adopted by CAFE CBA (23).

### Infant mortality

There is now substantial evidence that air pollution adversely affects a wide range of measures of fetal and infant health, including mortality (27). Infant mortality has been quantified in several studies, including those of Rabl (28) and Kaiser et al. (29). These base their quantification on Woodruff et al. (30), an American cohort study of four million infants, where post-neonatal infant mortality between the ages of one month and one year was associated with mean outdoor concentrations of  $PM_{10}$  in the first two months of life, giving a concentration–response function:

Change in (all-cause) infant mortality of 4% per  $10 \mu\text{g}/\text{m}^3 \text{PM}_{10}$  (95% CI 2–7)

This concentration–response function was used again in CAFE CBA (23); use of all-cause rather than cause-specific mortality is consistent with other choices made in CAFE CBA.

From one viewpoint, this concentration–response function may imply some overestimation of PM effects in infants. For example, as noted by Kaiser et al. (29), it is unclear whether effects attributed to  $PM_{10}$  (30) may be capturing some effects of other air pollutants. On the other hand, any overestimation of infant mortality is more than offset by the fact that any mortality effects of air pollution on people aged between 1 and 30 years are currently not quantified in CAFE CBA. Also, the cohort study concentration–response function (30) is supported by evidence from time series studies where the estimated relative risks are as high as, or higher than, those from Woodruff et al.,

e.g. in Bangkok (31) and in São Paulo (32). These differences may reflect differences between cities with different pollution mixtures, health status and health care of infants, and maternal age at birth compared with the United States. It may also reflect study design issues in that (a) time series studies may capture some aspects of mortality better than cohort studies; and (b) for infants, lifetime exposure as captured by cohort studies is necessarily short and much more similar to that captured by time series studies than is the case for adults.

More generally, Kaiser et al. (29) noted that problems of transferability may be more severe for infants than for adults. In Europe, Bobak and co-workers have studied the effects of air pollution on infant health, initially in the Czech Republic but also elsewhere (33–36). However, Rabl (28) found that estimated impacts using all-cause infant mortality were similar, regardless of whether impact estimation was based on coefficients taken from Woodruff et al. (30) or from Bobak & Leon (35) (use of the latter required “conversion” from TSP to  $PM_{10}$ ).

It is unclear to what extent infant deaths associated with and presumably attributable to air pollution occur among children who are already very frail and therefore unlikely to survive into adulthood. This complicates assessment both of public health importance and of monetary valuation. For that reason, both Kaiser et al. (29) and CAFE CBA (23) estimate attributable deaths rather than life expectancy, although this transfers the problem to valuation rather than solving it. Although the survival and health of young people are seen as particularly important, in terms of numbers of deaths the effects of ambient particles on infants are small compared with those on adults. It follows that uncertainties in the concentration–response function from Woodruff et al. (30) used by CAFE CBA and other quantifications are not of great practical importance either.

## 7.7 Morbidity – general methodological remarks

### General strategy

It is recognized (see Chapter 3) that ambient PM is associated with a wide range of non-lethal cardiopulmonary health effects, although the data base in

this respect is less complete and less consistent than the evidence on the effects of PM on mortality. Consequently, and as noted in an AIRNET report (37) and by CAFE CBA (23), there are two different traditions in quantifying these morbidity impacts, reflecting different purposes and uses. One approach, for example COMEAP (38) and APHEIS (22), quantifies only those end-points where there are strongly reliable data both for concentration–response functions and for background rates. This approach is useful in showing that there is a public health problem of at least the magnitude quantified and can help provide *general* (i.e. not policy-specific) motivation to protect health by reducing air pollution. Its main drawback is that, by including only those effects for which there is strong evidence for quantification, the approach systematically underestimates the overall effects of air pollution on health. When used in cost–benefit analyses, it is anti-precautionary.

The second approach (e.g. 12,21,39) aims to quantify all end-points where, on balance of probabilities, the relevant air pollutant has an effect. This implies quantification of a wider range of end-points than in the first approach and it also means that, for some of the effects included in the second quantification, there is greater uncertainty in the concentration–response function and/or in background rates than would be acceptable under the first, more restrictive, approach. Nevertheless, it can be argued that the approach as a whole gives a more realistic assessment of the overall effects of air pollution, and is thus the appropriate strategy when comparing the costs and benefits of *specific* policies or developments that affect air pollution. Therefore, Hurley et al. (23) adopted it for CAFE CBA. Also, they focused on studies of incidence rather than prevalence, so that the benefits of reducing pollution could more easily be expressed as annual benefits, for comparison with annual costs. Effects of the exposure on lung function were not included in the impact estimation because there are no suitable studies linking them with monetary valuation.

Discussion in the Task Force confirmed that, though confidence in the precision of morbidity outcome estimates is less than that for mortality, it is appropriate to include them in the report to indicate the possible range of pollution effects. The following

sections address current possibilities, and restrictions, in estimating effects of PM on morbidity in Europe.

### **Implementation, including impact functions**

The two approaches to health impact assessment use the same technical methods in estimating the effects of ambient PM on any particular morbidity end-point, methods that are very similar to those used in estimating attributable deaths. As described earlier, these methods use a concentration–response function expressed as percentage change in end-point per (10)  $\mu\text{g}/\text{m}^3$   $\text{PM}_{10}$  (or, more rarely,  $\text{PM}_{2.5}$ ) and link this with (a) the background rates of the health end-point in the target population, expressed as new cases (or events) per year per unit population; (b) the population size; and (c) the relevant pollution increment expressed in  $\mu\text{g}/\text{m}^3$  PM. Results are then expressed as estimated new or “extra” cases, events or days per year attributed to PM.

It is usual to base these calculations on annual average anthropogenic PM, even when estimating the aggregated annual effects of short-term exposures to air pollution, such as the effects on health of daily variations in pollution as identified through time series or panel studies. This approach is much simpler computationally than the alternative of calculating effects on each day of the year and then aggregating. In the absence of threshold, and assuming a linear concentration–response function, these two computational approaches give the same results. The use of the simpler approach, based on annual averages, should not be taken as implying or suggesting that what are being quantified are the effects of long-term exposure, although for PM some effects of long-term exposure are also quantified.

When the concentration–response function is derived from Poisson regression or related analyses, for example from time series studies of hospital admissions, it is easy and natural to express the relative risk as the percentage change in background rates of incidence (or prevalence). When the concentration–response function is derived from logistic regression analyses, such as in panel studies of daily occurrence of symptoms or individuals’ use of medication, then the estimated relative risk applies

to the *odds* of occurrence, when what is needed is the relative change in *probability*. When the occurrence is rare, with probability of (say) less than one in ten, odds and probability are interchangeable with little error. A percentage change in probability can also be derived for more frequently occurring events, but its value depends on the background rates used. For some examples see (23).

Note that the percentage change in probability can be combined with background rates to give a single *impact function* expressed as:

Number of (new) cases, events or days annually, per unit population (say, per 100 000 people), per  $(10) \mu\text{g}/\text{m}^3$  annual average  $\text{PM}_{10}$  (or  $\text{PM}_{2.5}$ ).

This impact function can then be linked, as before, with population size and the relevant pollution increment to give the estimated number of annual occurrences in the target population. This is convenient in implementation.

### Dealing with missing data on background rates

For many health end-points, reliable data on background rates of morbidity in the EU target population (25 countries) are not readily available. This is because they are not collected routinely or, if they are collected, may be subject to different protocols and standards at different locations or are not readily accessible. One strategy then is to use other general epidemiological studies of that health end-point – not necessarily studies of air pollution and health – to provide estimates of background rates. Examples of this are the International Study of Asthma and Allergies in Children (ISAAC) and, for adults, the European Community Respiratory Health Study (ECRHS).

Another approach is to estimate an impact function from where the relevant epidemiological studies were carried out and then transfer and use that impact function for quantification in the wider European target population. Clearly, the reliability of that quantification depends on how transferable the impact function is, i.e. how stable it is spatially. The approach is, however, well established in health impact assessment practice (12,21). The two approaches have been used (for different end-points) for CAFE CBA (23);

otherwise, few if any morbidity end-points would have been quantifiable.

## 7.8 Morbidity from long-term (chronic) exposure

### Chronic cardiovascular disease

The effects on mortality of long-term exposure to ambient PM apply to cardiopulmonary mortality generally, and probably to lung cancer also. Reanalysis by Krewski et al. (16) of the original ACS study data (15) pointed to effects on cardiovascular deaths in particular. It is to be expected then that ambient PM also affects the development and/or worsening of chronic cardiovascular disease. However, we have not found suitable studies of long-term exposure to quantify these impacts. (Some cardiovascular effects of short-term exposures are noted below.)

