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Health effects of transport-related air pollution

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Health effects of transport-related air pollution

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Michal Krzyzanowski, Birgit Kuna-Dibbert
and Jürgen Schneider

Abbreviations

Organizations, other entities and studies

ADAC	Allgemeiner Deutscher Automobil Club
AIRNET	Thematic Network on Air Pollution and Health
APHEA2	Air Pollution and Health: a European Approach 2
CAFE	Clean Air for Europe
CANTIQUE	project on concerted actions on non-technical measures and their impact on air quality and emissions
CEN	European Committee for Standardization
EC	European Commission
EEA	European Environment Agency
EECCA	eastern Europe, the Caucasus and central Asia
EFTA	European Free Trade Association
EPEFE	European Programme on Emissions, Fuels and Engines Technologies
EU	European Union
EXPOLIS	study of air pollution exposure distributions of adult urban populations in Europe
IARC	International Agency for Research on Cancer
ISAAC	International Study of Asthma and Allergies in Childhood
HEAVEN	project on a healthier environment through the abatement of vehicle emissions and noise
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
PEACE	study of acute pollution effects on asthmatic children
SAVIAH	Small Area Variations in Air Quality and Health study
THE PEP	Transport, Health and Environment Pan-European Programme
TRAPCA	project on transport-related air pollution on childhood asthma
UNECE	United Nations Economic Commission for Europe

Technical terms

1-OH-pyrene	1-hydroxypyrene
8-OHdG	8-hydroxy-2'-deoxyguanosine

8-oxodG	8-oxo7,8-dihydro-2'-deoxyguanosine
CAPs	concentrated ambient particles
CI	confidence interval
CoPM	combustion and other particulate matter
DEPs	diesel exhaust particles
DNA	deoxyribonucleic acid
ECG	electrocardiogram
$FEF_{25–75\%}$	forced mid-expiratory flow
GIS	geographical information systems
GDP	gross domestic product
GM-CSF	granulocyte-macrophage colony stimulating factor
GNP	gross national product
ICAM-1	intercellular adhesion molecule 1
IFN-γ	interferon gamma
Ig	immunoglobulin
IL	interleukin
LFA-1	leukocyte function-associated antigen 1
MCP-1	monocyte chemoattractant protein 1
MCT	monocrotaline
OR	odds ratio
OSPM	Operational Street Pollution Model
OVA	ovalbumin
PAHs	polycyclic aromatic hydrocarbons
pkm	passenger-kilometres
PM	particulate matter
ppm	parts per million
RNA	ribonucleic acid
ROS	reactive oxygen species
RR	relative risk
SIR	standardized incidence ratio
SMR	standardized mortality ratio
SP	surfactant protein
SRM	standardized reference material
Th	T-helper
tkm	tonne-kilometres
TNF-α	tumour necrosis factor alpha
VCAM-1	vascular cell adhesion molecule 1
VOCs	volatile organic compounds

Foreword

Transport plays a fundamental role in the lives of societies and individuals: how people interact, work, play, organize production, develop cities, and get access to services, amenities and goods is inextricably linked with the development of mobility and the choices people make about it. In societies that rely heavily and increasingly on private motorized transport, vehicles are expected to become safer, more luxurious and powerful, and to be driven more frequently. These expectations, however, often do not take account of the ensuing consequences: increased fuel consumption, greater emissions of air pollutants and greater exposure of people to hazardous pollution that causes serious health problems. The increased intensity of and reliance on transport also increase the risk of road-traffic injuries, exposure to noise and sedentary lifestyles. These risks are a disproportional threat to the most vulnerable groups in the population, such as children and the elderly, and they raise important questions about social inequalities.

An increasing body of evidence points to the magnitude of these adverse effects on health and to the need to identify solutions that both reduce risks to health and meet the requirement for mobility. This creates a major challenge to governments, public health organizations and environmental authorities, to urban and transport planners, and to all citizens. Efforts to meet the challenge are reflected in a number of policy initiatives. Among them are the international implementation of the WHO/United Nations Economic Commission for Europe (UNECE) Transport, Health and Environment Pan-European Programme (THE PEP), the European Commission's Clean Air for Europe (CAFE) programme, which addresses transport-related air pollution, and the Environmental Strategy for Eastern Europe, Caucasus and Central Asia agreed at the 5th Ministerial Conference "Environment for Europe" in 2003.[1]

Properly understanding the risks is a prerequisite to addressing them and to eliminating or reducing them. One of WHO's key roles is to analyse the scientific evidence on health risks and to present the conclusions to governments, policy-makers, experts and the public, with the aim of protecting health. To help assess the health risks of

[1] Steering Group on Environmental Strategy for Countries of Eastern Europe, Caucasus and Central Asia (2003). *Environmental partnerships in the UNECE region: Environmental Strategy for Eastern Europe, Caucasus and Central Asia*. Geneva, UNECE (ECE/CEP/105/Rev. 1; http://www.unece.org/env/proceedings/files.pdf/Item%207/7a/7aDocuments/ece.cep.105.rev.1.e.pdf, accessed 12 December 2004).

transport-related air pollution presented in this book, WHO invited experts from a wide range of disciplines to help address different aspects of this complex issue.

Transport-related air pollution must be reduced before its effects on health can be prevented, and this requires:

- *combining the development of cleaner transport technologies with the implementation of effective policies to manage the demand for transport; and*
- *selecting modes of transport that are safer for health and the environment.*

The activities of populations, the planned use of spaces, individual behaviour and the choices available to transport users – all these affect people's exposure to pollution and the related health risks. Research on the effects on health of transport-related air pollution identifies hazards and indicates vulnerable groups. The participation of experts dealing with all these issues in the development of this book increased the value of the risk assessment it presents and should facilitate the use of its conclusions in implementing effective actions and policies. We at the WHO Regional Office for Europe are grateful for the contributions of the authors and reviewers and are confident that their efforts to ensure the best possible scientific standard for this publication will serve its readers well.

We hope that a better understanding of the health risk of transport-related air pollution will aid WHO Member States in their efforts to protect public health and the environment, and in their efforts to build a stronger evidence base. This could lead not only to the technological improvement of vehicles and fuels but also to changes in public behaviour and better management of transport demands and urban planning, allowing a wider introduction of healthy means of transport. Such changes would both reduce the health risks of transport-related air pollution and bring other health benefits, such as reduced risks of traffic accidents and the positive effects of walking and bicycling. These would greatly amplify the benefits and cost–effectiveness of investments in reducing pollution. The benefits to public health of such an integrated approach would be the most welcome result of this WHO book.

Marc Danzon
WHO Regional Director for Europe

Executive summary

The effects on health of transport-related air pollution are among the leading concerns about transport. Research in recent decades consistently indicates the adverse effects of outdoor air pollution on human health, and the evidence points to air pollution stemming from transport as an important contributor to these effects.

This book provides a systematic review of the literature on transport-related air pollution and a comprehensive evaluation of the health hazards of such pollution. It focuses on air pollution related to road transport (mostly from urban and suburban passenger and freight transport) and the risks it presents to human health. It also considers the entire chain of relevant issues: from patterns and trends in activities that determine the intensity of emissions from transport, to primary emissions and the formation of secondary pollutants by means of transportation, and finally through to patterns of human exposure to such pollutants. The discussion of the adverse effects on health considers the results of both epidemiological studies and toxicological assessments of biological mechanisms.

Factors determining emissions

In the coming decades, road transport is likely to remain a significant contributor to air pollution in cities. Many urban trips cover distances of less than 6 km. Since the effectiveness of catalytic converters in the initial minutes of engine operation is small, the average emission per distance driven is very high in urban areas. Also, poorly maintained vehicles that lack exhaust aftertreatment systems are responsible for a major part of pollutant emissions.

Contribution of traffic to pollution levels

Traffic contributes to a range of gaseous air pollutants and to suspended particulate matter (PM) of different sizes and composition. Tailpipe emissions of primary particles from road transport account for up to 30% of fine PM (less than 2.5 μm in aerodynamic diameter or PM2.5) in urban areas. Other emissions related to road transport (such as those from resuspended road dust, and wear of tyres and brake linings) are the most important source of the coarse fraction of PM (2.5–10 μm in aerodynamic diameter or PM10–2.5). Road transport is also the main contributor to emissions of nitrogen dioxide and benzene in cities and is the major reason for non-compliance with current European Union (EU) limit values for these pollutants.

In so-called street canyons (where pollutants are trapped) with heavy traffic, concentration levels of all transport-related pollutants are much higher than in areas not affected directly by pollution sources – that is, with urban background pollution levels. In a 0.5-km-wide belt along major urban highways, concentrations of nitrogen dioxide, black smoke (or soot) and ultrafine particles (PM0.1) are markedly higher than in areas with less traffic. Several other transport-related pollutants, however, spread more uniformly over large areas of a city.

Current policies should result in reduced concentrations of transport-related pollution and in improved air quality. The concentration in 2010 is expected to be roughly 50% of that in 1995. Also, in 2010, 90% of the urban population in the 15 countries belonging to the EU before 1 May 2004 are expected to live in areas meeting the EU air-quality limit values for nitrogen dioxide (hourly value), carbon monoxide, benzene and lead.

Technological improvements and stricter emission standards will decrease vehicle-specific emissions. Nevertheless, several factors – the growth of transport, an increased number of diesel cars on the market, the large number of short trips and traffic congestion – may offset the benefits derived from these improvements. The present trends in transport patterns in the central and eastern parts of the WHO European Region follow the patterns in the western part, posing the risk of traffic making an increased contribution to air pollution. In the next decade, alternative vehicle technologies are unlikely to make important inroads in the market or to have a significant impact on air quality. Also, a large proportion of the population is expected to continue living in areas where current EU standards for PM and long-term average nitrogen dioxide are exceeded, owing mainly to road-traffic emissions.

Other factors are likely to contribute to inhibiting or preventing the reduction in people's exposure to transport-related air pollution; these include expansion of urban areas, increases in commuting time and greater traffic congestion. The trend in these contributing factors may also counteract the average improvement of air quality, particularly with respect to the levels of some gaseous pollutants.

Human exposure

The volume and spatial distribution of the emissions, as well as dispersion conditions, affect pollution levels. Several other factors also play a part in determining the exposure of a population.

Pollution intake is also determined by the number of people in polluted areas, how long they stay there and what they do. Time–activity patterns, particularly residence or work near busy roads (or both), and time spent in traffic are critical for population exposure. Travellers are often exposed to levels that are three times the background levels. In-vehicle exposures are especially high for primary exhaust gases and PM. Groups with high levels of exposure include people who live near busy roads or who ventilate their

residences with air from road canyons with heavy traffic, road users (such as drivers, commuters and pedestrians) and people whose jobs require them to spend a long time on the roads.

Urban planning and development also strongly shape exposure; they determine not only patterns of residence and mobility but also the availability of public transport and non-motorized transport options. Although the available data and models restrict the possibility of making precise estimates and predictions of exposure patterns, traffic can still be said to be responsible for an increasing proportion of the population's exposure to air pollution.

Studies on health effects

The epidemiological and toxicological evidence on the effects of transport-related air pollution on health has increased substantially in recent decades. Although this includes epidemiological and toxicological evidence, it is only a fraction of the total evidence on the effects on health of urban air pollution.

A review of this evidence indicates that transport-related air pollution contributes to an increased risk of death, particularly from cardiopulmonary causes. It increases the risk of respiratory symptoms and diseases that are not related to allergies. Experimental research indicates that the effects are linked to changes in the formation of reactive oxygen species, changes in antioxidant defence, and increased inflammation, thus providing some indication of mechanisms of susceptibility. Laboratory studies indicate that transport-related air pollution may increase the risk of developing an allergy and can exacerbate symptoms, particularly in susceptible subgroups. The evidence from population studies, however, does not consistently support this notion. While only a few studies have been conducted on the effects of transport-related air pollution on cardiovascular morbidity, they report a significant increase in the risk of myocardial infarction following exposure. Other studies and the experimental evidence indicate that exposure results in changes in autonomic nervous system regulation and increased inflammatory responses. A few studies suggest an increased incidence of lung cancer in people with long-term exposure to transport-related air pollution. Some studies suggest that it also causes adverse outcomes in pregnancy, such as premature birth and low birth weight, but the available evidence is inconsistent.

Few reported studies analyse the effects of specific interventions, and even fewer focus on transport-related air pollution. They indicate that reducing this pollution may directly reduce acute asthma attacks in children and the related medical care. Long-term decreases in air-pollution levels are associated with declines in bronchial hyperreactivity, in the average annual trend in deaths from all causes, and in respiratory and cardiovascular diseases. Such decreases are also associated with gains in life expectancy.

Often, the effects observed in epidemiological studies cannot be attributed to the specific pollution indicator used in the study, but can be attributed to a mixture of

pollutants. Fine PM (including black smoke) and ozone are associated with increased risks of mortality and respiratory morbidity, while exposure to nitrogen dioxide, ozone and PM has been linked to allergic responses. Other indicators of exposure to transport-related air pollution – such as distance to or residence near major roads and, partly, self-reported traffic intensity at a residence – are associated with several adverse health outcomes.

This accumulated evidence allows the hazards of transport-related air pollution to be identified, but makes only a limited contribution to the qualitative assessment of its adverse effects on health and to the prediction of the benefits of reducing this part of the total air-pollution mix.

Initial estimates show that tens of thousands of deaths per year are attributable to transport-related air pollution in the Region, similar to the death toll from traffic accidents. The research database still needs improvement, however, to allow a more precise evaluation of the effects of and changes in exposure. More research is needed on the patterns and adverse health effects of population exposure and on the role of the different components of the pollution mix. The relevance of emissions from various transport-related sources (such as heavy- and light-duty diesel cars) to health issues also needs further investigation. More studies that assess the public health benefits of various measures to improve air quality – particularly through interventions that address transport-related air pollution – are needed to support policies.

Despite the need for further research, the expected health benefits thoroughly justify measures to reduce exposure to transport-related air pollution. Traffic management is one of the instruments that can significantly reduce the exposure of residents of urban areas. In addition, the integration of environmental and health considerations into urban planning can be improved. In particular, urban planning may aim at integrative measures that lower emission rates, such as the promotion of highly efficient, service-oriented and clean public transport and improvements in the flow of traffic. Several technologies show promise in lowering emission levels from conventional vehicles, and their development should be promoted, along with effective control mechanisms (such as mandatory car inspections) for eliminating gross polluters and badly maintained vehicles. Finally, alternative vehicle technologies and fuel substitutes could lead to substantial future reductions in emissions of hazardous air pollutants.

Introduction

Transport is a vital part of modern life. The freedom to travel short and long distances opens the horizons for personal development and professional activities, increases the options for leisure and holidays, and allows better contact and understanding between people. The economic development of entire regions depends on the easy access to people and goods ensured by contemporary transport technology. Owing to its flexibility, road transport is a major transport mode, and cars are objects of desire and pride in many societies.

Unfortunately, these positive aspects are closely associated with the hazards to the environment and human health caused by transport, particularly road transport (Dora & Phillips, 2000). One of the leading concerns is the adverse effect on health of air pollution emitted by transport. Research in recent decades consistently indicates that outdoor air pollution harms health, and the evidence points to air pollution that stems from transport as an important contributor. The present trend towards increasing transport volume, and the associated risk of harm to air quality and health, threaten the policy objective of many countries, also stated by the European Union (EU) in its 6th Environment Action Programme: to achieve pollution levels that do not give rise to harmful effects on human health and the environment (European Commission, 2001).

A multitude of air contaminants of varying toxicity comes from road transport. These contaminants originate from the tailpipes of vehicles with internal combustion engines, from other vehicle components (such as brake and clutch linings and pads, tyres and fuel tanks), and from road-surface wear and treatment materials. Road traffic can be labelled the most important source for some pollutants of great concern, such as nitrogen oxides, benzene and carbon monoxide. Until recently, leaded petrol was an important contributor to exposing the population to lead. Recently, emissions of particulate matter (PM) have attracted much attention, owing mainly to epidemiological findings that suggest that it is a major risk to human health. Besides the pollution sources already mentioned, PM is also formed in the atmosphere, as a secondary pollutant from gases such as nitrogen oxides, sulfur dioxide and volatile organic compounds (VOCs). Atmospheric reactions that involve nitrogen oxides and VOCs lead to the formation of tropospheric ozone, a well-known air pollutant.

The mixture of air pollution varies in time and space, depending on several characteristics, such as proximity to roads, the composition of the vehicle fleet, traffic patterns and the presence of other pollution sources. The pattern of population exposure depends on both pollution levels and population activities. Both the short-term pattern and long-term average of exposure, along with individual susceptibility, lead to adverse effects on health, which may occur either immediately or years later.

Understanding the complex chain of events – from transport demand and traffic activities to emissions, ambient air quality, exposure and effects – requires information from a variety of scientific disciplines, often involving research on complex relationships. Decision-makers and risk managers often ask: what is the significance of the various components of the pollution emitted by transport that produce adverse health effects? Identifying such components would help risk managers to focus their efforts and enable a more forceful reduction of adverse effects on health. The elimination of lead from petrol is an example of this approach; it has resulted in a substantial reduction in exposure to lead and its harmful effects on the neurobehavioural development of children.

Reducing risk also requires knowledge of the significance of short-term exposure to high levels of pollution (which is often experienced in dense traffic), in contrast to the risks from long-term exposure to low levels (which may be experienced by large populations). Various risk-reduction measures may have both positive and negative effects; for example, reducing carbon dioxide emissions by increasing the proportion of diesel-powered cars may lead to increased PM emissions. Scientific evidence should therefore play an increasingly important role in making decisions on transport development and in evaluating its benefits and costs to society. As postulated by the Transport, Health and the Environment Pan-European Programme (THE PEP) (UNECE & WHO Regional Office for Europe, 2002), the integration of environmental and health aspects into policies and decisions on transport should be one of the principles of relevant decision-making.

Preliminary assessments indicate that diseases related to the air pollution caused by road transport affect tens of thousands of people in the WHO European Region each year (Künzli et al., 2000). The effects range from short-term aggravation of respiratory symptoms to a reduction in life expectancy by a year or more. While these assessments clearly indicate the need to substantiate intensive action to reduce transport-related air pollution, a better understanding of the role of exposure to various pollutants in producing adverse effects on health and of the determinants of this exposure may improve the effectiveness of further action. An example of policy demand for such action is the recently published resolution of the European Parliament that explicitly calls for a better link to be established in the European environment and health strategy "between the traffic, transport and air pollution, on the one hand, and asthma and respiratory diseases on the other …" (European Parliament, 2004).

This book provides a systematic review of the literature and a comprehensive evaluation of the health hazards of transport-related air pollution. This review focuses on pollution related to road transport (mostly urban and suburban, and passenger and freight transport) and the risks it poses to human health. It omits or briefly mentions other transport modes – such as rail, water and air transport – that may also contribute significant emissions of air pollutants. Further, the review does not cover other aspects of traffic relevant to health – such as noise pollution, traffic accidents, socioeconomic issues and the effects of congestion and climate change – even though they may be linked significantly with air pollution.

This review considers and addresses two topics:

- the entire chain of relevant issues, from patterns and trends of activities that determine emissions (such as the demand for freight and passenger transport) to the determinants of the intensity of pollution emissions from transport (such as fuel quality and additives, engine and aftertreatment technologies and transport patterns); and
- primary emissions from transport, the formation of secondary pollutants, and patterns of human exposure.

In discussing the adverse effects on health of exposure, the review considers the results of both epidemiological studies and toxicological assessments of biological mechanisms. It profited from a parallel WHO project for the systematic review of health aspects of air pollution (WHO Regional Office for Europe, 2004) in support of the European Commission (EC) programme Clean Air for Europe (CAFE). The WHO project covered the main air pollutants – PM, nitrogen dioxide and ground-level ozone – from all sources and helped to ensure that comprehensive literature was available to the present review.

Based on accumulated evidence, this review identifies key facts emerging from the available evidence, suggesting the action necessary to reduce the health risks created by road traffic. The elaboration of specific action plans, however, is beyond its scope.

This book identifies the strengths and weaknesses of the evidence; these characteristics are highly relevant to both quantifying the effects of traffic-generated pollution on the population and evaluating the possible benefits of particular interventions. The former, however, is also beyond the scope of this book. Such quantification must be made for a specific purpose, must be relevant to a specific population and requires the selection of appropriate concentration–response functions and data on exposure (WHO Regional Office for Europe, 2000, 2001).

This review points out the complexity of the causal chain and the limitations in the available knowledge of the links between transport emissions, population

exposures and adverse effects on health. The authors and other contributors gave a good deal of attention to seeking an indicator of the mixed composition of air pollution related to transport, which could be valuable at various stages of the causal chain and in health impact assessments. They agreed, however, that an indicator that adequately covers all relevant aspects has yet to be identified.

How to read this report and how it was written

This book is aimed at two main audiences: policy-makers and experts in the field of transport-related air pollution and public health. Accordingly, it provides an executive summary, plus summary information (called key points) at the start of each chapter, for policy-makers, and a full discussion, primarily for experts. In addition, a separate summary for policy-makers is also available (Krzyzanowski, 2005).

The WHO Regional Office for Europe initiated the preparation of this report early in 2002, through discussions of its scope and outline with potential contributors and external advisers. The outline was presented for comment to the members of AIRNET (Thematic Network on Air Pollution and Health) (IRAS, 2004), which is funded by the EC. After these discussions, WHO recruited the main contributors to the first draft of the review and identified a wider group of reviewers. The text was drafted and reviewed three times in 2003/2004. The editors carefully considered reviewers' comments in finalizing the report. The discussions and reviews aimed to make complete use of the existing evidence, based on peer-reviewed published material, and to reach a consensus on the interpretation and synthesis of the evidence.

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1. Factors determining emissions in the WHO European Region

Alois Krasenbrink, Giorgio Martini, Urban Wass, Edward Jobson, Jens Borken, Reinhard Kuehne, Leonidas Ntziachristos, Zissis Samaras and Menno Keuken

Key points

Facts

Collectively, internal combustion engines and conventional fuels are the dominant contributor to transport-related air pollution. To counter this, current regulations (such as the EU emission standards called Euro 0 through Euro IV) and future legislation (such as Euro V and Euro VI) will further reduce tailpipe emissions of regulated pollutants.

Compared with the very high volumes of transport in western Europe, the volumes in central Europe are much lower – currently, a third of passenger transport and a tenth of freight transport. Central European levels, however, were expected to increase soon after the enlargement of the EU. In the 12 countries of eastern Europe, the Caucasus and central Asia (EECCA), long-distance public and freight transport broke down between 1990 and 1998.

In the 15 countries belonging to the EU before May 2004, passenger cars cover 80% of their mileage on urban and suburban roads, while lorries cover about 80% of their mileage on suburban roads and motorways. To meet growing demands, the motorway network in the EU expanded by 2.7% annually in the 1990s. Though urban and suburban road extension has been marginal, traffic is generally increasing, leading to higher traffic density and congestion in cities. Volumes of urban public transport, however, have stagnated as a result of urban development. Although motorcycles and mopeds have the potential to increase traffic volume flow in cities, they also have high emissions of hydrocarbons, carbon monoxide and PM.

In urban areas, the large number of short trips in congested traffic and vehicles operating under cold-start conditions have offset the decrease in vehicular emissions. Many urban trips by private cars cover distances of less than 6 km. This leads to very high average emissions per distance driven, owing to the ineffectiveness of catalytic converters in the initial minutes of engine work. About 90% of gaseous pollutants are emitted within the first 200 seconds after initial ignition, when the catalytic converter has yet to reach its full operating temperature. The absolute emission values are higher at lower ambient temperatures. Also, due to poor

maintenance, lack of exhaust aftertreatment systems or both, a relatively small number of vehicles is responsible for a major share of the emissions.

Trends

Over the past 15 years, a number of trends have become apparent. In the next few decades, road transport will continue to grow in the 15 countries belonging to the EU before May 2004. The eastern half of the WHO European Region seems to be following the transport pattern of western Europe: more private cars and more goods transported by lorries.

For at least the next decade or two, conventional diesel and petrol engines will be the dominant technology. The market share for diesel-powered vehicles will increase further, and gram emissions per vehicle kilometre driven will decrease. Alternative vehicle technologies – fuel cells, electric vehicles, and hybrid vehicles – are unlikely to have a significant presence in the market before 2015.

A number of promising technologies are candidates for lowering vehicle emissions, including particle traps, a system to reduce nitrogen oxide emissions, preheated catalytic converters and electronic vehicle controls. For further emission reductions, new engine and aftertreatment technologies may require fuels that are free of metals and have zero sulfur content and a low content of polycyclic aromatic hydrocarbons (PAHs).

By 2020, in the EU, 20% of conventional fuels should be replaced by such substitutes as biofuels, natural gas and hydrogen. The main driving force for this initiative is the policy on climate change.

Conclusions

In urban areas, congestion and the large number of short trips under cold-start conditions have offset the decrease in emissions per vehicle. In coming decades, road transport is likely to remain a significant contributor to air pollution in cities.

Introduction

In western Europe, the transport of people and freight has dominated road traffic for many decades. The tremendous increase in the volume of people and goods transported during the last 100 years would not have been possible without the development of transportation technologies. For this analysis, the vehicles participating in road traffic are classified as passenger cars, lorries (including vans) and others (buses, motorcycles and mopeds). Although rail and public transport dominated the transport system in central Europe in the early 1990s, road traffic is now increasing rapidly (EEA, 2002, 2003a). For example, between 1990 and 1999, the total motorway length almost doubled in the 10 new EU Member States while increasing by almost a third in the 15 countries belonging to the EU before May 2004.

Emissions from road traffic, from both combustion and friction processes, result in a complex mixture of air pollution, which is known to have adverse effects on health (Hoek et al., 2002). The pollutants of greatest concern at present, because of their impact on human health, are PM, ground-level ozone and nitrogen dioxide. The transport sector is an important contributor of all three.

Air pollution by particles is characterized by the particle size, mass concentration, number concentration and chemical composition. Among other things, the chemical composition pertains to the soot content (expressed either as black smoke or as elemental carbon and organic carbon), the concentration of PAHs (such as benzo[a]pyrene), and the concentration of heavy metals (such as cadmium, chromium, copper and zinc). Lead is still a problem in some EECCA countries because of its continued use in transport fuels.

This chapter gives a brief overview of the transport patterns, fuels and propulsion technologies that dominate the market today; assesses their further development; addresses some emerging technologies that may become important in the future; and discusses the anticipated impact of future fuels, engines and exhaust aftertreatment technologies on toxic emissions from new vehicles.

Transport patterns

Transport, which is the physical movement of people and goods from one place to another, is determined by many interacting factors: economic activity, prices and disposable income, regulations, organizational structures, and spatial patterns – all based on the particular sociocultural background. These different factors vary greatly in the WHO European Region and continue to undergo dynamic changes. A broad classification of the Region into three geographical areas is necessary to accommodate the major differences in total and per person transport volumes, modal shares, patterns of use, vehicle technology, and specific and total emissions:

1. western Europe, including Austria, Belgium, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Luxembourg, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland and the United Kingdom;
2. central Europe, including Bosnia and Herzegovina, Bulgaria, the Czech Republic, Croatia, Hungary, Poland, Romania, Serbia and Montenegro, Slovakia, Slovenia and The former Yugoslav Republic of Macedonia; and
3. EECCA: Armenia, Azerbaijan, Belarus, Georgia, Kazakhstan, Kyrgyzstan, the Republic of Moldova, the Russian Federation, Tajikistan, Turkmenistan, Ukraine and Uzbekistan) and the Baltic states (Estonia, Latvia and Lithuania).

Data were not available on the eight remaining countries in the Region (Albania, Andorra, Cyprus, Israel, Luxembourg, Malta, Monaco and San Marino), but they have minimal impact on the trends discussed below.

The sections that follow provide figures on the total transport volume in these regions. This puts them in perspective with respect to both transport's share of the total emissions of air pollutants and the relative strength and modes of emissions in the regions. Figures on transport intensity and modal shares, per person and per gross national product (GNP), are also provided; they indicate the different

levels for the different regions. They also indicate potential future developments in central Europe, EECCA and the Baltic states as they approach western Europe's levels, which appears to be the path they are taking.

Western Europe

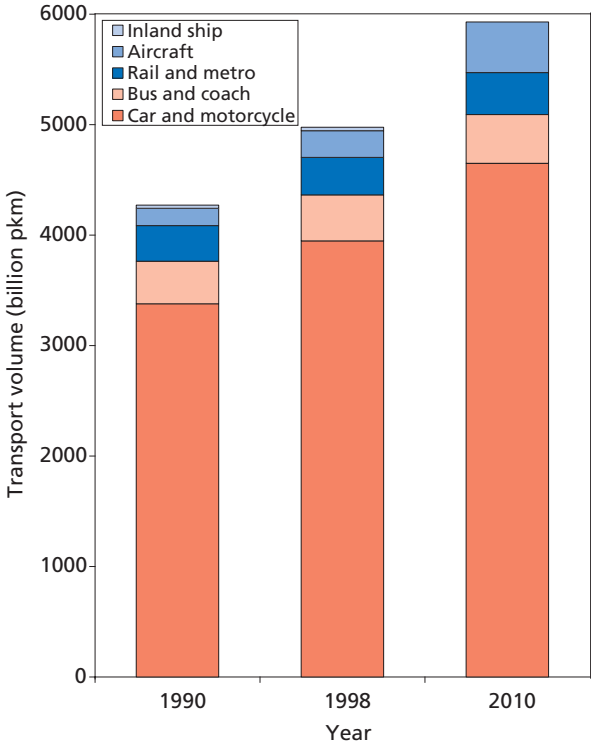
Passenger travel in western Europe has increased continuously in annual mileage and has shifted travel volumes to individual motorized vehicles. While both real income and disposable income have increased, both fuel prices and the purchase price of cars have decreased. Car ownership has risen continuously, with car trips increasingly replacing trips on public transport, by bicycle and on foot. Increasing distances per trip reflect greater distances between, for example, home, work, school, shopping and leisure locations – that is, lower settlement densities – and a deliberate choice of more distant options, which in turn have become accessible by faster vehicles. This is particularly true for leisure and holiday travel, with a move towards more frequent, though shorter, trips and more distant destinations, which are accessible by airplane (EEA 2003c; Eurostat, 2001).

Between 1970 and 2000, the total volume of passenger transport in western Europe more than doubled, to about 5000 billion passenger-kilometres (pkm) (Fig. 1.1). This growth was driven mainly by a 140% increase in passenger car travel, with an additional 630% increase in air travel. Cars now have about an 80% share of the total transport volume (EC, 2002). The share of air travel is now 6%, slightly less than rail, whose share dropped from 10% to 7% (Eurostat, 2001). On average, each citizen travels about 13 800 km annually, with levels in Greece, Portugal and Spain at around 10 500 km and in Denmark, France and the Netherlands at above 15 000 km (Eurostat, 2001). By 2010, total passenger travel volume is expected to grow by an additional 24%, and gross domestic product (GDP), 43% (EC, 2001).

In general, freight transport strongly depends on economic activity and production infrastructure. Western Europe has seen a significant extension of trade relations; among other contributions, this is due to the increasing economic integration of the EU and the movement to market economies farther east (EC, 2002; EEA, 2003d), a diversification of production processes and sites, and a continued trend away from heavy industry and towards refinement industries and services. Trends indicate more frequent and more distant consignments of freight. The significant growth of container transport, of just-in-time logistics and express delivery is enigmatic (EEA 2003e), however. These developments in both production and goods have been propagated to a large extent by lorry transport; as lorry transport has proven to be flexible and ubiquitously available at moderate costs, many of these production structures have come to depend increasingly on it (Borken et al., 2004).

As a consequence, the strong growth in land-based freight transport volume is due entirely to the increase in road freight volume; this is matched by a similar

Fig. 1.1. Volume of passenger transport in the 15 countries belonging to the EU before May 2004, in 1990, 1998 and 2010 (projection)



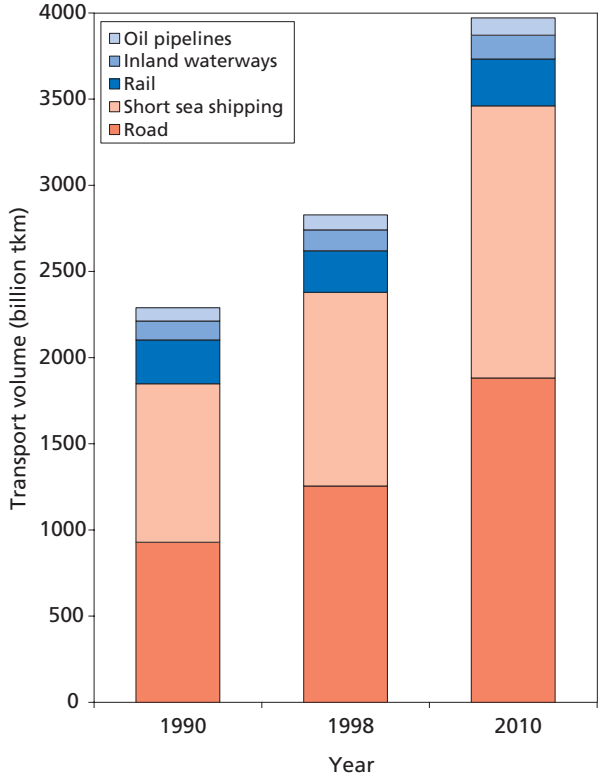
Note. The share of motorcycle transport is over 4%; that of light rail is about 17%. No data are projected for motorcycle and inland shipping in 2010.

Sources: Eurostat (2001) and EC (2001, 2002).

increase in short sea shipping. Rail and inland ships transport predominantly bulk materials (minerals, building material, coal, ore, scrap metal and petroleum products), and their transport volumes have stagnated in western Europe (EEA, 2003e Eurostat, 2001).

Overall, freight transport volumes have more than doubled since 1970, to about 3000 billion tonne-kilometres (tkm), with road and short sea shipping having shares of 44% and 41%, respectively, in 2000 (Fig. 1.2). Transport volume per person has similarly increased, to 7400 tkm per person. Short sea shipping is the backbone of external trade in the 15 countries belonging to the EU before May 2004: ore and other raw materials, as well as crude oil and petroleum products, are imported by ship, while high-value products are exported. Thus maritime shipping accounts for 70% (or 1.3 billion tonnes) of all tonnage traded with other countries and for 40% (or almost €2000 billion) of the value of external trade.

Fig. 1.2. Freight transport in the 15 countries belonging to the EU before May 2004, in 1990, 1998 and 2010 (projection)



Note. 1998 data on short sea shipping came from estimated 1997 data.
Sources: Eurostat (2001) and EC (2001, 2002).

Trade within the 15 countries belonging to the EU before May 2004 moves mostly by road (44% of tonnage traded), followed by short-sea and inland shipping (28% and 12%, respectively) (EC, 2002).

The intensity of freight transport increased slightly, to 466 tkm per €1000 GDP in 1998. Within the EU, it was greatest in the Netherlands (700 tkm per €1000 GDP), signifying the high share of low-value bulk goods, and least in Ireland (320 tkm per €1000 GDP) (Eurostat, 2001). In the past few years, transport volume has grown more than GDP; past freight transport trends, notably increased long-distance road transport, are expected to continue until 2010 (EEA, 2003d).

Central Europe

Since the 1990s, central Europe has been marked by a transition to market economies, by an increased freedom of movement, an increased number of trips and

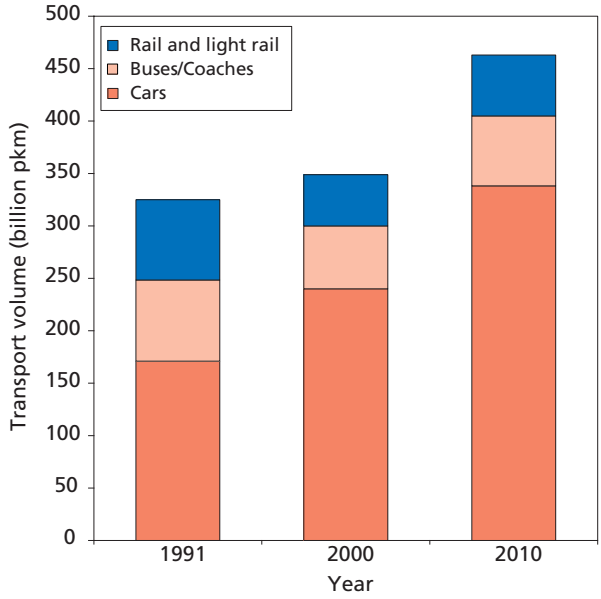
means of transport (with formerly state-owned transport companies undergoing major changes) and, in some cases, rapid technical change (Pucher, 1999; Berger, 2002). Differences between the countries in this group, however, are large and more pronounced than in western Europe.

Before December 1991, passenger transport for the general population in central Europe meant public transport by rail and bus (Pucher, 1999; TACIS Technical Dissemination Project, 1996). This declined drastically in the 1990s – for example, rail declined by more than 40% (EEA, 2003b) and urban transport volumes declined even more (Pucher, 1999). In contrast, private car travel grew. Best documented is the rise in car ownership, roughly correlated with GDP growth. Levels range from 430 cars per 1000 population in Slovenia to 300 in the Czech Republic, respectively, compared with 550 cars per 1000 in western Europe (EEA, 2003a). Car ownership rates appear to have peaked – contrary to those in western Europe, but from a much lower level – in the capital cities: such economic centres as Budapest, Prague and Warsaw (Pucher, 1999). Actual car use, however, also depends on fuel costs and disposable income, and therefore is not necessarily proportional to ownership (TACIS Technical Dissemination Project, 1996; Cherp et al., 2003).

The statistics available are not reliable, but they suggest that the decline in public transport volume has levelled off and that passenger car travel is now strongly driving the growth in volume. For the year 2000, the estimated total travel volume (Prognos, 2002) for the Czech Republic, Hungary and Poland – the largest economies in central Europe – was about 375 billion pkm (Fig. 1.3), with about a 70% share for car travel. A further one-third increase by 2010 has been estimated (Prognos, 2002). Per person, this translates to about 5000 km annually (EC, 2002; EEA, 2003e), which is about a third of the level in western Europe. There are, however, large discrepancies in passenger transport between central European countries, for reasons including armed conflict.

In the past, freight transport in central Europe was planned centrally, with greater emphasis on heavy industry and agriculture than on consumer products. A particularly high share for rail characterized freight transport. With declining economic activity in the 1990s, the total freight transport volume decreased by about 20–25%, in comparison with 1990 levels in the Czech Republic, Hungary and Poland (Fig. 1.4) (European Conference of Ministers of Transport, 2002a). It has now stabilized in these countries at around 300 billion tkm, which is about a tenth of the current transport volume in the 15 countries belonging to the EU before May 2004 (EEA, 2003c). The growth was entirely driven by road transport, which doubled its volume to its current 50% share of freight transport. Also, Turkey's estimate freight transport volume in 1998 was about 150 million tkm, of which road transport comprised 90% (European Conference of Ministers of Transport, 2002a). The reader should note, however, that the data used for Fig. 1.4 are inconsistent with some other data. (The Prognos (2002) data are inconsistent

Fig. 1.3. Total passenger transport volume (excluding aviation) in the Czech Republic, Hungary and Poland, 1991, 2000 and 2010 (projected)



Note. Data for the Czech Republic in 1991 are estimated to be two thirds of the value for Czechoslovakia.

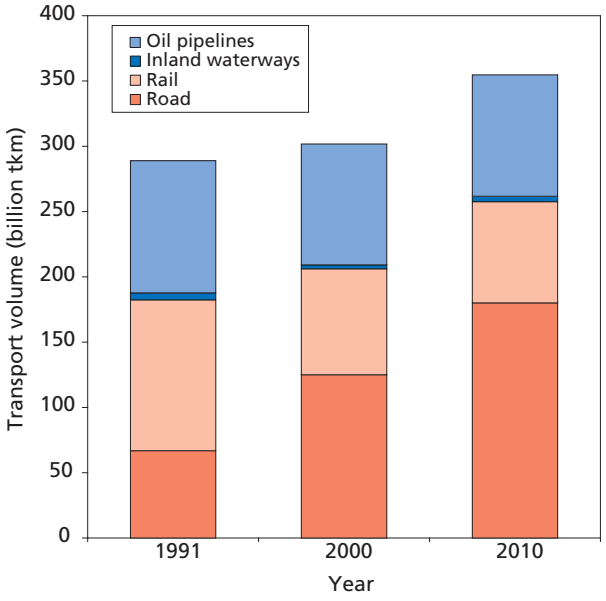
Source: Prognos (2002).

with those of European Conference of Ministers of Transport (2002a), which in turn are inconsistent with other data from the European Conference of Ministers of Transport (2002b) and those of the EC (2002). Prognos appears to have underestimated public/mass transport volumes in 1991, and to have overestimated the volume in 2000.)

Rail transport and rail companies' severe financial, technical and institutional problems in trying to maintain their service contribute to the growth in road transport of freight (Berger, 2002); rail's share of freight transport dropped from 70–80% in the early 1990s to about 40% recently (European Conference of Ministers of Transport, 2002a). Also, pipelines are twice as important in central Europe as in western Europe, with about a 10% share of freight transport. The average intensity of freight transport in central Europe reveals significant differences among countries' economies, spanning a factor of five from Slovenia to Slovakia with, respectively, about 250 tkm per US$ 1000 GDP and about 1250 tkm per US$ 1000 GDP (EEA, 2003d).

Central Europe's trade relations have now shifted towards western Europe, and its industries seem to have restructured, stabilized and recovered (Berger, 2002),

Fig. 1.4. Total freight transport volume (excluding short sea shipping) in the Czech Republic, Hungary and Poland, 1991, 2000 and 2010 (projected)



Note. Data for the Czech Republic in 1991 are estimated to be two thirds of the value for Czechoslovakia. Data for pipelines are from European Conference of Ministers of Transport (2002a); the value for 2010 equals the value for 2000 (as an indication).

Source: Prognos (2002).

from a relatively low level. GDP is estimated to show 4.4% real annual growth by 2010, and a strong growth in passenger and freight transport is expected – driven mostly by road transport (Prognos 2002; Zachariadis & Kouvaritakis, 2003). In joining the EU, the 10 new Member States are adopting its extensive environmental legislation, which limits specific vehicular emissions and defines fuel characteristics. Hence, improved vehicles and an upgraded transport infrastructure will drive the expected strong growth in transport volume. The overall pollutant emissions from transport might rise, as Zachariadis & Kouvaritakis (2003) indicate.

EECCA and the Baltic states

Before 1991, passenger transport in EECCA was geared even more towards highly subsidized public transport than it was in central Europe, and travel by private car was a limited and expensive privilege. Consequently, the deep and lasting economic recession since the 1990s and the general decline in public transport

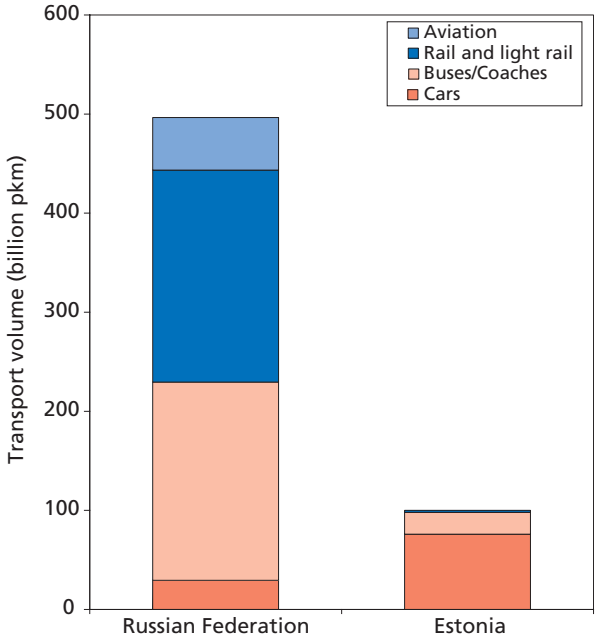
services have led to a dramatic drop in movement using rail, the previous backbone of long-distance travel. Passenger travel fell, indicatively, by 40–50% in the Russian Federation and Ukraine, 50–70% in Azerbaijan, Kazakhstan and Uzbekistan, and up to 80% in the Baltic states (TACIS Technical Dissemination Project, 1996; EEA, 2003a; World Bank Infrastructures and Energy Services Department Europe and Central Asia Region, 2002). Public urban transport, which had been an essential service while having a bad reputation, surged; its recovery is now hindered by the greater number and wealthier share of previous passengers' travelling by car. Increased road traffic has led to both congestion and urban sprawl; public transport is having difficulty in coping with this situation, and its service quality and hence attractiveness have deteriorated further (World Bank Infrastructures and Energy Services Department Europe and Central Asia Region, 2002).

Levels of car ownership have increased, but are still moderate, even when compared with those in central Europe. In 1999, car ownership ranged from about 300–330 cars per 1000 population in Estonia and Latvia to 150 cars and fewer per 1000 in central Asia (EEA, 2003a). Car ownership rates appear to peak in major cities: economic centres such as Riga in Latvia and Moscow and St Petersburg in the Russian Federation (TACIS Technical Dissemination Project, 1996). Actual car use, however, does not seem to show a similar extension, because disposable incomes are small; this is the case even though transport fuels (of which diesel is most important) cost about US$ 0.25–0.35 per litre (for diesel), only a few cents above the estimated production costs in December 2002 (TACIS Technical Dissemination Project, 1996; Cherp et al., 2003; Metschies, 2003).

Transport statistics are sketchy and inconsistent. No time series was available for passenger transport EECCA and the Baltic states. For the Russian Federation, 20 billion pkm was estimated for cars (Fig. 1.5), assuming on average 1000 pkm per car and 135 cars per 1000 inhabitants. No data on aviation were available for many countries. For 1998, a total passenger travel volume of around 500 billion pkm is estimated for the Russian Federation, which translates into 3500 pkm per person, of which urban transport comprises 50% (EC, 2002). In Estonia, however, cars are already estimated to account for 75% of the passenger travel volume, which is a typical western European level.

The drop in economic activity has been more pronounced and longer in EECCA than in central Europe. Compared with 1990 levels, freight transport volume fell drastically in EECCA, to only 40% between 1996 and 1998; it has recently recovered (European Conference of Ministers of Transport, 2002a, 2002b). According to the statistical data (Fig. 1.6), rail transport has decreased to less than half its previous volume, but still has about an 85% share of ground transport (European Conference of Ministers of Transport, 2002a). In the Baltic states, whose ports are busy with transit between western Europe and the Russian Federation, road freight transport is expanding – though rail still has 50%, 64% and 75% shares in Lithuania, Estonia and Latvia, respectively. The Russian

Fig. 1.5. Total estimated passenger transport volume by mode in the Russian Federation and Estonia, 1998



Note. The transport volume of Estonia is multiplied by a factor of 10 so that it can be shown alongside that of the Russian Federation.

Sources: EC (2002), Prognos (2002) and European Conference of Ministers of Transport (2002b).

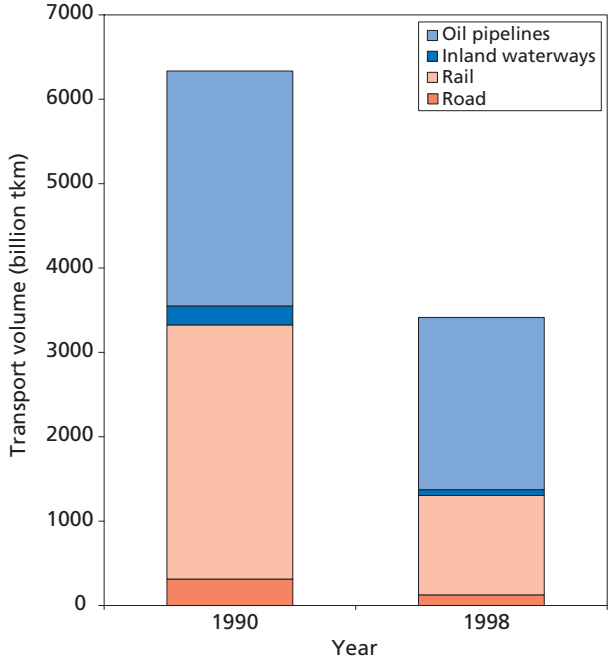
Federation accounts for 90% of the freight transport volume in both the Federation and Ukraine. In 1998, total freight transport volume in EECCA was about 1400 billion tkm; this was about five times the total volume in central Europe and almost as much as the freight transport volume in the 15 countries belonging to the EU before May 2004, in each case excluding pipelines and short sea shipping (EC, 2002; European Conference of Ministers of Transport, 2002a).

Pipelines are not discussed here in detail because of their low share in transport volume (5% in western Europe and 10% in central Europe) and negligible emissions of pollutants. In 1998, however, their transport volume of about 2000 billion tkm in the Russian Federation and Ukraine exceeded that of all surface transport modes together by a third and had dropped only to 75% of 1990 levels (European Conference of Ministers of Transport, 2002a).

Road transport

Transport is the second largest energy consumer in the Region, accounting for 30% in western Europe and 22% in central Europe in 1999 (EEA, 2003a). An

Fig. 1.6. Total freight transport volume by mode (excluding short sea shipping) in the Russian Federation and Ukraine, 1990 and 1998



Note. Transport by pipelines and inland waterways in Ukraine for 1998 is scaled according to development in the Russian Federation.

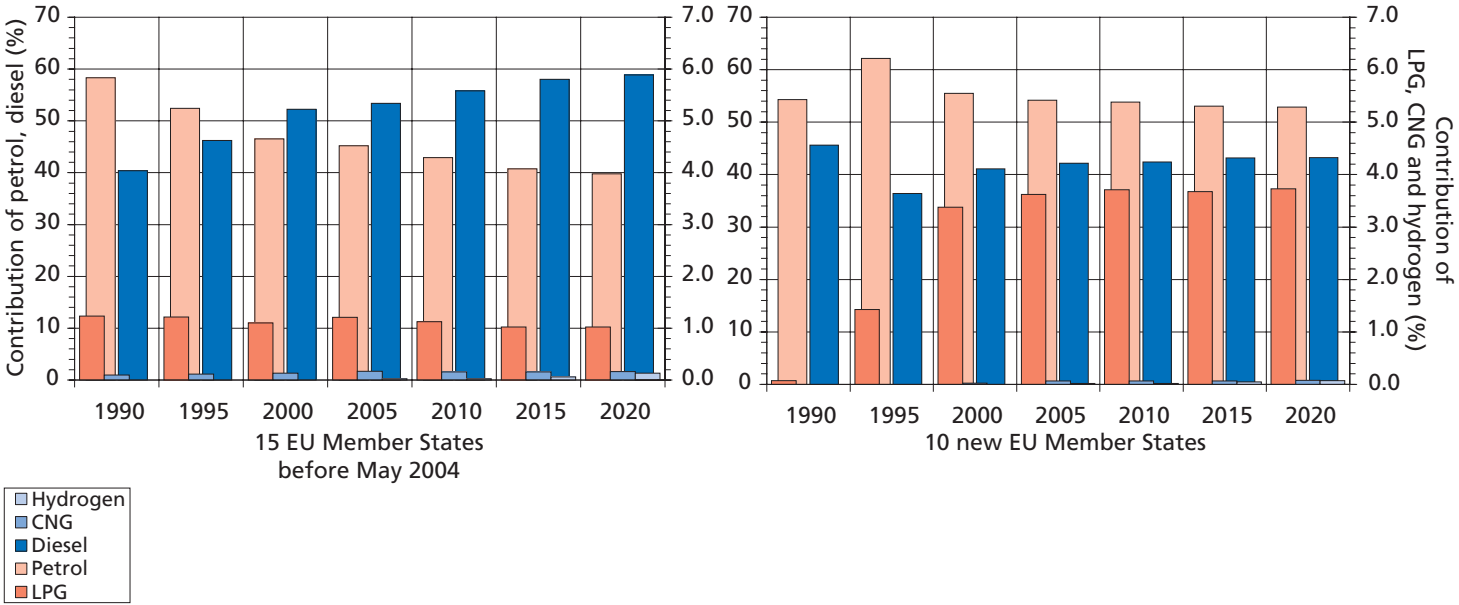
Sources: EC (2002), Prognos (2002) and European Conference of Ministers of Transport (2002b).

annual growth of about 2% and 3% in western and central Europe, respectively, was recorded for transport energy consumption in the decade 1990–1999.

The baseline scenario of CAFE (2002) has recently provided data on the evolution of fuel use in road transport. Fig. 1.7 provides fuel-use data for the EU. In the period 1990–2020, an annual growth of 1.33% and 2.23% in road transport energy demand is predicted for the 15 countries belonging to the EU before May 2004 and the 10 new EU Member States, respectively. In the former, diesel's share is expected to increase from 40% to 59% at the expense of petrol's share. No particular trend is expected in the 10 new EU Member States. Alternative fuels (compressed natural gas and hydrogen, collectively) show modest penetration rates and are not expected to exceed 0.3% of the total road energy consumption in 2020 (Fig. 1.8). The share of alternative fuels appears negligible in the 10 new EU Member States.

This scenario clearly demonstrates that internal combustion engines using conventional fuels will continue to be the main propulsion system in road

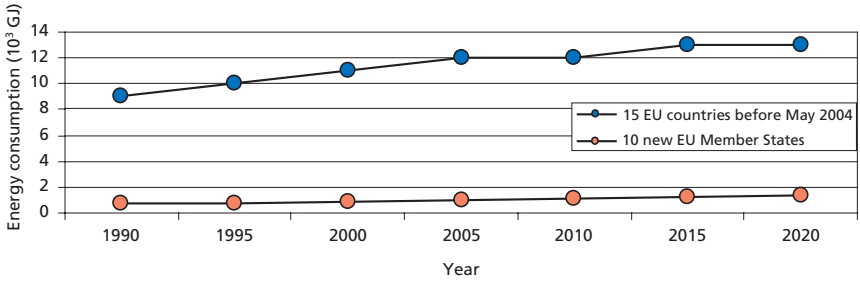
Fig. 1.7. Fuel use in the road transport sector in the EU, 1990 to 2020 (projected)



Note. LPG = liquified petroleum gas; CNG = compressed natural gas.

Source: CAFE (2002).

Fig. 1.8. Total energy consumption in the EU, 1990 to 2020 (projected)

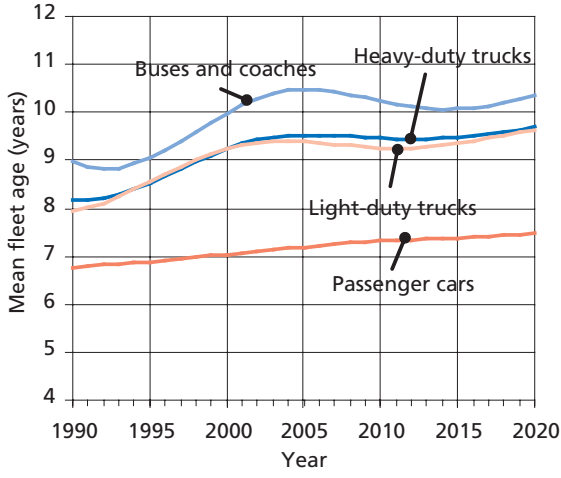


Source: CAFE (2002).

transportation. The 15 countries belonging to the EU before May 2004, however, are likely to shift significantly towards more efficient diesel engines, but not towards alternative fuels, whose small fraction mainly reflects their use only in dedicated applications, such as urban fleets and company cars. As to individual fleets, the shift from petrol-burning to diesel-burning engines in passenger cars is consistent with the fuel-use mix predicted by the CAFE programme (see Fig. 1.7).

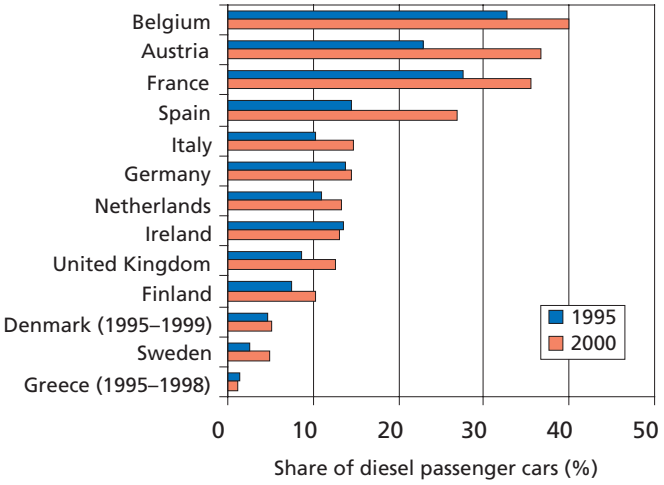
Fleet ageing (Fig. 1.9) may have a significant effect on mean emission levels. Fig. 1.10 shows historical data (Eurostat, 2002) on the share of diesel cars in the total passenger car fleet for most of the 15 countries belonging to the EU before

Fig. 1.9. Mean age of on-road vehicles in the EU according to vehicle category, 1990 to 2020 (projected)



Note. No data are available for Luxembourg and Poland.
Source: Eurostat (2002).

Fig. 1.10. Shift of passenger cars to diesel engines, 1995–2000



Source: Transport and Environment Database System Project (2003).

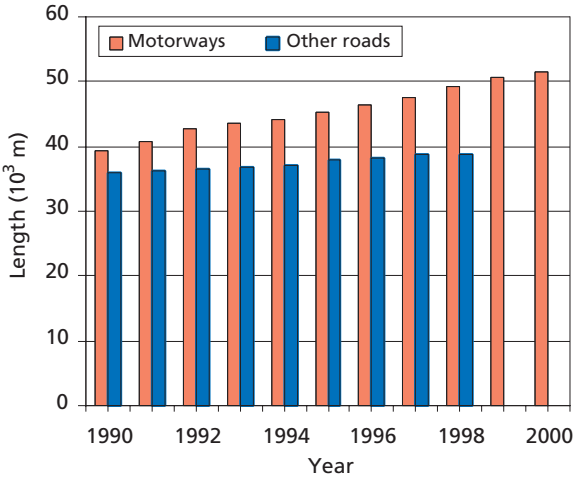
May 2004. In several, almost half of passenger cars use diesel, and diesel cars are increasingly penetrating the market in all but a few countries. Despite the introduction of new vehicles in the fleet, the mean age seems to be increasing over time. Models predict a moderate but constant increase in the age of passenger cars (from 6.7 years in 1990 to 7.4 years in 2020) and a more significant increase for commercial vehicles in 1995–2005. The mean age of such vehicles, however, seems to stabilize at about 10 years.

The European Environment Agency (EEA, 2003a) reported that, in the decade 1990–1999, the motorway network expanded by an annual rate of about 2.7% in the 15 countries belonging to the EU before May 2004 (Fig. 1.11), and by 7.2% in the 10 new EU Member States. Expansion in the latter is expected to continue as a function of improving economies and the need to expand the road network westward. The extension of the urban and suburban network is only marginal in both cases.

Fig. 1.12 shows the proportions on passenger and freight volumes on highways and rural and urban roads. Urban and rural driving make up more than 80% of total passenger transport, while highways are more significant for freight transport. No significant shift in modal shares is predicted in the coming years.

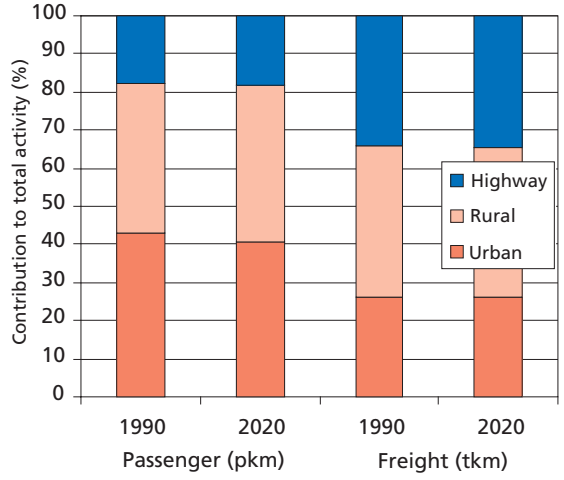
Road transport varies not only in spatial distribution but in temporal patterns. Fig. 1.13 presents traffic density as a function of time of day for a number of European cities. Traffic is much heavier in the daytime and peaks in most of the cities around the opening and closing times for offices. This affects travelling conditions and, consequently, as will be shown, the values for emissions. André & Hammarström (2000) found that, in most European cities, cars travel at a mean

Fig. 1.11. Length of motorways and other roads in the 15 countries belonging to the EU before May 2004, 1990–2000



Source: Eurostat (2002).

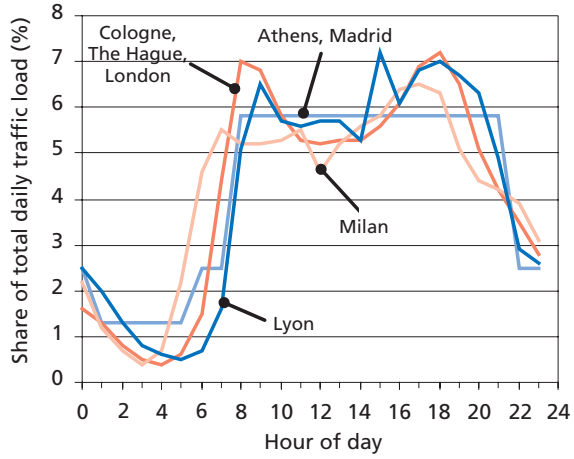
Fig. 1.12. Shares of passenger and freight activity share on different types of road, 1990 and 2020 (projected)



Source: Transport and Environment Database System Project (2003).

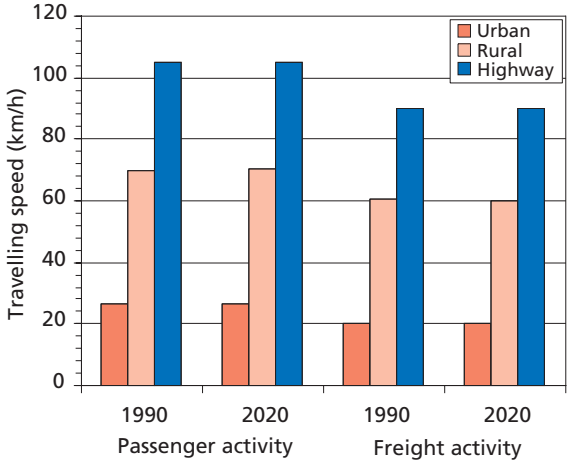
speed of 18–29 km/h with a range of ±7 km/h, depending on the hour and the city. In congested traffic, speeds are as low as 5 km/h. Fig. 1.14 shows weighted mean speeds for all the countries belonging to the EU before May 2004, predicted by the Transport and Environment Database System Project (2003). No particular change in travelling speeds is predicted for the coming years.

Fig. 1.13. Typical hourly profiles of traffic density for some European cities, 1990



Source: EC (1996).

Fig. 1.14. Mean travelling speeds for passenger and freight activity in the 15 countries belonging to the EU before May 2004, 1990 and 2020 (projected)



Source: Transport and Environment Database System (2002).

The use of motorcycles, scooters and mopeds is a special topic. For climatic reasons, these two-wheelers are very popular in southern European cities, where they also contribute to an improvement of the overall traffic flow. As to emissions, however, a small two-stroke scooter can have very much higher (up to three orders of magnitude) specific emissions of hydrocarbons and high levels of PM,

consisting mainly of VOCs, with only a small fraction of solid particles. The introduction of several measures – for example, the use of aftertreatment systems and new direct injection technologies – has reduced the emissions from two-stroke mopeds. Also, the right choice of lubrication oil can significantly reduce PM emissions.

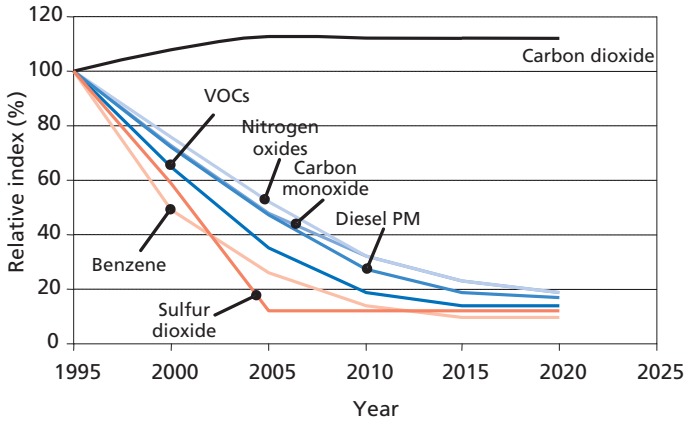
Road transport emissions in the EU

To assess future trends in emissions and air quality and the cost–effectiveness of different policy measures, Auto-Oil II (1999) established a baseline scenario for the evolution of emissions from road transport in the 15 countries belonging to the EU before May 2004 (Fig. 1.15). This forecast showed that, despite the increasing demand for transport, conventional pollutant emissions should decrease in the coming years as a result of stringent EU regulations for vehicle emissions and fuel quality. Carbon dioxide emissions, however, will increase modestly, despite the voluntary commitment of the European automotive industry to achieve an average 140 g/km carbon dioxide for the fleet of new passenger cars sold in the EU in 2008 (ACEA, 1998). This picture is well established today among policy-makers, the industry and researchers in the field. The following sections provide an insight into how road transport sources contribute to total emissions.

Emission patterns

Emissions from road transport are distinguished by four main components:

Fig. 1.15. Baseline scenario for evolution of emissions from road transport in the 15 countries belonging to the EU before May 2004



Note. The reference year is 1995 (1995 = 100%).
Source: Auto-Oil II (1999).

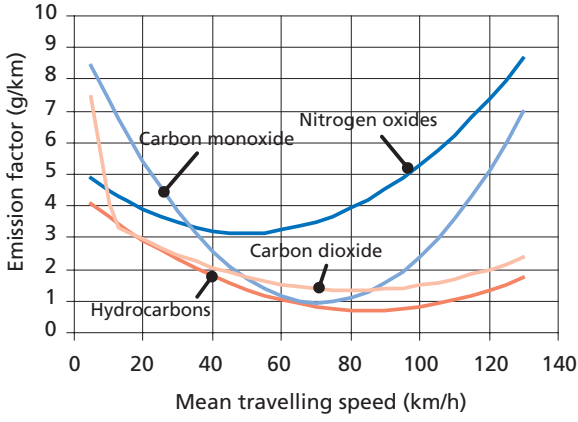
- exhaust emissions under thermally stabilized engine operation (hot emissions);
- exhaust emissions during transient operation after engine start-up (cold-start emissions);
- emissions originating directly from fuel evaporation; and
- non-exhaust PM emissions produced by wear on vehicle components (such as tyres, brakes and clutch) and road abrasion.

Hot emissions depend mainly on vehicle technology (emission control and aftertreatment) and driving conditions. Fig. 1.16 shows the effect of driving conditions on emission levels for a fleet of Euro-I petrol passenger cars. Mean vehicle speed is an important parameter, because it can be used to characterize driving patterns. The slow speeds encountered in cities usually include stop-and-go conditions, which may significantly increase emissions. On the other hand, the high speeds encountered on motorways demand high power output, which again increases emission levels. With respect to emissions, moderate speeds are the most favourable.

Mean speed, however, is not the only relevant parameter. Fig. 1.17 compares driving with the same mean speed but with different driving dynamics – that is, frequency and intensity of accelerations. Calm driving is obviously better than more aggressive vehicle use.

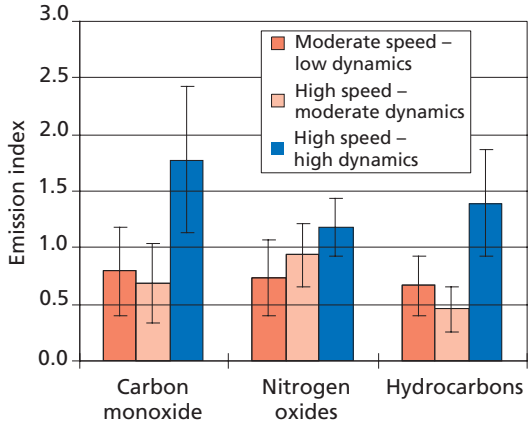
Emissions associated with cold starts are mostly significant for catalyst spark-ignition vehicles, because of the low catalyst efficiency before light-off temperature

Fig. 1.16. Effect of mean travelling speed on emission levels from passenger cars with catalysts



Note. So that they can be shown on the same graph, the figures for nitrogen oxides and hydrocarbons have been multiplied by 10 and those for carbon dioxide, divided by 100.
Source: Ntziachristos & Samaras (2000).

Fig. 1.17. Effects of driving dynamics on emission performance



Note. On the emission index, 1 = moderate speed and dynamics.
Source: Ntziachristos & Samaras (2000).

(the temperature at which 50% conversions of carbon monoxide or nitric oxide is achieved) is reached and because of the engine over-enrichment needed to sustain drivability despite incomplete fuel volatilization. Obviously, exhaust emissions increase with decreasing ambient temperature (Joumard & Sérié, 1999). The performance observed depends on the type of pollutant. Most cold-start over-emission of pollutants, which depends on fuel enrichment, occurs mainly in the earliest fractions of the trip. Estimation of the over-emission of engine cold start at a fleet level is a function of the pollutant considered, ambient temperature, vehicle technology, mean speed and average trip distance (Ntziachristos & Samaras, 2001). The effect of cold starts is concentrated mainly in urban areas, where most passenger cars are started and where many trips are shorter than 6 km. As a consequence, the aftertreatment system does not work under optimum conditions most of the time; this leads to relatively high emissions per distance driven, compared with long-distance driving (even at high speed) on roads outside of urban areas and on highways.

Evaporative losses are another source of non-methane VOC emissions from petrol-fuelled vehicles. Fuel evaporation occurs as diurnal losses (due to the temperature variation of the fuel-tank content during the day), as hot and warm soak losses in the mixture-preparation system (due to the elevated engine temperature) and as running losses during vehicle operation. For a fleet of passenger cars, evaporation losses depend on the ambient temperature variation, fuel volatility and mean trip distance. Evaporation losses, however, have been significantly reduced in modern passenger cars, which are equipped with sealed fuel injection systems and active carbon canisters in their tank vents.

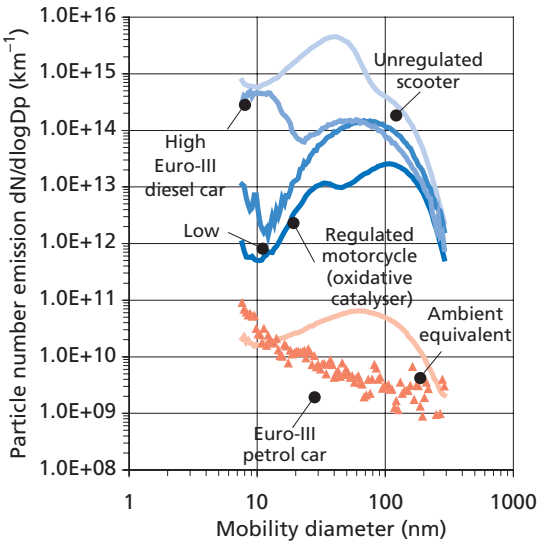
PM emissions

As a direct source of airborne particles from exhaust and component wear, road transport is a significant source of primary emissions of PM in urban areas. It is also a source of secondary particles formed by condensation of gaseous species (sulfates and nitrates) emitted in the exhaust and by resuspension of debris accumulated on the road surface.

Emissions of exhaust particles have received much attention lately, because of suspected adverse effects on human health. Fig. 1.18 shows typical size distributions of exhaust particles emitted by different vehicles, compared with a typical urban background distribution. Airborne particle emissions from vehicles form either a monomodal lognormal distribution with a peak in the range of 50–100 nm (accumulation mode) or a bimodal distribution with an additional mode in the range 10–50 nm (nucleation mode). The accumulation mode consists mainly of soot particles formed in diesel engines, due to incomplete combustion. In some cases, such as uncontrolled two-stroke engines, the accumulation mode may also form by condensation of volatile material. When the nucleation mode appears, it consists almost exclusively of volatile material (hydrocarbons, hydrated sulfuric acid and salts), and its appearance is also a function of dilution conditions.

By mass, the most significant non-exhaust sources are brake and tyre wear (UNECE/EMEP Task Force on Emissions Inventories and Projections, 2004). Airborne brake debris consists of particles with an aerodynamic diameter of 3–10 µm and is composed of metals (such as iron, copper and lead) and organic

Fig. 1.18. Typical size distributions of exhaust particles of different vehicle types



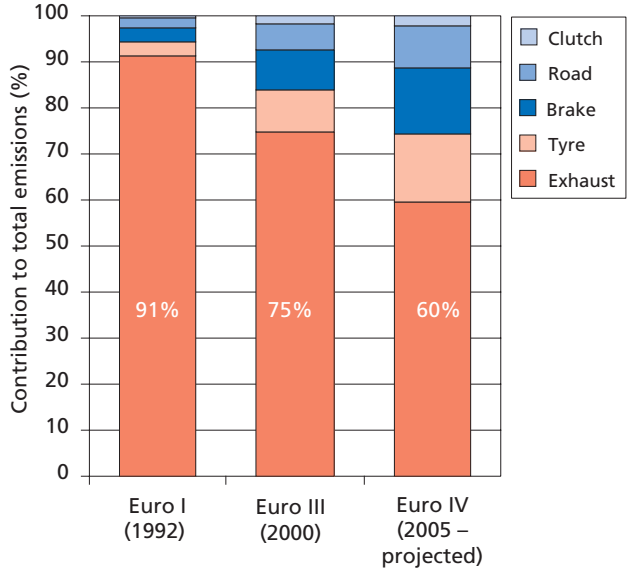
Source: Ntziachristos et al. (2003).

material and silicon components, which are used in the brake pads as binders. Tyre debris appears as particles up to several hundreds of micrometers in diameter, with a significant airborne fraction above 10 µm. Tyre particles consist of various rubbers (such as styrene butadiene rubber, natural rubber and polybutadiene). Organic zinc has been widely used as an indicator of tyre particles, because zinc is used in tyre production as a vulcanizing agent. Non-exhaust particle sources become increasingly important as exhaust emissions decrease. Fig. 1.19 shows the contribution of non-exhaust particle sources to total PM emissions from a diesel car. Also, a look at the emission levels of a well-maintained modern petrol car (see Fig. 1.18) shows that its major contribution to PM pollution is from non-exhaust sources.

Gross polluters and other factors

Emissions from vehicles and all other combustion processes have been reduced significantly over the past years. A number of other factors, however, devour part of this technological improvement. In cities, for example, traffic jams at peak hours drastically increase emissions per driven distance, and the search for parking places increases the distance driven. Still more important are the vehicles with poor or no maintenance or with malfunctioning aftertreatment systems – all of which result in increased emissions. The European Auto-Oil Programme Working

Fig. 1.19. Contribution of exhaust and non-exhaust particle sources to total PM emissions from diesel passenger cars of improving technology



Source: Ntziachristos & Samaras (2000).

Group 4 on Inspection and Maintenance (Auto-Oil II, 2003) estimated that the most polluting cars in the whole fleet (3%) are responsible for 27% of its emissions. Although this example is directed towards a worst-case scenario, it shows that only a few gross polluters might be responsible for large parts of vehicular emissions. This was confirmed by some experimental studies that used remote-sensing technologies to detect on-road single-vehicle tailpipe emissions.

Transportation technologies

The combustion engine or, more precisely, the internal combustion engine is by far the dominant source of power for on-road and off-road vehicles. In principle, fuel and air are fed to a chamber where combustion releases the chemical energy stored in the fuel and converts it to heat and mechanical power. This section is gives a brief overview of the engine and exhaust aftertreatment technologies that dominate the market today, and discusses how they may be developed further.

Emission regulations in the EU

Recent decades have been characterized by two major trends: lower emissions from new vehicles put on the market and the introduction of more environmentally friendly fuels, including the phasing out of leaded fuels and the reduction of sulfur concentrations in fuels. Emission regulations for new vehicles in the EU (called Euro I, Euro II, etc.) have been a major driving force for the reduction in emissions observed in recent years. A further tightening of controls on emissions from vehicles will be implemented through the Euro IV standard for light-duty vehicles (passenger cars) and Euro IV and V standards for heavy-duty engines (EU, 1998; EU, 1999). Also, discussions have begun on stricter limits for light-duty vehicles (Euro V) and heavy-duty lorry and bus engines (Euro VI).

Table 1.1 shows the EU emission standards for passenger cars and Table 1.2 shows the corresponding standards for heavy-duty engines. They regulate exhaust emissions of carbon monoxide, hydrocarbon, nitrogen oxides and PM. The Euro II to Euro IV standards for passenger cars differ for diesel fuel and petrol (lower carbon monoxide, but higher nitrogen oxides for vehicles that use diesel fuel). Vehicles that use petrol are exempted from PM standards, since port-injected petrol vehicles emitted very low levels of PM. The 2000 and 2005 standards are accompanied by fuel quality rules – for example, a maximum diesel-fuel sulfur content of 50 parts per million (ppm) in 2005. Fig. 1.20 shows comparisons of emission limits for nitrogen oxides and PM from passenger cars and heavy-duty vehicles, respectively, in the EU, Japan and the United States of America.

The reader should note that emission tests that use a chassis dynamometer on engine test benches are not performed primarily to deliver emission factors for subsequent use in modelling. These emission tests are executed under well-controlled laboratory conditions and simulate only a small fraction of all possible driving patterns and meteorological conditions.

Table 1.1. EU emission standards for passenger cars

Standard	Year	Diesel cars (g/km)				Petrol cars (g/km)			
		Carbon monoxide	Hydro-carbons and nitrogen oxides	Nitrogen oxides	PM	Carbon monoxide	Hydro-carbons	Hydro-carbons and nitrogen oxides	Nitrogen oxides
Euro I	1992	2.72	0.97	–	0.14	2.72	–	0.97	–
Euro II	1996	1.00	0.90	–	0.10	2.20	–	0.50	–
Euro III	2000	0.64	0.56	0.50	0.05	2.30	0.20	–	0.15
Euro IV	2005	0.50	0.30	0.25	0.025	1.00	0.10	–	0.08

Source: DieselNet (2004).

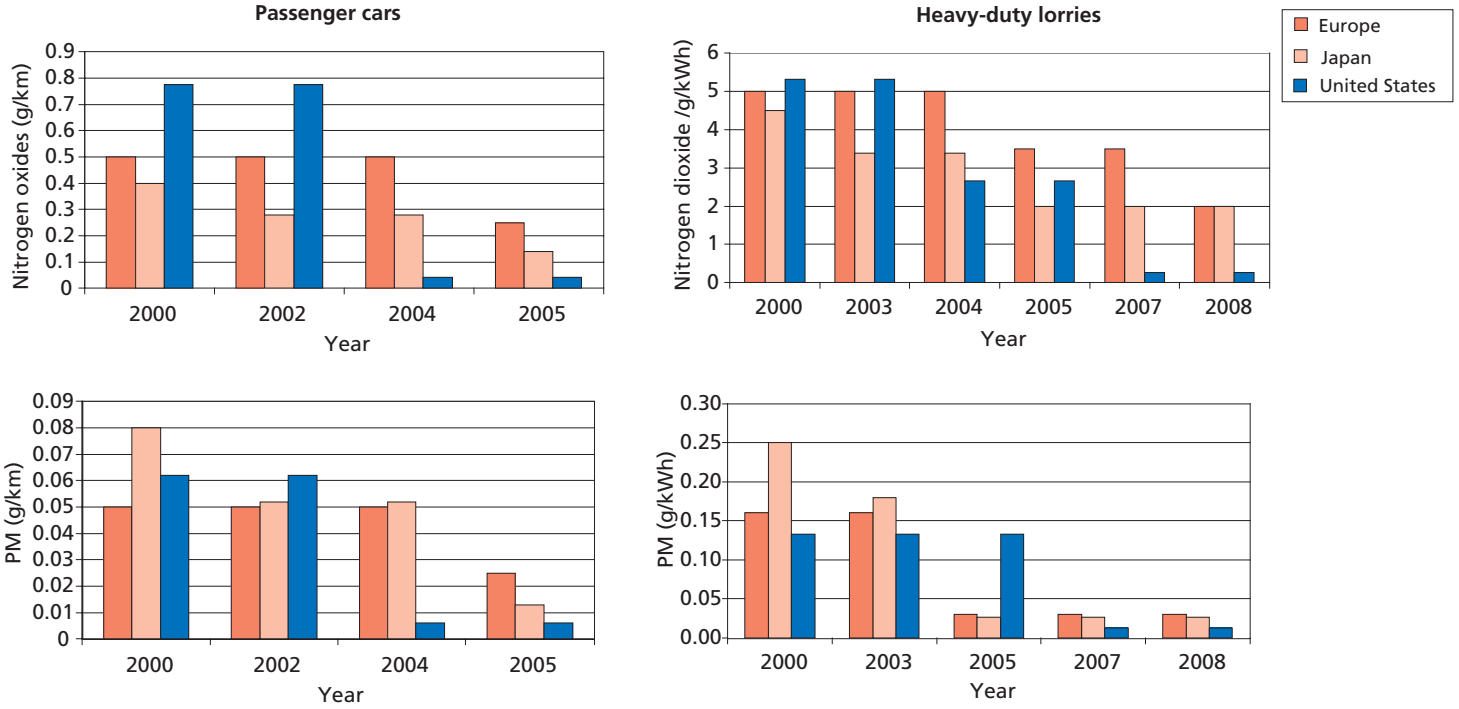
Table 1.2. EU emission standards for heavy-duty-vehicle diesel engines

Standard	Year	Carbon monoxide (g/kWh)	Hydrocarbons (g/kWh)	Smoke (m^{-1})	Nitrogen oxides (g/kWh)	PM (g/kWh)
Euro I	1992	4.5	1.1	–	8.0	0.36
Euro II	1996	4.0	1.1	–	7.0	0.25
Euro II	1998	4.0	1.1	–	7.0	0.15
Euro III	2000	2.1	0.66	0.80	5.0	0.10
Euro IV	2005	1.5	0.46	0.50	3.5	0.02
Euro V	2008	1.5	0.46	0.50	2.0	0.02

Note. Euro III introduced changes in test cycles for heavy-duty engines.