### Chronic respiratory disease

Results from the Adventist Health and Smog (AHSMOG) study (40–42) enable quantification of the effects of PM on the development (i.e. increase in new cases) of chronic respiratory disease, including chronic bronchitis. The AHSMOG study examined people on two occasions, about 10 years apart, in 1977 and 1987/1988. Chronic bronchitis was defined as reporting at survey the symptoms of chronic cough or sputum, on *most* days, for at least three months of the year, for at least two years – a somewhat milder definition than is used in many other studies. New cases of chronic bronchitis were defined as those that met the criteria in 1987/1988 but not in 1977.

Using a concentration–response function from Abbey et al. (41) and a background incidence rate (adjusted for remission of chronic bronchitis symptoms) of 0.378% estimated from Abbey et al. (41,42), Hurley et al. (23) derived an estimated impact function of:

New cases of chronic bronchitis per year per 100 000 adults aged  $\geq 27 = 26.5$  (95% CI 1.9–54.1) per  $10 \mu\text{g}/\text{m}^3 \text{PM}_{10}$ .

In considering reliability, note that:

- These incidence results are based on one study only (AHSMOG), although other cross-sectional studies (e.g. 43) and the literature as a whole provide strong supporting evidence of an effect.
- Similar results are found if the AHSMOG concentration–response function in  $PM_{2.5}$  is used, together with conventional conversion factors between the two PM metrics.
- Although many of the relationships between  $PM_{10}$  and chronic respiratory symptoms reported by Abbey et al. (41) are statistically significant at the 5% level, those based on mean concentrations of  $PM_{10}$  or  $PM_{2.5}$  (i.e. those used in CAFE CBA) were not quite statistically significant at the 5% level.
- There are issues of transferability that are greater than those for chronic mortality. The AHSMOG study is not only American-based but it refers to one part of the country (California) and to a population of distinctive lifestyle.
- Nevertheless, the AHSMOG background incidence of 0.378% seems reasonably transferable: it is higher than estimates of the attack rate of COPD in Europe from the Global Burden of Disease estimates for 2000 (44) but lower than estimates from particular European studies quoted in the *European lung white book* (45).

The effects of variations in background rates, concentration–response function and monetary valuation are assessed quantitatively, using Monte Carlo probabilistic methods, in Volume 3 of the CAFE CBA series (46).

## 7.9 Morbidity from short-term exposure (“acute effects”)

### Cardiac hospital admissions

European data on effects of PM are available for cardiac (ICD 390–429) rather than cardiovascular hospital admissions (26). CAFE CBA therefore quantified cardiac admissions, using a concentration–response function based on APHEA-2 results from eight cities in western and northern Europe (47) and a Europe-wide annual rate of emergency cardiac admissions estimated as the arithmetic mean of rates from eight

European cities derived from the appendices to the APHEIS-3 report (22). Together these imply an impact function:

Annual rate of attributable emergency cardiac hospital admissions = 4.34 (95% CI 2.17–6.51) per  $10 \mu\text{g}/\text{m}^3 PM_{10}$  per 100 000 people (all ages).

As for chronic bronchitis, the effect of quantifiable uncertainties (in background rates, concentration–response function and monetary valuation) is explored quantitatively, using Monte Carlo methods, by Holland et al. (46). The main unquantifiable uncertainty is the extent to which the extra hospital admissions in days following higher air pollution are genuinely additional admissions, rather than the bringing forward in time of admissions that might soon have occurred in any case (38).

### Respiratory hospital admissions (ICD 460–519)

WHO meta-analyses gives European risk estimates for age 65+ only (26). Since age-specific background rates were not easily available for CAFE CBA, Hurley et al. (23) used all-ages data, both for concentration–response function and for background rates, derived from APHEIS-3 (22). These were also based on eight European cities. Together they imply an impact function:

Annual rate of attributable emergency respiratory hospital admissions = 7.03 (95% CI 3.83–10.30) per  $10 \mu\text{g}/\text{m}^3 PM_{10}$  per 100 000 people (all ages).

Uncertainty issues are similar to those for cardiac hospital admissions.

### Restricted activity days and associated health end-points

Most major health impact assessments of air pollution and health include estimates of the effect of air pollution on days when normal activities are restricted, typically using concentration–response functions derived from Ostro (48) or Ostro & Rothschild (49). Both of these studies used data on adults aged 18–64 years from six consecutive years (1976–1981) of the US National Health Interview Survey (NHIS). NHIS

is a multi-stage probability sample of 50 000 households from metropolitan areas of all sizes and regions throughout the United States (49). Within NHIS, restricted activity days (RADs) are classified according to severity as (a) bed disability days; (b) work or school loss days; and (c) minor restricted activity days (MRADs), which do not involve work loss or bed disability, but do include some noticeable limitation of “normal” activity.

Ostro (48) studied both RADs and work loss days (WLDs) among adults aged 18–64 years in separate analyses for each of the six years 1976–1981. Results for RADs, based on about 12 000 subjects per year from 68 metropolitan areas, showed a consistent relationship with  $PM_{2.5}$ : the coefficient for each of the six years was positive and highly statistically significant ( $P < 0.01$ ) (48). A weighted mean coefficient was linked to estimated background rates of an average of 19 RADs per person per year (50) to give an estimated impact function of:

Change of 902 RADs (95% CI 792–1013) per 10  $\mu\text{g}/\text{m}^3$   $PM_{2.5}$  per 1000 adults at age 15–64.

In the main analyses of CAFE CBA, this impact function was applied to people aged 15–64 years, as in the original study. In sensitivity analyses, the same impact function was used but applied to all ages, on the grounds that it is unlikely that health-related restrictions on activity do not cease at age 65.

As an alternative, Hurley et al. (23) also derived impact functions for WLDs from Ostro (48) and MRADs from Ostro & Rothschild (49) to give, respectively:

Change of 207 WLDs (95% CI 176–238) per 10  $\mu\text{g}/\text{m}^3$   $PM_{2.5}$  per year per 1000 people aged 15–64 in the general population

and

Change of 577 MRADs (95% CI 468–686) per 10  $\mu\text{g}/\text{m}^3$   $PM_{2.5}$  per year per 1000 adults aged 18–64.

Issues of uncertainty are addressed, as for end-points, in CAFE CBA Volume 3 (46), in UNICE’s concerns about the CAFE methodology, and in the CAFE CBA

team’s response (25). The main uncertainty issues are the following.

- The estimates are based on the HIS study only, although this is a large-scale countrywide study with separate analyses of each of six years of data;
- There may be transferability problems from the United States to Europe, because:
  - RADs and WLDs are culture-related variables (although background rates used in the United States are similar to those from similar surveys in Canada and lower than those in the United Kingdom); and
  - the pollution mixture in the United States in 1976–1981 is quite different from that in Europe now (although relationships in PM seem quite robust to different ambient mixtures).
- $PM_{2.5}$  was derived from airport visibility data rather than measured directly. However, exposure misclassification tends to lead to underestimation of risks.

### Medication (bronchodilator) usage

WHO (20) concludes that there is sufficient evidence to assume a causal relationship between air pollution exposure and aggravation of asthma in children; one way that such a relationship may show is through increased medication usage. It is well understood that air pollution leads to exacerbation of asthma, and thus for the sake of completeness estimates of relationships between bronchodilator usage and PM were included in CAFE CBA, despite their lack of statistical significance.

### Children with asthma

For children with asthma, the WHO meta-analysis (26) gives a pooled estimate of the odds ratio that is not at all significant statistically. The meta-analysis was dominated by the PEACE study, whose generally negative results may at least in part be due to short study periods that included an influenza epidemic (51). Using data from various individual studies in Europe, and noting wide variation between studies, Hurley et al. (23) estimated the mean daily prevalence of bronchodilator usage *among panels of schoolchildren who met the PEACE study criteria* as about 10%.

These data were linked with the meta-analysis concentration–response function to give an impact function of:

Annual change in days of bronchodilator usage = 180 (95% CI -690–1060) per 10  $\mu\text{g}/\text{m}^3$   $\text{PM}_{10}$  per 1000 children aged 5–14 years meeting the PEACE study criteria.

European data from ISAAC (52) were used to give an estimate that approximately 15% of children in northern and eastern Europe and 25% in western Europe met those inclusion criteria.