Source: DieselNet (2004).

Fig. 1.20. Comparison of emission limits of PM and nitrogen oxides for passenger cars and heavy-duty lorries in the EU, Japan and the United States



Note. Test cycles have changed over time and differ between countries; a direct comparison of numbers is therefore difficult.
Source: DieselNet (2004).

Engine technology

Two major types of internal combustion engines have emerged: the diesel engine (invented by Rudolf Diesel in the late nineteenth century) and the spark ignition petrol engine (often called the Otto engine, after the inventor Nicolaus August Otto (1832–1891), or just petrol engine, after the fuel that is most suitable for this combustion principle). The following are some important differences between these two engine types.

- The petrol engine uses a spark plug to ignite the fuel, while combustion in a diesel engine is started by auto-ignition, due to the heat created during compression.
- The ratio between fuel and air in a petrol engine is almost stoichiometric (neither an excess nor a deficit of air), whereas a large excess of air characterizes the diesel engine.
- In a petrol engine, fuel is evaporated and mixed with air to form a more or less homogenous gas mixture. In contrast, air is compressed to high pressures in the cylinder of a diesel engine, and small droplets of fuel are then injected.

Low fuel consumption and high reliability are high priorities in heavy-duty vehicles, while weight, price and power output are more important in private cars. For these reasons, the diesel engine is dominant in heavy-duty vehicles. Both petrol and diesel engines are used, however, in passenger cars.

Two other combustion concepts may be seen as combinations of the diesel engine and petrol engine. These concepts are called gasoline direct injection and homogenous charge compression ignition. These new combustion principles are expected to lead to improved engines that will meet future demands for low emissions and low fuel consumption. Cars using gasoline direct injection are already on the market, and this engine type has the potential to combine the low energy consumption of diesel engines with the low exhaust emissions of spark ignition engines. Energy consumption, however, is still higher than that for a corresponding diesel engine and emissions of PM tend to be higher than those from a spark ignition petrol engine (ACEA, 2002). Homogenous charge compression ignition is still under development, but it may have a role to play in the future for both heavy-duty engines and light-duty passenger cars.

The fulfilment of emission requirements is not the only driver of engine development. Fuel consumption and the corresponding carbon dioxide emissions are also of utmost importance, and will probably attract even greater attention in the future. This is an issue because less than half of the energy of the fuel in a combustion engine is converted to useful mechanical energy (the energy used to make a vehicle move). Diesel engines are, in general, considerably more efficient than petrol engines.

Engines that use more than one fuel (such as petrol and methane, or petrol and ethanol) are on the market, and an increasing demand is expected for alternative fuels and particularly carbon-dioxide-neutral fuels. Examples of alternative fuels

include methanol, ethanol, dimethyl ether, liquefied petroleum gas (butane and propane) and biodiesel (such as methyl ester of rapeseed oil). The quality of the fuel may significantly influence the composition of the exhaust. It has been shown that the aromatic and sulfur contents of diesel fuel have a strong influence on the emissions of PAHs and PM. The widespread introduction of new alternative fuels might result in regulations that limit currently unregulated pollutants, such as aldehydes; in general, this would give engine manufacturers the ability to optimize their engines to a certain fuel, so that the legal emission requirements are met. Moreover, it is usually easier to achieve low emissions of toxic pollutants with a less complex fuel.

Exhaust aftertreatment

Petrol

The fact that petrol engines use a nearly stoichiometric ratio between fuel and air is important for exhaust aftertreatment. Because the air–fuel mixture (measured by a lambda sensor) is tightly controlled, a so-called three-way catalyst can be used. This device operates properly above 250 °C. Once this temperature is reached, the conversion rate of the pollutants – into carbon dioxide, water and nitrogen – approaches 100%. During the first 10–30 seconds of operation, however, the catalyst cleans the exhaust very poorly or not at all. About 90% of the emissions that reach the atmosphere during real-life driving are therefore produced during the cold-start phase and the following minute.

Diesel

For cars using diesel fuel, there is no practical way to use the three-way principle for exhaust aftertreatment. In a typical diesel exhaust, the oxygen content is about 10% (compared with 21% in the air and 1% in the petrol exhaust just after the engine), and the reduction of, for instance, nitric oxide is hard to achieve in this oxygen-rich environment.

The most critical toxic emissions from diesel engines are PM and nitrogen oxides. Diesel PM can be separated from the exhaust by filtration and the collected soot fraction removed by combustion. The combustion of soot generally starts at temperatures above 500 °C, but the exhaust gases do not often reach such high temperatures. Several techniques are therefore applied to regenerate particle filters:

- oxidation of nitric oxide to nitrogen dioxide (the continuous regeneration trap) and ensuing oxidation of soot by nitrogen dioxide (oxidation starts at about 300 °C);
- the use of fuel additives, such as cerium oxide and copper oxide, to lower the combustion temperature needed; and
- active regeneration by manipulation of the engine parameters, to increase the exhaust temperature.

If the soot is not properly regenerated, the uncontrolled combustion that occurs when the backpressure rises will damage the filter by overloading it. The demand for intensive servicing to remove ash that has been collected in the particulate filter (and the costs involved) may, in certain cases, complicate the use of such filters. Nonetheless, one automaker has sold diesel cars equipped with particle filters since May 2000. This system requires the use of a cerium-based fuel additive.

The German automobile association (Allgemeiner Deutscher Automobil Club – ADAC) has tested four vehicles equipped originally with particle traps and two vehicles with different retrofit solutions (ADAC, 2004). All the particle traps reduced PM emissions during the modified cold-start procedure (called the new European driving cycle) by more than 90%, to around 0.001 g/km. A fuel consumption increase of 5–9% was observed in comparison with similar vehicle models without a trap. This increase in fuel consumption was explained by the modified engine tuning that was chosen also to obtain the 40–50% reduction in nitrogen oxide emissions observed for the vehicles tested in the study. One of the retrofit systems reached a similar PM reduction without a significant fuel consumption increase. In all cases, no losses in vehicle performance were measured.

Interest in PM emissions from diesel engines has increased in the last few years. In general, engines using older technology produce significantly more PM (especially soot), but the characteristic shapes of the size distribution curves do not differ greatly between older and more advanced technology (ACEA, 2002).

To meet future demands for lower emissions of nitrogen oxides, several techniques are being considered, including selective catalytic reduction with the addition of urea, storage of nitrogen oxides and reduction of nitrogen oxides by the use of diesel fuel. For the removal of nitrogen oxides, the intrinsic problem is the reaction of the reducing agent needed with oxygen, instead of nitric oxide and nitrogen dioxide, which leads to an unwanted increase in fuel consumption. Because of these difficulties, improved engine technology has often been applied to lowering nitrogen oxide emissions, rather than adding exhaust aftertreatment devices.

Expected future development

There are many views on the future development of vehicles and fuels. Different assumptions about political, economic and technological developments lead to different outlooks. Nevertheless, conventional diesel and petrol engines still seem likely to dominate the market for new cars, lorries and buses in the next decade or two. The market share for these engines, however, is expected to decrease from today's figure of close to 100%.

Work will continue on a number of alternatives. Alternative drivelines will continue to be improved (see Box. 1.1). Hybrid concepts are likely to be developed further – driven by the need for lower fuel consumption. The improvement

Box. 1.1. Alternative drivelines

Electric vehicles

Electric vehicles offer a number of advantages, such as low noise levels and virtually no toxic emissions during operation. Emissions occur elsewhere, however: for instance, during the production of the batteries and the electricity needed to recharge them. Still, extensive use of such vehicles has been inhibited by the current limitations of battery technology, such as limited driving range, and by high costs, restricting their use to special niche applications. A breakthrough for battery-powered vehicles seems unlikely in the near future, even though batteries are constantly becoming cheaper and more efficient. It would be better if the electricity were produced aboard the vehicle, which is one of the reasons why many automakers appear to be focusing instead on hybrid electric vehicles or on fuel cells.

Hybrid electric vehicles

Hybrid electric vehicles combine the internal combustion engine of a conventional vehicle with the battery and electric motor of an electric vehicle. A hybrid is more expensive and heavier than the corresponding conventional vehicle, but might cut fuel consumption and toxic emissions by about half in urban driving. Some hybrid vehicles can recover energy when they slow down during braking. Vehicles that use a smaller engine in combination with an electric motor are sometimes called light hybrids, while full hybrids also have a battery pack that enables the car to be driven at low speeds with the engine off.

City applications benefit somewhat more from hybrid solutions than, for example, long-haul transport applications, since losses during low load conditions and idling are large contributors to fuel consumption during urban driving. Hybrids are commercially available, and a major Japanese automaker has already put more than 100 000 on the road.

Fuel cells

A fuel cell converts the chemical energy of the fuel directly to electricity. A fuel-cell vehicle can therefore be seen as an electric vehicle in which a fuel cell has replaced the battery. Many see fuel cells as a future option for motor vehicles, especially passenger cars and buses, and the level of research and development in this field has increased in recent years.

Fuel cells consist of a solid polymer membrane covered by thin layers of electrode material on each side. Electricity is produced when hydrogen is fed to one side of the cell (anode) and oxygen or air to the other side (cathode). Several types of fuel cells, including proton exchange membrane fuel cells, produce electricity with high efficiency from gaseous hydrogen.

The barriers to building a hydrogen infrastructure vary according to the application. City buses, because of relatively lower barriers, might be among the first fuel-cell-equipped vehicles used on a larger scale. Building a general hydrogen infrastructure would take a long time and involve substantial costs. The introduction of fuel cells for vehicles would be facilitated by the use of a liquid fuel instead of hydrogen gas, since the infrastructure for

Box. 1.1. (contd)

liquids is mostly in place, and some fuel cells already utilize liquid fuels directly. Examples include the direct methanol fuel cell and the solid oxide fuel cell. The latter was tested for stationary generation of electrical power and might find, in the far future, use in motor-vehicle auxiliary power units (for instance, for heating or cooling) or in propulsion systems. Many find that a more likely solution for road vehicles is to use fuel cells that utilize hydrogen produced from methanol or petrol aboard the vehicle, by means of a reformer. Ultimately, cost reductions must occur before fuel-cell vehicles become a competitive option for the mass market.

Automotive gas turbines

Gas turbines for lorries and buses have a long history. One company in the United States, for instance, presented a prototype lorry as early as 1964. Since then, various projects have tried to optimize the performance of the system by changing such factors as the turbine-inlet temperature, the compressor-inlet temperature and pressures. Advanced materials (such as ceramics) have also been developed to cope with higher turbine-inlet temperatures, thus increasing the thermal efficiency of gas turbine engines. Gas turbines have also been used in hybrid drive trains.

Automotive gas turbines are highly reliable (the turbine is simple, with the rotor as the only moving part), can potentially produce low levels of emissions of nitrogen oxides and PM and can run on a variety of fuels. Because of these qualities, they have been under development, particularly in Japan. On the other hand, they are expensive to manufacture, do not perform well during acceleration and deceleration, and are inefficient when idling and under low load conditions. For the time being, the technical challenges involved appear to limit the automotive industry's interest in gas turbines.

of fuel cells and batteries is expected to continue. High-volume production of light-duty vehicles powered by fuel cells, however, seems unlikely to begin before 2015 (DeCicco, 2001). Also, battery-powered electric vehicles appear to have a less positive future than was envisaged a few years ago.

Summary of transportation technology

Since the 1980s, exhaust emissions of carbon monoxide, nitrogen oxides, hydrocarbon and PM from new vehicles have been reduced by about a factor of 10 to 100. In the coming decade, another order-of-magnitude reduction seems to be technically possible. The pace of reduction will probably be determined by the gradually stricter emission standards set by the EU and the United States. One of the most important actions taken to reduce pollution from road traffic and consequently to improve air quality, is reducing emissions of PM and nitrogen oxides from conventional diesel engines and, to some extent, petrol engines. Also, diesel engines and the corresponding aftertreatment devices need to be developed

further to fulfil coming emission requirements in the EU and the United States. Several options exist for reducing nitrogen oxides, and a rapidly growing demand for particle filters in diesel passenger vehicles is expected. Heavy-duty vehicles may need particle filters to meet the Euro V standards in 2005. One challenge is to achieve reliable particle trap regeneration under all possible driving conditions.

The extensive use of electronics and software to control engines is a prerequisite for the development of engines with low emissions. To gain fully from improved engine technology, however, vehicles should not emit significantly more pollutants under normal operations for the particular type of engine than what was measured during the approval procedure. Owners must, of course, guarantee that their vehicles are properly maintained. In addition, future legislation should not leave room for interpretation – for instance, about how emissions are controlled outside the speed and load combinations used during approval testing for the particular type of engine. Action may also be needed to prevent illegal manipulation of the engine control unit; for example, by changing the software, an engine can be made to deliver higher power and lower fuel consumption simultaneously. This, however, is possible only if the emission requirements are completely put aside. Furthermore, such manipulation is likely to reduce the durability of emission control devices.

Fuels and additives

Fuel quality in Europe[2]

In 1988, the EU mandated the European Committee for Standardization (CEN) to develop specifications for premium and regular grades of unleaded petrol, diesel fuel and automotive liquified petroleum gas. Member States were required to adopt the specifications proposed as national standards by September 1993 and withdraw conflicting national standards by the same date. All standards have been revised to incorporate new regulations on fuels introduced after 1993.

In the early 1990s, on the initiative of the EC, the Auto-Oil I Programme began. Both the oil and automotive industries were involved, and the Programme's main objectives were to assess the cost–benefit ratio of the measures to reduce pollution from traffic and to improve air quality. It also included an experimental phase: the European Programme on Emissions, Fuels and Engines Technologies (EPEFE), which provided reliable data on the effects of engine technology and fuel quality on emissions. The EPEFE results represented the basis for the development of a new fuel regulation: as a consequence, Directive 98/70/EC (EU, 1998b) set out stringent new specifications for automotive fuels, particularly for parameters relevant to the environment. The first phase was introduced in 2000, and the next step was planned for 2005. Directive 98/70/EC can be summarized as follows.

[2] *Source:* CONCAWE (2001a, 2001b).

- All petrol must be unleaded from 1 January 2000; exceptions to this ruling might be possible up to 2005 if a Member State could show that eliminating lead from petrol would result in technical difficulties.
- From the year 2000, all petrol must comply with the new limits on petrol volatility, PAHs (including benzene), olefins and sulfur. Further reductions in PAHs and sulfur content would be mandated in 2005.
- From the year 2000, diesel fuels must comply with new specifications that introduce a higher ignition quality requirement and a limitation on PAHs and sulfur content. In 2005, the sulfur content would be cut further.

Directive 2003/17/EC (EU, 2003a) amended Directive 98/70/EC and introduced modifications to the maximum sulfur content in fuels. The limit of 50 mg/kg after 1 January 2005 was confirmed, but, from that date on, sufficient quantities of petrol and diesel fuels with a maximum sulfur content of 10 mg/kg had to be available on an appropriately balanced geographical basis, to permit the free circulation of new vehicles requiring these fuels. Finally, in 2009 no petrol and diesel fuel may contain more than 10 mg/kg sulfur. The Directive also states that the EC must evaluate the need for further modification of fuel specifications (other than those for sulfur) and to confirm the final date of entry into force of the 10 mg/kg limit for diesel by the end of 2005.

In general, fuel quality is lower outside the EU. For example, the maximum content of benzene in petrol is higher and leaded petrol is still on the market in some countries; in some cases, there is no limit on PAH content. With regard to diesel fuel, the ignition quality (minimum cetane number, which is a measure of diesel fuel combustion properties, similar to the use of octane ratings for petrol) is lower and the maximum sulfur content and density are higher.

Main environmentally relevant parameter: evolution of legislation

Petrol

Several EU directives have been issued to regulate the lead content of petrol; in particular, as a consequence of the introduction to the market of passenger cars equipped with three-way catalysts, Directive 85/210/EEC allowed unleaded petrol (0.013 g/l max.) to be marketed and encouraged Member States to provide incentives (for example, through taxation) to promote the use of unleaded petrol (Table 1.3).

Directive 85/210/EEC, which allowed the introduction of unleaded petrol, also specified a benzene level of 5% by volume (v/v) maximum; this value was cut to 1% v/v by Directive 98/70/EC. The latter also set a maximum PAH content in all petrol of 42% v/v from the year 2000, and 35% v/v from 2005. Before Directive 98/70/EC was issued, however, several European countries had already

Table 1.3. EU legislation to regulate lead content in petrol

Directive	Content	Maximum lead content (g/l)
78/611/EEC	Limits on maximum lead content	0.15–04
85/210/EEC	Unleaded petrol allowed to be marketed Introduction of a premium unleaded grade of 95 research octane rating/ 85 motor octane rating	0.013 (unleaded only)
87/416/EEC	Ban of leaded regular petrol allowed	–
98/70/EC	Ban of leaded petrol from 2000	0.005

Table 1.4. European legislation to regulate benzene content in petrol

Country	Maximum benzene content (% v/v)	Date
Austria	3	1 September1990
Italy	3	1 January 1993
	1	1 July 1999
Germany	1	October 1995
Finland	3	January1993
	1	April 1994

Note. Adherence in Germany was voluntary. Italy also set a maximum content of 40% v/v for PAHs in petrol from 1998.

introduced more stringent specifications; Finland used tax incentives to encourage the diffusion of reformulated petrol (Table 1.4).

Oxygenated compounds can have beneficial effects on emissions, especially in cars without catalysts. Directive 85/536/EEC specified that, by 1988, Member States had to ensure that there were no legal and administrative obstacles to the sale of petrol blends containing oxygenates suitable for use in spark ignition engines.

Sulfur in petrol does not affect regulated pollutant emissions directly, but it somewhat reduces the efficiency of a three-way catalyst. Moreover, such new engine technologies as gasoline direct injection, which require aftertreatment devices other than the three-way catalyst (nitrogen oxide adsorbers), require very low sulfur levels. For this reason, the maximum sulfur content will be reduced further in the next few years (Table 1.5).

Diesel

Directive 93/12/EEC fixed a maximum limit of 0.2% sulfur by mass (m/m) to be applied to all gas oils, including diesel fuel, from 1 October 1994, and a maximum limit of 0.05% m/m for diesel fuel from 1 October 1996. In the interim, Member

States were required to ensure the progressive availability of a diesel fuel with a sulfur content of 0.05% m/m by 1 October 1995. All EU countries adopted the 0.05% m/m limit from 1 October 1996.

Directives 98/70/EC and 2003/17/EC further reduced the sulfur content to 350 mg/kg from 1 January 2000, to 50 mg/kg from 1 January 2005. From 2009 (a date to be confirmed by the review process expected to finish in 2005), the maximum sulfur content allowed will be 10 mg/kg (Table 1.5). Before the entry into force of Directive 93/12/EEC, some European countries introduced national limits on the sulfur content of diesel. Others (Denmark, Finland, Sweden, and the United Kingdom) used tax incentives to promote special grades of diesel with lower sulfur contents.

Table 1.5. Development of sulfur in petrol and diesel fuels

Directive/Norm	Implementation date	Maximum sulfur content (mg/kg)	
		Petrol	Diesel
CEN EN 228:1993	September 1993	500	–
93/12/EEC	1 October 1994	–	2000
	1 October 1996	–	500
98/70/EC	1 January 2000	150	350
	1 January 2005	50	50
2003/17/EC	1 January 2005	10	10
	1 January 2009	10	10

Basic fuel quality and fuel properties[3]

Fuel properties are often divided into two classes: enabling properties and enhancing properties. The enhancing properties are those fuel properties that change emissions by a small degree compared with the changes brought about by engine technology. Fuel properties are called enabling when the changes they bring about enable the use of new engine technologies that can greatly reduce pollutant emissions (see Box 1.2).

Alternative fuels[4]

In a green paper on the security of the EU energy supply (EC, 2000), the EC proposed the replacement of 20% of conventional fuels for road transport with

[3] *Source*: CONCAWE (1999).

[4] *Source*: EC (2001).

Box 1.2. Enhancing and enabling properties

Enhancing properties

The effect of fuel quality on each regulated and unregulated pollutant depends on the engine technology considered. The following subsections consider the following pollutants: nitrogen oxides, hydrocarbon and carbon monoxide, PM, benzene, aldehydes and 1,3-butadiene, and PAHs.

Nitrogen oxides

In modern conventional petrol vehicles equipped with a three-way catalyst, petrol quality can influence emissions, but the effects are generally very small. The main fuel properties that can influence nitrogen oxide emissions are concentrations of sulfur, PAHs and, to a lesser extent, olefins. High levels of sulfur can reduce catalyst efficiency, while higher levels of PAHs can either increase or reduce nitrogen oxide emissions, owing to competing effects on the combustion process and catalyst efficiency. Density, T95 distillation temperature and PAH content are the diesel fuel properties that have an effect on nitrogen oxide emissions; in this case, however, the effects are quite small. Moreover, the effects are different in magnitude and sign for light-duty and heavy-duty vehicles.

Hydrocarbons and carbon monoxide

Petrol composition has an almost negligible effect on hydrocarbon and carbon monoxide emissions, which are reduced very effectively by the catalyst. Diesel fuel quality, however, has a much larger impact on these emissions from both light- and heavy-duty diesel vehicles. Density, T95 distillation temperature and, mainly, cetane number can influence the emissions to a large extent but, in general, diesel vehicles already emit very low levels of these pollutants. The effects can therefore be important in relative terms, but not significant in absolute terms.

PM

Some properties of diesel fuel – mainly density, T95 distillation temperature, PAH content and sulfur content – can significantly influence the mass of PM emissions from diesel vehicles. In the case of PM emissions, the effects of fuel quality changes differ for light- and heavy-duty engines.

A recent study on PM emissions states that the effects of fuel on the heavy-duty vehicle nucleation mode[a] are greater than the effects of the technology. They report 80% lower PM emissions for the Euro-III compressed natural gas heavy-duty vehicle than for the Euro-II diesel heavy-duty vehicle, with a reduced accumulation mode and a lower nucleation mode. Heavy-duty compressed natural gas vehicles and passenger cars with diesel particulate filters had emission values for the number of particles (number emission values: total number of particles emitted per driven kilometre) that were an order of magnitude lower. No nucleation mode was found for light-duty vehicles; the multi-point-injection light-duty

Box 1.2. (contd)

vehicle had number emission values similar to those for vehicles with diesel particulate filters – an order of magnitude lower than for conventional diesel vehicles. Particle mass and size were similar for liquified petroleum gas and petrol. Total particle number emissions at 120 km/h were higher for vehicles with diesel particulate filters than for vehicles using petrol and liquified petroleum gas, but in the same range for all three technologies (Anderson & Wedekind, 2001).

Benzene

Mainly petrol-fuelled engines emit benzene, and it comes from fuel benzene that has escaped the combustion process and from partially burned heavier PAHs. Limiting the content of benzene and PAHs in the petrol can reduce benzene emissions, but using a very efficient three-way catalyst remains the most effective means.

The benzene emissions of Euro-II passenger cars and light-duty vehicles were studied under various conditions (Heeb et al., 2002). When the benzene in fuel was reduced from 2% to 1%, a pre-catalytic reduction for urban driving of 23–29% was observed. At speeds above 100 km/h, the reduction percentage was 6–18%. At speeds above 130 km/h the post-catalytic benzene emissions were independent of the benzene content of the fuel. Under a number of distinct conditions, the authors observed benzene formation in the catalytic converter.

Aldehydes and 1,3-butadiene

These unregulated pollutants are influenced by the fuel quality similarly to hydrocarbon emissions. For petrol vehicles, the influence of a catalyst is much more important than that of fuel parameters; for diesel-powered vehicles, aldehydes and 1,3 butadiene emissions decrease when density and levels of PAHs are reduced and when the cetane number is increased.

PAHs

Vehicles emit PAHs by several pathways (CONCAWE, 1998); a fraction of the exhaust PAHs comes from the fuel PAHs that survive combustion, while other exhaust PAHs can be created during the combustion process from non-PAH fuel components. The lubricating oil may also contribute to the exhaust PAHs.

Enabling properties

The effects of fuel properties on pollutant emissions are significant, but they are usually very small when compared with the effects of changes in engine technology. Some fuel properties, however, can have a very important effect when they enable the use of new engine technologies that make an important contribution to reducing pollutant emissions; good examples of this are the introduction of unleaded petrol, which enables the use of three-way catalyst technology, and low-sulfur fuels, which are required by some new

aftertreatment devices (such as nitrogen oxide storage catalysts and some particulate traps).

A diesel fuel with a low content of sulfur and PAHs was tested in medium-duty lorries equipped with diesel particulate filters. Compared with emissions produced with a baseline fuel and no filter, reductions were found in PM (89–98%), total hydrocarbons (72–80%) and carbon monoxide (81–90%). The effect of the fuel was much greater when it was combined with the filter. Also, when the filter was used, there were large reductions for alkenes, alkynes and PAHs (Durbin et al., 2003).

[a] The terms nucleation mode and accumulation mode refer to the mechanical and chemical processes by which most aerosol particles are produced. In the nucleation mode, the smallest aerosols (radius of 0.001–0.1 μm) are produced principally by gas-to-particle conversion, which occurs in the atmosphere. In the accumulation mode, larger aerosols (radius of 0.1–1 μm) are generally produced by the coagulation of smaller particles and by the condensation of gas vapour onto existing aerosol particles.

substitute fuels by 2020 (see Box. 1.3). Substitutes exist for use in the medium, long and very long terms. The most promising forms are biofuels in the short and medium terms, natural gas in the medium and long terms and hydrogen in the very long term. The spread of biofuels could help reduce the EU's energy dependence, improve the environment and diversify agricultural production and jobs.

The EC has put forward two specific measures to promote biofuels.

1. The first, Directive 2003/30/EC (EU, 2003b), adopted on 8 May 2003, established the gradual introduction of a minimum percentage of compulsory biofuel consumption in each Member State: a 2% rate would be proposed as a first stage (2005), with total flexibility as to whether this objective is achieved by mixing biofuels with fossil fuels or by using pure biofuels. In this way, unforeseen effects on engines and the environment would be avoided. At the same time, it would create a stable market and should increase the production capacity of existing biofuels fivefold. The second stage would need to aim at achieving a biofuel penetration rate of about 5.75% by 2010.
2. The second measure is a proposal that aims to set new EU rules on tax reductions for biofuels. While meeting the need to coordinate the national arrangements on biofuel taxation, the proposal would also help Member States create the necessary economic and legal conditions for achieving or even exceeding the objectives in Directive 2003/30/EC. The proposal would give Member States the option of introducing tax reductions consistent with their budgetary constraints, local circumstances (for example, for agricultural crops) and technological choices.

Box 1.3. Alternative fuels, fuel blends and additives

The Auto-Oil II programme provided an overview of the advantages and disadvantages of several fuels and fuel blends (Auto-Oil II, 2000).

Compressed natural gas

Compressed natural gas has a number of advantages. It has a low carbon-to-hydrogen ratio, is nearly free of sulfur and contains no toxic components. Also, it results in low emissions of PM, lower emissions of carbon dioxide per gigajoule of fuel, low cold-start emissions, and lower emissions of nitrogen oxides, and has a very low summer smog potential. On the other hand, it results in relatively high emissions of methane.

Liquefied petroleum gas

Liquefied petroleum gas can optimize the emission performance of passenger cars by reducing the emissions of nitrogen oxides, hydrocarbon and carbon monoxide by 80–95% relative to a petrol-fuelled vehicle. It has low sulfur content and a negligible amount of toxic components. Emissions of benzene, toluene and xylene and summer-smog potential are lower than for petrol-powered cars, and emissions of PM, PAHs, aldehydes and formaldehyde, and winter smog formation potential are lower than those of diesel-powered vehicles.

Dimethyl ether

Dimethyl ether can be produced from a variety of feedstocks, including natural gas, heavy crude oil, coal, waste and biomass. Emissions of nitrogen oxides and PM engines powered by dimethyl ether are comparable to those of lean-burn heavy-duty engines using liquified petroleum gas and natural gas or to light-duty Otto engines equipped with three-way catalysts. Dimethyl ether is non-toxic and, because of the absence of carbon–carbon bonds, emissions of hydrocarbons (such as PAHs and benzene) are very unlikely (Verbeek & Van Der Weide, 1997). Dimethyl ether can be used as fuel in dedicated engines or as an additive to biofuels.

Biodiesel

Biodiesel (fatty acid methyl ester), mainly produced from rapeseed oil, and used in up to 30% blends with normal diesel, is biodegradable and non-toxic. It contains almost no sulfur and no PAHs. As a result, it gives lower emissions of PM and reduced PAH content of exhaust particles. Its cold-start behaviour, however, is inferior, and the relatively higher level of nitrogen oxide emissions would require some engine adjustments. Its biggest disadvantage is a high level of aldehyde emissions.

Bioethanol

Ethanol can be fermented and distilled from biomass (such as sugar cane, cereals, sugar beet and wood). Bioethanol contains no sulfur, is biodegradable and is less toxic than methanol

or biomethanol. Petrol fuels blended with ethanol can reduce emissions of carbon monoxide and nitrogen oxides slightly and can reduce emissions of hydrocarbons more significantly. High ethanol content, however, results in higher emissions of unburned ethanol and aldehydes. The conversion rate of the three-way catalyst for unburned ethanol is low and, as a consequence, tailpipe emissions are high, while aldehydes are converted effectively. Also, bioethanol and blends with petrol and diesel fuel give rise to lower emissions of PM, benzene and 1,3 butadiene. It was observed that benzene emissions decrease with increasing bioethanol content in petrol. Moreover, the ozone formation potential is lower than for emissions from petrol- or diesel-powered vehicles. Major disadvantages, however, are very high hydrocarbon evaporative emissions and increased formation of acetaldehyde. Compared with pure petrol-powered vehicles, formaldehyde emissions are reduced in petrol blended with bioethanol (He et al., 2003; Hsieh et al., 2002; Bucksch & Egebäck, 1999).

Biomethanol

Biomethanol and blends with petrol, diesel and dimethyl ether are highly corrosive and toxic. A significantly high level of aldehydes was formed in such blends.

Diesel/Water emulsions

These emulsions have a water content of about 10–13%, plus some 2–3% additives to stabilize the mixture. A reduction in nitrogen oxides and PM was observed in such emulsions, especially for heavy-duty engines.

Methylcyclopentadienyl manganese tricarbonyl

Methylcyclopentadienyl manganese tricarbonyl, a manganese-containing additive in diesel, has been used in the United Kingdom since 1995. It has also been used as an anti-knock additive in Canada since 1976. The environmental and health consequences, however, are not well understood, and its use as a fuel additive has raised several concerns over the last 25 years about its effect on human health.

Methyl tertiary butyl ether, ethyl tertiary butyl ether and methyl tertiary amyl ether

These are oxygenated compounds used as octane enhancers that mix with petrol perfectly. Among them, methyl tertiary butyl ether is the most cost-effective, because of its low production costs. An offshoot of using such oxygenates as a petrol component is the lowering of emissions of carbon monoxide from mature technology vehicles, due to the so-called natural leaning effect provided by their inclusion. A small decrease in total hydrocarbons is also commonly seen in emissions from mature technology vehicles, while the effect on emissions of nitrogen oxides is more variable and seldom beneficial. Methyl tertiary butyl ether, like all other oxygenates, increases aldehyde emissions (Graupner et al., 1996; Poulopoulos & Philippopoulos, 2000). Ethyl tertiary butyl ether as a petrol blend (15–17%) might replace methyl tertiary butyl ether as an oxygenated compound; it can

Box 1.3. (contd)

be produced from bioethanol and generates no problems with toxicity and ecotoxicity. The effects on air quality of using these oxygenates in petrol fuel blends were evaluated for the Strasbourg area; modelling results showed reductions for carbon monoxide (30%) and VOCs (45%), and a slight increase of nitric oxide (0–5%).

Methanol-containing additives

Methanol-containing additives were tested in mixtures of up to 15% v/v. There were moderately decreased PM emissions, and decreased emissions of nitrogen oxides. In general, emissions of total hydrobarbons and carbon monoxide were found to increase (Chao et al., 2001).

Cerium oxide

As a catalytic diesel-fuel additive, cerium oxide reduces emissions of hydrocarbons and carbon monoxide; no effect on emissions of PM has been observed (Lin & Chao, 2002). Cerium-based additives have been proposed and marketed as fuel-borne catalysts, to reduce emissions of PM from diesel-powered vehicles and to reduce the regeneration temperature of diesel particle filter systems.

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2. Contribution of traffic to levels of ambient air pollution in Europe

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Key points

Facts

In the 1990s, the introduction of technological measures – for example, improvements in fuel quality, abatement technology such as catalytic converters and refinements in engine design – improved the overall air quality in most urban areas, even though road traffic increased. Average urban background concentrations of ozone, however, increased in the 1990s, probably owing to the reduced titration of ozone caused by decreasing emissions of nitric oxide gas.

In most urban areas of the countries belonging to the EU before May 2004, however, road transport is the most important source of ambient air concentrations of the following pollutants: nitrogen oxides, carbon monoxide, benzene, black smoke (except in cities where coal is widely used for domestic heating) and ultrafine particles (those whose aerodynamic diameter is smaller than 1.0 µm – PM1.0). In these areas, tailpipe emissions of primary particles by road transport contribute up to 30% of PM2.5, and non-tailpipe pollutants (such as resuspended road dust and brake-lining wear) are the most important source of the coarse fraction (those whose aerodynamic diameter is 2.5–10 µm – PM2.5–10). Overall, in the countries belonging to the EU before May 2004, emissions from road transport are mainly responsible for the non-compliance of some urban areas with the limit values of the EU air-quality directives for benzene, nitrogen dioxide and PM10 (aerodynamic diameter smaller than 10 µm). A similar conclusion cannot be drawn for the 10 new EU Member States, owing to insufficient data on PM, ozone and nitrogen dioxide.

Concentrations of transport-related air pollutants vary between areas of a city. In a belt as wide as 0.2–0.5 km along major urban highways, concentrations of nitric oxide, black smoke (soot) and PM0.1 are much higher than those of the urban background. Also, the gradients for nitrogen dioxide are less pronounced, while PM2.5 and PM10 have an even smoother spatial distribution. Moreover, in traffic-intensive street canyons (areas where dispersion is limited by

[5] The authors thank Steinar Larssen, Norwegian Institute for Air Research, Kjeller, Norway, for his comments and suggestions on this chapter.

tall buildings), limited dispersion ensures that levels of all traffic-related pollutants are much higher than those of the urban background.

Trends

Current policies should result in improved air quality. By 2010, levels of traffic-related pollutants in most urban areas in the countries belonging to the EU before May 2004 are forecast to be roughly 50% of those in 1995, and an estimated 90% of the urban population will live in areas meeting the EU limit values for nitrogen dioxide (hourly value), carbon monoxide, benzene and lead. Without additional measures for annual nitrogen dioxide and 24-hour and annual PM10, an estimated 20%, 70% and 50%, respectively, of urban populations will live in areas where limit values are not attained – mainly because of road transport emissions.

Increasing traffic congestion and the growth of traffic volume in urban areas are fuelling non-attainment and undermining the benefits to urban air quality of the introduction of cleaner fuels, more stringent emission limits for new vehicles, and road management. Also, at urban and rural background locations, average ozone concentrations are expected to increase further, owing to a decrease in emissions of nitrogen oxides, which act as ozone sponges, and possibly owing to an increase in hemispheric background concentrations of ozone. Further, the relative contribution of road-traffic non-tailpipe emissions to PM10 is expected to increase in the coming years.

The likelihood that transport development in central and eastern Europe will follow the patterns in western countries poses the risk of traffic's increasing its contribution to air pollution in the WHO European Region.

Conclusions

Owing to continuing urbanization and expansion of urban areas, an increasing share of the population is likely to be exposed to elevated levels of traffic-related pollutants, in spite of the general decrease in overall air pollution levels. Also, exposure to traffic-related air pollutants is probably increasing, as a result of the increasing share of passenger vehicles in commuting and the increasing amount of time vehicles spend in congested traffic on high-volume roads.

Introduction

Road-traffic emissions come from a number of sources. They include exhaust pipe emissions and contributions from friction processes and resuspended road dust. This results in a complex mixture that includes PM and gaseous pollutants, such as nitrogen oxides (nitric oxide and nitrogen dioxide), carbon monoxide and VOCs, all of which pose risks to health (WHO Regional Office for Europe, 2003). This chapter:

- analyses the extent, spatial distribution and time trends of transport-related air pollution;
- examines the determinants of the ambient concentration of main pollutants; and

- gives examples of the effects of measures to reduce the impact of traffic on air quality, and discusses the possibility of an additional indicator for traffic-related pollution.

The chapter focuses mainly on data from the countries belonging to the EU before May 2004, but the general tendencies and conclusions should be applicable to a broader range of countries in the WHO European Region.

Despite the increase in the volume of road transport (EEA, 2002c, 2003), emissions of transport-related air pollution decreased in the last two decades throughout the EU (EEA, 2002b). This was mainly the result of policy-driven technological improvements (such as the introduction of three-way catalysts for passenger cars in the mid-1990s), fleet renewal and improvements in fuel quality. Consequently, the parameters of air quality affected by traffic-related emissions have generally improved over the last two decades. Air pollution, however, remains a problem: the air-quality limit and target values for ozone, PM10 and nitrogen dioxide that are to be met by 2005–2010 are currently exceeded extensively in European cities. This is also true of the air-quality limit and target values for ozone and, to some extent, PM10 in rural areas (WHO Regional Office for Europe, 2003; EEA, 2001).

PM pollution may be the greatest potential threat to health (Hoek et al., 2002). Exposure to concentrations above the EU limit values for PM10 is expected for a significant proportion of the urban population. Also, urban areas are still expected to exceed the EU limit value for nitrogen dioxide (annual average) in 2010, especially in street canyons and near highways (EEA, 2001). On suburban, regional and Region-wide levels, emissions by road traffic contribute to the formation of ground-level ozone. At urban background locations, ozone concentrations show an increasing trend, which also raises concern about health (WHO Regional Office for Europe, 2003).

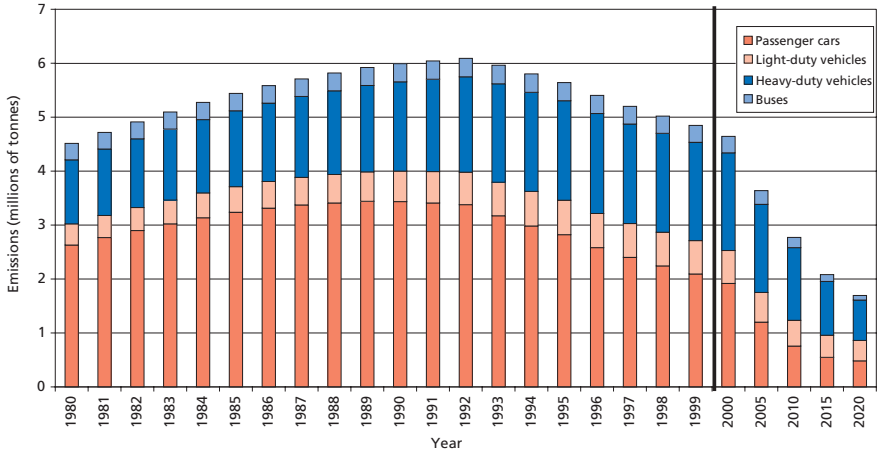
Traffic emissions of nitrogen oxides, carbon monoxide and VOCs in Europe

Fig. 2.1–2.4 present emissions of nitrogen oxides, carbon monoxide, VOCs and PM by road traffic for the period 1981–2020. Projections begin with the year 2000.