### Adults with asthma

For adults with asthma, the WHO meta-analysis (26) used results from three locations in the Netherlands (all involving bronchodilator usage, and only one of which was statistically significant) to give a pooled odds ratio that was positive but not nearly statistically significant. The *mean daily prevalence of bronchodilator use by people with asthma* was estimated for CAFE CBA as about 50% among adults with established asthma, based on several European studies that gave various results; Hurley et al. (23) acknowledges that this estimate is only approximate. Data from the ECRHS were used to estimate that, across Europe, about 4.5% of adults have asthma of a severity comparable to that of the Dutch panels on which the concentration–response function was based (53). These data were linked to give an estimated impact function:

Change in bronchodilator usage days: 912 (95% CI -912–2774) per year per 10  $\mu\text{g}/\text{m}^3$   $\text{PM}_{10}$  per 1000 adults aged  $\geq 20$  with well established asthma (say 4.5% of the adult population).

### Lower respiratory symptoms (LRS), including cough, in children

Cough in children with chronic respiratory disease was one of the end-points considered in the WHO meta-analysis of air pollution studies in Europe. Results, dominated by the PEACE study, were generally negative and the WHO meta-analysis (26) gave no consistent or overall evidence of an adverse effect.

However, a more recent systematic review (54) very strongly suggests that effects of PM on respira-

tory symptoms should be quantified for children in general and not be confined to those with chronic symptoms. This reviewed studies of both cough and LRS (wheezing, chest tightness, shortness of breath and possibly cough). Results showed the same pooled relative risk (random effects model) for cough (12 studies) and for LRS in general (16 studies). For CAFE CBA (23) these were combined and linked with an estimate of the mean daily prevalence of LRS, including cough, based on two Dutch studies of children from the general population (55,56), to give an estimated impact function:

Change of 1.86 (95% CI 0.92–2.77) extra symptoms days per year per child aged 5–14 per 10  $\mu\text{g}/\text{m}^3$   $\text{PM}_{10}$ .

### LRS, including cough, in adults with chronic respiratory disease

The WHO meta-analysis of studies in Europe examined cough in relation to  $\text{PM}_{10}$  among adults with respiratory disease (26). Six studies were identified, the panels studied including adults with asthma, chronic respiratory symptoms, chronic obstructive pulmonary (airway) disease and bronchial hyperresponsiveness. Following further review of these studies, CAFE CBA considered LRS rather than cough in five panels from four of the studies (57–60) identified for the WHO meta-analysis. A random effects meta-analysis of results from all five panels was linked to both (a) estimates of the mean daily prevalence of LRS (including cough) in symptomatic panels, based on the studies underlying the concentration–response function; and (b) estimates of the percentage of people qualifying for such panels, using data from ECRHS (53) (there are important country-related differences in prevalence) to give an estimated impact function:

Annual increase of 1.30 (95% CI 0.15–2.43) symptom days (LRS, including cough) per 10  $\mu\text{g}/\text{m}^3$   $\text{PM}_{10}$  per adult with chronic respiratory symptoms (approx 30% of the adult population).

### Other possible indicators of health impacts of air pollution

Several other indicators of ill health could be considered in describing the effects of PM on health. How-



ever, the existing data base is too weak to allow meaningful quantification of the impacts. The following indicators deserve attention in future studies.

- *Consultations with primary care physicians (general practitioners).* There is evidence, for example from studies in London (61–63), that daily variations in ambient PM, ozone and other gaseous pollutants are associated with consultations with primary care physicians. CAFE CBA used the London studies to quantify a relationship between ambient PM<sub>10</sub> and consultations for asthma and for upper respiratory diseases, excluding allergic rhinitis (23). Because of differences in primary health care systems, it is difficult to know to what extent these relationships are transferable within Europe. CAFE CBA therefore used them in sensitivity analyses only, to help assess whether these end-points were important.
- *Acute respiratory symptoms in adults.* CAFE CBA considered if it might be better to examine symptoms in the general population rather than among adults with chronic respiratory disease. An impact function estimating the effect of PM on symptom days in the general population was derived (23), based on an American study (64) that has previously been used in health impact assessment quantification studies for PM (e.g. 21,50). This may lead to an overestimate of effects, and so it was included in CAFE CBA with the intention that it be used *only* for sensitivity analyses to indicate how large an effect *might* be.
- *Visits to hospital emergency departments.* For CAFE CBA, Hurley et al. (23) did not attempt to quantify a European impact function for emergency department visits and PM.

## References

1. Brunekreef B. Air pollution and life expectancy: is there a relation? *Occupational and Environmental Medicine*, 1997, 54:781–784.
2. Cohen AJ et al. Mortality impacts of urban air pollution. In: Ezzati M et al., eds. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Geneva, World Health Organization, 2004:1353–1433.
3. Hurley JF et al. *Towards assessing and costing the health impacts of ambient particulate air pollution in the UK*. Edinburgh, Institute of Occupational Medicine, 2000.
4. Künzli N. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or cohort studies? *American Journal of Epidemiology*, 2001, 10:1050–1055.
5. Ostro B. *Outdoor air pollution: assessing the environmental burden of disease at national and local levels*. Geneva, World Health Organization, 2004 (WHO Environmental Burden of Disease Series, No. 5).
6. *Quantification of the health effects of exposure to air pollution. Report of a WHO Working Group, Bilthoven, Netherlands 20–22 November 2000*. Copenhagen, WHO Regional Office for Europe, 2001 (document EUR/01/5026342) (<http://www.euro.who.int/document/e74256.pdf>, accessed 2 November 2005).
7. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2003 (document EB.AIR/WG.1/2003/11) (<http://www.unece.org/env/documents/2003/eb/wg1/eb.air.wg1.2003.11.pdf>, accessed 1 October 2005).
8. *Modelling and assessment of the health impact of particulate matter and ozone*. Geneva, UNECE Working Group on Effects, 2004 (document EB.AIR/WG.1/2004/11) (<http://www.unece.org/env/documents/2004/eb/wg1/eb.air.wg1.2004.11.e.pdf>, accessed 1 October 2005).
9. *Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide. Report on a WHO working group*. Copenhagen, WHO Regional Office for Europe, 2003 (document EUR/03/5042688) (<http://www.euro.who.int/document/e79097.pdf>, accessed 2 November 2005).
10. American Thoracic Society. What constitutes an adverse health effect of air pollution? *American Journal of Respiratory and Critical Care Medicine*, 2000, 161:665–673.

11. de Hollander AE et al. An aggregate public health indicator to represent the impact of multiple environmental exposures. *Epidemiology*, 1999, 10:606–617.
12. Künzli N et al. Public health impact of outdoor and traffic-related air pollution: a European assessment. *Lancet*, 2000, 356:795–801.
13. McMichael AJ et al. Inappropriate use of daily mortality analyses to estimate longer-term mortality effects of air pollution *International Journal of Epidemiology*, 1998, 27:450–453.
14. Pope CA et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 2002, 287:1132–1141.
15. Pope CA et al. Particulate air pollution as predictor of mortality in a prospective study of US adults. *American Journal of Respiratory and Critical Care Medicine*, 1995, 151:669–674.
16. Krewski D et al. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of particulate air pollution and mortality*. Cambridge, MA, Health Effects Institute, 2000 (<http://www.healtheffects.org/Pubs/Rean-ExecSumm.pdf>, accessed 3 November 2005).
17. Jerrett M et al. A cohort study of air pollution and mortality in Los Angeles. *Epidemiology*, 2004, 15: S46.
18. Samet JM et al. *National morbidity, mortality and air pollution study*. Cambridge, MA, Health Effects Institute, 2000 (HEI Report 94, Part 2).
19. Katsouyanni K et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: Results from 29 European cities within the APHEA 2 project. *Epidemiology*, 2001, 12:521–531.
20. *Health aspects of air pollution – answers to follow-up questions from CAFE. Report on a WHO working group meeting, Bonn, Germany, 15–16 January 2004*. Copenhagen, WHO Regional Office for Europe, 2004 (document EUR/04/5046026) (<http://www.euro.who.int/document/E82790.pdf>, accessed 3 November 2005).
21. *Externalities of Energy (ExternE) Volume 2: Methodology – Method for estimation of physical impacts and monetary valuation for priority impact pathways. Part 1: Impact assessment. Part 2: Economic valuation*. Brussels, ExternE Project, European Commission, 1995 (<http://www.externe.info>, accessed 3 November 2005).
22. *Health impact assessment of air pollution and communication. Third year report, 2002–2003*. Brussels, APHEIS Programme, European Commission, 2004 (<http://www.apheis.net/Apheis3NEW1.pdf>, accessed 3 November 2005).
23. Hurley F et al. *Methodology for the cost–benefit analysis for CAFE. Volume 2: health impact assessment*. Brussels, European Commission, 2005 ([http://europa.eu.int/comm/environment/air/cafe/pdf/cba\\_methodology\\_vol2.pdf](http://europa.eu.int/comm/environment/air/cafe/pdf/cba_methodology_vol2.pdf), accessed 13 December 2005).
24. Krupnick et al. *Peer review of the methodology of the cost–benefit analysis for the clean Air for Europe Programme*. Brussels, European Commission, 2004 (<http://europa.eu.int/comm/environment/air/cafe/activities/pdf/krupnick.pdf>, accessed 13 December 2005).
25. Hurley et al. *CAFE CBA Team response to UNICE concerns with key aspects of the CBA Methodology*. Didcot, AEA Technology Environment, 2005 (<http://cafe-cba.aeat.com/files/CAFE%20CBA%20response%20to%20UNICE%20concerns%205%20May%202005.pdf>, accessed 13 December 2005).
26. Anderson HR et al. *Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O<sub>3</sub>). Report of a WHO task group*. Copenhagen, WHO Regional Office for Europe, 2004 (document EUR/04/5042688) (<http://www.euro.who.int/document/e82792.pdf>, accessed 3 November 2005).
27. *Effects of air pollution on children's health and development: a review of the evidence*. Copenhagen, WHO Regional Office for Europe, 2005 (<http://www.euro.who.int/document/E86575.pdf>, accessed 3 November 2005).