Emissions of nitrogen oxides (Fig. 2.1) increased steadily during the 1980s, by about 25%, owing to increasing road traffic. They fell by 20% from 1991 to 1998, mainly owing to the introduction of three-way catalysts in new passenger cars. The gradual increase in sales of diesel-powered passenger cars in some countries (Austria, Belgium, France, Germany and the Netherlands) may result in higher nitrogen oxide emissions. The increasingly tighter limits for new vehicles (Euro III and Euro IV) were expected to reduce emissions significantly, starting from 2000.

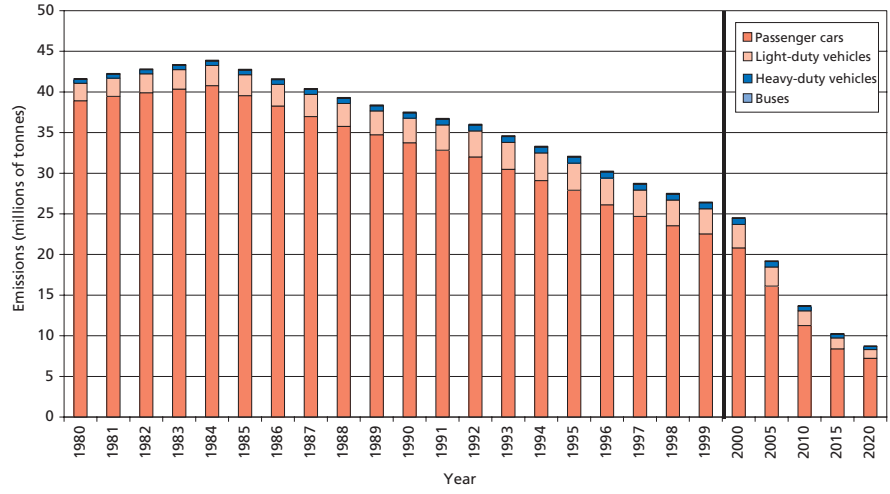
Emissions of carbon monoxide (Fig. 2.2) in general decreased from 1981 to 1998, mainly owing to the introduction of three-way catalysts. This trend is expected to continue.

Fig. 2.1. Total traffic emissions of nitrogen oxides in the countries belonging to the EU before May 2004, 1981–2020 (projected)



Source: Transport and Environment Database System Project (2003).

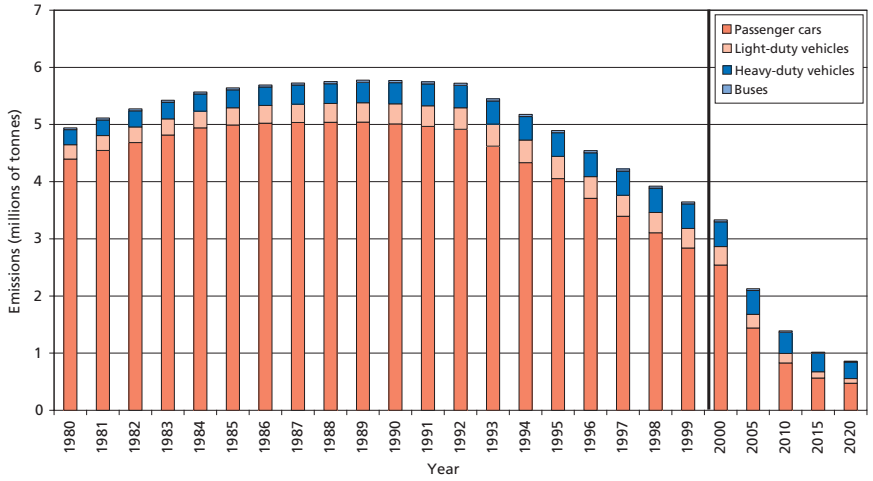
Fig. 2.2. Total traffic emissions of carbon monoxide in the countries belonging to the EU before May 2004, 1981–2020 (projected)



Source: Transport and Environment Database System Project (2003).

Passenger cars contribute about 80% of the total emissions of non-methane VOCs from traffic (Fig. 2.3). As a result of tighter emission standards, a drastic decrease in these was projected, starting in 2000. The decrease would also indicate a reduction in benzene emissions. Emission tests measure benzene as part of the mixture of VOCs. In addition to the effect of catalytic converters on reducing emissions, the benzene content in petrol was recently reduced from 5% to 1%.

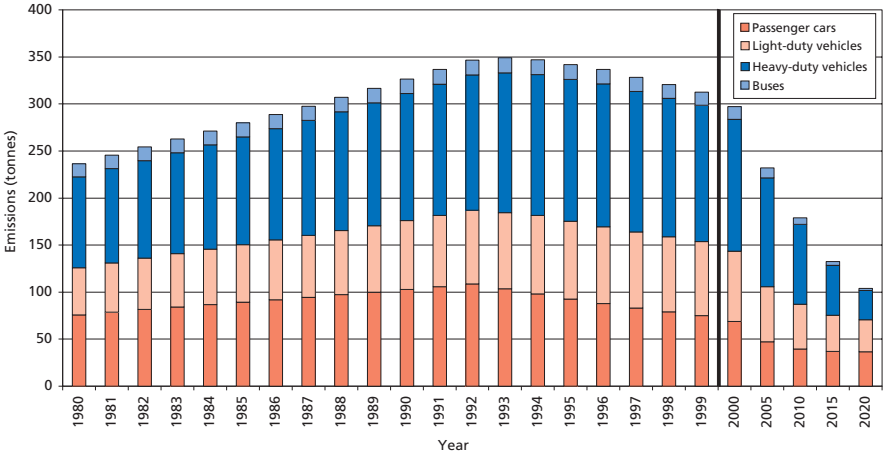
Fig. 2.3. Total traffic emissions of VOCs in the countries belonging to the EU before May 2004, 1981–2020 (projected)



Source: Transport and Environment Database System Project (2003).

Similarly to the other three pollutants, emissions of PM – characterized as the mass of particles – decreased in the last decade (Fig. 2.4). Little information, however, is available on the trend in the number of particles and the chemical composition of PM in exhaust emissions. In addition to exhaust emissions, road traffic also emits PM from tyre wear, brake wear, road abrasion and resuspension

Fig. 2.4. Total traffic emissions of PM in the countries belonging to the EU before May 2004, 1981–2020 (projected)



Source: Transport and Environment Database System Project (2003).

of road dust. Fig. 2.4 does not consider these, although in view of the increasing number of vehicles, their contribution is expected to increase. Neither does it consider the contribution of exhaust emissions by road traffic to the formation of secondary particles: mainly nitrogen oxides, but also sulfur dioxide and ammonia.

The traffic emissions shown in Fig. 2.1–2.4 are based on dynamometer tests that use standardized driving cycles. These tests result in emission factors – the amount of a pollutant emitted per kilometre driven by a certain type of vehicle at a certain speed (discussed in Chapter 1) – that relate only to exhaust pipe emissions and exclude others. In addition, lack of maintenance and the ageing of vehicles, and actual driving behaviour (for example, in congested traffic) influence emission factors. Fig. 2.1–2.4 therefore present a trend that should be regarded with some caution. In general, for dispersion modelling of traffic pollution, emission factors are modified in accordance with the results of monitoring road-traffic pollution in road tunnels or along motorways.

Across the 31 countries that submitted data to the EEA in 2000, the greatest contributors to emissions of primary PM10[6] and gases leading to the formation of secondary PM10 were the energy-production (30%), road-transport (22%), industrial (17%) and agricultural (12%) sectors (EEA, 2003). The sectoral breakdown of emissions is similar for the countries belonging to the EU before May 2004. Primary particulate emissions, however, are of lower relative significance, because secondary particles formed from nitrogen oxides and sulfur (both emitted mainly from combustion sources) and ammonia (emitted mainly by agricultural activities) are the greatest contributors to the formation of PM. Total PM emissions in the 25 Member States in the EU fell by 35% during the period 1990–2000; those across the four countries of the European Free Trade Association (EFTA – Liechtenstein, Iceland, Norway and Switzerland) decreased by 7%. Neither EU nor international legislation has targets or ceilings for primary PM emissions.

Contribution of traffic emissions to ozone and nitrogen oxides

Ozone and most of nitrogen dioxide are not primary emissions of road traffic. Ozone is formed in the lower atmosphere by complex reactions that involve VOCs, carbon monoxide and nitrogen oxides in the presence of sunlight. This may result in episodes of summer smog with high concentrations of ozone, such as occurred in the summer of 2003 over large areas of Europe. Ozone can be transported over long distances and is regarded as a Region-wide air pollution problem. Typically downwind of large urban conglomerates (such as Milan,

[6] The data collected do not include emissions from tyre wear, friction processes and resuspension of road dust.

Athens and the Ruhr area), plumes of high concentrations of ozone are encountered in the summer. High concentrations of ground-level ozone lead to increases in the frequency of respiratory symptoms and in deaths (WHO Regional Office for Europe, 2003).

In general, cold weather in the winter results in higher levels of pollutants in ambient air, owing to reduced atmospheric dispersion. Although industrial and traffic-related emissions are relatively constant over the course of a year, the additional input of pollutants such as carbon monoxide and nitrogen oxides from domestic heating sources may also contribute to elevated concentrations. In contrast, ozone levels are normally higher in warmer weather, due to the enhanced photochemical formation of ozone, especially during smog events typical of warm, stable atmospheric conditions.

Nitrogen oxides are mainly emitted as nitric oxide: usually 90–95% of nitrogen oxides. Freshly emitted nitric oxide reacts rapidly with ozone to form nitrogen dioxide. The sum of the concentrations of ozone and nitrogen dioxide is called oxidant and is expressed in $\mu g/m^3$ ozone equivalents. The oxidant concentration is conserved in the reaction of nitric oxide, ozone and nitrogen dioxide. Subsequently, at much greater distances from the sources, nitrogen dioxide acts as a precursor for the formation of ozone as well as for secondary particles – that is, ammonium nitrate. In view of these reactions, nitric oxide is a better marker for traffic air pollution near roads, though nitrogen dioxide is a more relevant marker for adverse health effects.

Composition of primary traffic-related PM emissions

PM emissions from road traffic come from exhaust pipes, tyre wear, brake linings and resuspension of road dust. The physical and chemical characteristics of PM emitted from each of those sources differ substantially. Also, emission factors for these emissions vary substantially between locations and over time. For example, the wear of the road surface is much greater when special tyres are used to drive on ice and ground frost. Resuspension of road dust depends on several factors, such as road surface, humidity, intensity of traffic and wind speed. Traffic-generated turbulence – and thus the amount of resuspension – increases with the speed and weight of the vehicle. The use of sand as a friction-inducing material on slippery roads, as well as the cleaning regime, is also important for the amount of resuspension. Box 2.1 presents background information on the physical and chemical aspects of PM.

Coarse particles (PM2.5–10) settle more quickly after formation or emission than do fine particles (PM2.5). The highest concentrations of coarse particles are generally found in street canyons. Few data, however, are available on emissions or concentrations. Primary road-traffic emissions of PM may be characterized by different parameters.

Box 2.1. Physical and chemical aspects of PM in ambient air

Sources

PM in ambient air comes partly from natural sources, such as wind-borne soil, sea spray and emissions of biogenic, organic compounds. Anthropogenic sources include not only fossil-fuel combustion, but also mining, agriculture and industry. PM is emitted directly (primary emissions) or formed in the atmosphere by conversion of gaseous precursors – that is, nitrogen oxides, sulfur dioxide, VOCs and ammonia – into secondary particles. PM may be transported in the atmosphere over hundreds to thousands of kilometres, depending on the particle size.

Physical and chemical characteristics

PM in ambient air ranges in size from a few nanometres to tens of micrometres. Particles smaller than 1 µm have number concentrations in the range of 100–100 000 per cubic centimetre, while those exceeding 1 µm have concentrations less than 10 per cubic centimetre. PM of various sizes differs in number, surface and volume/mass distribution. Moreover, different sources result in different types of PM, and the chemical and physical aspects may change during transport in the atmosphere (Palmgren et al., 2003).

- First is the **mass** concentration of PM10, PM2.5 and PM1.0; particles larger than 10 µm are less likely to enter the human respiratory tract, and are not considered harmful to health.
- The ultrafine particles dominate the **number** concentration of particles in ambient air, but hardly contribute to the mass. At polluted sites, particles consist mainly of carbonaceous material – that is, elemental carbon and organic compounds.
- As to **chemical composition**, primary exhaust emissions of PM consist mainly of a mixture of elemental carbon and organic compounds, and traces of heavy metals and sulfur. Tyre wear also contains carbonaceous materials, while brake wear is rich in heavy metals. Mineral components originate from road wear and resuspension.

The EU air-quality standard for PM is expressed in PM10 and PM2.5. There is no standard for the number of particles or the chemical composition. Many monitoring programmes for air quality include measurements of so-called black smoke or soot content. The black-smoke method is traditionally used to measure the so-called blackness of PM. This simple qualitative method is based on the light reflectance of PM and provides an indication of the blackness of particles. In general, no relationship exists between the results of black-smoke measurements of PM and PM10/PM2.5 concentrations in ambient air. Measurements of soot content indicate the mass of elemental carbon in PM.

Black smoke, soot content and elemental carbon in PM are usually highly correlated. The black-smoke method has been applied for many decades. In the

1950s and 1960s, it provided information on air pollution related to the combustion of coal. Starting in the 1970s, it was used as an indicator of diesel emissions from road traffic. In view of the long availability of the data, the simplicity of the method and the possible adverse effects on health of so-called black particles, the continued use of black-smoke measurements is recommended. They could also be used to monitor the effect on air quality of black-particle emissions from stationary combustion sources that use fossil fuels. Black smoke is relevant for road traffic with diesel engines, non-road transport by ships and trains (with diesel locomotives) and power generation by oil and coal. Box 2.2 gives background information on the PAH content of PM, including benzo[a]pyrene.

Improving air quality by decreasing traffic emissions

Decreasing traffic emissions have resulted in improved air quality. AirBase (ETC/ATC, 2003) – the EC air-quality database maintained by EEA – reports data from a large number of monitoring stations across the Region, including rural, urban, street and industrial locations. Since the revision of the exchange of information decision in 1997 (EU Council of Ministers, 1997), the availability of air-quality data all over Europe, particularly in the EU, has improved considerably. There are numerous time-series for nitrogen dioxide, sulfur dioxide, carbon monoxide and ozone. Since 1997, the number of stations that report on PM10 has increased substantially. EEA (2004) has shown that annual average nitrogen dioxide concentrations at all monitoring stations tended to decrease between 1990 and 2000. Carbon monoxide concentrations also showed a decreasing trend, which illustrates the significant benefit of reducing traffic emissions to air quality in general. The situation for ozone, however, is less clear: the annual average values for ozone seemed to increase in Europe over the period 1995–2000. The probable causes are the non-linear chemistry by which ozone is formed, the increasing hemispheric background concentration and less scavenging of ozone by nitric oxide emissions.

Fig. 2.5 shows the recent trends in pollutant levels at urban background locations in a selection of cities across Europe. For PM10, only data for 1995 and 2000 were available. These locations are the best sites for monitoring the impact of decreasing traffic emissions. Fig. 2.5 shows decreasing concentrations of nitrogen dioxide and carbon monoxide, for which traffic is the main contributor at urban background locations. For ozone the situation is more complicated. At all monitoring stations, the ozone concentrations seemed to increase from 1990. Fig. 2.5 also shows that the oxidant concentrations (expressed as $\mu g/m^3$ ozone equivalents) remained relatively constant in 1990, 1995 and 2000. Hence, decreasing nitrogen dioxide concentrations are directly related to increasing ozone concentrations (see the section on ozone and nitrogen dioxide in urban areas below).

Box 2.2. PAHs and benzo[a]pyrene

PAHs are a group of organic chemical compounds that contain two or more aromatic benzene rings fused together. More than 500 PAHs have been detected in the air: the lighter PAHs are found in the gas phase, while PAHs with five or more rings, such as benzo[a]pyrene, are usually adsorbed onto fine PM. In the past, the major urban source of PAHs in Europe was the domestic burning of coal. Today, vehicle emissions are the dominant source in most urban areas, though wood combustion may be important in some areas, such as Scandinavia and eastern Europe.

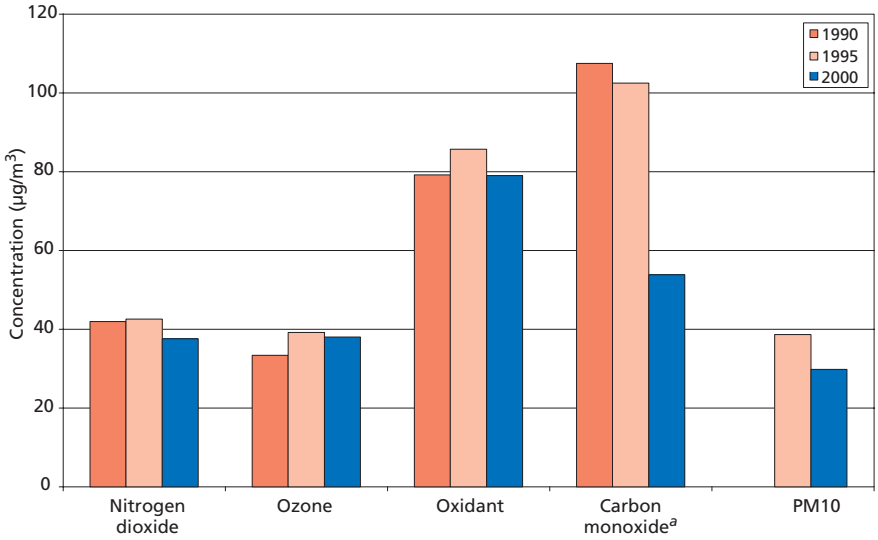
In general, roadside PAH concentrations are higher than urban background concentrations, which in turn are higher than rural concentrations. Some PAHs are carcinogenic, and benzo[a]pyrene is often used as a marker of the carcinogenic potency of ambient total PAHs. The particulate fraction of PAHs is usually of greatest concern, since it contains the majority of the carcinogenic compounds and can be transported over long distances. Though routine measurement of PAHs has been scarce in the past, PAH concentrations appear to be an order of magnitude lower in urban areas today than they were when coal burning was commonplace (EC, 2000).

The economic evaluation of air quality targets for PAHs, initiated by the EC Directorate-General for the Environment (2001), rated diesel and petrol road-transport emissions as major and moderate sources of PAHs, respectively. Their contributions to background levels in European countries have been estimated to be in the range of 1.8–39.0% and 0.0–0.5%, respectively. These estimates, however, entail considerable uncertainty and do not represent the elevated contribution of traffic in hot spots (street canyons). Nevertheless, urban and urban background levels have been shown to be consistently higher than levels in suburban and rural locations (DG Environment, 2001; Smith & Harrison, 1998).

In summary, traffic is a big contributor to urban levels of PAHs, and concentrations can be especially high close to busy roads (EC, 2001). For example, roadside levels of benzo[a]pyrene in hot spots are in general three times higher than background levels when emissions from other sources are minimal (DG Environment, 2001). In regions with high emissions (such as western Germany and Poland), the mean annual concentrations of benzo[a]pyrene can exceed 3 ng/m^3; the proposed EU air-quality objective is 1 ng/m^3 as an annual mean. In background locations in many European countries, benzo[a]pyrene concentrations vary within the range 0.1–1.0 ng/m^3 (Shatalov et al., 2003).

This is of concern to health. Decreasing PM10 concentrations are difficult to relate directly to decreasing emissions by traffic, because many other sources contribute directly or indirectly to PM10 concentrations. Because of weather variations, the annual average concentration may fluctuate strongly from year to year. Hence, the change between annual values provides only an indication of the actual trend. The most recent data from EEA indicate that PM10 concentrations remained about the same or even slightly increased in 1999–2003.

Fig. 2.5. Annual average concentrations of nitrogen dioxide, ozone, oxidant, carbon monoxide and PM10 at urban background locations in Athens, Berlin, London, Utrecht and Prague, 1990, 1995 and 2000



[a] The scale for carbon monoxide extends to 1000, so carbon monoxide concentrations are presented in tens of µg/m³.

Source: ETC/ATC (2003).

Contribution of traffic emissions to urban air quality

To discuss the impact of decreasing traffic emissions on future urban air quality, this section discusses the projected regional and urban background air quality for 2010, using the results of the Auto-Oil II Programme (Auto-Oil II, 2000), which covered almost 50% of the urban population in western Europe. Its report concluded that, while air quality is expected to improve substantially towards 2010, limit values are likely to continue to be exceeded, especially the objectives for annual average nitrogen dioxide and 24-hour and annual average PM10.

The third assessment of Europe's environment (EEA, 2003) called air pollution among the most serious environmental problems faced by cities in eastern Europe and central Asia. Lack of monitoring data, however, precludes an in-depth assessment in these countries, and more specific data on the effects of transport emissions on air quality are not available. The remaining sections in this chapter therefore address the effect of traffic on urban air quality in western Europe.

Projected regional and urban background air quality

The objectives for air quality to be attained in 2005 and 2010 are formulated in the EU air-quality directive and its daughter directives (DG Environment, 2004).

In this respect, the directives on the national emission ceilings, on fuel quality and the further introduction of vehicle emission limits (Euro IV and Euro V), are also relevant. Table 2.1 lists the limit values for the protection of health. Because adverse effects on health are still associated with concentrations in ambient air below the limit values, these values do not comprise a no-effect protection level of air quality.

In a follow-up study of the Auto-Oil II Programme modelling calculations, the EEA (2001) computed the size of the urban population in the EU living in areas not in compliance with the air-quality objectives (Fig. 2.6), and estimated the additional emission reductions needed to reach compliance. This was performed for about 200 cities in the EU for the reference years 1995 and 2010, taking account of the Auto-Oil II Programme base scenario. The air pollutants considered were sulfur dioxide, nitrogen dioxide, PM10, lead, carbon monoxide, ozone and benzene. Both the Auto-Oil II Programme modelling effort and the EEA study address urban background air pollution. Populations living near urban highways or in street canyons with extensive traffic, however, are exposed to higher concentrations of transport-related air pollution. Thus, the Auto-Oil II Programme modelling method may underestimate the size of the population exposed to air

Table 2.1. EU air-quality objectives valid in 2004

Pollutant	Averaging period	Air-quality standard/objectives
Sulfur dioxide[a]	1 hour	350 µg/m³, not to be exceeded more than 24 times in a calendar year
Sulfur dioxide[a]	24 hours	125 µg/m³, not to be exceeded more than 3 times in a calendar year
Nitrogen dioxide[a]	1 hour	200 µg/m³, not to be exceeded more than18 times in a calendar year
Nitrogen dioxide[a]	Calendar year	40 µg/m³
PM10[a]	24 hours	50 µg/m³, not to be exceeded more than 7 times in a calendar year
PM10[a]	Calendar year	20 µg/m³
Lead[a]	Calendar year	0.5 µg/m³
Carbon monoxide[b]	8 hours	10 mg/m³
Ozone[c]	Daily 8-hour maximum	120 µg/m³, not to be exceeded more than 20 days per calendar year
Benzene[b]	Calendar year	5 µg/m³
Benzo[a]pyrene[d]	Calendar year	1 ng/m³

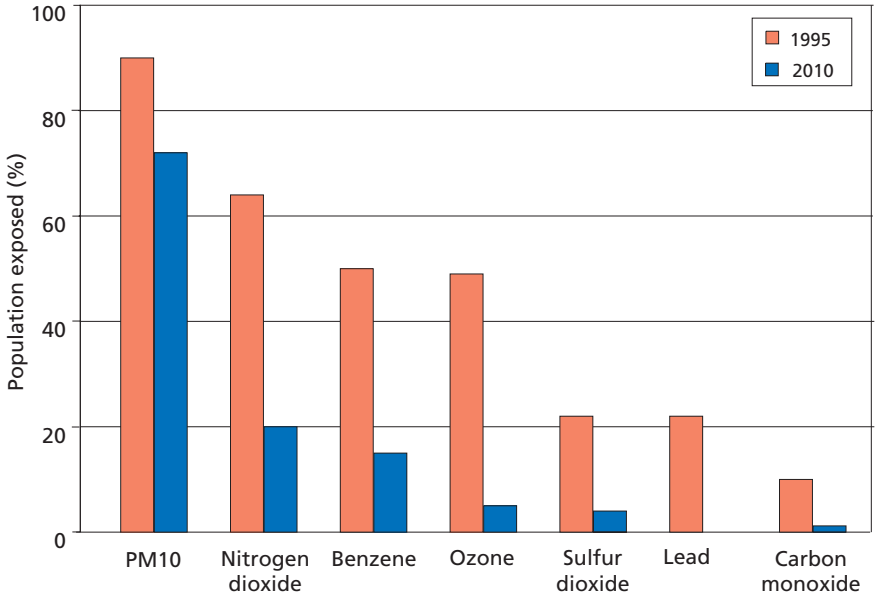
[a] Limit values for the protection of human health are from Directive 1999/30/EC on sulfur dioxide, nitrogen dioxide, PM10 and lead (EU Council of Ministers, 1999).

[b] Limit values for the protection of human health are from Directive 2000/69/EC on carbon monoxide and benzene.

[c] The target value for the protection of human health is from the proposed daughter directive on ozone (Directive 2002/3/EC).

[d] EC proposes this target value for benzo[a]pyrene.

Fig. 2.6. Percentage of urban population in Europe living in background areas exposed to pollutant levels not in compliance with EU standards, 1995 and 2010 (projected)



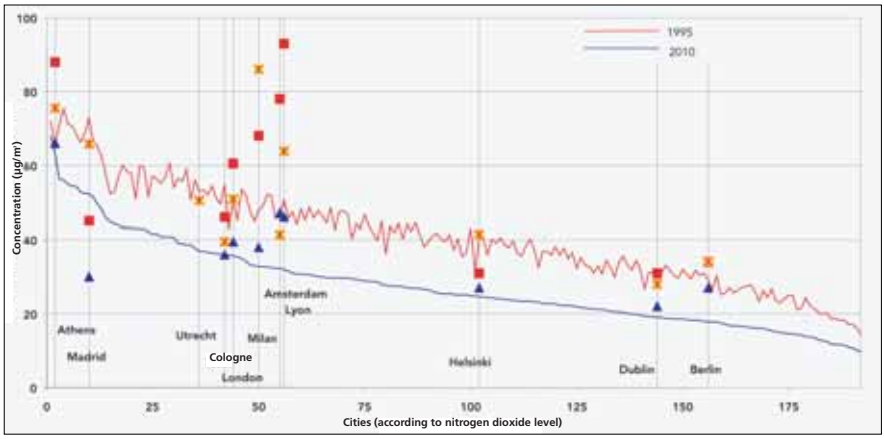
Source: EEA (2001).

quality not in compliance with EU standards and may underestimate exposure to PM emitted by traffic.

Fig. 2.6 shows the improvements expected for 2010 in urban background air quality, as a result of planned emission-reduction measures. The quality of urban air is expected to improve, in terms of both numbers of people exposed and severity of exposure. For example, in 1995 about 25 million people lived in cities where the objectives for four pollutants were exceeded simultaneously. In 2010, this number is expected to be reduced to less than 4.5 million, according to the Auto-Oil II Programme modelling results and the EEA study (2001).

The model calculations in the Auto-Oil II Programme and the results of the EEA study may be compared with measurements performed by EuroAirnet, the European-wide air-quality monitoring network coordinated by EEA (2000b) under Council Decision 97/101/EC on the exchange of information (EU Council of Ministers, 1997). EuroAirnet covers rural, industrial, urban and traffic locations, and a total of 431 million people in 29 countries. This includes 126 million people in urban populations, which is about 56% of the total population living in cities with more than 20 000 inhabitants. Data from EuroAirnet are reported to AirBase. Fig 2.7 presents measured and modelled results for the annual urban background concentrations of nitrogen dioxide in (in descending order) Athens,

Fig. 2.7. Measured and modelled annual mean urban background concentrations of nitrogen dioxide in European cities, 1995 and 2010 (projected)



Source: EEA (2001).

Madrid, Utrecht, Cologne, London, Milan, Amsterdam, Lyon, Helsinki, Dublin and Berlin (EEA, 2001). The measurements in Lyon and Milan seem to overestimate (or the model underestimates) the urban background, but in general the modelled air quality is in acceptable agreement with the measurements for the urban background. In Fig. 2.7, the upper line corresponds to the 1995 results, and the lower line, to the 2010 estimates obtained with the general empirical approach; squares and triangles correspond to the maximum concentration in the urban domain for 1995 and 2010, respectively; asterisks indicate observed annual means (1992–1996).

About 45 million of the 100 million inhabitants of the cities covered by AirBase data may have received exposures above the annual limit value of 40 µg/m^3 of nitrogen dioxide in 1999 and 2000. For PM10, a smaller population is covered: about 65 million inhabitants. Of these, about 28 million may have received exposures above the daily limit value (see Table 2.1 for limit values). For ozone the urban population coverage of AirBase was about the same as that for nitrogen dioxide, some 94 million people. Of these, about 18 million were exposed to ozone levels above the target value (WHO Regional Office for Europe, 2003).

The EEA study projected that, in 2010, less than 12% of the urban population would be exposed to air exceeding the limits for sulfur dioxide (10%), hourly nitrogen dioxide (0%), carbon monoxide (1%), ozone (6%), benzene (12%) and lead (0%). For nitrogen dioxide and PM10, however, the projections are less optimistic, as the objectives for air quality are expected to be exceeded for annual nitrogen dioxide (19%), 24-hour PM10 (71%) and annual PM10 (52%). Again, these percentages refer to the population exposed to air pollutants at urban

background locations. The levels of pollution for people living in street canyons or near urban highways may be higher. Fig. 2.8 gives a schematic view of urban air pollution near traffic hot spots and at urban background locations, on the spatial scale of an idealized city structure: that is, with low residential buildings on the outside and tall buildings in the centre.

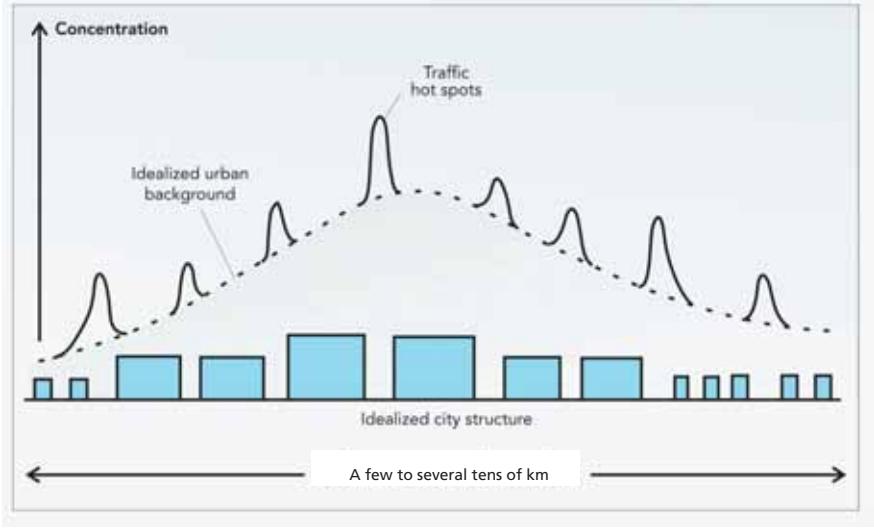
Fig. 2.8 shows that air quality may vary within a city, depending on traffic intensity, physical topography (such as the width of a street versus the height of buildings) and weather conditions. The next section focuses on traffic-related hot spots, where people are exposed to higher concentrations, and emphasizes traffic-related PM and nitrogen dioxide. Box 2.3 gives background information on hot spots not related to road transport.

Contribution of traffic to PM in urban areas

EEA (2000a) reported that transport dominated PM10 emissions in 1990. An update of the overall inventory of PM emissions in 1999 suggests that road and other transport accounted for 39% of PM10 emissions in the EU (EEA, 2002a). Between 1990 and 1999, advances in the reduction of PM from transport contributed to the overall reduction of PM10 emissions. Traffic-related PM, however, still represents a large source of PM10, especially in urban areas with large volumes of road traffic.

Source apportionment techniques have also been used to track components of air pollution to their possible sources (Pio et al., 1996; Harrison et al., 1997;

Fig. 2.8. City structure and pollution levels: urban background and some traffic-related hot spots



Source: EEA (2001).

Box 2.3. Air-quality hot spots from sources other than road transport

Locations with locally increased air pollution are called hot spots. The causes may be the limited dispersion of pollutants (as in a street canyon) or high local emissions (for example, near a highway). Hot spots that are not related to road transport include railway stations, airports and harbours. In addition to heavy road traffic near these facilities, diesel locomotives, airplanes and ships may emit considerable amounts of air pollutants in a short time, and emissions may be confined to the area. Few studies have examined the adverse effects on health of pollution near such hot spots.

Railway stations

Measurements of the air quality in and around railway stations and of the apportionment of sources have been published for Gare de l'Est in Paris (100 000 travellers on an average working day) – a dense urban area (AIRPARIF, 2001, 2003). On average, within a 1000-m radius of the station, the diesel locomotives (80 movements per day) emit about 16% of total nitrogen oxides and 9% of primary PM in the area; the rest is due to heavy road traffic. During peak operating periods (that is, three locomotives operating simultaneously), however, they contribute about 50% of nitrogen oxides and 33% of PM. Their strongly enriched pollutant plumes can lead to peak concentrations of nitrogen oxides of 750–1200 μg/m³ at 200–400 m from the rail tracks (that is, at the surrounding housing), depending on wind direction and speed. These peak values decline after train departures, but they remain recognizable (at above 25% of the total concentration) for up to 9 minutes. Modifications of the operation and remotorization could substantially reduce these emissions.

Coastal areas and ports

Ship emissions contribute an estimated 10–20% to the overall PM10 concentration for most coastal areas in western Europe (Fagerli & Tarrasson, 2001). Ports are potential hot spots when their traffic volume is high. For instance, the world's largest port by mass turnover, Rotterdam (about 320 million tonnes in 2001), has about 30 000 sea-going and 133 000 inland vessels calling annually. The measured annual PM10 concentrations are elevated (36–46 μg/m³) in the total Rijnmond area, including the city of Rotterdam. Dust from the turnover of ore and coal appears to be the predominant source, while about 5–10 μg/m³ has been attributed to emissions from ships (TNO, 2003). Besides their contribution to PM10, ships emit significant amounts of soot. With soot comprising 40–80% of the primary PM mass (Mangelsdorf, 1999), emissions from ships might give rise to elevated ambient soot concentrations.

Airports

Europe's 10 largest airports, all in western Europe, have about 770–1400 take-offs and landings daily, with an average of about 60 000–175 000 passengers (EC, 2003). This traffic volume, which is comparable to that of a medium-sized city, entails high and

canalized traffic flows on the ground for passengers, staff, supplies and services. At the four of Europe's top five airports for which data were available (London's Heathrow Airport, Frankfurt-am-Main Airport, Paris' Charles de Gaulle International Airport and Amsterdam's Schiphol Airport), the current annual average EU limit value of 40 µg/m³ nitrogen dioxide was exceeded in 2001/2002 by up to 13 µg/m³. These concentrations, however, do not seem directly attributable to aircraft emissions. For Schiphol Airport, the contribution from aircraft emissions to local air quality within a 10-km radius was estimated to be below 10% for nitrogen dioxide and PM10, with road traffic providing the dominant contribution (Franssen et al., 2002); similar estimates are reported for airports in the United States (GAO, 2003).

Pinto et al., 1998; Hosiokangas et al., 1999; Querol et al., 2001; Manoli et al., 2002; Nava et al., 2002; Vallius et al., 2003). Overall, these studies suggest that primary traffic emissions can contribute about a quarter to a half of the fine PM mass in a typical urban area. As mentioned, traffic also contributes to secondary PM, from sulfates and nitrates formed in the atmosphere and resuspended road dust, which is associated mainly with PM10. Owing to limitations and uncertainties in characterizing source profiles and in clearly interpreting the results of the statistical methods, however, these observations should be regarded as a rough indication of possible sources of PM.

A good deal of research in the past few years has investigated the relationship between elemental carbon, PM mass and the soot fraction. Near traffic locations, PM10, PM2.5 and PM1.0 are highly correlated with black smoke and with elemental carbon (Roemer & van Wijnen, 2001; Cyrys et al., 2003). Traffic contributes a large part of the elemental and organic carbon content of PM in urban areas and in close proximity to major roadways, especially in the smaller fractions (Pakkanen et al., 2000; Ruellan & Cachier, 2001; Viidanoja et al., 2002). In some cases, black smoke may be a more sensitive measure for monitoring changes in traffic-related PM (Roemer & van Wijnen, 2001). This is important because, in a variety of situations, black smoke can be linked specifically to diesel-fuelled vehicular traffic, in the absence of other major sources. Box 2.4 provides a more detailed discussion of traffic-related indicators of air quality.

A draft CAFE position paper (CAFE, 2003) and recent reports on the phenomenology of PM (Van Dingenen et al., 2004; Putaud et al., 2004) provide more insight into the differences between concentrations at rural and urban background sites and at kerb sites in street canyons. Data collected by EuroAirnet (CAFE, 2003) and by various research projects (Van Dingenen et al., 2004; Putaud et al., 2004) on PM10/PM2.5, particle number and size distributions and chemical composition were also considered.

One conclusion from these reports is that at all urban sites and thus all kerb sites, the EU indicative limit value for 2010 (annual average of 20 µg/m³ PM10)

Box 2.4. Traffic-related markers for air-quality and exposure assessment

Due to the complex mixture of air pollution from road traffic, it is impractical or impossible to measure all relevant components. Markers or proxies are sought that correlate well with target pollutants, but they are not necessarily of concern to health. The selection of a marker, such as PM or a gaseous pollutant, depends on the information requirements. In general, traffic-related markers are used for a variety of applications: air-quality management, source apportionment, exposure assessment and epidemiological studies. Chapter 4 covers the issue of indicators used to study adverse effects on health in more detail.

Nitrogen oxides and carbon monoxide are often used as markers for transport-related air pollution, and nitric oxide is a marker for combustion processes and an indicator of fresh exhaust-pipe emissions near roads. Nitrogen dioxide is formed rapidly in the atmosphere from nitric oxide. Compared with nitric oxide, the background concentrations of nitrogen dioxide are relatively high. Consequently, nitrogen dioxide is a less sensitive marker for transport-related air pollution. Improved engines and fuels are reducing the relevance of carbon monoxide as a traffic marker.

Traffic exhaust emits considerable amounts of VOCs, such as alkanes, alkenes and aromatic compounds (toluene, benzene and o-, m- and p-xylene). The ratios of the concentrations of VOCs are quite specific profiles of traffic exhaust. Benzene is a marker of transport-related air pollution in general, and a more specific indicator of emissions from two-wheeled vehicles, which are widely used in cities in southern Europe. Also, near roads, the concentration of benzo[a]pyrene – a marker for the concentration of PAHs in ambient air – is an indicator of the influence of traffic on air quality in locations where the impact of other sources is negligible.

Since lead and bromine additives were removed from petrol in Europe and North America, traffic-generated PM has been more difficult to identify. For traffic-related particles, PM2.5 is probably a better marker than PM10 (Harrison et al., 1997; Viidanoja et al., 2002). In less polluted areas in northern Europe, however, regional background sources (sulfates and nitrates) contribute a high proportion of PM2.5 and road traffic, a low proportion. In areas with road dust generated by studded tyres or sanding or both in winter, PM10 is better correlated with traffic-related PM than PM2.5. Vehicles are also an important source of ultrafine particles; at sites near roads, the concentrations of ultrafine particles are significantly higher than in background locations. These examples show that traffic-generated PM is site- and season-specific and that no general PM marker has been identified for traffic emissions.

Perhaps the most adequate marker for traffic-related PM today (in the absence of other combustion sources in urban areas) is black smoke. In spite of being a result of a semiquantitative method, its advantages include ease of analysis and low cost. Also, its long use in many countries warrants continuation of this marker. Black smoke is particularly useful for assessing the dispersion of traffic-related PM in urban areas, because small-scale burning of domestic and industrial solid fuel is becoming rare and modelling can become

quite complex. The ratio of black smoke concentration measured near traffic locations to background concentration is significantly higher than those for PM10 and PM2.5 (Pakkanen et al., 2000; Roemer & van Wijnen, 2001; Cyrys et al., 2003). Thus, black smoke is a more sensitive marker of traffic-related PM, with emphasis on diesel-fuelled traffic, than PM10 or PM2.5 (Roemer & van Wijnen, 2001).

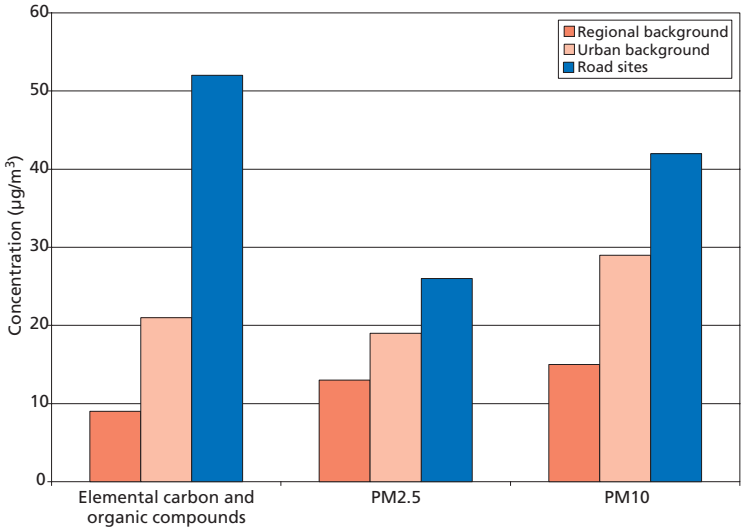
Because markers for traffic pollution are not necessarily the most health-threatening compounds and because many technologies for pollution control are quite compound specific, the regulation of traffic-related markers may bring few or no health benefits. For example, nitrogen dioxide is a traffic-related marker for which a WHO air-quality guideline value has been set (WHO Regional Office for Europe, 2000), but from a health perspective it may be less harmful than the fresh exhaust mixture for which it is a marker. Thus, controlling nitrogen dioxide concentrations does not necessarily control the associated harmful pollutants from traffic exhaust.

and the United States Environmental Protection Agency standard (annual average of 15 μg/m^3 PM2.5) are exceeded. Observations indicate that traffic is responsible for high concentrations of PM10 and PM2.5 in urban areas. The ratio of PM2.5 to PM10 varies between 0.5 and 0.8, depending on the site. No correlation exists between PM mass concentration and total particle number concentration. Fig. 2.9 illustrates the contribution of traffic to PM by showing the mass concentration of PM10 and PM2.5, and the sum of the mass concentrations of elemental carbon plus organic compounds.

One conclusion that can be drawn from Fig. 2.9 is that the ratio of urban to rural background mass concentrations for PM10, PM2.5 and black carbon is roughly about 2, while the ratios of kerb-site to urban-background mass concentrations for PM10, PM2.5 and the sum of elemental carbon and organic compounds are about 1.5, 1.5 and 2.5, respectively. Hence, both PM10 and PM2.5 are equivalent indicators of traffic emissions, while the sum of the concentrations of elemental carbon and organic compounds (and thus also black smoke) may be a more sensitive indicator of PM traffic emissions. The EuroAirnet data also indicate that the highest concentrations for PM10 and PM2.5 are encountered in southern Europe and that the ratio of PM2.5 to PM10 is different for northern, western and southern European countries.

Table 2.2 shows the average chemical composition of PM10 and PM2.5 at rural background, urban background and kerb sites in street canyons (Van Dingenen et al., 2004; Putaud et al., 2004). Table 2.2 indicates that secondary aerosol constitutes the largest fraction of PM10 and PM2.5 at all three sites. It is transported over long distances and across national boundaries. In addition, traffic emissions at kerb sites in street canyons result in a significantly higher percentage of mineral dust in the composition of PM10 – that is, resuspended road dust – and in a higher percentage of elemental carbon in the composition of both PM10 and PM2.5 than at urban background sites.

Fig. 2.9. Mass concentrations of elemental carbon and organic compounds, PM10 and PM2.5 at rural and urban background sites and kerb sites



Note. Concentrations of elemental carbon and organic compounds are multiplied by 3 to enable them to be shown on the same scale as PM.

Source: CAFE (2003).

Table 2.2. Average chemical composition of PM10 and PM2.5 at rural and urban background sites, and at kerb sites in street canyons

Component	PM10 composition (%)			PM2.5 composition (%)		
	Rural back-ground	Urban back-ground	Kerb site	Rural back-ground	Urban back-ground	Kerb site
Elemental carbon	6	5	13	8	8	17
Organic matter	16	20	22	23	22	29
Mineral dust	10	9	19	5	7	8
Secondary aerosol[a]	32	35	24	42	43	30
Sea salt	8	4	3	3	3	1
Unknown	28	27	19	19	17	15

[a] Sum of ammonium nitrate and ammonium sulfate.

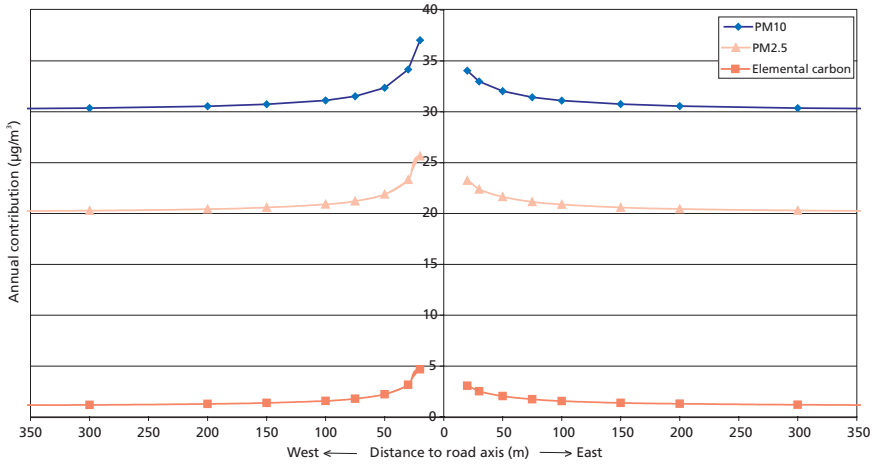
Sources: Van Dingenen et al. (2004) and Putaud et al. (2004).

Fewer data are available for ultrafine particles, but research at kerb sites and urban background locations indicates that traffic emissions significantly increase concentrations of particles of 10–100 nm. These data show that, owing to limited dispersion in street canyons, traffic at such sites may present a special urban health risk. More research is needed on the adverse effects on health associated with urban populations living in street canyons with large volumes of traffic.

Research in the Netherlands provides information on the dispersion of pollutants from traffic on an urban highway into residential areas (Buringh et al., 2002; TNO, 2002). On this highway, more than 120 000 vehicles a day (with 5–10% heavy-duty lorries) pass through a residential area. Fig. 2.10 presents the modelled air quality in urban areas adjacent to the highway for the average concentrations of PM10, PM2.5 and elemental carbon in 2001. The urban background concentrations in 2001 for PM10, PM2.5 and elemental carbon were 30 μg/m^3, 20 μg/m^3 and 1 μg/m^3, respectively. Similarly, Fig. 2.11 presents the estimated number of particles as a function of the distance to the highway. The urban background level for ultrafine particles is 15 000 per cm^3.

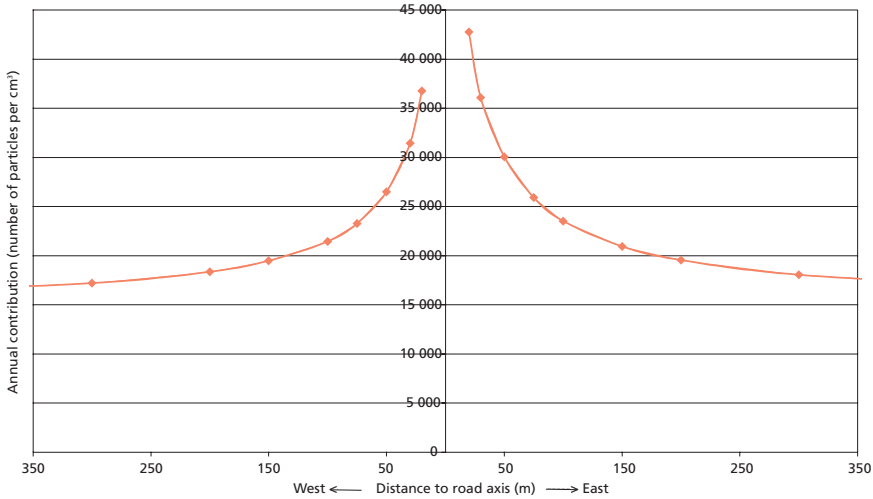
As shown, PM10, PM2.5 and elemental carbon concentrations are elevated up to 150 m from the urban highway, and ultrafine particles have significantly higher concentrations up to 250 m from the highway. These findings coincide with associated adverse effects on health experienced by residents living up to 100 m from a highway (Hoek et al., 2002). These residents are exposed to significantly higher traffic-related PM, than those in urban and rural (regional) background areas.

Fig. 2.10. Annual contribution of PM and elemental carbon from highway traffic to the air quality in Rotterdam, the Netherlands as a function of the distance from the highway



Source: TNO (2002).

Fig. 2.11. Annual contribution of ultrafine particles from highway traffic to the air quality in Rotterdam, the Netherlands as a function of the distance from the highway



Source: TNO (2002).

Concentrations of ozone and nitrogen dioxide in urban areas and streets

Fig. 2.12 shows the diurnal profile for nitrogen dioxide and ozone for traffic stations in Finland, Germany and Greece. The nitrogen dioxide concentrations show maxima during rush hours, while ozone concentrations are reduced; the concentration of oxidant (the sum of nitrogen oxides and ozone) is a good indicator of the background ozone concentration upwind from an urban area. While oxidant levels are similar in Mannheim and Helsinki, they are much higher in Athens, due to more sunlight and high emissions of ozone precursors. On average, ozone is lower in Athens than Helsinki and Mannheim; this is related to the higher levels of nitrogen oxides in Athens.

Due to the non-linearity in the chemistry, the contribution from traffic to the concentrations of nitrogen dioxide and ozone in urban areas and busy streets is variable, depending on weather conditions, background concentrations of ozone and nitrogen oxides, and emissions of nitrogen oxides. As mentioned, downwind of large urban agglomerations, traffic emissions contribute to the formation of ozone, in relation to the concentrations of other precursors, such as VOCs and carbon monoxide; in urban areas, however, traffic usually results in decreasing ozone concentrations, owing to the scavenging effect of freshly emitted nitric oxide. Further evidence for this conclusion comes from the variation of the concentration with day of the week: on weekends, ozone concentrations are higher and nitrogen dioxide concentrations lower than on working days (Fig. 2.13).

Fig. 2.12. Diurnal variation of average ozone, nitrogen dioxide and oxidant concentrations at traffic stations in Helsinki, Mannheim and Athens

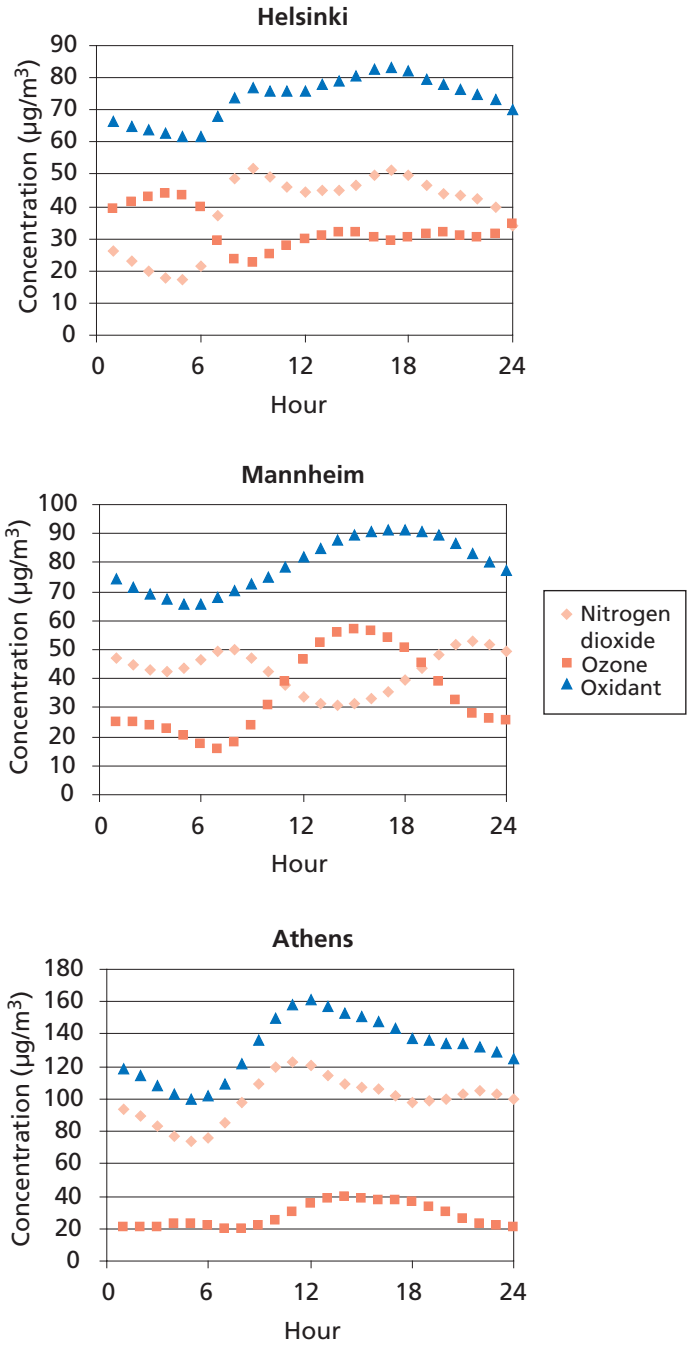
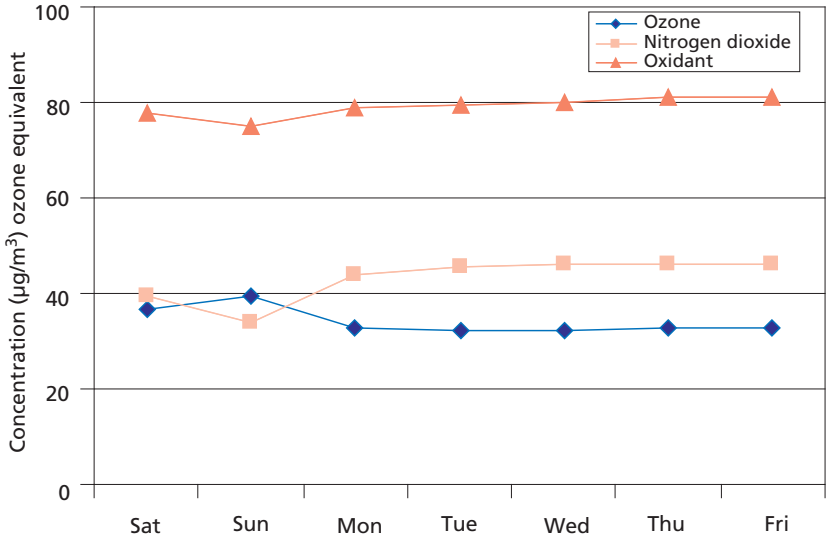


Fig. 2.13. Changes in concentrations of ozone, nitrogen dioxide and oxidant according to the day of the week, average values for 23 stations, 1990–2000



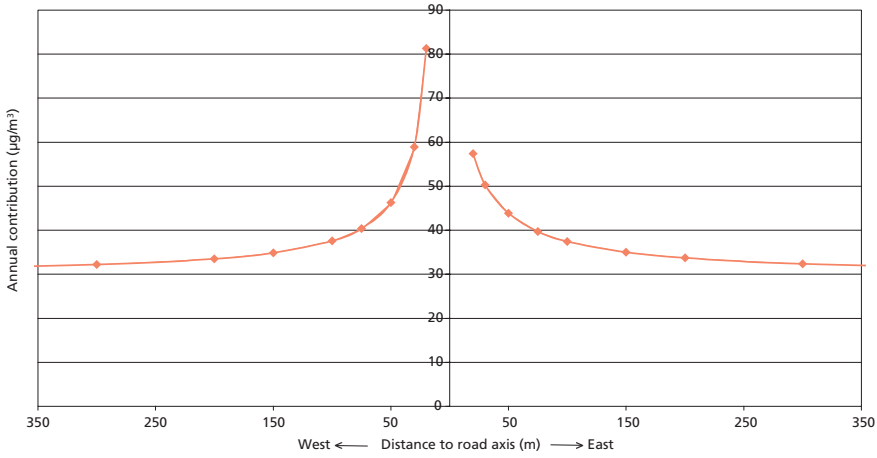
In the last 10 years, nitrogen dioxide concentrations measured at traffic stations have decreased, probably owing to reduced emissions of nitrogen oxides. The concurrent increase in average ozone concentration and a weak negative correlation between trends in ozone and nitrogen dioxide suggest that the trends in nitrogen oxides at least partly explain the trend in ozone. Probably for the same reason, trends in ozone for rural monitoring stations are in general increasing less than those for urban and traffic stations.

As with PM, the contribution of traffic to nitrogen dioxide levels in areas adjacent to a highway in Rotterdam has been measured and modelled. As an example, Fig. 2.14 shows the annual nitrogen dioxide concentration in adjacent residential areas. As with traffic-related PM, residents living up to 250 m from an urban highway are exposed to significantly higher nitrogen dioxide concentrations than those in urban and rural background locations. A comparison of Fig. 2.10 and 2.14 shows that increased concentrations of nitrogen dioxide, a secondary pollutant, extend farther from the highway (250 m) than concentrations of primary pollutants, such as PM (150 m). Hence, nitrogen dioxide is a more conservative indicator of transport-related air pollution than PM.

Effect of traffic management on urban air quality

Technological measures – such as catalytic converters, and improved fuels and engine designs – have effectively reduced exhaust emissions and the evaporation of

Fig. 2.14. Annual contribution of nitrogen dioxide from highway traffic to the air quality in Rotterdam, the Netherlands as a function of the distance from the highway



Source: TNO (2002).

fuels. In general, these measures have improved air quality, despite the increasing volume of road traffic. Owing to traffic volume and the limited dispersion of air pollution, however, limit values for PM and nitrogen dioxide are not expected to be attained in urban areas. Also, emissions of VOCs and nitrogen oxides need to be further reduced to control ozone levels. Finally, air pollution caused by traffic harms health – even at levels below the limit values – which requires further control of the effect of traffic on the urban environment.

Examples

In addition to the technical measures described in Chapter 1, measures to reduce road-traffic volume are effective for all emissions mentioned. These include economic (incentives), fiscal (taxes and road use fees), voluntary, regulatory (bans), informative (campaigns) and educational measures.

Reducing traffic volume also benefits the quality of life, by reducing the nuisance of noise, improving safety, reducing emissions of greenhouse gases and improving the landscape in inner cities. The EC Fourth Framework Programme project on concerted actions on non-technical measures and their impact on air quality and emissions (CANTIQUE) recommended non-technical measures to limit the effect of traffic on urban air quality and climate change. Little research has yet evaluated the effectiveness of these measures on urban air quality. In the United Kingdom, the imposition of congestion charges in London in 2003 resulted in lower traffic volume; the effect on air quality is still being evaluated.

The EC project on a healthier environment through the abatement of vehicle emissions and noise (HEAVEN – IST, 2004) introduced traffic management measures and demonstrated their effect on air quality.

- In Paris, separate bus lanes improved traffic circulation and thus limited road-traffic emissions. This resulted in a 2–10% improvement in air quality for nitrogen oxides, carbon monoxide and PM10. Also, car-free days improved the overall air quality by 10–16%, while the air quality of restricted zones improved by up to 30–47%.
- In Berlin, banning lorries in urban areas improved the air quality for nitrogen dioxide by as much as 50%.
- In Rotterdam, a speed limit of 80 km/h on an urban highway improved traffic dynamics and thus reduced traffic emissions for nitrogen oxides by 25%. This improved annual air concentrations of nitrogen dioxide in urban areas near the highway by 5–10%.

These examples show that traffic management is an effective instrument for urban and regional policy-makers who implement local measures.

Conclusions and the action needed

Mobility in general and road transport in particular are expected to increase throughout the European Region. Technological improvements, along with more stringent exhaust-emission legislation (for example, on exhaust aftertreatment and reformulated fuels) will, however, contribute to a further substantial reduction in emissions of air pollutants per vehicle-kilometre for passenger and freight vehicles. The greenhouse gas carbon dioxide is an important exception.

Despite the increase in road traffic, the air quality in urban areas in the EU improved in the 1990s; for traffic-related pollutants, it is projected to improve from 1995 levels by more than 50% by 2010, as shown in Fig. 2.6. Technological improvements in vehicle engines and fuels in response to EU standards are the main drivers for this development. Road traffic, however, is still the most important source of nitrogen oxides, carbon monoxide and benzene in most cities in the EU. Traffic is also a large contributor to urban PAH levels. Moreover, source apportionment studies suggest that between a quarter and a half of the fine PM mass in urban areas comes from primary traffic emissions. The relative contribution of traffic to elemental carbon is usually even larger.

Studies performed within the framework of the Auto-Oil Programme and by EEA conclude that, in urban background locations, in 2010, more than 90% of the urban population in western Europe will live in areas in compliance with the air-quality objectives for sulfur dioxide, hourly nitrogen dioxide, carbon monoxide, benzene and lead. For annual nitrogen dioxide concentrations and for both the 24-hour and annual PM10 concentrations, however, 19%, 71% and 52%,

respectively, of the urban population will still live in areas not attaining the objectives. Also, the levels of pollution will be higher, especially in street canyons and near urban highways. Additional measures are required to correct this situation through, for example:

- traffic management
- greater use of transport means other than cars and lorries
- physical planning that, among other things, reduces traffic in residential areas, optimizes travel distances and enables safe walking and cycling for everyday transport.

In contrast, ozone concentrations are lower in busy streets and in urban areas than they are downwind (or even upwind) of cities in the EU. The local depletion of ozone by reaction with freshly emitted nitric oxide tends to be more important than the formation of new ozone; hence, the local contributions from traffic are often negative – that is, reducing nitrogen oxide emissions from traffic will in general cause local ozone concentrations to increase.[7] At urban background locations, the levels of ozone indeed increased in the 1990s, while nitrogen dioxide levels decreased. From a health perspective, this trend needs further attention, because ozone has a considerable adverse affect on health.

Whether reduced emissions by road traffic will be sufficient to bring air quality into compliance with EU legislation in some urban environments heavily affected by traffic is uncertain. EU legislation on air quality requires a maximum concentration, on an annual basis, of 40 μg/m³ for both nitrogen dioxide (in 2010) and PM10 (in 2005). In view of the more intense use of space in urban environments and the growing use of cars, it is questionable whether technological improvements alone will be sufficient to achieve these standards or to provide adequate protection against adverse effects on health. Further, it is unclear whether standards for PM10 alone will be sufficient to reduce the adverse effects on health due to PM concentrations in the ambient air.

A number of conclusions can be drawn from studies of transport-related air pollution.

- Near urban highways (with more than 100 000 vehicles a day and more than 5% heavy-duty lorries), levels of nitrogen dioxide and ultrafine particles are significantly increased up to 250 m away, as compared with levels at urban background locations.
- The concentrations of PM10, PM2.5 and elemental carbon are significantly increased up to 150 m from urban highways.

[7] Nitrogen oxide emissions from traffic, however, lead to an increase in regional ozone levels and, therefore (among other things), to high background concentrations.

- In street canyons with intense traffic and limited dispersion, levels of all traffic-related pollutants (nitrogen dioxide, PM10, PM2.5, ultrafine particles, and the mineral and elemental carbon content of PM) are significantly higher than those in urban background locations.
- Downwind of urban agglomerations, ozone concentrations can be increased owing to VOC, carbon monoxide and nitrogen oxide emissions, for which road traffic is an important contributor.
- At urban background locations, the trend in ozone concentrations is increasing. This calls for further reduction in emissions of VOCs and nitrogen oxides from road traffic.

To maximize the health benefits of implementing European legislation on air quality, more research is required on the effect of PM-related traffic emissions on health. It could address a combination of PM10 (an indicator of road dust resuspended by traffic), nitrogen oxides (an indicator of traffic emissions, in general, including ultrafine particles) and elemental/black carbon (an indicator of diesel-related traffic emissions of PM).

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3. Human exposure to transport-related air pollution

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Key points

Facts

Assessing the population's exposure to transport-related air pollution requires the consideration of a range of factors. Levels of pollutants vary between cities and even over short distances; this has implications for the assessment of exposure. Where populations and individuals spend their time living and working is also important. The pollutants of concern to health include nitrogen dioxide, carbon monoxide, PM10, PM2.5, black smoke, benzene, PAHs and metals (such as lead). Among the factors that determine the level of population exposure are:

- *emissions, which depend on volume and composition of passing traffic;*
- *the distance between roads and housing and workplaces;*
- *dispersion conditions, which are influenced mainly by orography (the average height of land over a certain domain), local circumstances (such as street canyons versus open areas) and weather conditions; and*
- *time spent in different microenvironments, particularly in different traffic modes, such as mopeds, cars and public transport.*

Some groups may receive much higher exposures than others, such as people who live and work near busy roads and those who travel or commute in heavy traffic. Also, the intake of pollutants by road users varies between drivers, bicyclists, and pedestrians. Further, exposure to transport-related air pollution is difficult to separate from exposure to total air pollution.

Trends

Trends in exposure are difficult to establish, because programmes to measure repeated exposure do not exist. As a result, population-level estimates do not adequately reflect extreme individual exposures. Owing to trends in urban emissions, however, traffic may conceivably be responsible

for exposing an increasing proportion of the population to air pollution, even though air-pollution levels have decreased.

Introduction

Despite their obvious importance to sustaining health, remarkably few studies specifically assess the general public's exposure to transport-related air pollution. Instead, most evidence on exposure is a by-product of epidemiological studies investigating associations with health or of baseline studies carried out for environmental impact assessments. Neither approach is an ideal basis for estimating exposures at the population level, since the geographical areas considered may not represent the wider population. For example, estimates of personal exposure to transport-related air pollution often come from measurements collected at fixed ambient monitoring stations. Because most people are mobile, these values do not reflect the distribution of a population's exposure.

Using the WHO environmental health criteria for assessing human exposure as a guideline, exposure is defined as "contact over time and space between a person and one or more biological, chemical and physical agents" (MacIntosh, 2000). As to transport-related air pollution, the most important aspects of this exposure include:

- concentration, in $\mu g/m^3$ or another equally valid metric;
- duration in seconds, minutes, hours, days, weeks, months and years;
- settings, such as location of residence and workplace and transport mode (for example, commuting); and
- exposed population, whether this comprises the general population, subgroups and/or individuals.

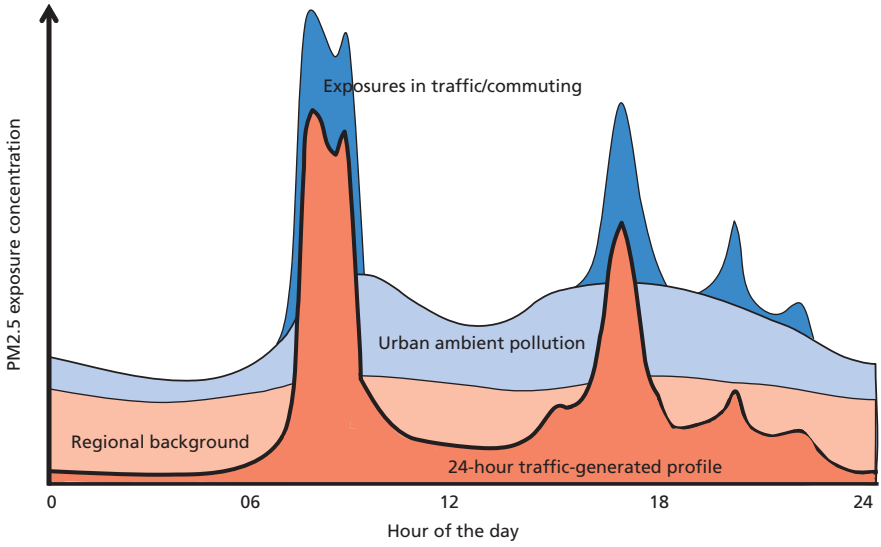
Assessments are made to identify and define the exposures that occur or are expected to occur in human populations. A description of exposure for a particular transport mode or microenvironment should include at least the exposure concentration and duration of contact.

Population exposure to traffic pollution can be classified into three general categories (Fig. 3.1):

1. the contribution of traffic emissions to rural and urban background air pollution;
2. the locally elevated levels of traffic pollution near busy roadways and city zones; and
3. the still further elevated exposure concentrations in commuting/transport.

The categories in Fig. 3.1 are superimposed on each other in this order. The exposure profile will differ according to the pollutant and population of interest.

Fig. 3.1. Relative exposure concentration of PM2.5 and the influence of traffic, according to the hour of the day



Note. The section bounded by thick lines presents a 24-hour traffic-generated exposure profile for PM2.5. The sections bounded by thin lines show additive total PM2.5 contributions from the regional background (bottom), urban ambient pollution (above) and exposures in traffic/commuting (peaks). Exposure while commuting represents about half of total exposure to PM from traffic.

In general, people who spend more time in traffic with elevated concentrations are expected to have higher exposures to transport-related air pollution than those living in rural areas with little direct exposure to traffic exhaust gases and other related pollutants. This is not always the case, however. Exposure to ozone – a secondary component formed in the atmosphere at some distance from traffic – is widespread and not very different for people living in rural and urban areas, although it depends largely on the time spent outdoors.

This chapter reviews the available evidence on exposures at the population level and outlines some of the issues that need to be considered in interpreting its relationship to human health.

Methods of assessing exposure

Exposure to transport-related air pollution is seldom measured directly and completely, because of the high costs of personal monitoring, the wide range of pollutants involved and the availability of suitable monitoring technologies for only a small number of these pollutants. Also, none of the commonly studied pollutants is wholly specific to transport.

Information on exposure to transport-related air pollution comes from studies that use a wide variety of designs. In many cases, information is gathered as part

of an epidemiological study. Rather more rarely, studies have been undertaken specifically to quantify exposures, in either the population at large or specific subgroups. One of the largest and most comprehensive of these studies was the EU-funded study of air pollution exposure distributions within adult urban populations in Europe (EXPOLIS), which involved measurements in 10 different cities across the EU in the 1990s; it provided comparative data for different countries, microenvironments and pollutants.

Both epidemiological studies and studies of exposure use a wide range of methods to assess exposure. Exposures are often measured indirectly: inferred from pollutant concentrations, in either the ambient or indoor environment. Also, in many epidemiological studies, participants are asked to estimate their own exposures, for example, by classifying the level of road traffic on or degree of nuisance from the nearest streets. More rarely, modelling techniques are used to predict exposures at a residence or in various microenvironments. By weighting these estimates, according to the time spent in each microenvironment, some measure of overall exposure can be obtained. Two approaches tend to be taken:

1. spatial analysis techniques (often with the aid of geographical information systems – GIS), to model pollution patterns on the basis of the available monitoring sites; and
2. dispersion models, to simulate the transport of pollutants from their source through the environment.

Many methods for spatial analysis or dispersion modelling have been developed, but their applicability and reliability are uncertain in many cases. Stringent validation and comparisons of methods are essential before results are widely used to assess exposure to traffic. Box 3.1 gives examples of these two methods in European epidemiological studies. In a few cases, exposures have been measured directly, by using personal monitoring devices or samplers. These give explicit measurements of exposures for identifiable individuals, but their cost, bulkiness and (for the simpler and more readily transportable samplers, at least) long averaging times often limit their use. In addition, they only provide information on exposure to air pollution from all sources. No matter what approach is ultimately used, the estimation or measurement of exposures to transport-related air pollution can be studied in a variety of ways. One way is to compare the exposures of people who live in urban areas, where transport-related air pollution is intrinsically higher, with populations in rural areas. Others compare people who live close to busy roads with those who live farther away or who live in areas with less heavy traffic.

Each of these approaches helps to show the share of exposure attributable to road traffic. More detailed studies, however, focus on exposures while people are travelling. These studies typically compare either exposures in a specific transport

Box 3.1. Exposure indicators based on measurements and models used in epidemiological studies

Traditionally, investigators in epidemiological studies have used data from the nearest air pollution monitoring stations to assess exposures. In recent years, a range of modelling techniques has been developed to provide more specific estimates of small-area or individual exposure. Two main types have been used: statistical regression models and physical dispersion models.

Statistical regression models

In the EU-funded Small Area Variations in Air quality and Health (SAVIAH) study, Briggs et al. (1997) and Pikhart et al. (2000) pioneered the use of regression-based methods to assess exposure to transport-related air pollution. They developed models by establishing relationships between nitrogen dioxide, monitored at a dense network of monitoring sites, using passive samplers) and relevant environmental variables, measured using GIS. Three main sets of variables gave good predictions of nitrogen dioxide concentrations:

- traffic volume within a 300-m radius of each site studied, weighted by distance;
- the area of intensively built-up land in the same radius; and
- the altitude of the site.

Models were validated against data from a separate set of monitoring sites.

This approach has since been used in other situations. Briggs et al. (2000), for example, showed how a regression model developed in one city could be applied – with local calibration, using just a few monitoring sites – to estimate pollution levels in others. Brauer et al. (2002, 2003) and Gehring et al. (2002) used a similar approach to model PM2.5 reflectance and nitrogen dioxide concentrations in the Netherlands, and the cities of Munich and Stockholm. Hoek et al. (2001, 2002) also used a regression model, based on urban density, to assess regional contributions of black smoke and nitrogen dioxide concentrations in the Netherlands. In all these situations, the approach provided good approximations of long-term average exposures. For estimating shorter-term exposures (for example, as part of time-series or cohort studies), however, the models are likely to be less reliable, because they do not account for varying weather conditions. In these situations, dispersion models probably give better estimates.

Pollutant dispersion models

A wide range of line-source models has been developed over recent years, though as yet few of these have been used to assess exposure in epidemiological studies. Larssen et al. (1993) adapted and applied one of the earliest, HIWAY 2, to estimate hourly carbon monoxide concentrations, and the EPISODE model (Walker et al., 1999) was used in a number of studies to estimate nitrogen dioxide and fine PM concentrations (Bartonova et al., 1999; Clench-Aas et al., 1999, 2000).

Box 3.1. (contd)

Most models combine data on emissions from traffic with data on weather conditions (such as wind speed, wind direction and mixing height) to simulate dispersion processes. The accuracy of the models tends to vary, depending on the pollutants and types of dispersion environments, so the use of different combinations of models is often advisable. Thus, in Sweden, Pershagen et al. (1995) combined the HIWAY 2 model with a more recent dispersion model, CALINE 4 (Benson, 1992), to estimate time-weighted nitrogen dioxide concentrations at the residences of children. In Denmark, Raaschou-Nielsen et al. (2001) coupled a photochemical model with the Danish Operational Street Pollution Model (OSPM), developed by Hertel & Berkowitz (1989), to estimate nitrogen oxide and nitrogen dioxide concentrations. In France, OSPM was linked with the CALINE model to derive an air pollution index (ExTra) as part of a case–control study on transport-related air pollution (Zmirou et al., 2002; Reungoat et al., 2003).

mode with the background ambient concentration, or assess and compare exposures in different modes: for example, while following the same or equivalent routes. The studies that use different transport modes are especially helpful in ranking them according to their exposures and possible risks to health. Nevertheless, an understanding is needed of what contribution transport environments make to total exposures. In this case, studies need to assess the exposures of individuals or population groups as they go about their lives, and identify what part of overall exposure is attributable to time spent in transport. Different designs thus help to address different questions. The following sections report evidence from these various study designs.

Overall, the wide range of methods used in different studies makes direct comparisons difficult. For this reason, considerable uncertainty remains about population-level exposures to transport-related air pollution. Several issues need to be considered when selecting a transport-related marker for air quality and exposure assessment (discussed in Chapter 2). A marker is often chosen because of the relative ease of measurement using low-cost techniques, and in part because it is largely derived from transport sources. Many of these markers have not been fully studied, and most environmental concentrations and exposures in the general population to transport sources remain poorly quantified at best. Also, the extent to which these pollutants are specific to transport sources varies considerably from one area to another, depending on the existence and characteristics of other sources, such as industrial activity.

Exposures in urban versus rural regions

In reality, comparisons of urban and rural regions can be seen as assessing the range of exposures within the general population. Also, exposures to transport-related air pollution are nested within this range/variability: a product of a complex mix

of contributions from different local pollution sources and people's behaviour. Several studies show that air pollution from traffic is higher in urban areas than in rural or non-urban areas. These studies calculated exposures using data from personal and microenvironmental monitoring, often based on passive samplers (Linaker et al., 1996; Raaschou-Nielsen et al., 1996), dispersion modelling (Oosterlee et al., 1996) and GIS-based methods (Jensen et al., 2001; Kousa et al., 2002). Studies related specifically to carbon monoxide, VOCs, PM and metals from traffic sources, however, are uncommon. Moreover, differences in the classification of sampling stations used by various monitoring networks or of personal measurements of selected individuals can underestimate the range of exposure between urban and rural locations.

Chapter 2 discusses the differences between urban and rural sites in the concentration of various pollutants and the possible contribution from traffic in detail. In general, most information on transport-related air pollution and estimates of population exposure for urban-versus-rural (or urban background) areas are for nitrogen dioxide. The multicentre study of acute pollution effects on asthmatic children (PEACE) that was carried out simultaneously in 14 European countries in the winter of 1993/1994, however, provides information on some other pollutants. The study summarized the city/background ratios for all 14 countries: 1.2, 1.4 and 1.8 for PM10, black smoke and nitrogen dioxide, respectively (Hoek et al., 1997). Moreover, a multimode assessment of transport users' exposure PM2.5 in London showed that mean personal exposure levels in road transport modes were about double the concentrations measured at an urban background site (Adams et al., 2001).

In a study in Huddersfield, United Kingdom, Collins (1998) compared estimates of population exposure to nitrogen dioxide, based on dispersion modelling, a regression-based method and two GIS-based interpolation techniques. Estimated mean annual exposures varied relatively little (26.9–31.6 μg/m^3), with 75th-percentile values showing slightly greater variability (28.5–34.8 μg/m^3), while the share of the population estimated as being exposed to concentrations above the guideline (37 μg/m^3) ranged from 2.9% to 8.0%. Unpublished work by Briggs and colleagues, comparing results from dispersion modelling and a regression-based model gave mean concentrations (± standard deviation) of 27.0 (±13.9) μg/m^3 and 50.6 (±5.6) μg/m^3 in London (for n = 11 395 home locations) and 16.7 (±11.9) μg/m^3 and 44.2 (±9.6) μg/m^3 in Sheffield (for n = 10 383 homes), respectively. Regional background concentrations probably contribute an additional 10–15 μg/m^3 to the estimates from the dispersion model, implying mean exposures of about 40 μg/m^3 and 30 μg/m^3 in London and Sheffield, respectively. The differences in these estimates emphasize the need for care in comparing exposure data from different areas and studies.

Gauvin et al. (2001) reported personal exposures to nitrogen dioxide for 79 children from three cities in France. Average exposures were 25 ± 11 μg/m^3

in Toulouse, 30.7 ± 7.7 μg/m^3 in Grenoble and 32.3 ± 9.9 μg/m^3 in Paris. Studies in Munich reported average monitored nitrogen dioxide concentrations of 65.8 μg/m^3 at 18 specifically selected high-concentration urban sites, compared with 32.2 μg/m^3 for 16 school sites in areas with moderate concentrations (Carr et al., 2002).

Separating the contribution from transport in most of these situations is difficult, though some indication of the influence of local traffic sources can be gleaned by comparing nitrogen dioxide exposures in locations with more or less heavy traffic, or by considering distance from major roads. In the SAPALDIA study, for example, nitrogen dioxide exposures were measured for about 50 people in each of 8 regions in Switzerland; passive samplers were deployed for 4 weeks on 3 occasions during 1992/1993 (Monn et al., 1998). Mean exposures ranged from about 13 ± 3 μg/m^3 in the rural and mountain regions of Montana to about 42 ± 11 μg/m^3 for the city of Geneva. The difference can be largely attributed to urban road-traffic sources.