28. Rabl, A. Interpretation of air pollution mortality: number of deaths or years of life lost? *Journal of the Air & Waste Management Association*, 2003, 53:41–50.
29. Kaiser R et al. Air pollution attributable postneonatal infant mortality in U.S. metropolitan areas: a risk assessment study. *Environmental Health: a Global Access Science Source*, 2004, 3:4 (<http://www.ehjournal.net/content/3/1/4>, accessed 3 November 2005).
30. Woodruff TJ, Grillo J, Schoendorf KC. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives*, 1997, 105:608–612 (<http://ehp.niehs.nih.gov/members/1997/105-6/woodruff.html>, accessed 3 November 2005).
31. Ostro B et al. The impact of particulate matter on daily mortality in Bangkok, Thailand. *Journal of the Air & Waste Management Association*, 1999, 49:100–107.
32. Conceicao GMS et al. Air pollution and child mortality: a time series study in Sao Paulo, Brazil. *Environmental Health Perspectives*, 2001, 109(Suppl. 3):347–350.
33. Bobak M, Richards M, Wadsworth M. Air pollution and birth weight in Britain in 1946. *Epidemiology*, 2001, 111:207–216.
34. Bobak M. Outdoor air pollution, low birth weight and prematurity. *Environmental Health Perspectives*, 2000, 108:173–176.
35. Bobak M, Leon DA. The effect of air pollution on infant mortality appears specific for respiratory causes in the postneonatal period. *Epidemiology*, 1999, 10:666–670.
36. Bobak M, Leon DA. Air pollution and infant mortality in the Czech Republic, 1986–88. *Lancet*, 1992, 340:1010–1014.
37. Hurley F, Sanderson E, eds. *Final report of AIRNET Work Group 4: health risk and health impact assessment*. Brussels, European Commission, AIRNET Programme, 2005 (<http://airnet.iras.uu.nl/>, accessed 4 November 2005).
38. Committee on the Medical Effects of Air Pollutants. *The quantification of the effects of air pollution on health in the United Kingdom*. London, H.M. Stationery Office, 1998 (<http://www.advisorybodies.doh.gov.uk/comeap/statementsreports/airpol7.htm>, accessed 13 December 2005).
39. Ostro B, Chestnut L. Assessing the health benefits of reducing particulate matter air pollution in the United States. *Environmental Research*, 1998, 76:94–106.
40. Abbey DE et al. Long-term inhalable particles and other pollutants related to mortality of non-smokers. *American Journal of Respiratory and Critical Care Medicine*, 1999, 159:373–382.
41. Abbey DE et al. Estimated long-term ambient concentrations of PM<sub>10</sub> and development of respiratory symptoms in a nonsmoking population. *Archives of Environmental Health*, 1995, 50:139–152.
42. Abbey DE et al. Long-term ambient concentrations of total suspended particulates, ozone and sulfur dioxide and respiratory symptoms in a non-smoking population. *Archives of Environmental Health*, 1993, 48:33–46.
43. Schwartz J. Particulate air pollution and chronic respiratory disease. *Environmental Research*, 1993, 62:7–13.
44. *The world health report 2002: reducing risks, promoting healthy life*. Geneva, World Health Organization, 2002.
45. Loddenkemper R, ed. *European lung white book. The first comprehensive survey on respiratory health in Europe*. Lausanne, European Respiratory Society, 2003.
46. Holland M et al. *Methodology for the cost-benefit analysis for CAFE. Volume 3. Uncertainty in the CAFE CBA: methods and first analysis*. Brussels, European Commission, 2005 ([http://europa.eu.int/comm/environment/air/cale/activities/pdf/cba\\_method\\_vol3.pdf](http://europa.eu.int/comm/environment/air/cale/activities/pdf/cba_method_vol3.pdf), accessed 13 December 2005).

47. Le Tertre A et al. Short-term effects of particulate air pollution on cardiovascular diseases in eight European cities. *Journal of Epidemiology and Community Health*, 2002, 56:773–779.
48. Ostro BD. Air pollution and morbidity revisited: a specification test. *Journal of Environmental Economics and Management*, 1987, 14:87–98.
49. Ostro BD, Rothschild S. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. *Environmental Research*, 1989, 50:238–247.
50. Oak Ridge National Laboratory/Resources for the Future. *Estimating externalities of the coal fuel cycle. Report 3 on the external costs and benefits of fuel cycles. A study by the US Department of Energy and the Commission of the European Communities*. McGraw Hill, 1994.
51. Roemer W et al. Pollution effects on asthmatic children in Europe: the PEACE study. *Clinical and Experimental Allergy*, 2000, 30:1067–1075.
52. International Study of Asthma and Allergies in Childhood Steering Committee. Worldwide variations in the prevalence of asthma symptoms: the international study of asthma and allergies in childhood (ISAAC). *European Respiratory Journal*, 1998, 12:315–335.
53. Variations in the prevalence of respiratory symptoms, self-reported asthma attacks, and use of asthma medication in the European Community Respiratory Health Survey (ECRHS). *European Respiratory Journal*, 1996, 9:687–695.
54. Ward DJ, Ayres JG. Particulate air pollution and panel studies in children: a systematic review. *Occupational and Environmental Medicine*, 2004, 61(4):e13.
55. van der Zee S et al. Acute effects of urban air pollution on respiratory health of children with and without chronic respiratory symptoms. *Occupational and Environmental Medicine*, 1999, 56:802–812.
56. Hoek G, Brunekreef B. Effect of photochemical air pollution on acute respiratory symptoms in children. *American Journal of Respiratory and Critical Care Medicine*, 1995, 151:27–32.
57. Dusseldorp A et al. Associations of PM<sub>10</sub> and airborne iron with respiratory health of adults near a steel factory. *American Journal of Respiratory and Critical Care Medicine*, 1995, 152:1932–1939.
58. Neukirch F et al. Short-term effects of low-level winter pollution on respiratory health of asthmatic adults. *Archives of Environmental Health*, 1998, 53:320–328.
59. Hiltermann TJN et al. Asthma severity and susceptibility to air pollution. *European Respiratory Journal*, 1998, 11:686–693.
60. van der Zee S et al. Acute effects of urban air pollution on respiratory health of 50–70 yr old adults. *European Respiratory Journal*, 2000, 15:700–709.
61. Hajat S et al. Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London. *Thorax*, 1999, 54:597–605.
62. Hajat S et al. Association between air pollution and daily consultations with general practitioners for allergic rhinitis in London, United Kingdom. *American Journal of Epidemiology*, 2001, 153:704–714.
63. Hajat S et al. Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. *Occupational and Environmental Medicine*, 2002, 59:294–299.
64. Krupnick AJ, Harrington W, Ostro B. Ambient ozone and acute health effects: Evidence from daily data. *Journal of Environmental Economics and Management*, 1990, 18:1–18.



## 8. Risk estimates

### KEY MESSAGES

- According to RAINS model estimates, current exposure to PM from anthropogenic sources leads to the loss of 8.6 months of life expectancy in Europe. The impacts vary from around 3 months in Finland to more than 13 months in Belgium.
- The total number of premature deaths attributed to exposure amounts to around 348 000 annually in the EU (25 countries).
- Apart from effects on mortality, some 100 000 hospital admissions per year can be attributed to exposure. Several other impacts on morbidity are also expected to occur but the existing database is weak, affecting the precision and reliability of the estimates.
- Current legislation related to the emission of pollutants is expected to reduce impacts by around one third. A further reduction, down to around 50% of those estimated for current pollution levels, could be achieved by implementing all currently feasible emission reduction measures (the maximum feasible reduction (MFR) scenario).

### 8.1 Introductory remarks

The main purpose of integrated assessment modelling is to identify cost-effective strategies for reducing exposure to PM in Europe. Since nonanthropogenic PM sources are clearly not under human control, it is not necessary to include such sources in the framework of RAINS. The main aim is not to provide a complete analysis of total health impacts but to identify those European sources that contribute significantly to PM exposure and that can be controlled.

As shown in Chapter 5, not all sources are currently included in the EMEP model, which is the basis for atmospheric dispersion in RAINS. As a consequence, PM concentrations predicted by the RAINS model are usually lower than those measured at monitoring sites. The main nonanthropogenic sources include marine aerosols (consisting mainly of sea salt) and natural dust (such as dust from the Sahara), secondary aerosols resulting from natural emissions of non-methane VOC (e.g. monoterpenes), sulfur dioxide (e.g. from volcanoes), nitrogen oxides, ammonia and natural biological PM (such as debris from plants and microorganisms). Taken together, these sources can contribute several  $\mu\text{g}/\text{m}^3$  and more as an annual mean to  $\text{PM}_{10}$  mass (1–3).

Secondary organic aerosols originating from

anthropogenic emissions of non-methane VOC are also currently not included in the model. This leads not only to an underestimation of total anthropogenic PM levels (and therefore possibly the effects) but also has potential implications for the identification of cost-effective control strategies, since emissions of non-methane VOC as precursors of secondary organic aerosols are not considered.

The model of  $\text{PM}_{2.5}$  concentrations used for the risk assessment includes primary particles from anthropogenic sources as well as secondary inorganic aerosols. Contributions from natural sources and secondary organic aerosols are not considered in the risk assessment.

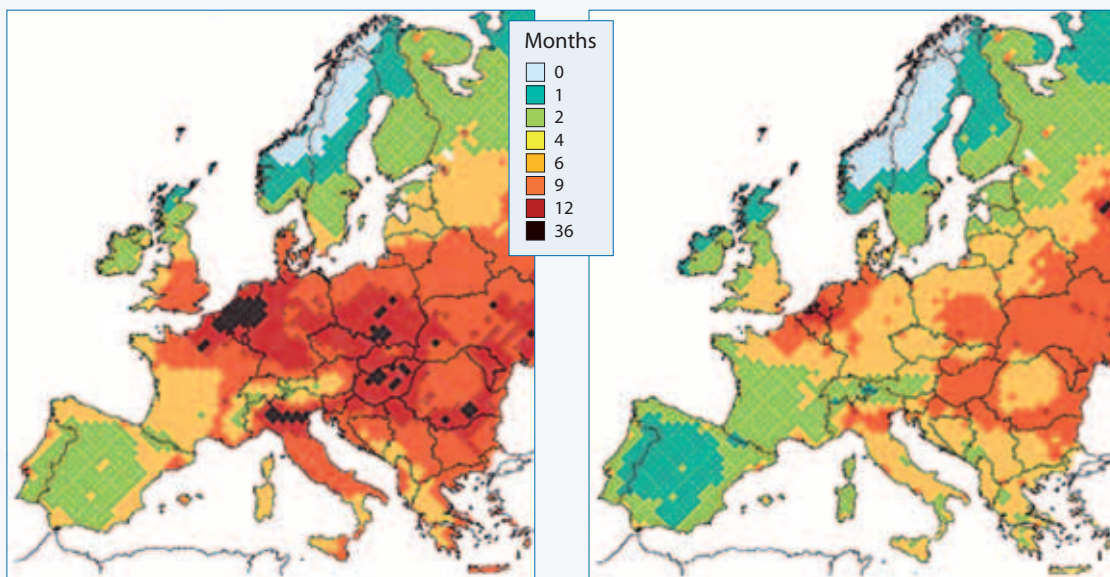
### 8.2 Impact of PM on life expectancy as estimated by the RAINS model

The RAINS model has been used to quantify the impacts of PM exposure on the statistical life expectancy of the European population, as outlined in Chapter 7. Based on the concentration–response function derived from the cohort studies, the RAINS model estimates health impacts for a given air pollution control scenario through the following steps.

1. Obtain, for all European countries, information (a) on current mortality rates from UN popula-

- tion statistics and (b) on future baseline mortality rates that are implied by the United Nations world population projections.
- Estimate exposure of the European population to PM pollution using the EMEP model, assuming a 25% increment in  $PM_{2.5}$  concentration over the grid estimate in the cities located in the grid cell (“city effect”). Such an increment, resulting from local low-level emission sources, has been suggested by the results of the CITY-DELTA project for continental-scale risk assessment (4). Several exposure estimates were calculated to analyse the impact of emission reduction policies: (a) for 1990; (b) for 2000; (c) for 2010, assuming implementation of presently decided emission controls; and (d) for different emission reduction scenarios (up to 2020), including the lowest PM levels that could hypothetically be achieved by full application of present-day technical emission controls. This requires spatially explicit information on population densities and on PM levels resulting from the three emission scenarios.
  - Using associations between PM pollution and mortality found by epidemiological studies (the linear concentration–response function, with risk coefficient 6% per 10  $\mu\text{g}/\text{m}^3$  annual mean  $PM_{2.5}$ ), determine the modification of mortality rates due to PM pollution.
  - Calculate changes in life expectancy (compared to the baseline United Nations scenario) resulting from the modified exposures to PM pollution of the three emission scenarios for each of the EMEP  $50 \times 50$ -km grid cells. Country-specific changes in life expectancy can also be calculated.
  - Examine how sensitive these estimates are to changes in the underlying assumptions.
- These calculations are carried out for all of Europe with a spatial resolution of  $50 \times 50$  km, corresponding to the grid system defined by EMEP. While the dispersion model used for this analysis calculates ambient concentrations of PM at this resolution, the present spatial distribution of population in Europe had to be compiled from a variety of sources. For 2010, the assumption was made that, within each country, the spatial distribution of population will

**Fig. 8.1. Loss in statistical life expectancy that can be attributed to the identified anthropogenic contributions to  $PM_{2.5}$  (in months) for emissions in 2000 (left) and emissions of the CAFE baseline scenario for 2020 (right) (average of calculations for the four meteorological years 1997, 1999, 2000 and 2003)**



**Table 8.1. Estimates of loss in statistical life expectancy that can be attributed to the identified anthropogenic contributions to PM<sub>2.5</sub> (in months) for emissions in 2000 and the “no further climate measures” scenario for 2010 and 2020**