A study in the Netherlands (Rijnders et al., 2001) measured the personal exposure of children to nitrogen dioxide in three schools in a major city (highly urbanized: about 4000 addresses per km^2), a smaller city (moderately urbanized: about 1500 addresses per km^2) and a village (not urbanized: about 200 addresses per km^2). Averaged over the year, the personal exposures were 30.2 μg/m^3 for the children from the city, 19.9 μg/m^3 for those from the smaller city and 16 μg/m^3 for those in the village. For the school near a busy road, the average exposure was 26.4 μg/m^3, compared with 22.2 μg/m^3 and 20.0 μg/m^3, respectively, for the schools adjacent to a fairly busy road and a quiet road. In addition, the nitrogen dioxide concentrations at the rear façade of the children's homes were measured; the average concentrations showed similar differences: those of the city and small city were 11 μg/m^3 and 4 μg/m^3, respectively, above that of the village. In a study in Helsinki, Finland, personal nitrogen dioxide exposures of children in daycare centres were about 9 μg/m^3 higher in downtown than in suburban areas (Alm et al., 1998). In Germany, Krämer et al. (2000) estimated annual personal and outdoor nitrogen dioxide concentrations; these were5–7 μg/m^3 and 12–17 μg/m^3 higher, respectively, in two urban areas than in a suburban area.

Much of the variation in exposure in urban areas certainly occurs in the near-field zone, close to major roads. Using regression techniques, for example, Briggs et al. (1997, 2000) found that traffic within 40 m of the measurement site accounted for much of the variation in monitored nitrogen dioxide concentration. Gilbert et al. (2003) deployed passive samplers along a 1300-m transect perpendicular to a major highway, with traffic volumes of over 100 000 vehicles a day. Concentrations of nitrogen dioxide ranged from 22.6 μg/m^3 to 55.7 μg/m^3, declining logarithmically with distance. Concentrations were also significantly lower upwind than downwind of the highway.

Further, variations in exposure over time that result from changes in meteorological conditions and impacts of other local sources of air pollution need to be recognized. This is equally valid for the entire range of exposure categories. For example, with respect to transport-related emissions, marked variations in modelled nitrogen dioxide exposures were found in Helsinki at different times of the day, in response to both varying traffic activity and population distribution (Kousa et al., 2002). Klaeboe et al. (2000) used dispersion modelling to compare exposure distributions in Oslo, Norway for about 1000 people taking part in environmental studies in three different years: 1987, 1994 and 1996. A general reduction in exposures was observed over time, with median exposures falling from about 50 μg/m^3 in 1987 to about 36 μg/m^3 in 1996, perhaps largely because of improved vehicle design and emission controls. Conversely, modelling using the Airviro dispersion model in Stockholm, Sweden gave population-weighted average exposures of about 24 μg/m^3 for transport-related nitrogen dioxide in 1990, compared with about 15 μg/m^3 in 1955 (Bellander et al., 2001). The growth in exposures was a result of increased traffic flows, which more than offset reductions in unit emission rates.

Exposure of people living near busy traffic routes

Since vehicle emissions by definition take place on roads, people who live close to busy roads might be expected to be exposed to higher concentrations of transport-related air pollution and at greater risk of adverse effects on health. A considerable body of research already supports these assumptions. For example, several studies have demonstrated higher rates of respiratory illness and symptoms, and reduced lung function in people living near major roadways (such as: Brunekreef, 1997; English et al., 1999; Hoek et al., 2002; Livingstone et al., 1996; Venn et al., 2001). Nevertheless, patterns of exposure are often more complex than anticipated, and vary substantially, depending on the pollutant concerned and the lifestyle and behaviour of the particular population group.

Primary and secondary pollutants differ in their patterns of exposure near roads. In general, exposures to primary transport-related pollutants, such as carbon monoxide, benzene and elemental carbon, show consistently raised levels in people who live close to major roads. Raised exposures may also be attributed to materials derived from road dust and wear on vehicle parts. For secondary pollutants, such as ozone and some PM fractions, however, this relatively simple association often fails to hold true. Instead, the highest exposures may occur at a considerable distance from roadways. Concentrations of nitrogen dioxide may also show quite complicated geographical and temporal patterns, owing to the constant interaction between nitrogen oxides emitted by vehicles and ozone in the atmosphere, though exposures from sources near roads are often higher. Studies therefore typically show greater contrasts in exposures to carbon monoxide, soot,

benzene and nitrogen dioxide between near-road and more distant locations than for particle mass concentrations or other secondary pollutants (Roorda-Knape et al., 1998; Roemer & van Wijnen, 2001; Janssen et al., 2001). Marked differences in exposure due to traffic volume on nearby roads also occur, though again patterns may vary between pollutants, depending on such factors as traffic composition (for example, the balance between petrol- and diesel-fuelled vehicles) and speed.

Unlike the exposure levels of people living close to roadways, those of people living further afield are rarely assessed directly. Usually they are inferred from measurements of either ambient or indoor concentrations at the place of residence. Most primary pollutants typically show steep gradients with distance from roads. In general, the highest exposures are found within the first 50–100 m from roadways, and exposures often fall to background levels by 300 m or more. Inevitably, the extent of raised exposures varies, depending on local topography and the configuration of buildings. Exposures are greater where pollutants are trapped in street canyons or deep valleys. Indoor concentrations also typically show weaker associations with distance and traffic volume, because the entry of pollutants is often restricted, while indoor sources may distort any relationship to distance from roads.

Table 3.1 summarizes the results from a number of recent studies on a variety of primary pollutants. It shows that exposures or concentrations for most pollutants are 2–3 times higher near busy roads than in background environments. While the ratios are remarkably constant across these studies, absolute concentrations vary, reflecting the specific circumstance of traffic intensity and emission characteristics. Weather conditions may also be important. In particular, wind direction affects the distribution of exposures on either side of a roadway. When winds are blowing from the road, exposures downwind tend to be much higher (Hitchins et al., 2000; Janssen et al., 2001). In a study near busy motorways in Zuid-Holland province, in the Netherlands, for example, nitrogen dioxide and black smoke varied by factors 1.5 and 2, respectively, with distance to the motorway. The gradient with respect to the distance to the road was higher, however, during periods when the wind blew for at least 33% of the time from the road towards the measuring site (Roorda-Knape et al., 1998).

In the case of PM, different measures of the spatial variation of concentrations show marked, contrasting differences. Concentrations of fine dust (PM10, PM2.5, PM1.0) in ambient air tend to be only marginally higher near busy roads; measurements of black smoke, elemental carbon and absorbance show more marked contrasts (Bloemen et al., 1993; Janssen et al., 1997; Fischer et al., 2000; Roemer & van Wijnen, 2001). Compared with PM mass concentrations, the higher spatial variability of black smoke or elemental carbon seems to be a common phenomenon and is noted in studies in France, Germany, Switzerland, Sweden and the United States (Pfeffer, 1994; Zagury et al., 2000; Kinney et al., 2000; Röösli et al., 2001; Cyrys et al., 2003). Overall, exposures of people

living close to busy main roads might be expected to be about 25% higher than background exposures for the mass of PM10 or PM2.5, while the excess is likely to be at least 50% for elemental carbon or absorbance measures. For measures of particle number, the contrasts may be much greater (up to sevenfold or more in some studies), though there are few studies from which to draw data.

These differences between different particle measures have considerable significance for both epidemiology and policy. They suggest, for example, that measurements of mass may underestimate contrasts in exposure attributable to road traffic; elemental carbon or measurements of absorbance may be more informative. Similarly, coarse particles settle more quickly than fine particles, which result in road-dust concentrations declining relatively steeply with distance from the road. The highest concentrations are usually found in street canyons, especially when wet periods are followed by dry conditions. Since the coarse fraction of PM also shows low site-to-site correlations within a city or region, coarse particles may not be a good indicator of the average concentrations or exposure levels in a larger neighbourhood (Wilson & Suh, 1997). In addition, coarse particles (including road dust) do not infiltrate indoors as efficiently as fine particles, and they are more rapidly removed by deposition.

As noted, exposure differs in relation to traffic volume and location in either the nearest street or surrounding neighbourhood. Table 3.2 summarizes results from a number of studies. All else being equal, exposures appear to vary more or less in proportion to traffic volume or density. In a study in the Netherlands (Rijnders et al., 2001), for example, wintertime personal exposures to nitrogen dioxide were: 36.0 μg/m^3 for children living and going to school near a motorway with an average flow of 170 000 vehicles a day, 29.7 μg/m^3 for children near a motorway with 126 000 vehicles a day and 25.1 μg/m^3 for those near a road with 45 000 vehicles a day. Summertime exposures, however, showed far less consistent variations with traffic flow. In a study in Amsterdam, both indoor and outdoor concentrations of PM10, soot and PAHs were consistently higher on busy streets than quiet ones (Fig. 3.2). Again, there are some variations between different pollutants. In general, contrasts are greater for primary pollutants, such as carbon monoxide, benzene, PAHs and elemental carbon, which are something like twice as high in areas with heavy traffic, compared with streets with light traffic. For the mass of PM, the contrasts tend to be less; typically the ratio is of the order of 1.5.

Journey-time exposures

Although people spend relatively little time travelling, they may receive substantial exposures during these periods. Ultimately, there are two main contributing factors. First, many transport microenvironments are relatively more heavily polluted than others. Second, most journeys are made during rush hours, when the increased volume of traffic results in high ambient pollution levels. In the United

Table 3.1. Reported concentrations and exposure ratios for people living near busy streets, compared with more distant locations, in six cities

Pollutant (and measurement unit)	City	Near-road		Distant		Ratio	Source
		Distance from road (m)	Concentration	Distance from road (m)	Concentration		
Carbon monoxide (ppm)	Los Angeles	17	2.3	150	0.4	5.8	Zhu et al. (2003)
Benzo[a]pyrene (ppb)[a]	Huddersfield	< 31	0.00087	62–920	0.00454	1.0	Kingham et al. (2000)
	Huddersfield	< 31	1.98	62–920	1.15	1.1	Kingham et al. (2000)
Benzene (μg/m³)	Delft	15	2.6	305	1.9	1.4	Roorda-Knape et al. (1998)
	Overschie	15	1.8	133	1.9	1.0	Roorda-Knape et al. (1998)
PAH (μg/m³)	Huddersfield	< 31	0.108	69–920	0.221	1.0	Kingham et al. (2000)
	Huddersfield	< 20	39.0	20–160	27.4	1.4	Smallbone (1998)
	Delft	15	47.8	305	30.6	1.6	Roorda-Knape et al. (1998)
Nitrogen dioxide (μg/m³)	Overschie	15	44.8	260	32.1	1.4	Roorda-Knape et al. (1998)
	Tokyo	< 20	60	> 150	32	1.9	Nakai et al. (1995)
	Huddersfield	< 31	1.25	62–920	1.06	1.2	Kingham et al. (2000)
	Osaka	5	44.0	150	36.0	1.2	Funasaka et al. (2000)
PM10 (μg/m³)	Delft	15	32.2	305	30.6	1.1	Roorda-Knape et al. (1998)
	Overschie	15	32.1	260	32.3	1.0	Roorda-Knape et al. (1998)
PM2.5 (μg/m³)	Delft	15	20.1	305	18.5	1.1	Roorda-Knape et al. (1998)
	Overschie	15	20.8	260	19.6	1.1	Roorda-Knape et al. (1998)
	Huddersfield	< 31	17.8	62–920	19.5	1.1	Kingham et al. (2000)
PM1.0 (μg/m³)	Osaka	5	44	150	36	1.2	Funasaka et al. (2000)
Soot/elemental carbon/ black smoke (μg/m³)	Delft	15	14.9	305	7.4	2.0	Roorda-Knape et al. (1998)
	Overschie	15	12.2	260	8.7	1.4	Roorda-Knape et al. (1998)
	Osaka	5	12.3	150	8.1	1.5	Funasaka et al. (2000)
	Los Angeles	17	21.7	150	6.5	3.3	Zhu et al. (2003)

PM2.5 absorbance ($m/10^5$)	Huddersfield	< 31	1.67	62–920	1.54	1.3	Kingham et al. (2000)
Particle number ($x\ 1^{-5}/cm^3$)	Los Angeles	17	2.0	150	0.61	3.3	Zhu et al. (2002)

[a] ppb = parts per billion.

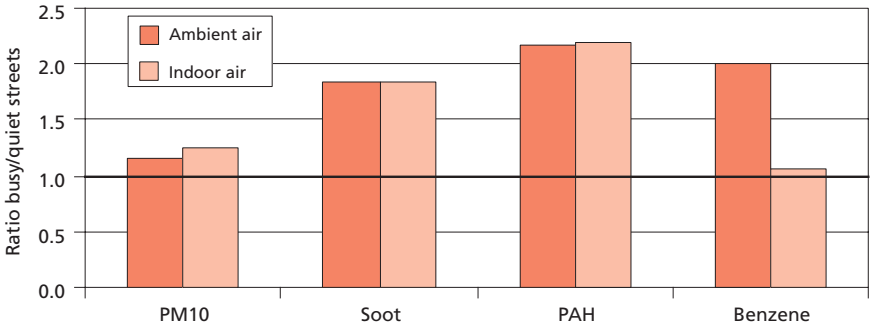
Note. The cities mentioned are located in Japan (Tokyo, Osaka), the Netherlands (Delft, Overschie), the United Kingdom (Huddersfield) and the United States (Los Angeles). Ratios are not necessarily equivalent to average near-road/average distant concentrations, because they are based on the averages of paired estimates.

Table 3.2. Reported concentrations and exposure ratios locations varying in traffic volume, covered by five studies

Pollutant (and measurement unit)	City or country	Location				Ratio	Source
		High traffic volume		Low traffic volume			
		Description	Concentration	Description	Concentration		
Benzo[a]pyrene (ng/m³)	Amsterdam	High traffic	0.67	Low traffic	0.29	2.3	Fischer et al. (2000)
Benzene (µg/m³)	Amsterdam	High traffic	5.2	Low traffic	3.1	2.0	Fischer et al. (2000)
	Hanover	High traffic	4.0	Rural	2.9	1.4	Ilgen et al. (2001)
Toluene (µg/m³)	Hanover	High traffic	22.2	Rural	24.8	0.9	Ilgen et al. (2001)
Ethylbenzene (µg/m³)	Hanover	High traffic	2.8	Rural	2.4	1.2	Ilgen et al. (2001)
Xylene (µg/m³)	Hanover	High traffic	9.7	Rural	7.7	1.3	Ilgen et al. (2001)
PAH (µg/m³)	Amsterdam	High traffic	0.33	Low traffic	0.15	2.2	Fischer et al. (2000)
VOCs (µg/m³)	Amsterdam	High traffic	10.8	Low traffic	6.0	2.1	Fischer et al. (2000)
Nitrogen dioxide (µg/m³)	Huddersfield	400–800 vehicles/hour	36.9	100–400 vehicles/hour	27.3	1.3	Smallbone (1998)
	Netherlands (winter)	Very busy (169 637 vehicles/day)	36.0	Not busy (45 129 vehicles/day)	25.1	1.4	Rijnders et al. (2001)
	Netherlands (summer)	Very busy (169 637 vehicles/day)	15.6	Not busy (45 129 vehicles/day)	17.9	0.9	Rijnders et al. (2001)
PM10 (µg/m³)	Amsterdam	High traffic	41	Low traffic	31	1.2	Fischer et al. (2000)
PM2.5 (µg/m³)	Munich	Traffic	14.3	Urban background	13.3	1.1	Cyrys et al. (2003)
	Stockholm	Traffic	13.8	Urban background	10.2	1.4	Cyrys et al. (2003)
	Netherlands	Traffic	19.9	Urban background	17.8	1.1	Cyrys et al. (2003)
Soot/elemental carbon/ black smoke (µg/m³)	Munich	Traffic	2.5	Urban background	2.1	1.4	Cyrys et al. (2003)
	Stockholm	Traffic	3.9	Urban background	1.4	1.8	Cyrys et al. (2003)
	Netherlands	Traffic	2.1	Urban background	2.1	1.8	Cyrys et al. (2003)
PM2.5 absorbance (10^{-5}/m)	Munich	Traffic		Urban background	1.7	1.3	Cyrys et al. (2003)
	Stockholm	Traffic	2.1	Urban background	1.2	1.8	Cyrys et al. (2003)
	Netherlands	Traffic	2.6	Urban background	1.5	1.8	Cyrys et al. (2003)
	Amsterdam	High	2.8	Low	1.57	1.8	Fischer et al. (2000)

Note. The cities mentioned are located in Germany (Hanover, Munich), the Netherlands (Amsterdam) and Sweden (Stockholm). Ratios are not necessarily equivalent to average near-road/average distant concentrations, because they are based on the averages of paired estimates.

Fig. 3.2. Ratio of concentrations of transport-related air pollution in indoor and outdoor air for houses near busy roads compared with houses near quiet roads in Amsterdam, the Netherlands



Source: Fischer et al. (2000).

Kingdom, for example, 12% of adults were found to be travelling at 08:00 and 16:00, compared with 9% at 12:00, 7% at 20:00 and 2% at 24:00 (Office for National Statistics, 2003). As a result, journey-time exposures often contribute disproportionately to the total, and account for the main peaks in exposure for many people. A study in Copenhagen, Denmark found that, although participants only spent about 45 minutes per day in their cars (3% of their time), this contributed about 10% of total benzene exposure (Skov et al., 2001).

Research on the exposure of car drivers dates back to the 1960s. Most early studies focused on carbon monoxide, largely because of its ease of measurement. More recently, interest has expanded to PM, VOCs, PAHs and metals, and study designs are varied. A large proportion of available studies has focused on cars; a smaller one has considered other modes, such as buses, trains, underground railways, bicycling or walking. Most studies have compared exposures or concentrations in individual transport modes with those measured at a background monitoring site. Fewer have directly compared different transport modes, and only a few, mainly recent studies have attempted to compare exposures by different modes on the same or equivalent routes at the same time. Partly owing to these differences in design, results vary somewhat and are difficult to compare.

Subgroups at risk of high transport-related exposure

Although reliable data on travel behaviour are sparse, the available statistics are remarkably consistent (Table 3.3). In general, people seem to spend 1–1.5 hours a day travelling, though this varies with occupation, age, gender and socioeconomic status. Subgroups with a risk of high exposure are especially important, because they may be subject to the highest exposure levels. This high exposure may be determined by the actual means of transport and the places where people spend the majority of their time – that is, where they work or live.

Table 3.3. Time spent travelling, based on time–activity surveys

Location	Travelling time (hours/day)	Population group	Source
Northampton, United Kingdom	1.25	Adults	Office of National Statistics (2003)
	1.2	Adults	Briggs et al. (2002)
	1.3	Students	Briggs et al. (2003)
	1.0	Schoolchildren	Briggs et al. (2003)
Germany	1.7	Adults	Seifert et al. (2000) not on list
	1.4	Children	Seifert et al. (2000)
United States	1.3	Adults	Klepeis et al. (2001)
	1.4/1.6	Adults	Leech et al. (2002)[a]
	0.9/1.2	Children (aged 11–17 years)	Leech et al. (2002)
	0.9/0.9	Children (aged < 11 years)	Leech et al. (2002)
Canada	1.3/1.6	Adults	Leech et al. (2002)
	0.8/0.7	Children (aged 11–17 years)	Leech et al. (2002)
	0.6/1.0	Children (aged < 11 years)	Leech et al. (2002)
Prague, Czech Republic	2.0	Adults	Jantunen et al. (1999)[b]
Milan, Italy	1.9	Adults	Jantunen et al. (1999)
Athens, Greece	1.9	Adults	Jantunen et al. (1999)
Helsinki, Finland	1.8	Adults	Jantunen et al. (1999)
Basel, Switzerland	1.8	Adults	Jantunen et al. (1999)
Grenoble, France	1.5	Adults	Jantunen et al. (1999)

[a] Travelling times from Leech et al. (2002) comprise winter/summer values.

[b] Travelling times reported by Jantunen et al. (1999) are based on the 50th percentile and exclude cycling and walking.

People whose jobs require travel clearly spend the most time in traffic, and young and elderly people tend to spend the least. Women, who often have less access to a car and who are less likely to be commuters, also tend to have lower travel times. Poor people may be less likely to own or drive a car, but are more likely to use public transport, and thus spend longer periods in buses, trains or underground railways.

Occupation

When compared with the general population, people with outdoor occupations in urban areas are typically exposed to higher concentrations of transport-related air pollution or are in contact with it for longer periods, or both. Such people include postal workers, rubbish collectors, street sweepers, traffic wardens, police officers, tollbooth workers, taxi/bus/tram drivers, underground-railway operators and attendants, street vendors, bicycle messengers and delivery-lorry drivers. The extent of exposure to transport-related air pollution for many of these occupations is poorly understood or unknown. Nevertheless, some research is available on exposure related to underground railways and the exposure of police, traffic wardens and drivers of taxis, buses and trams.

Socioeconomic status

A recent review of health, wealth and air pollution by O'Neill et al. (2003) suggests that socioeconomic status and its relationship to poor health may be partially explained by related exposure differentials. For example, using traffic density on the level of a census block group in California indicates that low-income people and non-White children may have higher exposure to vehicle emissions (Gunier et al., 2003). Possible explanations for the different distributions of air pollution by socioeconomic status include: housing-market dynamics, racism and class bias in land-use decisions (O'Neill et al, 2003). If proximity to areas of dense traffic depresses property values, the lower prices for dwellings are likely to attract people of lower socioeconomic status, who often have higher personal exposures as a result. This effect is in addition to the likely impact of socioeconomic status on the mode of transportation that people use.

Comparisons between in-vehicle exposures and background concentrations

Comparisons between in-vehicle exposures and concentrations at background measurement sites have generally demonstrated increased exposures for travellers to almost all the pollutants studied (see Table 3.4). A summary of 16 studies conducted in the United States between 1966 and 1994, for example, showed that the carbon monoxide concentration in vehicles was generally about 3.5 times the concentrations in the ambient air. A number of European studies provide a similar picture (van Wijnen & van der Zee, 1998): a study conducted in

Table 3.4. Reported concentrations and exposures in different transport modes and in roadside location

Pollutant (and measurement unit)	City or country	Mode					Roadside location	Source
		Car	Bus	Underground railway	Bicycle	Walking		
Carbon monoxide (ppm)	Athens	21.4	10.4	–	–	11.5	–	Duci et al. (2003)
	Paris	3.8	–	–	–		1.9–3.8	Zagury et al. (2000)
Benzene (μg/m³)	Amsterdam	60	–	–	20	20	12	van Wijnen et al. (1995)
	Birmingham[a]	7.7	–	–	–	–	2.9	Leung & Harrison (1999)
	Copenhagen	5.2	–	–	–	–	2.9	Skov et al. (2001)
	Copenhagen	14.4	–	–	5.2	–	–	Rank et al. (2001)
	Republic of Korea	33.1	20.7	–	–	–	–	Lee & Jo (2002)
Toluene (μg/m³)	Birmingham	31.0	–	–	–	–	5.5	Leung & Harrison (1999)
	Copenhagen	69.3	–	–	20.6	–	–	Rank et al. (2001)
	Republic of Korea	233	153	–	–	–	–	Lee & Jo (2002)
Ethylbenzene and xylene (μg/m³)	Copenhagen	66.7	–	–	18.1	–	–	Rank et al. (2001)
Nitrogen dioxide (μg/m³)	Paris	139	–	–	–	–	84–117	Zagury et al. (2000)
Benzo[a]pyrene (μg/m³)	Berlin (summer)	1.0	–	0.7	–	–	–	Fromme et al. (1998)
	Berlin (winter)	3.2	–	4.0	–	–	–	Fromme et al. (1998)
PAHs (μg/m³)	Berlin (summer)	10.2	–	30.2	–	–	–	Fromme et al. (1998)
	Berlin (winter)	28.7	–	67.5	–	–	–	Fromme et al. (1998)
PM10 (μg/m³)	Northampton	43.2	–	–	–	38.2	26.6	Gulliver & Briggs (2005)
PM2.5 (μg/m³)	London (summer)	37.7	39.0	247.2	34.5	–	15.0	Adams et al. (2001)
	London (winter)	33.7	38.9	157.3	23.5	–	13.0	Adams et al. (2001)
	London	33	–	246	–	–	–	Pfeiffer et al. (1999)
	Northampton	15.5	–	–	–	15.1	–	Gulliver & Briggs (2005)
PM1.0 (μg/m³)	Northampton	7.0	–	–	–	7.1	–	Gulliver & Briggs (2005)

Black smoke/	Paris	168	–	–	–	–	89–141	Zagury et al. (2000)
elemental carbon	London (summer)	26.1	16.3	–	15.4	–	5.8–18.6	Adams et al. (2002)
(μg/m^3)	London (winter)	34.4	24.7	–	19.2	–	–	Adams et al. (2002)
PM2.5 absorbance (10^{-5}/m)	Huddersfield	5.7	7.6	–	6.3	–	–	Kingham et al. (2000)
Manganese (μg/m^3)	London	0.020	–	2.281	–	–	–	Pfeiffer et al. (1999)
Magnesium (μg/m^3)	London	0.436	–	0.872	–	–	–	Pfeiffer et al. (1999)

[a] Unit for benzene concentrations in Birmingham is ppb.

Note. The cities listed in this table are in Denmark (Copenhagen), France (Paris), Germany (Berlin), Greece (Athens), the Netherlands (Amsterdam) and the United Kingdom (Birmingham, Huddersfield, London and Northampton).

Amsterdam in 1990 indicated that the carbon monoxide concentration in a car was about four times that at a city background measuring site (van Bruggen et al., 1991; van Wijnen et al., 1995). Benzene shows even greater contrasts, with in-car exposures typically 5–8 times the city background concentrations (den Tonkelaar & van der Tuin, 1983; Chan et al., 1991; van Bruggen et al., 1991; van Wijnen et al., 1995). For nitrogen dioxide, however, contrasts appear to be smaller. Studies carried out in Amsterdam and Delft, the Netherlands, Frankfurt-am-Main, Germany and elsewhere reported in-car exposures of drivers to nitrogen dioxide to be about 1.5 times the city background concentrations (van Bruggen et al., 1991).

In all cases, these differences are not wholly due to the accumulation of pollutants inside cars; they also reflect the marked reductions in ambient concentrations with distance from the road. The contrasts also vary substantially, depending on a range of local conditions, including traffic density, vehicle speed, weather and road configuration. Table 3.4 shows results from comparisons of exposures or concentrations in different transport modes. Absolute exposure levels and their distribution by various transport modes vary considerably between different studies, reflecting not only different microenvironmental conditions but also, perhaps, different measurement methods and study designs. Few studies have compared more than two or three transport modes, so generalizations are difficult. Nevertheless, a broad ranking of these microenvironments is evident, at least in western European cities, with exposures often being:

- highest in underground railways
- intermediate in buses and cars
- somewhat less in cyclists and walkers.

It is crucial to recognize that average exposures in different transport modes may be highly misleading. Different modes imply different journey times and, many travellers spend time in transport at the expense of time in less polluted microenvironments, such as the home, workplace or school. Longer journey times by some modes, such as bicycling and walking, may thus compensate to a great extent for the reduced average exposures. Under some conditions, therefore, bicycling and walking can actually increase exposures, in comparison with motorized modes. In addition, increased breathing rates while bicycling and walking may mean that larger volumes of pollutant are inhaled. A study in Amsterdam indicated that bicyclists breathe on average more than twice as much air as car drivers. Car drivers inhaled only marginally higher amounts of carbon monoxide and benzene, while bicyclists inhaled a significantly higher amount of nitrogen dioxide (van Wijnen et al, 1995). Thus, simple comparisons between exposures implied by concentrations in these different transport microenvironments, or even by personal monitoring, need to be interpreted with caution.

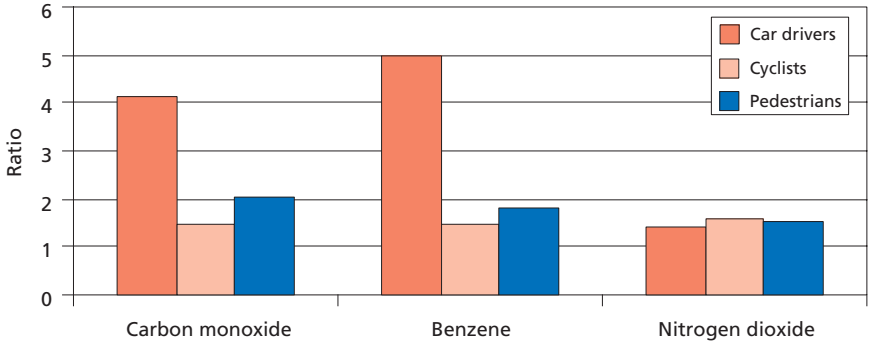
Patterns

Across these different pollutants and transport environments, however, several important patterns stand out.

1. Exposures to primary exhaust gases and PM are especially high, presumably because these penetrate the vehicle either directly from the exhaust system or by recirculation from outside. Fig. 3.3 shows the varying ratios for carbon monoxide, benzene and nitrogen dioxide for three different transport modes in Amsterdam. In a study of Paris taxis, Zagury et al. (2000) found nitric oxide concentrations to be more than 11 times higher than at the city background measuring point; in contrast, those of nitrogen dioxide were 1.9 times higher. Similarly, exposures to elemental carbon, measured by reflectance of PM2.5 filters, were up to 11 times higher in London than urban background concentrations reported in other studies, while the mass of PM2.5 was only some 2.3 times higher (Adams et al., 2002).
2. Exposures in underground railways are excessively high, and those in traffic tunnels can be much higher than on open streets. Both warrant further study for their effects on health, and are discussed in more detail below.

In addition, these patterns vary according to vehicles' characteristics. Leung & Harrison (1999), for example, reported marked differences in in-vehicle benzene and toluene exposures in different makes of car, with significant self-contamination occurring in vehicles with poor seals and petrol leaks. In-vehicle furnishings and solvents may also be a source of exposure to some pollutants, such as toluene and xylene. In contrast, gas-powered vehicles seem to result in greatly reduced in-vehicle exposures, at least to PM. In a study in Slovenia, average elemental carbon concentrations inside a natural-gas bus were found to be about three times lower than those inside diesel buses on the same day and location

Fig. 3.3. Ratio of road users' exposure to city background concentrations in Amsterdam, the Netherlands



Source: van Wijnen et al (1995).

(Bizjak & Tursic, 1998). Many other factors also affect these relationships. In a Finnish study of urban commuter exposure inside a car, the effects of wind speed, driving speed and relative humidity were seen to vary between coarse and fine particles (Alm et al., 1999).

Exposure studies on underground railways

As mentioned, exposures on an underground railway (whether it is called a metro, subway or underground) can be considerably higher than those above ground. For example, PM2.5 concentrations in the London Underground have been shown to be up to 10 times higher than those for surface transport modes (Pfeifer et al., 1999; Sitzman et al., 1999; Adams et al., 2001, 2002), and a study of Stockholm's underground railway found similar results (Johansson & Johansson, 2003). Elemental analysis of samples in London showed that PM was composed mainly of iron and silica from brakes and train tracks (Sitzman et al., 1999). These patterns, however, are not repeated everywhere. In China, one study found PM10 levels were 1.2–3 times higher for roadway transport than in the subway, and carbon monoxide levels about 2.6–9.3 times higher (Chan et al., 2002). Similarly, carbon monoxide was higher in minibuses during morning rush hour than in the subway in Mexico City (Gomez-Perales et al., 2004).

In New York City, elevated exposures to manganese, chromium and iron from steel dust was observed in teenagers commuting to school (Chillrud et al., 2004). The ratio of iron to manganese in these exposures was essentially constant, yet distinctly different from typical crustal material and ambient air PM, but agreed fully with the samples collected from the subway system. The PM2.5 (iron) exposures of the young people commuting by subway were an order of magnitude higher than the exposures of those using another transport mode.

In Finland, Tervahattu et al. (2004) identified the PM2.5 particles found in underground and aboveground stations, and underground railway cars of the Helsinki Metro. PM2.5 levels in the underground stations were 3–20 times those above ground. In the trains, the concentrations increased rapidly as the cars entered a tunnel, and decreased rapidly when they exited. Commuter exposure therefore depends on the proportion of each trip spent in tunnels. Above ground, the particles were similar to other urban ambient air particles, but a high proportion of the underground particles were dominated by iron, along with crustal minerals. Overall, these studies warrant further study on the possible adverse effects on health of underground railway exposures.

Special situation: tunnels

As traffic intensity continues to increase in densely populated areas, road tunnels are built to alleviate traffic congestion. Levels of transport-related air pollutants – such as nitrogen oxides, PM, VOCs and metals – can be several times higher in road tunnels than on open streets (City of Stockholm Environment and Health

Administration, 1995; Barrefors, 1996; Indrehus & Vassbotn, 2001; Wingfors et al., 2001; Sternbeck et al., 2002; Valiulis et al., 2003).

Several factors influence air pollution levels in road tunnels, such as the length, width and ventilation of the tunnel; traffic intensity; the types, speed and fuel of vehicles using the tunnel; and the pollutant concentration in incoming air. Pollution concentration, transit time and vehicle ventilation determine the individual exposure of people using the tunnel. Several of these factors differ for various types of tunnel and some, including tunnel ventilation, may vary with time and depend on decisions about and local standards for what is acceptable (City of Stockholm Environment and Health Administration, 1995; Indrehus & Vassbotn, 2001).

Compared with other microenvironments, concentrations in tunnels are high and closely related to traffic density (Kirchstetter et al., 1999; Swietlicki et al., 1999; Indrehus & Vassbotn, 2001). For example, inside the Stockholm tunnel, concentrations of nitrogen oxides, nitrogen dioxide and carbon monoxide were 4–5 times higher at 1000 m from the entrance than at the entrance and 5–10 times higher than levels in streets with heavy traffic (City of Stockholm Environment and Health Administration, 1995). A study in the United States on exposure of road construction workers to carbon monoxide in a tunnel 1.6 km long found a linear increase in concentration with distance from the tunnel entrance (Kamei & Yanagisawa, 1997). In Stockholm, tunnel exposures were compared with a control exposure in a clean environment nearby (Svartengren et al., 2000). The concentrations of nitrogen dioxide in the tunnel were similar outside and inside the car. The median levels of nitrogen dioxide, PM10 and PM2.5 concentrations in the tunnel were about 26 times, 18 times and 19 times those of controls during the 08:00–09:00 rush hour (Monday to Friday).

The concentration and composition of air pollution in road tunnels vary through the day, depending on traffic intensity. In Stockholm, the ratio of nitrogen dioxide to nitrogen oxides is generally lower in the tunnel than in the street, and varies according to time and ventilation through the tunnel (Johansson et al., 1996; Indrehus & Vassbotn, 2001). PM data have rarely been used to assess air quality in tunnels, but measurements in the Caldecott Tunnel, located in the San Francisco Bay Area, United States indicated that the fine particle mass distribution peaked in the size range of 0.1–0.18 μm (aerodynamic diameter). Elemental carbon and organic matter were the largest components of particle mass in all size ranges studied. The fraction of elemental carbon, however, might differ for other road tunnels (Allen et al., 2001).

Non-exhaust emissions and resuspension of coarse particles are also important (Sternbeck et al., 2002; Valiulis et al., 2003). In Lithuania, Valiulis et al. (2003) found that trace metal concentrations were lower on fine particles than those on coarse particles for a road tunnel in Vilnius, suggesting that re-emitted road dust was highly enriched in trace metals owing to past emissions in the tunnel. In contrast, Sternbeck et al. (2002) found that the ratio of PM2.5 to PM10 was

low, and elemental carbon correlated better with the smaller particles. Overall, the tunnel studies suggest that acute exposures differ in composition and magnitude from those in other traffic microenvironments, and this warrants further study of the possible effects on human health.

Assessment of traffic exposure in a variety of microenvironments

As discussed, an assessment of total exposure over a wide range of microenvironments offers an excellent description of the factors, including traffic modes, that affect personal exposure. Kousa et al. (2002) provide a useful review of exposure assessments, according to the different methods used, and provide results from the EXPOLIS study in several regions of Europe. A large database of published exposure data has been collected and reported for the study (KTL, 2004). As expected, reported exposures vary widely, reflecting not only the different methods of analysis but also the regional and local variations in source intensity, dispersion environment (such as weather, topography and building configuration), and the time–activity patterns and behaviour of the target population group.

Nevertheless, the total microenvironmental assessment of transport-related exposure usually offers the best and perhaps most realistic opportunity to assess integrated exposure to each pollutant for a given population or group. Often, techniques used as part of an exposure assessment in epidemiological studies also provide valuable evidence for total exposure to transport-related air pollution. As a result, these various types of study design may help highlight subgroups of the population with a high risk of transport-related exposure. Unfortunately, assessment studies of total microenvironmental exposure are rare, and only a relatively small number of representative individuals of the population are realistically sampled, providing little information on the distribution of exposure in the general population. Nevertheless, the following is a summary of information gathered, on a per-pollutant basis, to help understand the impact of traffic on total exposure.

Nitrogen dioxide

In an epidemiological study in five French metropolitan areas, Zmirou et al. (2002) analysed nitrogen dioxide exposures of 217 pairs of matched cases and controls of children aged 4–14 years. Average personal exposures were 31.4 (±13.9) μg/m^3, while indoor concentrations averaged 36.1 (±21.4) μg/m^3; the calculated average lifelong exposure to transport-related emissions of nitrogen oxides was 62.6 (±43.1) μg/m^3. Similarly, results from the EXPOLIS study provide average personal exposures of 25 μg/m^3 in Helsinki, 30 μg/m^3 in Basel and 43 μg/m^3 in Prague (Kousa et al., 2001). Variation in the ambient, fixed-site monitored concentration of nitrogen dioxide, however, explained only 11–19% of the variation in 48-hour personal exposure. Kousa et al. highlight that, although

microenvironmental analysis of exposure concentrations affected by traffic may give higher exposure estimates, owing to related impacts on exposure, other determinants, such as use of a gas appliance and workplace location, also significantly influence personal exposure.

VOCs

Analysing the EXPOLIS data on benzene exposure from Helsinki, Edwards & Jantunen (2001) observed that, after eliminating people exposed to environmental tobacco smoke from the sample population, daily exposures were significantly increased in people who lived in houses with attached garages, spent time in the garage or visited filling stations and refuelled cars. Time spent in a car, however, showed only a modest impact on the fit of a least-squares regression exposure model. The determination coefficient R^2 improved from 0.29 to 0.39, when the model included time spent in a car as a variable.

The relative impact of car transport and traffic on exposure to benzene depends on two factors:

1. the benzene concentration in petrol, which increases the absolute contribution of traffic; and
2. indoor sources of benzene, particularly tobacco smoke, which decrease the relative contribution of traffic to exposure.

In addition, biological monitoring provides an integrated assessment of exposure based on the uptake of certain pollutants (such as PAHs) from all contributing microenvironments (Box 3.2).

Traffic is also a source of exposure to a multitude of VOCs other than benzene, formed in fuel evaporation and incomplete combustion. Edwards et al. (2001) used a principal components analysis[8] on simultaneously sampled, personal, indoor (home and workplace) and outdoor VOCs to identify source factors of exposure in Helsinki. Interestingly, the chemicals that are commonly associated with traffic were split into two distinct components. The first component explained 19% of the variation in personal exposure, 20% of the variation in outdoor air VOCs and 24% of the variation in workplace concentrations. It was dominated by nonane, decane, undecane, trimethylbenzene and propylbenzene and, to a lesser extent, by xylene and trimethylbenzene. This first component was identified as the local traffic source. The second component was dominated by xylene, ethylbenzene styrene and toluene. This component explained 17%

[8] A principal-components analysis is a mathematical procedure that transforms a number of possibly correlated variables into a smaller number of uncorrelated variables called principal components. The first principal component accounts for much of the variability in the data, and each succeeding one accounts for part of the remainder.

Box 3.2. Biomarkers of exposure to PAHs

Biomarkers of exposure measure internal dose (as metabolites in blood or urine) or biologically effective dose (as deoxyribonucleic acid (DNA) adducts, protein adducts or DNA breaks by the Comet assay). In many cases, studies on environmental exposure to mutagens and carcinogens lack data on exposure. For example, personal exposures to PAHs are often below or near detection limits for environmental exposure. Also, it is sometimes difficult to relate the effects observed with biomarkers to pollution alone, if information on ambient exposure and lifestyle are not fully presented for each individual.