EU Member State	2000			2010			2020		
	Central estimate	Range		Central estimate	Range		Central estimate	Range	
Austria	8.0	7.4	9.0	5.9	5.5	6.8	4.8	4.5	5.5
Belgium	13.6	11.7	15.4	9.9	8.5	11.3	8.8	7.6	10.0
Denmark	7.3	6.6	8.7	5.8	5.2	7.0	5.3	4.8	6.4
Finland	3.1	2.6	3.7	2.7	2.2	3.2	2.4	2.0	2.9
France	8.2	7.0	9.3	5.9	4.9	6.7	5.1	4.3	5.8
Germany	10.2	8.9	11.6	7.5	6.5	8.6	6.4	5.6	7.3
Greece	7.1	7.0	7.3	5.8	5.7	5.9	5.2	5.1	5.3
Ireland	3.9	2.9	5.1	3.0	2.2	4.0	2.7	2.0	3.6
Italy	9.0	8.5	9.6	6.6	6.2	7.1	5.6	5.3	6.0
Luxembourg	9.7	8.0	11.2	7.1	5.6	8.2	6.0	4.8	7.1
Netherlands	12.7	10.9	14.6	9.7	8.2	11.2	9.0	7.6	10.2
Portugal	5.2	4.9	5.4	3.4	3.2	3.6	3.2	3.0	3.4
Spain	5.1	5.0	5.4	3.5	3.4	3.7	3.2	3.1	3.3
Sweden	4.3	3.9	5.2	3.4	3.1	4.2	3.2	2.9	3.8
United Kingdom	6.9	5.5	8.7	4.9	3.8	6.4	4.5	3.5	5.7
Total, 15 Member States	8.2	7.4	9.3	6.0	5.4	6.8	5.3	4.7	5.9
Cyprus	—	—	—	—	—	—	—	—	—
Czech Republic	10.1	9.2	11.2	7.2	6.5	8.1	5.7	5.1	6.4
Estonia	4.4	3.7	5.2	3.8	3.2	4.6	3.4	2.9	4.2
Hungary	12.4	11.6	13.6	8.9	8.3	9.8	7.1	6.6	7.9
Latvia	5.1	4.4	6.1	4.4	3.7	5.3	3.9	3.3	4.7
Lithuania	6.9	6.2	8.1	5.9	5.3	7.0	5.2	4.6	6.0
Malta	7.7	7.4	8.0	6.8	6.5	7.1	7.4	7.0	7.8
Poland	10.7	9.9	11.8	8.1	7.4	9.0	6.4	5.9	7.2
Slovakia	10.4	9.6	11.4	7.7	7.1	8.6	6.2	5.7	6.9
Slovenia	9.3	8.7	10.3	6.9	6.4	7.7	5.7	5.3	6.3
Total, new Member States	10.3	9.5	11.4	7.7	7.1	8.6	6.2	5.7	6.9
Grand total	8.6	7.7	9.6	6.3	5.6	7.1	5.4	4.9	6.1

Note: The central estimates present the average of four calculations for four meteorological years (1997, 1999, 2000, 2003), while the range indicates the variation across individual meteorological conditions.

Source: CAFE (5).

remain unchanged. The age group distribution, as well as the life tables for the population in a grid cell, was deduced from the United Nations national data set.

For each country, the age-specific baseline non-accidental mortalities contained in such life tables are calculated from population statistics as the quotient

of deaths to population for five-year periods between 2000 and 2050. These calculations were carried out at national level, using statistics and projections of cohort sizes and numbers of deaths provided by the United Nations Population Division (2000). For estimating losses in life expectancy, all cohorts aged at least 30 years in 2010 are followed over their whole



lifetime, i.e. from 2010 to 2075. The mortality rates projected by the United Nations scenario for 2050 were assumed to be constant between 2050 and 2075.

The RAINS model estimates changes in the loss in statistical life expectancy that can be attributed to changes in anthropogenic emissions (ignoring the role of secondary organic aerosols). This calculation is based on the assumption that health impacts can be associated with changes in PM<sub>2.5</sub> concentrations, but no health impacts are calculated for PM from natural sources and for secondary organic aerosols. Since the risk function used in this analysis is based on the cohort study conducted in the population over 30 years of age, the assessment in RAINS does not quantify infant mortality and thus underestimates overall effects.

Results from these provisional estimates are presented in Fig. 8.1, based on the average of four calculations conducted for four different meteorological years. The reductions in baseline emissions will significantly reduce calculated losses in life expectancy in Europe (although even in 2020, for large sections of the population, life expectancy losses attributable to anthropogenic PM are calculated to exceed six months). Obviously, these calculations are sensitive to the meteorological conditions assumed in the analysis. While by definition these calculations address long-term exposure to PM, there is uncertainty about the meteorological conditions that are most representative for present and future climates.

Estimates for each of the 25 EU countries are presented in Table 8.1

Fig. 8.2 compares the national estimates of loss in life expectancy in the 25 EU countries for the year 2000 and predictions for 2020 under the current legislation (CLE) scenario, with the impacts of PM reduced under the MFR scenario. For most countries, the reduction in health impacts expected under current legislation amounts to about half of the distance between the current impacts and those still existing under the MFR scenario.

### 8.3 Other estimates of the health impact of PM

This section summarizes the results of the CAFE CBA (6). The exposure information is based on the RAINS calculations presented above, assuming aver-

**Table 8.3. Main indicators of the damage to health and its expected reduction due to implementation of current emission-reduction legislation up to 2020, estimated by cost-benefit analysis for the 25 EU Member States**

Impact estimate for 2000	Expected reduction of the impact in 2020 (CLE scenario)
3.6 million life years lost	1.2 million
348 000 attributable deaths per year	76 000
100 000 hospital admissions annually	30 000

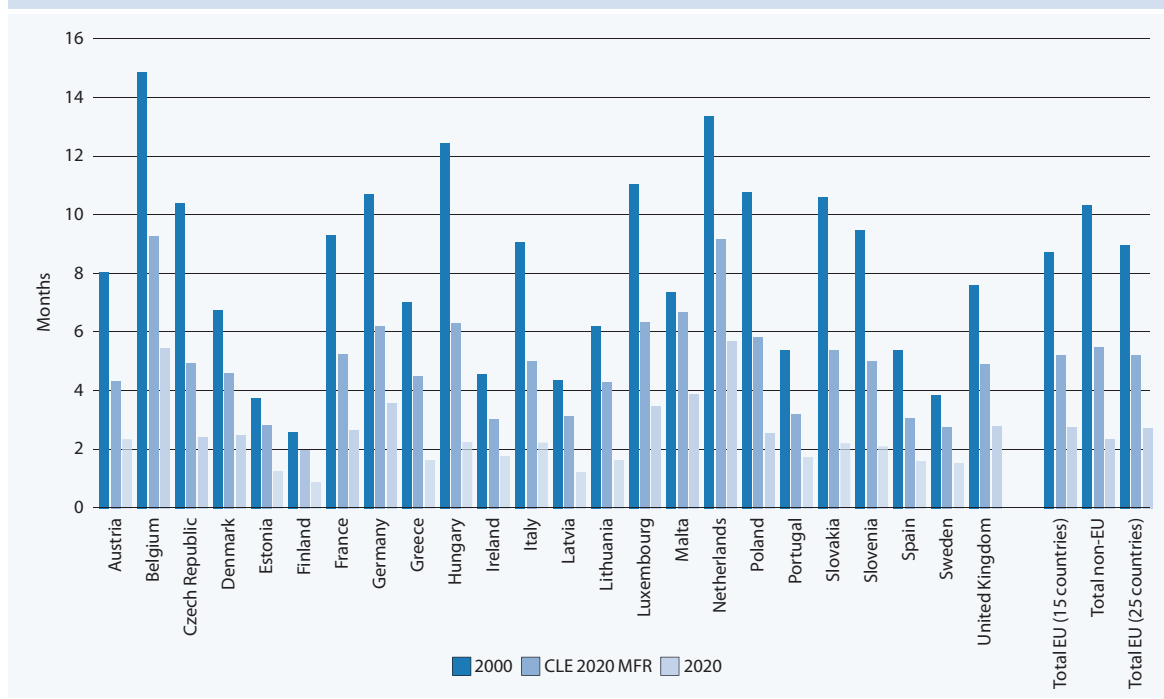
Source: Watkiss (7).

**Table 8.4. Annual number of deaths attributable to exposure to PM in the EU and the expected reduction owing to implementation of current emission-reduction legislation up to 2020, estimated by cost-benefit analysis for the 25 EU Member States**

Country	Baseline, 2000 (thousands)	Change, 2000-2020 (thousands)
Austria	5.5	0.9
Belgium	12.8	2.9
Cyprus	0.2	0.0
Czech Republic	9.1	2.6
Denmark	3.3	0.5
Estonia	0.6	0.2
Finland	1.3	0.0
France	42.1	7.4
Germany	75.0	12.5
Greece	7.2	0.3
Hungary	12.9	4.5
Ireland	1.2	0.2
Italy	50.7	12.8
Latvia	1.3	0.4
Lithuania	2.2	0.5
Luxembourg	0.3	0.0
Malta	0.2	0.0
Netherlands	15.5	1.6
Poland	32.9	8.0
Portugal	5.0	1.5
Slovakia	4.3	0.9
Slovenia	1.6	0.3
Spain	19.9	5.8
Sweden	3.3	0.6
United Kingdom	39.5	12.1
All EU countries	347.9	76.3

Source: Watkiss (7).

**Fig. 8.2. Loss in average statistical life expectancy that can be attributed to the identified anthropogenic contributions to PM<sub>2.5</sub> (in months) in Europe for emissions in 2000, the CLE scenario for 2020 and the MFR scenario for 2020 (calculated for the meteorological conditions of 1997)**



age weather conditions in 2000 and 2020. The meteorological year of 1997 has been selected to be the representative year for such weather conditions.