These considerations aside, tollbooth workers, traffic wardens, bus drivers and postal workers have usually been used as model groups for studying air pollution in big cities. In the Czech Republic, Binkova et al. (1995) studied the effect of personal exposure to carcinogenic PAHs on DNA adducts in women working outdoors as postal workers or gardeners in the polluted city of Teplice. The effect in nonsmokers was significant. In the follow-up study, Binkova et al. (1996) compared women in the polluted region of Teplice with controls in the district of Prachatice. They found a significant correlation between individual personal exposures to carcinogenic PAHs and DNA adducts.

The effect of environmental pollution has been measured in several groups of traffic police in Italy. Peluso et al. (1998) conducted a study in Genoa, determining benzo[a]pyrene exposure, using personal monitoring, and analysing the formation of DNA adducts in white blood cells, using a ^{32}P-postlabelling technique. A higher level of benzo[a]pyrene and DNA adducts was observed in the police officers during the summer; no effect was observed in the winter. Similar results for workers exposed to transport-related air pollution in Florence and Prague have been reported elsewhere (Palli et al., 2001; Binkova et al., 2002). In Prague, Binkova et al. (2002) studied the effect of environmental exposure to airborne, particulate-bound PAHs on DNA-adduct levels in male police officers spending more than 8 hours outdoors and in matched controls spending more than 90% of their daily time indoors. The total DNA adduct levels did not differ significantly between the two groups, but the level of benzo[a]pyrene-derived DNA adducts was significantly higher in the exposed group.

A large study in Denmark evaluated several biomarkers of exposure and of adverse effects on health in bus drivers and mail carriers, using 1-hydroxypyrene (1-OH-pyrene) in urine, and exposure dose by DNA and protein adducts (Hansen et al., 1998; Autrup et al., 1999; Loft et al., 1999). Significantly higher levels of bulky-aromatic DNA adducts were observed in bus drivers working in the central part of Copenhagen. The biomarker levels in mail carriers were similar to the levels in suburban bus drivers. Surprisingly, significantly higher levels of malondialdehyde in plasma and PAH-albumin adducts were observed in the suburban group of bus drivers, which was used as a control group because of the assumed lower exposure to ambient air pollutants. Autrup et al. (1999) recommended analysing oxidative damage to DNA, because pollution by diesel exhaust particles may induce such damage only. Damage in bus drivers was determined by urinary excretion of 8-oxo7,8-dihydro-2'-deoxyguanosine (8-oxodG) (Hansen et al., 1998). Drivers in the

centre of Copenhagen showed a significant difference in 8-oxodG excretion from those in the outlying rural or suburban areas. This suggests that exposure to ambient air pollution causes oxidative damage to DNA (Loft et al., 1999).

Overall, the results of various studies indicate the value of different biomarkers in different types of pollution scenarios. The classic 1-OH-pyrene seems to be an inconvenient biomarker for environmental exposure. DNA adducts measured by a ^{32}P-postlabelling procedure have become the most popular of biomarkers of exposure and are probably the biomarker of choice for evaluating exposure to PAH. For oxidative damage, 8-oxodG and the Comet assay seem to be convenient biomarkers. Nevertheless, all of these need reliable standardization and international validation for human biomonitoring in the future.

of the variation in personal VOC exposure, and the largest share, 33%, of variation of VOCs in outdoor air. As this component exhibited a strong dependence on the easterly wind, the conclusion was that it consists of VOCs from a nearby oil refinery and possibly St Petersburg's traffic. The other principal components identified factors in VOC exposure that originate indoors or from consumer products, most notably tobacco smoke.

Carbon monoxide

In the EXPOLIS study (Georgoulis et al., 2002), one-minute measurements were taken over 48 hours for 401 randomly selected study participants (mainly nonsmokers) in five cities in different countries: Athens, Basel, Helsinki, Milan and Prague. For the duration of the sampling, study participants were asked to complete a time–activity diary.

Marked differences between cities broadly reflected levels of traffic and urban density, with the highest exposures to carbon monoxide among nonsmokers in Milan (2.17 ± 1.44 mg/m^3) and Athens (1.68 ± 2.45 mg/m^3) and the lowest in Helsinki (0.45 ± 2.57 mg/m^3). An analysis of the time–activity data showed that exposures were significantly higher when participants spent time in street traffic and indoors in the presence of smokers. Overall, personal carbon monoxide exposures were about 100% higher when participants were in a car or taxi and about 50% higher when they were on a motorcycle than when they were simply outdoors. During periods of walking, bicycling and train travel, personal carbon monoxide exposures were closer to outdoor exposures.

PM

A number of studies have assessed the contribution of traffic to PM exposures in Helsinki and other EXPOLIS centres. For example, Ilacqua & Jantunen (2003) analysed EXPOLIS data on PM2.5 from Athens, Basel and Helsinki, where they incorporated PM2.5 constituents and some presumed traffic-specific VOCs (xylene, ethylbenzene and trimethylbenzene) in the same model. In Athens,

where leaded petrol was still widely used in 1996, these VOCs and black smoke were found with lead and bromine in the same factor. In Basel, lead in petrol was not completely phased out, because it was available in the neighbouring French city of Mulhouse; consequently, lead and bromine were still weakly found in the same factor, dominated by traffic VOCs and black smoke. In Helsinki, where leaded fuel was no longer available, the same VOCs and black smoke were still in one factor, along with some weakly loaded elements, but each of these elemental markers was more strongly loaded in some other factor; that is, none was more specific to traffic. The elemental carbon or black smoke index might be useful as a marker of PM from traffic, if the contributions of other local sources and the regional background are carefully considered.

Koistinen et al. (2004) conducted a chemical mass balance analysis on the EXPOLIS PM samples, using the source reconstruction method of Malm et al. (1994) and Brooke et al. (1997). After identifying the mass contributions of secondary aerosols, mineral dust from soil and construction, and sea and de-icing salt, a fraction remained that correlated with black smoke concentration; this fraction contributed an average 38% to the total personal exposure to PM2.5. These other particles in mass reconstruction analysis were named the combustion and other PM (CoPM) fraction and contained, among other things, all primary combustion and secondary organic particles, including but not limited to all traffic exhaust particles. In their analysis, Koistinen et al. went no further in identifying the contribution of traffic to exposure. Tainio et al. (2004), however, concluded – after subtracting the long-distance contribution and dividing the remaining CoPM fraction contributed by local combustion sources, according to emission inventory data and source-specific intake fractions – that the average contribution of local traffic to PM2.5 exposure in Helsinki was 1.7 $\mu g/m^3$, about 50% of which was acquired while commuting. On average, the level of PM2.5 during time in traffic was 10–20 $\mu g/m^3$ higher than that in ambient air and was due to both traffic-generated exhaust and street dust particles.

Metals

Koistinen et al. (2004) conducted a principal-component analysis using the elemental constituents and black smoke index of outdoor and indoor microenvironments and simultaneously sampled personal PM2.5 samples from Helsinki. The factor, which was strongly loaded with black smoke, was also loaded with sulfur, potassium and manganese, indicating sources other than traffic combustion. In outdoor air, the factor that was linked to local traffic was most strongly loaded with black smoke, zinc and iron; black smoke, however, was not loaded in this factor in personal exposure, and the factor explained only 12% of the variation in personal exposure. One must conclude that no clear traffic factor was found in this analysis, although the four strongest factors explained 71% of the variation in total personal exposure to PM2.5.

Conclusions

Assessing the population's exposure to transport-related air pollution is clearly important for epidemiological studies determining the relationship of this exposure to health outcomes and for both evaluating the risks to public health and identifying people at risk. Some indication of exposure distributions can be obtained from relatively small, but well-stratified, personal monitoring campaigns, as used, for example, in the EXPOLIS study. Some measure of overall exposure can be obtained by weighting various estimates according to the time spent in each microenvironment. Unfortunately, cost, lack of suitable technologies and the logistical difficulties involved inevitably mean that opportunities for direct measurement of exposure tend to be limited. Instead, reliance must often be placed on more indirect assessment methods, typically by using proxies, such as distance to the roads or source intensity, or by modelling exposures, using GIS or other techniques.

For most pollutants, exposure concentrations appear to be up to twice as high in urban as in rural areas, and over 2–3 times as high near busy roads as at background measurement sites. In general, comparisons between in-vehicle exposures and background concentrations have demonstrated increased exposures for travellers, often greater than threefold. This is especially true for primary air pollutants: carbon monoxide, benzene, nitric oxide and black smoke. There are large uncertainties in quantifying emissions of PM10 from traffic – for which most particles are formed by mechanical processes – but local and periodic resuspension of the particles can dominate them.

The contribution of traffic to PM mass concentrations is due both to the increase in primary PM and to an increased concentration of secondary components formed at some distance from the road. The latter show no clear gradients with the distance to the source. Stronger gradients are found for particle numbers and soot, which are indicative of diesel exhaust gases.

The pollution and therefore exposure levels in residential areas are determined mainly by the emissions (determined by quantity of traffic passing the road, traffic composition, and other contributing factors), the distance of an residence to the road, topography and weather conditions (such as the time during which the wind blows from the road towards the accommodation).

For car drivers, traffic conditions (such as speed, road type and traffic intensity), weather conditions (such as wind speed and temperature) and cars themselves are the main determinants of exposure levels. While bicyclists and pedestrians are exposed to lower average concentrations than car drivers, some factors offset some of the differences. Also, journey-time exposures often contribute disproportionately to the total and account for the main peaks in exposure for many people. In general, people seem to spend 1–1.5 hours a day travelling, although this period varies with occupation, age, gender and socioeconomic status. Moreover, knowledge is limited on the importance of real-life peak exposures, such as those measured in tunnels.

Needs for further information

Because too few detailed studies have been undertaken, it is difficult to make reliable statements about population-level exposures to the many pollutants of interest. Nevertheless, marked variations in exposure evidently occur both between cities and over quite short distances, such as a few hundred metres. Where people live and, even more important, where they spend their time can thus have a major effect on their exposure. Exposures appear to increase in proportion to traffic volume. While population-level estimates of exposures have unequivocal value, they may mask the extremes of exposure experienced by some subgroups.

Real attention and future exposure assessments thus need to focus on some of groups at particular risk, and on the time–activity patterns that determine their exposures. These groups include:

1. people (especially the elderly and very young) who live close to busy roads;
2. children whose schools lie close to major roadways; and
3. people who spend much of their time travelling through or working in environments with heavy traffic, such as traffic wardens and traffic police, street traders and some commuters.

Further, future studies need to address high-exposure sites (such as road tunnels), the contribution of road dust to air pollution levels and the value of specific biomarkers in expanding the knowledge base.

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4. Studies on health effects of transport-related air pollution

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Key points

Facts

Evidence from epidemiological and toxicological studies on the effects of transport-related air pollution on health has increased substantially, although it is only a fraction of the total evidence on the effects of urban air pollution on health. A review of this evidence indicates that transport-related air pollution affects a number of health outcomes, including mortality, non-allergic respiratory morbidity, allergic illness and symptoms (such as asthma), cardiovascular morbidity, cancer, pregnancy, birth outcomes and male fertility. Transport-related air pollution increases the risk of death, particularly from cardiopulmonary causes, and of non-allergic respiratory symptoms and disease. Experimental research indicates that the effects are linked to changes in the formation of reactive oxygen species (ROS), changes in antioxidant defence, and increased non-allergic inflammation, thus articulating some parameters of susceptibility. While laboratory studies indicate that transport-related air pollution may increase the risk of developing an allergy and can exacerbate symptoms, particularly in susceptible subgroups, the evidence from population studies that supports this conclusion is inconsistent.

Though only a few studies have been conducted, a significant increase in the risk of heart attack (myocardial infarction) following exposure to transport-related air pollution has been reported. Other studies and the experimental evidence indicate changes in autonomic nervous system regulation and increased inflammatory responses, as a result of exposure. Cancer, too, is a problem. A few studies suggest an increased incidence of lung cancer in people exposed to transport-related air pollution for a long time. An elevated incidence of cancer in children with high or prolonged exposure to air pollution cannot be excluded, though the supporting evidence is less consistent than that for adults. Certain occupational groups, such as professional drivers

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and railway workers show increased incidence of and mortality from lung cancer, especially in instances of long exposure. Finally, some studies suggest that transport-related air pollution has adverse outcomes on pregnancy, such as premature birth and low birth weight, but the available evidence is inconsistent.

Only a few intervention studies have been conducted, most of them not specific to transport-related air pollution. They show, however, that reducing such pollution may directly reduce acute asthma attacks and related medical care for children. They also show that long-term decreases in air pollution levels are associated with a gain in life expectancy and with declines in bronchial hyperactivity, the average annual trend in deaths from all causes, and respiratory and cardiovascular diseases.

Possible indicators of exposure

Often, the effects observed in epidemiological studies cannot be attributed to the specific indicators addressed, but to a mixture of pollutants. Fine PM (including black smoke) and ozone are associated with increased risks of mortality and respiratory morbidity, while exposure to nitrogen dioxide, ozone and PM has been linked to allergic responses. Other indicators of exposure to transport-related air pollution, such as residence near or distance to major roads and, in part, self-reported traffic intensity at a residence, were associated with several adverse health outcomes. A drawback of self-reported transport-related air pollution exposure, however, is that it might overestimate the effect of self-assessed health.

Introduction

This assessment of the health risks associated with air pollution is based on combined scientific evidence from epidemiology and toxicology. WHO's recent systematic review of the health aspects of air pollution in Europe assessed this evidence, focusing on the health effects of PM, ozone and nitrogen dioxide (WHO Regional Office for Europe, 2003, 2004a). This valuation provides the background for the analysis of transport-related air pollution, but does not offer a specific response to the risks created by transport. Though PM, ozone and nitrogen dioxide are produced by traffic, other emission sources also contribute to the population's exposure and the effects on health. Only some of the many studies conducted are specific to the assessment of transport-related air pollution. In addition, the WHO review project did not consider the variety of other pollutants, including carcinogens, emitted by motor-vehicle engines, tyres and brakes.

Both epidemiology and toxicology have advantages and limitations in studying the adverse effects of air pollution on health. The advantage of epidemiological studies is their relevance to real life, in terms of both exposure patterns and coverage of target populations. These studies may include subjects differing in age and health status, performing normal activities in their everyday environment, sometimes for a prolonged period. This characteristic, however, is also a limitation when exposure to pollution from a specific source, such as transport, is considered. The air-pollution mixture experienced by subjects in most epidemiological

studies is generated by a variety of sources, so it is difficult to attribute the effects of exposure on health to a particular source.

Specific epidemiological approaches reduce the problem of identifying the source and help indicate the extent to which transport contributes to the observed adverse effects on health. Each of the following examples of approaches to assessing exposure to transport-related air pollution has specific limitations and strengths. Some studies have concentrated on the components emitted by transport sources, although, in most cases, other processes also generate them. Measured traffic-related combustion pollutants (such as carbon dioxide, nitrogen oxides, benzene, black smoke, soot and PM2.5) or modelled exposure to these pollutants could be used to study possible adverse effects on health. Other studies have explored the spatial or temporal patterns of pollution's composition and level, to link the exposure to transport. Still others have used indirect indicators of transport-related air pollution, such as distance to roads of a certain traffic intensity and type. Several studies have used self-reported traffic intensity, street type or frequency of traffic jams at residences as exposure surrogates. The authors of this book consider these surrogates as subjective measures of exposure. Moreover, some studies have used data on traffic counts on roads nearest a residence, GIS-derived distances of homes from busy roads, or a combination of both, as proxies for exposure to transport-related air pollution. Consideration of the special situation of subjects with occupational exposure also contributes to the evidence on transport-related air pollution.

Toxicological studies comprise experimental studies of human volunteers, different animal species and strains and cell cultures derived from human or animal lungs or airways. Toxicological studies can examine the effects of specific components, such as those from transport emissions, combined exposures to defined pollutants or exposures to ambient mixtures. The human studies are usually limited in duration, concentration range and the assessment of the end-points of the effects of exposure. They often include healthy volunteers, but more recently have also tested subjects with some degree of disease. Most recently, they have employed novel techniques, using particle concentrators, to expose people to concentrated particles of ambient air pollution; several of these studies have focused on the effects of typical transport-related pollutants, such as diesel particles. Many experimental studies have identified the reactive effects of transport-related air pollution in the airways and cardiovascular system, and the sections that follow discuss the possible mechanisms of the effects indicated by these findings.

Both animal studies and cell-culture studies contribute to the body of evidence on the hazardousness of transport-related air pollution. Animal studies permit the investigation of larger concentration ranges, longer exposure times, more end-points and a wider range of pollutants. Extrapolating findings to the human situation, however, is difficult, whether healthy or diseased animals are used. The data reviewed here come from studies on concentrated PM collected from urban air, diesel particles, some gases and VOCs.

Cell-culture studies are useful for experiments to identify hazards and explore the mechanisms of disease development, and may support results from other model systems. The compounds reviewed include different types of particles (such as those emitted from diesel, diesel alternatives or petrol combustion, ultrafine particles or particle mix from urban air), gases, VOCs and PAHs. As with animal experiments, extrapolation from in vitro effects to the human situation is difficult, so the results are most useful in support of other approaches, such as hypotheses of mechanisms and plausibility arguments.

The different end-points of toxicological research – such as inflammation, production of IgE, cell death, DNA damage, fibrinogen and vasoconstriction – are discussed under different disease outcomes, but the reader should remember that toxicological outcomes are very often indicators of biological/pathological responses or of susceptibility, but not necessarily of disease. Further, many of the toxicological studies described below used exposure concentrations of pollutants well above the levels routinely experienced by people in Europe and North America. These concentrations are essential for identifying the hazards of pollutants, but they must be interpreted along with the evidence from epidemiological and exposure-assessment studies to yield a risk assessment.

This chapter summarizes the combined epidemiological and toxicological evidence on the possible links of various indicators of health to air pollution generated by transport, and focuses on the studies that specifically attribute the exposure to transport. The evidence from various types of studies is presented for each of the health outcomes considered, and the combined evidence is used to draw conclusions.

Mortality

Epidemiological studies on the adverse effects of air pollution on health most frequently use mortality as an indicator. It is routinely registered and reported in most populations, fairly well standardized and readily available. Numerous studies conducted in Europe, North America and other parts of the world indicate the association of death, particularly from cardiopulmonary causes, with various indicators of air pollution, and point to the important role of fine PM and ozone (WHO Regional Office for Europe, 2003). The effects of both long-term exposures, observed in cohort studies, and short-term (daily) changes in pollution levels have been reported.

Most of the evidence, however, comes from studies on the effects of the pollution mix generated by a variety of sources, which include traffic, communal and industrial combustion, and long-range transport of air pollution. Identifying the effects related specifically to the pollution created by transport is a challenge. In this respect, international and multi-city studies provide an opportunity to link disparate patterns of associations detected in various cities with differences between cities in the contribution of various sources to pollution.

Measured compounds

Studies have investigated the association between transport-related air pollution and mortality. The multinational, EC-funded APHEA2 (Air Pollution and Health: a European Approach 2) project included data from 29 European cities (Katsouyanni et al., 2001). It reported the combined estimate for an increase in the daily number of deaths associated with a 10-μg/m^3 increase in daily black smoke concentrations as 0.6% (95% confidence interval (CI): 0.3–0.8%). In the two-pollutant model, considering black smoke with nitrogen dioxide, the estimated effect of black smoke was lower, although it remained significant. The effect was slightly higher in cities with high concentrations of nitrogen dioxide. These results could be interpreted as meaning that nitrogen dioxide might serve as an indicator of the presence of more toxic particles, such as traffic-related particles. In contrast, the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the United States found no evidence for modification of the association between PM and daily mortality by nitrogen dioxide (Samet et al., 2000).

Other studies associated black smoke more strongly with respiratory and cardiovascular mortality than with other causes of death (Zmirou et al., 1998; Ballester et al., 2002; Le Tertre et al., 2002). In a case-crossover analysis of a group of men and women with pre-existing chronic obstructive pulmonary disease, who had died during 1990–1995, Sunyer et al. (2000) found that black smoke levels were associated with mortality for all causes. For an increase of 20 μg/m^3 (the interquartile change) in daily mean level of black smoke, the odds ratio (OR) adjusted for temperature, humidity and influenza was 1.112 (95% CI: 1.017–1.215). The association was stronger for mortality from respiratory causes (OR: 1.182; 95% CI: 1.025–1.365), but was not significant for cardiovascular causes (OR: 1.077; 95% CI: 0.917–1.264). Also, the risk of dying associated with black smoke was greater for older women admitted to intensive care units and for people with a higher rate of emergency room visits due to chronic obstructive pulmonary disease.

Modelled exposure

Modelled exposures have been used to associate transport-related air pollution with mortality. A recent reanalysis (Schwartz et al., 2002) of the data from the Six Cities study in the United States, using source apportionment techniques, showed that PM2.5 from traffic-related particles has a linear association with mortality. The relationship was a 3.4% increase in mortality per 10-μg/m^3 increase in traffic-related PM2.5, compared with no major effects for non-combustion-derived particles, with about half of that effect for PM2.5 in general. An earlier reanalysis of the Six Cities study (Laden et al., 2000) suggests that daily mortality was associated mostly with air pollution from such combustion sources as traffic, coal and residual oil. The study looked at pollution data obtained in the 1980s, when lead could still be used as a tracer for traffic exhausts, so the results are relevant for a

mixture of transport-related air pollution for which lead is a tracer. It is uncertain, however, whether these results are still representative of present-day mixtures.

Subjective and indirect indicators of exposure

The distance from a residence to a road was used as an indicator of traffic exposure in both time-series and cohort studies of mortality. A time-series study for Amsterdam estimated increased adverse effects of air pollution on people living close to major roads (Roemer & van Wijnen, 2001). A cohort study in the Netherlands showed an increased risk of death for people living close to major roads, in addition to the impact of the background level of black smoke and nitrogen dioxide (Hoek et al., 2002); the study also found that the effects on cardiopulmonary mortality were the most pronounced. The increased risk of death from cardiopulmonary causes was estimated to be 1.34 (95% CI: 0.68–2.64) per 10-μg/m^3 increment of black smoke and 1.95 (95% CI: 1.09–3.51) for living near a major road.

Occupational exposure

Several epidemiological studies showed associations between mortality and occupational exposure to emissions from transport, though not all found a statistically significant excess in risk. The studies were both retrospective and prospective.

A retrospective study of the effects of occupational exposure to carbon monoxide on mortality from heart disease (Stern et al., 1988) found that tunnel officers had higher mortality from arteriosclerotic heart disease than people in the general population of New York City (standardized mortality ratio (SMR): 135; 90% CI: 109–168). Tunnel officers also had a higher risk of mortality from arteriosclerotic heart disease than the less-exposed bridge officers, which leads to the hypothesis that motor exhaust might increase the risk of myocardial infarction.

A prospective study on mortality of professional drivers in London – particularly lorry drivers – showed excess deaths from stomach cancer, lung cancer, bronchitis, emphysema and asthma, although there were significantly fewer deaths than expected from all causes and circulatory diseases (Balarajan & McDowall, 1988). This pattern, however, could not be confirmed in taxi drivers. The possible relationship between occupational exposure to vehicle exhaust and cancer risk was studied in a Danish cohort (Hansen, 1993); lorry drivers were followed for cause-specific mortality for a ten-year period, and increased mortality from lung cancer (SMR: 160; 95% CI: 126–200) could be shown, indicating that exposure to diesel exhaust may have contributed to the observed increased risk of lung cancer. Alfredsson et al. (1993) compared mortality from myocardial infarction and other causes for all male bus drivers in Sweden was with that of other employed men over a fifteen-year period; they found a 50% increase in mortality from myocardial infarction among drivers in the counties with the largest cities. Another study identified a significantly increased risk of mortality from ischaemic heart disease in bus drivers working in an area with high traffic

intensity (Netterstrom & Suadicani, 1993). A cohort study in Rome explored the mortality pattern of taxi drivers exposed to vehicle exhaust (Borgia et al., 1994). An increased SMR was observed for lung cancer (123; 95% CI: 97–154). Owing to statistical uncertainty, however, the results did not clearly indicate an association between the risk of lung cancer and exposure to vehicle exhaust among taxi drivers.

In Italy, Lagorio et al. (1994) compared the mortality in a cohort of petrol-station attendants with that in the regional population. Data analysis indicated some increased risks for oesophageal cancer (SMR: 241; 90% CI: 82–551) and brain cancer (SMR: 195; 90% CI: 77–401); this also applied to the attendants of small stations with a small number of employees and those working at stations with a high number of sales of petrol per full-time employee (SMR: 351; 90% CI: 120–803 and SMR: 266; 90% CI: 105–559, respectively). A historical cohort study of police officers in Rome showed mortality from cardiovascular diseases that was higher than expected, though that from respiratory conditions was lower than expected; no statistically significant excess mortality from lung and other types of cancer was found (Forastiere et al., 1994).

In summary, the few available studies that focus on transport-related air pollution indicate that it contributes substantially to the increased risk of death (particularly from cardiopulmonary causes) from exposure to air pollution.

Respiratory morbidity

Non-allergic respiratory morbidity

Measured compounds

Studies have sought an association between measured transport-related air pollution compounds and non-allergic respiratory morbidity. In the Netherlands, Steerenberg et al. (2001) compared children attending a school located near a busy motorway in Utrecht (mean black smoke levels: 53 µg/m^3) with children in suburban areas attending a school located in the middle of a green area (mean black smoke levels: 18 µg/m^3). The former showed significantly higher mean levels of inflammatory nasal markers (interleukin-8 (IL-8 – +32%), urea (+39%), uric acid (+26%), albumin (+15%), nitric oxide metabolites (+21%)), as well as a slightly lower (although not significant) peak expiratory flow (–5.3 ml/min). Associations of several inflammatory markers and peak expiratory flow with measured black smoke level were more pronounced in the urban than suburban children.

Janssen et al. (2003) reported on 24 schools located within 400 m of busy motorways in the Netherlands; the study included 2503 schoolchildren aged 7–12 years. PM2.5, soot and nitrogen dioxide were measured in the schools for a year. Non-allergic respiratory symptoms (such as current phlegm and current bronchitis) were increased near motorways with high traffic counts of lorries, not cars. The adverse effects on health were mostly restricted to allergic, sensitized or

bronchial-hyperreactive children. Lung-function changes and bronchial hyperreactivity, however, were not related to the pollution.

A questionnaire-based survey of 6109 adults who lived close to 55 centrally located air-quality monitoring stations was conducted in Sweden (Forsberg et al., 1997b). Exposure to sulfur dioxide (range of six-month averages: 1.7–16.0 μg/m^3) was most consistently related with cough, phlegm, and upper-respiratory symptoms. While exposure to nitrogen dioxide was related significantly to cough and throat or nose irritation, black smoke was not. The strongest associations (statistically significant for all outcomes considered), however, were found between self-reported exposures to traffic and self-reported respiratory health parameters.

A study of 5421 children aged 5–11 years in a 1-km^2 grid in Dresden, Germany, showed that all measured traffic-related pollutants (such as nitrogen dioxide, carbon monoxide and benzene) increased the risks of morning cough and bronchitis (Hirsch et al., 1999). Impaired lung function was seen only in children with high exposure to benzene, while bronchial hyperreactivity was lower in children exposed to higher levels of nitrogen dioxide and carbon monoxide (Hirsch et al., 1999).

A study of 843 children from 8 Austrian communities measured nitrogen dioxide as a traffic indicator (Studnicka et al., 1997). Increased prevalence of cough (apart from cold), but not bronchitis, was associated with high levels of nitrogen dioxide.

Two Norwegian time-series studies demonstrated an increased risk of hospital admissions for respiratory diseases in days with higher levels of benzene, formaldehyde and toluene – the air pollutants that come mainly from traffic (Hagen et al., 2000; Oftedal et al., 2003).

Modelled exposure

Studies using models have also sought an association between transport-related air pollution and non-allergic respiratory morbidity. Exposure to transport-related air pollution at residences was assessed by a combination of measurements and GIS-based models in the Dutch part of the EU-funded international project on the effect of transport-related air pollution on childhood asthma (TRAPCA). In the project, the Dutch birth cohort of 4000 children showed significantly increased risks of ear, nose and throat infections, as well as influenza (Brauer et al., 2002), but a weak association with cough and bronchitis. The German sister study in the TRAPCA project included nearly 2000 children living in Munich. Children with higher estimated exposure to transport-related air pollution showed a statistically significant increase in risk of cough without infections and dry cough at night, but not of respiratory infections (Gehring et al., 2002).

Exposure to soot, benzene and nitrogen dioxide was modelled using traffic counts and GIS data in 7509 children in Munich (Nicolai et al., 2003). Morning cough was more common in children with higher estimated exposure to these

pollutants. After adjusting for hereditary influences and environmental tobacco smoke (including during pregnancy), Pershagen et al. (1995) found positive associations between modelled outdoor nitrogen dioxide concentrations and the respiratory health of young children. The adjusted relative risk (RR) for wheezing bronchitis was 2.7 (95% CI: 1.1–6.8) for the category of high exposure among girls less than 48 months old. This association, however, was not found among boys.

In adults, in Toronto, Canada, modelled exposure to transport-related air pollution was associated with bronchitis, chronic obstructive pulmonary disease, pneumonia and hospital admission (Buckeridge et al., 2002). In Norway, Clench-Aas et al. (2000) found associations between self-reported symptoms and traffic-related pollutant concentrations, estimated by using a source-oriented air-pollution model; in this study, the risk of a number of symptoms was increased by 20–40% for an interquartile range in indicator pollutants: nitrogen dioxide or PM2.5. In a Dutch study, only mild dyspnoea was more prevalent in adults living along busy streets than in residents of quiet areas, while such associations were seen for most respiratory symptoms in children (Oosterlee et al., 1996).

A study by Northridge et al. (1999) on diesel exhaust exposure and lung function among 24 adolescents in Harlem, New York City, showed that 76% of the children had detectable levels of 1-OH-pyrene, a marker of diesel exhaust exposure, 13% (3 children) had a forced mid-expiratory flow ($FEF_{25–75\%}$) of less than or equal to 80% of their predicted measurements, and 17% (4 children) had 80–90% of the predicted value; no relation was apparent between $FEF_{25–75\%}$ and urinary 1-OH-pyrene levels. The authors suggested that further studies of larger numbers of adolescents in multiple sites were needed for a better understanding of the relationship between the burden of asthma (exacerbation, predisposition or both) and diesel exhausts.

Subjective and indirect indicators of exposure

A number of studies reported the associations of bronchitis and cough with different self-reported and surrogate indicators of exposure to transport-related air pollution. High exposure to transport-related air pollution was associated with:

- increased prevalence of bronchitis in children (Braun-Fahrländer et al., 1992) and adults (Nitta et al., 1993);
- cough in children (Braun-Fahrländer et al., 1992; Brunekreef et al., 1997; van Vliet et al., 1997; Ciccone et al., 1998) and adults (Nitta et al., 1993); and
- wheeze in children (Venn et al., 2001).

A few studies, however, did not find associations between traffic-related exposures and specific non-allergic symptoms, such as bronchitis and cough in

adults or children (Wjst et al., 1993; Lercher et al., 1995) and wheeze in children (Venn et al., 2000), or did not report effects for these symptoms, even though the data were probably collected. The associations between traffic-related surrogate variables and non-allergic respiratory outcomes are more consistent in children than adults.

Occupational exposure

Several studies found an increased risk of respiratory symptoms or disease in people with occupational exposure to vehicle exhausts. A study of highway tollbooth workers reported an increased number of acute irritative symptoms in exposed people, such as headache, nasal congestion, eye irritation and dry throat (Yang et al., 2002). Bus drivers, conductors and taxi drivers in Shanghai showed higher prevalence of respiratory symptoms and chronic respiratory diseases than controls not exposed to vehicle emissions (Zhou et al., 2001). The adjusted ORs were 1.95 for throat pain (95% CI: 1.55–2.46), 3.90 for phlegm (95% CI: 2.61–5.81), 1.96 for chronic rhinitis (95% CI: 1.11–3.46) and 4.19 for chronic pharyngitis (95% CI: 2.49–7.06).

A questionnaire-based study in Denmark (Raaschou-Nielsen et al., 1995) investigated the prevalence of respiratory diseases and other disease symptoms in street cleaners in Copenhagen. The street cleaners showed a significantly higher prevalence of chronic bronchitis and asthma than cemetery workers, who are exposed to lower levels of pollution and served as controls. In the statistical analysis, adjusted for smoking and age, ORs for chronic bronchitis (2.5; 95% CI: 1.2–5.1) and asthma (2.3; 95% CI: 1.0–5.1) were significantly elevated for street cleaners.

A five-year survey of Swiss customs officers investigated the adverse effects on health of occupational chronic exposure to diesel-engine emissions on respiratory mucous membranes (Glück et al., 2003). It showed that officers that cleared diesel lorries had significantly higher goblet cell hyperplasia, with increased metaplastic and dysplastic epithelia, and an increase in leukocyte counts.

Controlled studies of human exposure

Using particle concentrators, several studies evaluated the impact of exposure to transport-related air pollution on respiratory symptoms or on indicators of inflammation of the respiratory system. Ghio et al. (2000) exposed 38 healthy exercising subjects, for 2 hours, to either filtered air or concentrated ambient particles (CAPs) originating mainly from motor-vehicle exhaust (CAPs: 207 $\mu g/m^3$) in an exposure chamber connected to a Harvard particle concentrator. The subjects were unable to identify any increased symptoms following the exposures, and lung function was not changed. Bronchoscopy was performed at 18 hours after exposure, and cell counts displayed a mild increase in neutrophils in both bronchial and alveolar fractions in the people with greatest CAP exposure, as

compared with those exposed to filtered air. The concentrations of inflammatory mediators IL-6, IL-8, prostaglandin E_2, α_1-antitrypsin and fibronectin were unchanged. This combination of markers indicates mild airway inflammation. In the quest to identify the chemical components in the ambient PM responsible for inflammatory responses to the exposures, a sulfate/iron/selenium factor was reportedly associated with an increase in the percentage of bronchoalveolar lavage neutrophils. These chemical components are usually associated with other pollution sources than traffic (Huang et al., 2003).

In another study, the larger particles were filtered out and the remaining particles, mostly primary particles originating from motor vehicles, were concentrated. Healthy and asthmatic subjects were exposed to these fine CAPs during rest and exercise intervals (Gong et al., 2003). The average PM2.5 concentration during exposures was 174 μg/m^3 (range: 99–224 μg/m^3). Relative to filtered air, CAPs tended slightly to worsen cardiorespiratory symptoms four hours and two days after the exposure. The changes were generally small. Inflammatory cells were unchanged, as were concentrations of IL-6 and IL-8. Analysis of induced sputum showed a decrease of columnar epithelial cells by about 50% in healthy and asthmatic subjects at one day after exposure, suggesting that CAPs induced a direct epithelial response. This was not accompanied by an increase in inflammatory cells one day after exposure. Methods that use induced sputum to detect epithelial and inflammatory cell infiltration may be less sensitive than those that use bronchoscopies with lavages and biopsies. As compared with diesel exhaust exposures, the response of healthy subjects in this experiment may be slightly less pronounced (Nordenhäll et al., 2000). An earlier occurrence of a transient inflammatory response, such as that shown by Salvi et al. (1999), cannot be excluded.

Other studies investigated the effect of exposure to freshly generated diesel exhaust by inhalation, or to resuspended diesel exhaust particles by nasal instillation; the effects were assessed by studying indices of airway inflammation in induced sputum, bronchoalveolar lavage and bronchial mucosal biopsies. Four studies that used inhalation of diesel exhaust demonstrated increases in inflammatory parameters (a significant increase in neutrophils, and $CD4^+$ and $CD8^+$ lymphocytes) in bronchoalveolar lavage several hours after exposure (Rudell et al., 1990, 1994, 1996, 1999). Symptoms in the eyes and nose, and unpleasant smells, increased during exposures. Both airway resistance and specific airway resistance increased significantly during exposures to diesel exhaust, as compared with exposures to filtered air. The presence of a ceramic particle trap reduced the particle numbers almost by half. This reduction, however, was insufficient to produce any significant beneficial effect in the population of 12 healthy young people investigated (Rudell et al., 1996). A follow-up study (Rudell et al., 1999) demonstrated that diesel exhaust, as compared with exposure to air, significantly increased the total number of alveolar macrophages and reduced their capacity to ingest yeast particles in vitro; also, the number of neutrophils increased while

the number of $CD3^+$ and $CD25^+$ lymphocytes decreased. Statistically, the ceramic particle trap did not result in a significant reduction in the inflammatory response in the airway after exposure to diesel exhaust, compared with exposure to unfiltered exhaust. The insignificant trends all pointed towards a slightly smaller response after exposure to the filtered exhaust. Because the number of subjects in the study was relatively small, the power to detect a protective effect of the use of the ceramic particle trap was also small (Rudell et al., 1999).

In a subsequent study, healthy human subjects, performing intermittent moderate exercise, were exposed for 1 hour to diesel exhaust with 300 μg /m^3 PM. Bronchial wash and bronchoalveolar lavage demonstrated increases in neutrophils and B-lymphocytes, as well as increased secretion of histamine and fibronectin. Bronchial mucosal biopsies demonstrated significant increases in neutrophils, mast cells, and $CD4^+$ and $CD8^+$ T-lymphocytes; they also demonstrated up-regulated intercellular adhesion molecule 1 (ICAM-1) and vascular cell adhesion molecule 1 (VCAM-1), as well as increased leukocyte function-associated antigen 1 (LFA-1)-positive cells in the bronchial mucosal tissue. Moreover, significant increases in neutrophils and platelets were demonstrated in peripheral blood after exposure (Salvi et al., 1999, 2000). In a follow-up study, Stenfors et al. (2004) investigated a lower exposure concentration of diesel exhaust (108 μg/m^3 PM) in 24 healthy and 15 mildly asthmatic subjects. Diesel exhaust and filtered air exposures were performed in random order, at least three weeks apart. Both mildly asthmatic and healthy subjects showed increased airway resistance of similar magnitude. The healthy subjects showed signs of airway inflammation, with an increase in IL-8 protein and up-regulation of endothelial adhesion molecules. In contrast, the asthmatic subjects did not show any increase in neutrophilic or basal asthmatic inflammation. Instead, they experienced a fivefold increase in the expression of anti-inflammatory cytokine IL-10 in the bronchial epithelium, compared with a reduction by half in the healthy subjects. It was suggested that this cytokine in the asthmatic subjects might possibly enhance later IgE-production and thus enhance the T-helper type-2 cytokine (Th-2) response (Stenfors et al., 2004).

Nordenhäll et al. (2001) published a study supporting that assumption, in which a group of asthmatics who inhaled corticosteroids (on average, 1200 μg per day) were exposed under similar conditions to diesel exhaust with PM10 (300 μg/m^3) for an hour. The asthmatic subjects showed an increase in bronchial hyperresponsiveness 1 day after exposure. Bronchial hyperresponsiveness is a key marker and symptom of asthma and relates to its exacerbation. Consequently, this could link the association of enhanced symptoms and exacerbations in asthmatics with periods of higher concentrations of PM in ambient air.

Nightingale et al. (2000) demonstrated an alternative model for diesel exhaust exposure. Cyclone collectors at the exhaust of a stationary diesel engine accumulated exhaust. A commercial powder disperser was then used to resuspend

the collected diesel powder in an exposure chamber. Ten healthy, non-atopic, nonsmoking volunteers (mean age: 28 years), with normal lung function and absence of bronchial hyperresponsiveness, participated. The subjects were exposed to resuspended diesel particles and clean air, with a four-week interval, in a randomized sequence. The diesel particle concentration was approximately 200 μg/m^3 PM10 during the two-hour exposure. Sputum was induced at 4 and 24 hours after exposure. At the time of the first induction after exposure to diesel particles, induced sputum neutrophils and myeloperoxidase increased significantly, as compared with clean air. No changes in lymphocytes, eosinophils or epithelial cells were detected, and lung function and peripheral blood markers of inflammation were unchanged. The level of exhaled carbon monoxide almost doubled after exposure.