The results are presented as annual environmental and health and impacts. The following scenarios have been analysed:

- The situation in 2000;
- The situation in 2020, assuming that current air pollution legislation is implemented and EU Member States carry out their climate policy obligations under the Kyoto Protocol and continue implementing greenhouse gas reduction policies consistent with the assumptions used by the RAINS model; and
- the difference between these years, i.e. the impact of current policies up to 2020.

The analysis estimated that over three million life years were lost in the EU (25 countries) in 2000 through exposure to PM (Table 8.3). This is equivalent to about 348 000 attributable deaths annually.

Both of these health impacts are based on, and are thus consistent with, the estimates from loss of life expectancy in the RAINS model, which calculates the total (not annual) change in life years. National estimates are presented in Table 8.4.

Further estimates of impacts of PM exposure in the year 2000 include an additional 680 attributable infant deaths, some 32 million respiratory medication use days, and several hundred million restricted activity days. Significant reductions in annual impacts over the period 2000–2020 are expected under the CLE scenario, but the remaining burden to health remains significant.

## References

1. Querol X et al.  $PM_{10}$  and  $PM_{2.5}$  source apportionment in the Barcelona Metropolitan Area, Catalonia, Spain. *Atmospheric Environment*, 2001, 35/36:6047–6419.
2. Querol X et al. *Levels and composition of  $PM_{10}$  and  $PM_{2.5}$  in Spain*. Madrid, Ministry of the Environment, 2003.
3. Buringh E, Opperhuizen A, eds. *On health risks of ambient PM in the Netherlands. Executive summary*. Bilthoven, National Institute for Public Health and the Environment (RIVM), 2002.
4. CITY-DELTA European Modelling Exercise. An inter-comparison of long-term model responses to urban-scale emission-reduction scenarios (<http://rea.ei.jrc.it/netshare/thunis/citydelta>, accessed 22 October 2005).
5. Amman M et al. Baseline scenarios for the Clean Air for Europe (CAFE) Programme. Final Report. Laxenburg, International Institute for Applied Systems Analysis, 2005 (CAFE Scenario Analysis Report No. 1) ([http://europa.eu.int/comm/environment/air/cale/activities/pdf/cale\\_scenario\\_report\\_1.pdf](http://europa.eu.int/comm/environment/air/cale/activities/pdf/cale_scenario_report_1.pdf), accessed 17 December 2005).
6. Hurley F et al. *Methodology for the cost–benefit analysis for CAFE. Volume 2: health impact assessment*. Brussels, European Commission, 2005 ([http://europa.eu.int/comm/environment/air/cale/pdf/cba\\_methodology\\_vol2.pdf](http://europa.eu.int/comm/environment/air/cale/pdf/cba_methodology_vol2.pdf), accessed 13 December 2005).
7. Watkiss P et al. *CAFE CBA: Baseline analysis 2000 to 2020*. Didcot, AEA Technology Environment., 2005 ([http://europa.eu.int/comm/environment/air/cale/activities/pdf/cba\\_baseline\\_results2000\\_2020.pdf](http://europa.eu.int/comm/environment/air/cale/activities/pdf/cba_baseline_results2000_2020.pdf), accessed 17 December 2005).

## 9. Conclusions and recommendations

### 9.1 Particulate air pollution causes significant human health effects in Europe

The analysis summarized by the previous chapters demonstrates that PM in ambient air currently creates a significant threat to human health in Europe. The most severe effects relate to an increased risk of premature death, expressed by the estimated 8.6 months' average reduction in life expectancy in the 25 countries of the EU. Some 3.6 million life years are lost through some 348 000 attributable deaths annually due to current PM exposure in the EU (emission data from 2000). PM exposure also has an impact on various less severe health end-points, including 100 000 hospital admissions per year due to respiratory and cardiac emergencies. These effects are not uniformly distributed across Europe, being three times higher in the most polluted than in the least polluted areas. By 2020, it is expected that loss of life expectancy in Europe will fall owing to the implementation of currently existing legislation, and this will lead to further reductions of emissions. Nevertheless, even with this reduction in exposure, statistical loss of life expectancy due to PM in Europe will still exceed five months on average across the population. Morbidity will also decrease owing to the expected reduction in PM levels, but the numbers of hospital admissions and of other, milder health problems attributed to PM exposure will still remain significant.

### 9.2 The health effects of long-range particulate pollution are approximately proportional to the fraction of ambient PM concentrations attributable to long-range sources

There is no clear, direct evidence identifying which of the many sources of PM are responsible for the effects and, in particular, to what extent these effects are caused by PM from long-range transport of pollution. WHO working groups recognized that some components of the PM mixture common in long-range-transported PM (ammonium salts, chlorides, sulfates and nitrates) have lower toxicity under laboratory conditions than other components. However,

it is important to note that PM from long-range transport also contains considerable concentrations of components identified as hazardous in toxicological studies, such as organic materials and transition metals. WHO concluded that it is currently not possible to quantify precisely, and differentiate, the contributions from different sources and different PM components to the effects on health caused by exposure to ambient PM. It is reasonable to assume that the magnitude of the health effects of PM from anthropogenic sources is proportional to the level of population-weighted exposure estimated by mass concentration of ambient  $PM_{2.5}$  in urban background and rural locations, together with data on population density. On that basis, and following WHO advice on the toxicity of components, the contribution of PM from long-range transport to the health effects of PM could be proportional to the fraction of population-weighted PM exposure attributable to long-range transport of pollution.

PM consists of a mixture of solid and liquid particles suspended in the air, which can either be directly emitted into the air (primary PM) or be formed secondarily in the atmosphere from gaseous precursors (mainly sulfur dioxide, nitrogen oxides, ammonia and non-methane VOC). Both primary and secondary particles originate from human activities and from natural sources. Anthropogenic sources of primary particles include particularly fuel combustion from transport, power generation and domestic heating, and also the handling of different materials during construction work, industrial production processes, mechanical abrasion of different materials and agricultural activities.

The aerodynamic diameter of particles is closely associated with the observed health effects. Particles with a diameter less than  $10\ \mu\text{m}$  ( $PM_{10}$ ) are able to penetrate the upper respiratory tract defences and have therefore become a common indicator for PM ambient concentrations and emission. Recent WHO evaluations point to the health significance of even smaller particles, those with a diameter less than  $2.5\ \mu\text{m}$  ( $PM_{2.5}$ ). In particular, the effects of long-term PM exposure on mortality (life expectancy)

seem to be attributable to  $PM_{2.5}$  rather than to coarser particles. The latter, with a diameter of 2.5–10  $\mu m$  ( $PM_{2.5-10}$ ) may have more visible effects on respiratory morbidity.

Particulates in the size range between 0.1 and a few micrometers have considerable potential for transboundary transport. These particles may remain for up to several days in the atmosphere and can thereby be transported 1000 or more kilometres. Pollution dispersion models, and in particular the EMEP model, indicate that the contribution of transboundary sources to average  $PM_{2.5}$  concentrations of anthropogenic origin ranges from around 30% in large European countries to 90% in the smaller ones. Correspondingly, a large part of pollution emitted by each country substantially affects air quality in neighbouring countries, and not only close to their common borders.

In the last few years,  $PM_{10}$  has become widely monitored in western parts of Europe, mainly owing to the requirements of EU directives. Annual average concentrations of  $PM_{10}$  vary widely between different countries and regions; however, typical annual mean concentrations are about 26  $\mu g/m^3$  in urban (background) areas and 32  $\mu g/m^3$  in street canyons, whereas in rural areas annual averages of approximately 22  $\mu g/m^3$  are reached. In most areas in Europe,  $PM_{10}$  levels are dominated by the rural background component, which also contributes 75% of the urban background concentrations.  $PM_{2.5}$  is measured to a much lesser extent than  $PM_{10}$ . Annual average concentrations of  $PM_{2.5}$  in urban areas reach 15–20  $\mu g/m^3$  at urban background stations, typically 20–30  $\mu g/m^3$  at traffic sites and 11–13  $\mu g/m^3$  at rural background stations. Though there are sources of pollution in rural areas, a substantial portion of the PM there arrives by long-range transport.

Although relevant studies are still scarce, the existing data indicate that population exposure to PM from outdoor sources is to a large extent influenced by ambient concentrations of PM, which therefore act as a good proxy measure of population exposure. Furthermore, source apportionment analyses of personal exposure have confirmed that ambient measurements of long-range transboundary PM, as estimated by secondary sulfate, are representative of population exposure to PM.

These observations, combined with the understanding that there is a considerable contribution by long-range transport to PM concentrations in cities also, confirm that the long-range transport of PM significantly affects exposure levels of the population in Europe. Thus impact assessment based on estimates of the ambient concentration of PM should also be relevant for the long-range transport fraction of the pollution.

### **9.3 Current policies to further reduce emissions will bring further improvements but nevertheless leave a significant health burden**

Anthropogenic emissions of  $PM_{10}$  and  $PM_{2.5}$  in the year 2000 reached about 5600 and 3800 kilotonnes, respectively, in the EMEP domain and fell by around half between 1990 and 2000. Emissions of PM precursors (ammonia, nitrogen oxides and sulfur dioxide) also fell significantly in the final decades of the last century. Further reductions of primary PM emissions and precursors (in particular nitrogen oxides and sulfur dioxide) of the same magnitude are expected in the EU owing to further implementation of existing legislation. In addition to an observed relative increase of transport emissions compared to emissions from the industrial sector between 1990 and 2000, the domestic sector is expected to become an increasingly more important source of PM emissions in the future. Moreover, in contrast to all other sources of primary PM, international shipping emissions are predicted to increase in the next 20 years. Trend analyses (e.g. from the United Kingdom) indicate that changes in emissions are, in general, well reflected by changes in ambient concentrations of  $PM_{10}$ . It can therefore be inferred that changes in emissions are also followed by changes in PM concentrations and subsequently in the effect of PM on population exposure and health.

### **9.4 Key uncertainties, research needs and current policy implications**

There are still a number of uncertainties accompanying the process of the evaluation of health effects caused by ambient PM.

- There is as yet only incomplete quantitative knowledge available about the sources of parti-

cle emission in the various European countries. The RAINS model estimates emissions based on national data of sectoral economic activities as well as emission factors from the literature and from national sources.

- Ambient concentrations of PM have to be calculated from emissions of this pollutant and its precursor substances, taking account of dispersion and chemical reactions in the atmosphere. This is especially difficult, since PM in ambient air originates from a number of different sources and the chemical and physical reactions are complex. Not all processes are yet fully understood, for example the formation of secondary organic aerosols. The calibration and validation of the models, in particular of the EMEP model used for the present analysis, is based on the EMEP network monitoring data, but the PM monitoring (including chemical speciation) is still limited and not sufficiently representative. The models are also sensitive to meteorological conditions and there is uncertainty about the meteorological conditions that are most representative of present and future climates.
- Source apportionment analyses for personal exposure rely on a very limited number of exposure studies on PM. Population-based source-specific exposure modelling has not been fully developed and long-term exposure to specific sources has not been investigated.
- Although some PM components are considered to be more hazardous than others, it is currently not possible to precisely quantify the contributions from different sources and different PM components as well as contribution of regional vs local pollutants to the observed health effects of short- and long-term exposure.
- There are still questions to be resolved about expressing the effects of PM on mortality in terms of life expectancy or attributable deaths.

### 9.5 Implications of the assessment

Reduction of these uncertainties requires further concerted efforts by scientists of various disciplines conducting basic research on PM and its health effects, improvements in the quality and spatial coverage of the data on emissions, more information from air quality monitoring, and a deeper understanding of those

components of PM that are crucial for the observed impacts. However, even with the current uncertainties, the scientific evidence indicating serious health effects from exposure to ambient PM now and in the coming years is sufficient to encourage policy action for a further reduction of PM levels in Europe. Since the long-range transport of pollution contributes a major part of the ambient levels of PM and of the population exposure, international action must accompany local and national efforts to cut pollution emissions and to reduce its effects on health.



# Annex 1.

## Participants in the 8th Meeting of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution

Markus Amann	International Institute for Applied Systems Analysis, Austria
Yevhen Berezhny	Main Department of the State Environmental Monitoring and Air Protection, Ukraine
Khatuna Chikviladze	Hydromet Department, Georgia
Geert de Jong <sup>a</sup>	Shell Health Services, Belgium
Frank de Leeuw	National Institute of Public Health and the Environment (RIVM), Netherlands
Richard Derwent	Rdscientific, United Kingdom
Norbert Englert	Federal Environment Agency, Germany
Elena Evstafjeva	Crimean State Medical University, Ukraine
Bertil Forsberg	Umeå University, Sweden
Larry Gephart	Exxon Mobil Biomedical Sciences Inc., Belgium
Heinz-Detlef Gregor	Federal Environmental Agency, Germany
Fintan Hurley	Institute of Occupational Medicine, United Kingdom
Marek Jakubowski	Nofer Institute of Occupational Medicine, Poland
Matti Johansson	United Nations Economic Commission for Europe, Switzerland
Rokho Kim	WHO European Centre for Environment and Health, Germany
Michal Krzyzanowski	WHO European Centre for Environment and Health, Germany
Birgit Kuna-Dibbert <sup>a</sup>	WHO European Centre for Environment and Health, Germany
Titus Kyrklund	Swedish Environmental Protection Agency, Sweden
Steinar Larssen	Norwegian Institute for Air Research, Norway
Sally Jane Liu	Institute of Social and Preventive Medicine, Switzerland
Corinne Mandin	Institut National de l'Environnement Industriel et des Risques, France
Irina Priputina	Institute of Urban Ecology, Russian Federation
Oliver Günter Schall	Federal Ministry of the Environment, Nature Conservation and Nuclear Safety, Germany
Jürgen Schneider	Federal Environment Agency, Austria
Wolfgang Schoepp <sup>a</sup>	International Institute for Applied Systems Analysis, Austria
Per E. Schwarze	Norwegian Institute of Public Health, Norway
David Simpson	Norwegian Meteorological Institute, Norway
John Stedman <sup>a</sup>	AEA Technology, NETCEN, United Kingdom
Peter Straehl	Swiss Agency for the Environment, Forests and Landscapes, Switzerland
Leonor Tarrasón	Norwegian Meteorological Institute, Norway
Barbara Toczko	Chief Inspectorate for Environmental Protection, Poland
Leendert van Bree	National Institute of Public Health and the Environment (RIVM), Netherlands
Andre Zuber	European Commission, Belgium

<sup>a</sup> Contributing to the report but not participating in the meeting.



Particulate matter is a type of air pollution that is generated by a variety of human activities, can travel long distances in the atmosphere and causes a wide range of diseases and a significant reduction of life expectancy in most of the population of Europe.

This report summarizes the evidence on these effects, as well as knowledge about the sources of particulate matter, its transport in the atmosphere, measured and modelled levels of pollution in ambient air, and population exposure. It shows that long-range transport of particulate matter contributes significantly to exposure and to health effects.

The authors conclude that international action must accompany local and national efforts to cut pollution emissions and reduce their effects on human health.

## **World Health Organization Regional Office for Europe**

Scherfigsvej 8, DK-2100 Copenhagen Ø, Denmark  
Tel.: +45 39 17 17 17. Fax: +45 39 17 18 18.  
E-mail: [postmaster@euro.who.int](mailto:postmaster@euro.who.int)  
Web site: [www.euro.who.int](http://www.euro.who.int)

### **The WHO Regional Office for Europe**

The World Health Organization (WHO) is a specialized agency of the United Nations created in 1948 with the primary responsibility for international health matters and public health. The WHO Regional Office for Europe is one of six regional offices throughout the world, each with its own programme geared to the particular health conditions of the countries it serves.

#### **Member States**

Albania  
Andorra  
Armenia  
Austria  
Azerbaijan  
Belarus  
Belgium  
Bosnia and Herzegovina  
Bulgaria  
Croatia  
Cyprus  
Czech Republic  
Denmark  
Estonia  
Finland  
France  
Georgia  
Germany  
Greece  
Hungary  
Iceland  
Ireland  
Israel  
Italy  
Kazakhstan  
Kyrgyzstan  
Latvia  
Lithuania  
Luxembourg  
Malta  
Monaco  
Netherlands  
Norway  
Poland  
Portugal  
Republic of Moldova  
Romania  
Russian Federation  
San Marino  
Serbia and Montenegro  
Slovakia  
Slovenia  
Spain  
Sweden  
Switzerland  
Tajikistan  
The former Yugoslav  
Republic of Macedonia  
Turkey  
Turkmenistan  
Ukraine  
United Kingdom  
Uzbekistan