Controlled animal exposure studies

Controlled animal exposure studies have also sought an association between transport-related air pollution and non-allergic respiratory morbidity. In the United States, Clarke et al. (1999) treated healthy rats and rats with chronic bronchitis (induced by over 500 mg/m^3 sulfur dioxide) with CAPs from Boston air. The CAP treatment (206–733 μg/m^3 for 5 hours a day, for 3 days) induced a significant increase in tidal volume (the volume of air inhaled and exhaled at each breath) in both sets of animals and an increase in peak expiratory flow in the bronchitic animals. The CAP treatment also induced inflammation in both groups, as indicated by neutrophil, lymphocyte and protein content of lavage fluid measured 24 hours after the exposure. In a study by the same group, Saldiva et al. (2002) also treated healthy and bronchitic rats with CAPs from Boston air. Short-term exposure again induced a significant inflammatory reaction, as indicated by neutrophils in bronchoalveolar lavage, and this inflammation was dose dependent, but varied according to the CAP composition. Of the parameters measured, only vanadium and bromine concentrations correlated with both neutrophil counts in the bronchoalveolar lavage and in the alveolar walls at the bronchoalveolar junction and periphery. In another study, Zelikoff et al. (2003) treated aged rats with a single dose of PM2.5 CAPs (about 65 μg/m^3) from New York City and investigated the effect on them of infection with *Streptococcus pneumoniae*. The study identified no significant effect in healthy animals treated subsequently with the bacteria. Exposure of previously infected animals, however, led to an increase in bacterial burden and a decrease in the lavageable neutrophils and cytokines from the lungs.

Inflammatory cell-derived oxidants have been implicated in the mutagenic effects of particles (Driscoll et al., 1997), and hydroxyl radicals have been detected in the lungs of rats exposed to diesel exhaust particles (DEPs) (Han et al., 2001). There is also evidence of 8-hydroxy-2'-deoxyguanosine (8-OHdG) adducts in rats chronically exposed to DEPs (Jing et al., 1996; Tsurudome et al., 1999;

Iwai et al., 2000). The source of the hydroxyl radical may be Fenton chemistry (the chemistry that occurs when metal ions in water interact with peroxide), as a consequence of the metals associated with the particles or the accumulation of endogenous iron around the DEPs in the lung tissue (Ghio et al., 2000). Cassee et al. (2002) demonstrated that treating rats with concentrated, freshly generated diesel PM induced oxidative stress, as indicated by the raised glutathione content in the bronchoalveolar lavage fluid.

In studies with various minerals that may be found as components of road dust, Schwarze and co-workers demonstrated the inflammatory potential of stone particles with differing mineral and metal composition (Becher et al., 2001; Schwarze et al., 2002). Of the different stone particles studied, mylonite (median size: 8 μm) caused a stronger inflammation than did quartz 20 hours after exposure, while the mineral plagioclase in feldspar caused the least inflammation. This suggests that some mineral components of road dust present in PM10 could be important in mediating inflammation.

Mechanistic cell-culture studies

The mechanisms by which transport-related air pollution induces respiratory morbidity have also been investigated. Inhaled particles encounter the epithelial lining fluid, which contains antioxidants that may alter the effects of these particles. The antioxidants are depleted in the presence of at least some particles, though this has not yet been shown for urban-air particles (Zielinski et al., 1999). As with many other particles, CAPs induce oxygen radical-mediated lesions in cell-free DNA and intact cells, as measured by different methods (Donaldson et al., 1997; Smith & Aust, 1997; Prahalad et al., 2001; Knaapen et al., 2002; Shi et al., 2003). For PM10 or finer particles collected in different locations, non-enzymatic and enzymatic antioxidants reduced the formation of oxidants in most samples. Some studies showed that trivalent cations are associated with the oxidative effect (Upadhyay et al., 2003). In addition to metal ions, organic compounds (such as semiquinone radicals derived from the PM2.5 fraction) seem to be able also to contribute to redox (oxidation–reduction) cycling (Dellinger et al., 2001). Such compounds would be expected on combustion particles, including diesel particles. Two studies showed that the coarse fraction was more potent in damaging DNA than was the fine fraction, but antioxidants ameliorated the particle-induced effect (Greenwell et al., 2002; Shi et al., 2003). Both soluble and insoluble metals seemed to contribute to the formation of radicals. Samples from Hettstedt and Zerbst, Germany, varied in potency over different weeks of sampling, indicating variations in emissions and PM composition over time (Shi et al., 2003). Also, CAPs induced oxidative DNA damage in epithelial cells. The DNA damage was much greater in the presence of particles of residual oil fly ash than in the presence of the CAPs tested (Prahalad et al., 2001). In line with the temporary variations of oxidative potential in the Hettstedt and Zerbst samples,

different PM10 particles from Mexico City exhibited spatial variation of concentration-dependent DNA damage. The particles from the southern part of the city were the least potent, while CAPs from the northern part were the most potent (Alfaro-Moreno et al., 2002). Particles from all regions of the city were found to induce apoptosis in different types of cells. In macrophages, the apoptotic effect of standardized reference material (SRM) 1648 (St Louis particles) seemed to be mediated by the activation of scavenger receptors, not by the soluble fractions of the particles (Obot et al., 2002).

Pro-inflammatory mediators are involved in the development of inflammation, which is an important factor in many diseases. Different epithelial cells and macrophages exposed to ambient particles up-regulate ribonucleic acid (RNA) levels, protein release of pro-inflammatory mediators or both; the pro-inflammatory mediators include IL-8, tumour necrosis factor alpha (TNF-α), IL-1β, IL-6, monocyte chemoattractant protein 1 (MCP-1), granulocyte-macrophage colony-stimulating factor (GM-CSF) and ICAM-1 (Stringer et al., 1996; Kennedy et al., 1998; Fujii et al., 2001; Soukup & Becker, 2001; Huang et al., 2003). The effects of the particles seem to be related to soluble factors in some cases (Huang et al., 2003; Kennedy et al., 1998), but not all (Fujii et al., 2001). In some studies, the coarse fraction elicited a stronger response than the fine fraction. This response was most prominent with the insoluble coarse fraction and was to some extent attributable to endotoxin. Another study found a response to both endotoxin and soluble metal (Bonner et al., 1998). In other cells, however, the PM1.0 fraction elicited a stronger response than did the larger fractions (Huang et al., 2003). Both the coarse and fine fraction of CAPs collected near a busy highway in Downey, California decreased the ratio of reduced-to-oxidized glutathione in macrophages. Also, haem oxygenase 1 was induced by the particles, and this response seemed to depend on PAHs, rather than on metals (Li et al., 2002). Long et al. (2001) compared CAPs from the Boston area, sampled indoors and outdoors; they found that endotoxin appears to play a significant role in eliciting cytokine release, but other components may also be involved. Also, particles in indoor air tended to be more potent than those in outdoor air.

The in vitro data on combined exposure to microbial factors and particles do not render a clear picture. Particles still might increase an inflammatory response to microbes to a degree that damages the lung cells or inhibit the inflammatory response, and thus facilitate microbial attack.

DEPS sampled from different engines and the standard diesel particles, SRM1650, have been used in in vitro studies, such as those of Steerenberg et al. (1998), Takizawa et al. (1999) and Boland et al. (2001). Boland et al. (2001) observed that the SRM1650 particles and their own DEPs elicited similar effects on airway epithelial cells, while DEPs from an engine with an oxidation catalyst seemed less toxic. DEPS induced cell death in normal human bronchial epithelial cells, which were more sensitive to them than the other cell types tested. The

cytotoxicity of DEPs increased with decreasing glutathione content in the cells. Antioxidants, metal chelators and inhibitors of nitrogen oxide synthase reduced DEP cytotoxicity (Matsuo et al., 2003). A comparison of the effects of DEPs indicated that epithelial cells were less protected against oxidant damage than macrophage cells.

The organic fraction of DEPs has induced more cell death in epithelial cells than in macrophages. In macrophages, this response was partly reversed in the presence of the antioxidant N-acetylcysteine (Li et al., 2002). Both the aromatic and the polar fraction appeared to contribute to the response. PAHs, which are abundant on DEPs, increase oxidative stress in several different cell types (Burchiel & Luster, 2001; Garcon et al., 2001). Benzo[a]pyrene increased the oxidative DNA damage induced by ultraviolet light in two different mammalian cell types (Shyong et al., 2003). On the other hand, neutrophils amplified the formation of benzo[a]pyrene-DNA adducts in human blood neutrophils (Borm et al., 1997). PAHs were found to induce apoptosis in lymphocytes, thus exerting an immunosuppressive effect (Yamaguchi et al., 1997; Page et al., 2002). Apoptosis induced by benzo[a]pyrene metabolites seemed to depend on the activation of a receptor and induction of a PAH-metabolizing enzyme. In addition, protein kinases involved in both survival and death pathways inside the cell were activated (Chen et al., 2003; Solhaug et al., 2004). In another study, the apoptosis induced by PAHs was found to be distinguishable from clonal deletion, since some signal proteins involved in clonal deletion were not activated (Ryu et al., 2003). Suppression of mitogenesis of lymphocytes and inhibition of differentiation of monocytes to macrophages have also been reported. These effects seemed to be receptor dependent (Davila et al., 1996; van Grevenynghe et al., 2003).

DEPS and organic compounds from them have elicited the release of pro-inflammatory cytokines (IL-6, IL-8, GM-CSF IL-1β and eotaxin) from different types of epithelial cells and macrophages (Ohtoshi et al., 1998; Steerenberg et al., 1998; Boland et al., 1999; Bonvallot et al., 2001; Li et al., 2002; Takizawa et al., 2003). In contrast, macrophages (in BALB/c mice) and monocytes (RAW264.7) exposed to DEPs (300 $\mu g/m^3$) exhibited reductions in protein or RNA levels of TNF-α and IL-12, with no changes in IL-18 (Saito et al., 2002). Li et al. (2002) found that the effect of the extracts depended on the induction of metabolizing enzymes and that the concentration-dependent changes in IL-8 production were modulated by the induction of apoptosis in the epithelial cells. Bonvallot et al. (2001) demonstrated that GM-CSF production was elicited most strongly by the organic fraction of DEPs, while stripped DEPs exhibited only a small effect. The effect appeared to depend on a ROS-sensitive signal pathway. Upon exposure to benzene extracts of DEPs, an immortalized human bronchial epithelial cell line, BEAS-2B cells, produced increased amounts of IL-8 RNA and protein. Also, this study indicated the involvement of the transcription factor NF-κB and ROS (Kawasaki et al., 2001). A DNA microarray analysis revealed the increase of four

oxidant defence-related genes in macrophages exposed to extracts of DEPs. An increase in enzymes possibly related to DNA repair was also noted (Koike et al., 2002).

The finest particles in ambient air, the ultrafine particles, have received attention only recently. Ultrafine CAPs from Los Angeles were found to generate ROS in epithelial cells and macrophages and to induce haem oxygenase, an enzyme involved in defence against ROS. The ultrafine particles and, to a lesser extent, the fine ones localized to mitochondria, where they might cause further damage (Li et al., 2003). In the Netherlands, ultrafine particles (50 μg/ml) induced considerably less IL-6 release from macrophages than the coarse fraction, and less than the fine fraction. Ultrafine particles did not affect CD11b expression, yeast-induced oxidative burst and phagocytosis, while these functions were reduced in the presence of the coarse fraction and, to a lesser extent, the fine fraction. The effects of the coarse and fine fractions seemed to be partly mediated by endotoxin (Becker et al., 2003). Similar samples from Bilthoven, the Netherlands induced a concentration-dependent increase (up to 400 μg/ml) in IL-8 and IL-6 release from A549 cells. Although there was no significant difference between the size fractions' ability to elicit IL-8 release, the ultrafine particles were most potent in inducing IL-6 release. At higher concentrations, the coarse and ultrafine particles were apparently more toxic than the fine fraction. Surprisingly, ultrafine particles were not able to induce cytokine release from primary rat type-2 cells, in contrast to the coarse and fine fraction or St Louis dust (Hetland et al., 2004). In New York, human bronchial epithelial cells responded to ultrafine ambient particles (up to 100 μg/ml) with an increased release of GM-CSF (Reibman et al., 2002). The ultrafine particles appeared to exert a stronger effect than the larger size fractions, and the effect varied with collection period. Activation of protein kinases involved in survival and death signalling seemed necessary for the increased release of GM-CSF. Another study, using ultrafine carbon and epithelial cells, described increases in certain transcription factors and the involvement of factors associated with apoptosis (Timblin et al., 2002). Stone et al. (2000) observed that the ultrafine carbon effect was related to the influx of extracellular ionic calcium.

In some areas, the use of studded tyres leads to greatly increased abrasion of the road pavement, which results in substantial increases in PM10 and a much smaller increase in PM2.5. Most of the abrasion-generated PM consists of mineral particles (Hetland et al., 2000). Such particles include a variety of different minerals, such as quartz and amphiboles. Hetland et al. (2000) and Becher et al. (2001) showed that a mineral type, such as plagioclase, had little potential to induce the release of pro-inflammatory cytokines in different human and rat epithelial cells and macrophages. In contrast, stone types, such as mylonite and gabbro, and PM from a tunnel in which the pavement consisted of these stone types, were very efficient in eliciting pro-inflammatory responses. Though some of the minerals in these stones were rich in metals and produced some ROS, these

factors could not explain the differences in inflammatory potential (Hetland et al., 2001). Thus, some pavement abrasion particles may elicit inflammation in the lungs. Other known components of dust generated by road transport are tyre debris, including latex, and vehicle wear particles, but no in vitro information on them is available.

The antioxidant defence system of such compounds as certain vitamins and the radical-removing enzymes, such as superoxide dismutase, may be important markers of susceptibility. The growing evidence of the involvement of ROS in particle effects corroborates this notion. ROS, such as those generated by particles, might exert their effects at the cell surface by lipid peroxidation, through activation of nicotinamide adenine dinucleotide phosphate hydrogen oxidase or other enzymes, or stimulation of mitochondrial ROS production; in these two cases, the effects of ROS might be secondary to some other reactions. These reactions may involve the activation of certain cell surface receptors, downstream signalling through different types of protein kinases (such as tyrosine kinases and mitogen-activated protein kinases) and transcription factors (Samet et al., 1999; Sauer et al., 2001; Baulig et al., 2003; Brown et al., 2004). Secondary effects might be elicited, including autocrine effects of released mediators. The components of the surfactant are other factors that modulate the inflammatory response to particles influencing susceptibility (Hohlfeld et al., 2002–2003). Höhr et al. (2001) observed a reduced release of inflammatory cytokines, when epithelial cells and macrophages were exposed to particles in the presence of a phospholipid component of surfactant. Surfactant proteins are also deemed important for lung defence, and reduced release of these proteins would conceivably exacerbate pathological conditions in the lungs (Bridges et al., 2000; Hohlfeld et al., 2002–2003; Augusto et al., 2003). Oxidant gases (such as ozone and nitrogen dioxide) and ROS (hydrogen peroxide and ferrous chloride) have been shown to reduce the normal activity of surfactant proteins – either the antimicrobial activity of surfactant protein (SP) A/D or the reduction in surface tension by the concerted activity of SP-B, SP-C and SP-A (Putman et al., 1997; Wu et al., 2003).

Conclusions

In summary, rather substantial evidence points to transport-related air pollution's increasing the risk of non-allergic respiratory symptoms and suggests that inflammatory processes are related to exposure to such pollution. Fine PM (especially black smoke) and ozone were associated with the risk of morbidity, and similar outcomes were seen in studies that used different indicators of exposure. All size fractions of PM and different types of transport-related air pollution elicited inflammatory responses, which are associated with different diseases. In experimental studies, the organic fraction of DEPs appeared to produce more important reactive responses than did other types of particles. In general, however,

the different effects of various particle types could not be attributed to specific components of PM. Antioxidants and possibly surfactant components may be important determinants of susceptibility.

Allergic illness/symptoms including asthma

Measured and modelled exposure

Studies have sought an association between transport-related air pollution and allergic respiratory illness or symptoms. Using models for exposure, Brauer et al. (2002) detected associations with increased incidence of asthma in the first two years of life. Although not statistically significant, this association was in general robust, because the ORs were not altered to any great extent by the inclusion of potential confounding variables in the regression models or the sensitivity analyses. One must consider that the children observed were too young to have a reliable diagnosis of asthma, but the determination of wheeze, and its association with transport-related air pollution (also detected in this study), supports this diagnosis. The German part of the TRAPCA project, in which only a few asthma cases were reported, however, found no association of asthma incidence with transport-related air pollution (Gehring et al., 2002).

Considering wheeze as a possible key asthma-related symptom, and less related to doctors' diagnoses, does not make the picture clearer. While some studies found positive associations between traffic surrogate variables and the prevalence of wheeze in children (Oosterlee et al., 1996; Studnicka et al., 1997; Venn et al. 2001; Nicolai et al., 2003) and adults (de Marco et al., 2002), others did not (Hirsch et al., 1999; Wyler et al., 2000; Venn et al., 2000). The reasons for these inconsistent results remain unclear. Even within a single study, results on asthma and wheeze are inconsistent.

Other studies of adults also give inconsistent results. While some found increased reporting of asthma in subjects exposed to transport-related air pollution (Edwards et al., 1994; Duhme et al., 1996; van Vliet et al., 1997; Guo et al., 1999; Lin et al., 2002; Zmirou et al., 2004), others found no increased prevalence (Braun-Fahrländer et al., 1992; Nitta et al., 1993; Lercher et al., 1995; Waldron et al., 1995; Ciccone et al., 1998; Wilkinson et al., 1999).

No consistent association was found in the few studies that explicitly analysed associations between measured or modelled traffic exposure (or both) and hay fever. Studies in metropolitan areas (Hirsch et al., 1999; Lee et al., 2003) or urban/metropolitan areas (Krämer et al., 2000) found increased reporting of hay fever, in relation to a high level of exposure to transport-related air pollution, Several other studies conducted in large communities, however, reported no statistically significant associations with hay fever (Forsberg et al., 1997a; Wyler et al., 2000; Janssen et al., 2003; Nicolai et al., 2003). Even when the methods of the International Study of Asthma and Allergies in Childhood (ISAAC) were

applied in two cities in one country, the results on hay fever and traffic exposure were different (Hirsch et al., 1999; Nicolai et al., 2003).

With the review restricted to studies that used measured or modelled exposure to indicators of transport-related air pollution and allergic sensitization assessed by antibody measurements or skin-prick testing, the overall results remain inconsistent. While some studies reported a positive association between allergic sensitization and exposure to nitrogen dioxide (Krämer et al., 2000; Wyler et al., 2000; Janssen et al., 2003) others did not (Hirsch et al., 1999; Nicolai et al., 2003).

Controlled exposure studies

Important insights can be gleaned from a tunnel study in which subjects were exposed for a relatively short time to a high concentration of a real traffic-related pollution mix; 20 allergic asthmatic subjects were exposed during rest for 30 minutes in a busy city road tunnel, to study the effects of air pollution on allergen responsiveness (Svartengren et al., 2000). In the tunnel, the median levels of pollutants were 303 μg/m^3 nitrogen dioxide (range: 203–362 μg/m^3), 170 μg/m^3 PM10 (range: 103–613 μg/m^3) and 95 μg/m^3 PM2.5 (range: 62–218 μg/m^3). Four hours after exposure, an allergen provocation was performed and lung function responses measured. Subjects exposed to nitrogen dioxide levels of 300 μg/m^3 or more had a significantly greater early reaction following allergen exposure, as well as lower lung function and more asthma symptoms during the late phase, compared to the reference exposure. Subjects exposed to 100 μg/m^3 PM2.5 or more also had a slightly increased early reaction, compared to control subjects. It was suggested that the enhanced response of the asthmatics to the allergens in the tunnel was related causally to the nitrogen dioxide and PM2.5 content of the tunnel air pollution. A response to other potentially reactive compounds generated by motor vehicles is possible.

A series of studies by Diaz-Sanchez and colleagues have clearly indicated a modulating effect on the nasal mucosa by DEPs instilled at several hundred micrograms locally. Because the local deposition of PM per surface area in the nose is clearly high, the data are very interesting. Also, the data suggest that DEPs potentiate an allergy-related (Th-2) response, IgE production, and neo-sensitization in human nasal mucosa. Four days after a local nasal instillation challenge with 0.30 mg DEPs, a significant increase in nasal IgE, but not in other immunoglobulin classes, was demonstrated (Diaz-Sanchez et al., 1994). The number of IgE-secreting cells in nasal lavage also increased, but no increase was observed in IgA-secreting cells. Healthy, nonsmoking human volunteers were exposed to DEPs by intranasal instillation, and cytokines in the nasal lavage were estimated after 18 hours by an indirect approach using messenger RNA (Diaz-Sanchez et al., 1996). Before the challenge, most subjects had detectable messenger RNA levels of only a few cytokines (interferon gamma (IFN-γ), IL-2 and IL-13). After

the challenge, the levels of these three and a number of additional cytokines (IL-4, IL-5, IL-6 and IL-10) increased. An increase in such nasal cytokine expression after exposure to DEPs could again contribute to enhanced local IgE production.

Diaz-Sanchez et al. (1997a) also suggested that a nasal provocation with DEPs could act as an adjuvant to an allergen. In a group of ragweed-sensitive subjects, a nasal challenge was performed with 0.30 mg DEPs, the ragweed allergen *Amb a I* or both. The provocation with ragweed caused an increase in IgE and IgG4, as well as ragweed-specific IgE in the nasal lavage. A challenge with allergen and DEPs induced a sixteenfold greater increase in ragweed-specific IgE than DEPs alone. DEPS alone increased total IgE but, in combination with the allergen, antigen-specific IgE increased, as did the expression of Th-0 and Th-2 type cytokines: IL-4, IL-5, IL-6, IL-10 and IL-13. These studies suggest that DEPs can enhance B-cell differentiation. Fujieda et al. (1998) obtained similar results. A nasal challenge with a combination of DEPs and the allergen induced larger ragweed-specific IgE and IgG4 responses, compared with DEPs alone, but with similar total IgE levels (Diaz-Sanchez et al., 1997b). The cytokine pattern also changed, favouring allergic sensitization. DEPS and natural allergens may have a synergism that is a key to increasing allergen-induced respiratory allergic disease. Recently, this group demonstrated a role for genetic polymorphism in DEP-enhanced allergen response (Gilliland et al., 2004). This was associated with a polymorphism in the detoxifying enzyme glutation S-transferase M1 and suggests a clearer focus for new genetic aspects.

Chronic inflammation in mice, caused by multiple installations of DEPs, showed similarities to asthmatic inflammation – inflammation of the airway wall, goblet cell hyperplasia, eosinophils and airway narrowing – even in the absence of a concomitant allergic stimulus (Sagai et al., 1996). Concomitant exposure to DEPs enhanced the allergic inflammatory response in the airways of mice primed with ovalbumin (OVA); this appeared as increased local production of cytokines, such as IL-5 and GM-CSF, as well as increased infiltration of eosinophils and lymphocytes into the airways, along with goblet cell hyperplasia (Takano et al., 1997). Similar adjuvant effects for IgG and IgE were found with the more common allergen timothy grass pollen (Steerenberg et al., 1999). Anthracene, fluoranthene and benzo[a]pyrene in DEPs also enhanced the IgE response to model allergens (Kanoh et al., 1996). Studies with instilled carbon black and DEPs, and their effect on OVA-specific IgE and IgG in a rat instillation model, demonstrated that the particles are more important than the organic component, as the effect could be mimicked by carbon black (Al-Humadi et al., 2002). In studies of mice, both CD4+ and CD8+ lymphocytes drove these effects, since depletion of either subset decreases the extent of the adjuvant effect of DEPs on OVA allergy (Lovik et al., 1997). In the mouse footpad/popliteal lymph node model, the dominant role of particles over organics was again demonstrated, since carbon black had complementary qualities similar to those of DEPs, and yet a very

low level of organic contamination (Lovik, et al., 1997). Selective depletion of organics from DEPs resulted in complex changes in the ability of DEPs to affect Th-1 and Th-2 pathways following intranasal instillation, and different organic fractions modulated the two different pathways. Whole body plethysmography identified the role of GM-CSF in mediating DEP-induced airway hyperresponsiveness to acetylcholine in mice (Ohta et al., 1999). The hyperresponsive effect was mediated by muscarinic receptors and could be decreased by a beta-2-adrenergic agonist. DEPs may act as a carrier for allergens and deliver them to the lungs, and so enhance any adjuvant effects (Sagai, et al., 1996; Knox et al., 1997).

An exposure of basophils to DEP extract and an allergen resulted in an increase in IL-4 release. DEP extract and allergen were not synergistic in their effects, but the effect of the former lasted longer than that of the latter (Devouassoux et al., 2002). Kepley et al. (2003) did not observe a release of IL-4 from basophils when exposed to DEPs extract alone, but observed an increased release in the presence of PAHs, when the IgE receptors were cross-linked. The particle component and the organic fraction of DEPs may exert somewhat different effects, but combined they stimulate a Th-2-mediated response with the production of allergen-specific IgE and an eosinophilic inflammation (Ma & Ma, 2002).

Conclusion

Rather substantial evidence from controlled human exposure studies and animal experiments indicates that transport-related air pollution can increase the risk of allergy development and exacerbate allergic reaction. The evidence from population studies to support this observation is weak, however, because the results of several available studies that focus on transport-related air pollution are inconsistent. Further, identifying the component(s) of transport-related air pollution responsible for the allergic responses is still not possible, though nitrogen dioxide and ozone have been linked to these responses.

Lung function

Only two studies reported an association between lung function and transport-related air pollution. A Dutch study of children aged 7–12 years showed a significant association between lung function and the density of lorry traffic in the area of residence, but only a weak association with total traffic volume (Brunekreef et al., 1997). Living close to motorways and measured high concentrations of black smoke in schools were also associated with diminished lung function. These effects were stronger in girls than in boys. The ISAAC study of children in Dresden aged 5–7 and 9–11 years measured transport-related air pollution (nitrogen dioxide, carbon monoxide and benzene) in school areas and residential areas, based on pollution means in 1-km^2 grid cells, and found lower lung function in children with a high level of exposure to benzene, but no such association for carbon monoxide or nitrogen dioxide (Hirsch et al., 1999).

A few studies investigated the relation of bronchial hyperreactivity to surrogate variables for traffic-related pollutants, and found no positive associations. Hirsch et al. (1999) reported a statistically significant negative association between bronchial hyperreactivity and nitrogen dioxide and carbon monoxide in a large study, while a further study on children in Munich did not find any association between bronchial hyperreactivity and traffic volume in residential areas (Wjst et al., 1993).

Cardiovascular morbidity

Measured compounds

Several time-series studies assessed the association of daily changes of urban, mostly traffic-related air pollution with the variation in hospital admissions for cardiovascular diseases. These associations, with black smoke used as the pollution indicator, were especially evident in elderly people (Atkinson et al., 1999) in the hotter semester (Ballester et al., 2001). Poloniecki et al. (1997) did not find any detectable association with all circulatory diseases and angina, but observed a significant association with acute myocardial infarction. In eight large European cities in the APHEA2 project (Le Tertre et al., 2002), the pooled percentage increases associated with a 10-μg/m^3 increase in black smoke concentrations were 1.1% (95% CI: 0.4–1.8%) in cardiac admissions for people of all ages, 1.3% (95% CI: 0.4–2.2%) in cardiac admissions for people aged over 65 years, and 1.1% (95% CI: 0.7–1.5%) in ischaemic-heart-disease admissions for people over 65. The effect of black smoke was unchanged when the analysis controlled for carbon monoxide, but somewhat reduced when it controlled for nitrogen dioxide.

A case-crossover study of hospital admission for myocardial infarction in Rome showed statistically significant effects for exposure to nitrogen dioxide and marginally significant effects for exposure to carbon monoxide (D'Ippoliti et al., 2003). Transport is the main source of both pollutants in the city.

A study in Helsinki, where traffic is a major source of particulate air pollution, found that increases of fine and ultrafine PM concentrations increased the risk of ischaemia two days after exposure, as indicated by ST-segment depressions on electrocardiograms (ECG) (Pekkanen et al., 2002).

A recently published panel study of nine healthy state troopers in North Carolina investigated the possible physiological effects of in-vehicle, roadside and ambient PM2.5 before, during and after a patrol shift (Riedecker et al., 2004). In-vehicle exposure was associated with a decrease in lymphocyte count (–11% per 10 μg/m^3) and increased red blood cell indices (+1% mean corpuscular volume), increased neutrophils (+6%), increased C-reactive protein (+32%), increased von Willebrand factor (+2%), increased length of the heart beat cycle next morning (+6%), and increased heart rate variability and ectopic beats (+20%). The authors concluded that in-vehicle PM2.5 might cause pathophysiological changes

that involve inflammation, coagulation and cardiac rhythm. While the finding on blood inflammatory markers is quite consistent with previous results on the effects of air pollution, as summarized by the WHO Regional Office for Europe (2003), the results on increased heart rate variability are not.

Indirect exposure indicators

A recent paper assessed whether time spent in traffic can trigger myocardial infarction (Peters et al., 2004). This prospective case-crossover study included 691 subjects who survived myocardial infarction for at least 24 hours. The time spent in traffic one hour before the onset of myocardial infarction was associated with the increased risk (OR: 2.9; 95% CI: 2.2–3.8). Time spent in cars, public transport and on bicycles was consistently connected with an increased risk. The authors concluded that transient time spent in traffic might pose a risk to people vulnerable to myocardial infarction. Other traffic-related factors than transport-related air pollution, however, might have contributed to the elevated risk observed in this study.

Occupational exposures

A number of studies investigated the risk of coronary heart disease in professional drivers. This risk seems to be elevated, but the underlying causes are not well understood. Studies that specifically investigate the role in coronary diseases of occupational exposure to motor exhaust are rare. A case-referent study investigated the risk of myocardial infarction from occupational exposure to motor-vehicle exhaust, other combustion products, organic solvents, lead and dynamite (Gustavsson et al., 2001). The RR of myocardial infarction was elevated for people exposed to combustion products from organic material, but the previously demonstrated increased risk of myocardial infarction in professional drivers, especially bus drivers, could not be clearly confirmed. Individual risk factors and the psychological demands of the profession may explain a large part of the increased risk in this group, making it difficult to assess the additional risk due to the increased exposure to transport-related air pollution (Bigert et al., 2003).

Controlled exposure studies

Several recent studies examined the response of the cardiovascular system to exposure to CAPs. A significant increase in blood fibrinogen was observed in subjects exposed to CAPs, as compared with controls exposed to air, but no evident dose–response relationship was noted (Ghio, 2000; Harder et al., 2001). No effect on lymphocyte subsets in peripheral blood was found but increased blood fibrinogen was associated with a copper/zinc/vanadium factor. This gives some support to the hypothesis that metals, which are also present in transport-related air pollution, may be important in the reactive and cardiorespiratory responses to air pollution.

Gong et al. (2003) reported on controlled exposures to concentrated fine particles, mostly from motor vehicles, in healthy and asthmatic subjects. In summary, exposures to CAPs elicited different biological end-points, with statistically significant differences between CAPs and filtered air. The observed changes in blood parameters and heart rate variability were consistent with systemic (rather than respiratory) effects of exposure.

Brook et al. (2002) measured cardiovascular changes due to exposure to PM and ozone. The subjects were exposed for two hours on two separate occasions with at least a two-day interval between exposures; they were exposed once to filtered air and once to CAPs with about 150 $\mu g/m^3$ PM2.5 and 240 $\mu g/m^3$ ozone. Within 10 minutes after the exposure to the pollutants, a significant vasoconstriction occurred in the brachial artery, compared with the exposure to filtered air.

Controlled animal response studies

Gordon et al. (1998, 2000) used normal rats and rats treated with monocrotaline (MCT) to investigate the effects of CAPs on the pulmonary and cardiovascular systems. The CAP treatment induced an increase in the percentage of neutrophils and a decrease in the percentage of lymphocytes in the blood of both groups of animals, which is indicative of systemic inflammation. Neither group showed changes in cardiac rhythm, and the changes in cardiac function observed in the study were not statistically significant.

In experiments with dogs, Godleski et al. (2000) observed the effects of CAPs from Boston air on the cardiac system. The findings are complex, but in essence they suggest that CAPs disturbed autonomic balance, as indicated by changes in heart rate variability, either directly or through the respiratory system. The study also involved occlusion of the coronary artery and found that exposure to CAPs decreased the time from occlusion to ST-segment elevation in an ECG. The authors suggest that these changes could be caused by endothelial dysfunction, altered cardiac metabolism and responses to organic aerosol components. The study identified considerable variation between animals and in the same animal from day to day, possibly owing to variations in particle composition and dose.

Several experimental studies have shown that a small portion of ultrafine particles of various materials can enter the bloodstream, following deposition in the lungs (Nemmar et al., 2001, 2002; Kreyling et al., 2002). In an early study, chronic exposure to high levels of diesel soot produced marked thickening of the pulmonary artery wall in exposed rats, compared with controls (Vallyathan et al., 1986), although no mechanism was advanced. In a hamster model, instilled DEPs caused an increase in peripheral thrombus formation and platelet aggregation in vivo (Nemmar et al., 2003). The doses used produced pulmonary inflammation, so it was impossible to determine whether the mechanism was inflammation-driven or a consequence of bloodborne particles. Rats exposed by inhalation to resuspended diesel soot showed an elevation of plasma endothelin-3 at 36 hours

after exposure (Vincent et al., 2001); a similar exposure to carbon black had no effect. Studies have been conducted on rats pretreated with MCT, to induce a model of pulmonary hypertension. Such animals demonstrate hypertrophy of the media of the pulmonary muscular arteries, but this was not affected by concentrated, freshly generated, DEPs (Cassee et al., 2002). The MCT-treated animals did demonstrate an increase in bromodeoxyuridine labelling of Clara cells in the terminal bronchioles, which was enhanced by treatment with DEPs. This treatment also induced a significant increase in plasma fibrinogen (Cassee et al., 2002).

Accumulated experimental evidence suggests that combustion-related particles can penetrate to the bloodstream and can directly affect the cardiovascular system, but there is no evidence that particles specifically from transport-related air pollution become bloodborne. The effects of transport-related air pollution particles on the cardiovascular system could also be caused by inflammatory mediators that originate in the lungs. Such particles could also affect the regulation of the nervous system.

Conclusion

Only recent studies have focused on PM and explored the effects of exposure to transport-related air pollution on diverse indicators of cardiovascular diseases or functions of the cardiovascular system. While an epidemiological study reports a strong, significant increase of risk of myocardial infarction following exposure, other studies and the experimental evidence show changes in various cardiovascular parameters without providing a consistent explanation of the possible mechanisms involved.

Cancer

Measured or modelled compounds

Several Scandinavian studies on cancer used nitrogen dioxide or benzene as indicators of transport-related air pollution. In a study of Stockholm residents, Nyberg et al. (2000) found that the risk of lung cancer was associated with retrospectively assessed long-term exposure to transport-related air pollution, indicated by nitrogen dioxide averaged over 30 years. An RR of 1.2 (95% CI: 0.8–1.6) was found for the top decile of exposure, adjusted for tobacco smoking, socioeconomic status, residential radon and occupational exposures. The authors suggest a latency period, since the RR for the top decile of average exposure to traffic-related nitrogen dioxide 20 years earlier was higher: 1.4 (95% CI: 1.1–2.0).

Nafstad et al. (2003) reported the results of the analysis of data from a twenty-seven-year follow-up study on cardiovascular risk factors among 16 209 men (aged 40–49 years in 1972/1973) from Oslo. Those data were linked with information from the Norwegian cancer and death registers. To derive average annual air pollution levels at the participants' home addresses from 1974 to 1998, concentrations

of nitrogen oxides and sulfur dioxide from industry and from heating and traffic sources were estimated, using a combination of models and monitoring data. Controlling for age, smoking habits and length of education, the adjusted risk ratio for developing lung cancer was 1.08 (95% CI: 1.02–1.15) per 10-μg/m^3 increase in average concentration of nitrogen oxides at a home address between 1974 and 1978. The corresponding figure per 10-μg/m^3 increase in sulfur dioxide was 1.01 (95% CI: 0.94–1.08).

Studies of children are less consistent than those of adults in indicating the association of cancer risk with exposure to transport-related air pollution or air pollution in general.

A study by Feychting et al. (1998) reported an increasing risk of all types of cancer, leukaemias and tumours of the central nervous system with increasing indicators of exposure to transport-related air pollution, though the increase in risk was not significant in most exposure categories. The RR, however, achieved a statistically significant level (3.8; 95% CI: 1.2–12.1) in a small group of children with the largest exposure, indicated by a nitrogen dioxide level over 80 μg/m^3, compared with that for children exposed to transport-related air pollution below median concentrations.

For estimated exposures to benzene or nitrogen dioxide during pregnancy or childhood, a study by Raaschou-Nielsen et al. (2001) of Danish children did not observe an increase in the risk of developing leukaemia, tumours of the central nervous system or all selected types of cancer combined. The risk of lymphomas increased by 25% (P (for trend) = 0.06) and 51% (P (for trend) = 0.05) for a doubling of the concentrations of benzene and nitrogen dioxide, respectively, during pregnancy. Analyses by morphologic subtype showed that the increased risk of lymphomas associated with exposure to benzene and nitrogen dioxide in utero was restricted to Hodgkin's disease. In the adjusted analysis, the highest categories of exposure to benzene and nitrogen dioxide resulted in 4.3 and 6.7 times higher risks of Hodgkin's disease, respectively, than those of the lowest exposure category.

Indirect indicators of exposure

Harrison et al. (1999) studied living close to (within 100 m of) a main road or a petrol station in relation to the onset of childhood leukaemia in the United Kingdom. In comparison with the incidence of leukaemia in the general population, that among those living close to transport-related air pollution sources was slightly elevated: the RR was 1.16 (95% CI: 0.74–1.72) for those living close to a road and 1.48 (95% CI: 0.65–2.93) for those living close to a petrol station, but neither reached the level of statistical significance. In a recently reported Californian study, no increased cancer risks (for all cancer sites combined or leukaemia) were found among the offspring of mothers living in areas with a high density of traffic (Reynolds et al., 2004).

Occupational exposure

Most epidemiological studies on the adverse effects on health of occupational exposure to transport-related air pollution have been performed for professional drivers, such as lorry, bus and taxi drivers. A major population-based case–control study conducted in the 1980s in the United States investigated the association between employment with potential exposure to motor exhaust and the risk of bladder cancer (Silverman et al., 1986). A statistically significant trend in cancer risk with increased duration of lorry driving was observed. This risk was highest for lorry drivers and delivery staff, and lower for taxi and bus drivers, indicating, overall, a role for exposure to motor exhaust in bladder cancer etiology.

In another major study, a pooled analysis of two case–control studies on lung cancer in Germany investigated the association between diesel motor emissions and elevated risk of lung cancer (Brueske-Hohlfeld et al. 1999). An elevated OR adjusted for smoking was found for professional drivers (1.43; 95% CI: 1.23–1.67). An even higher risk was found for the subgroup of heavy equipment operators (OR: 2.31; 95% CI: 1.44–3.70). The risk of lung cancer for tractor drivers increased with duration of employment and reached statistical significance for exposures above 30 years (OR: 6.81; 95% CI: 1.17–39.51). Drivers in other occupations exposed to traffic-related diesel motor exhausts also showed an elevated OR for lung cancer (1.53; 95% CI: 1.04–2.24).

Several studies investigated the risk of cancer in Danish professional drivers. Based on a nationwide case–control study, Hansen et al. (1998) reported an increased risk of lung cancer among professional drivers. Taxi drivers showed the highest risk (OR: 1.6; 95% CI: 1.2–2.2); bus and lorry drivers had a somewhat lower but still increased OR value (1.3; 95% CI: 1.2–1.5). The risk of lung cancer increased with the duration of employment as a driver, with a highest OR of 3.0 (95% CI: 1.2–9.8) for more than 10 years as an active taxi driver. Another study, based on a large retrospective cohort (Soll-Johanning et al., 1998), reported an increased incidence of lung cancer in bus drivers and tramway employees (standardized incidence ratio (SIR): 1.6; 95% CI: 1.5–1.8). It also reported increased incidence of other types of cancer – such as pharyngeal (SIR: 1.9; 95% CI: 1.2–2.8), kidney (SIR: 1.6; 95% CI: 1.3–2.0), liver (SIR: 1.6; 95% CI: 1.2–2.2) and bladder cancer (SIR: 1.4; 95% CI: 1.2–1.6) – in bus drivers and tramway operators employed for more than three months. A more recent case–control study conducted by the same research group found a decreasing risk of lung cancer with increasing years of employment as a bus driver (RR: 0.97; 95% CI: 0.96–0.99), when the analysis controlled for confounders relevant to lung cancer risk, such as smoking (Soll-Johanning et al., 2003). Thus, taking the findings of the two studies together, one cannot attribute the excess risk of lung cancer observed among bus drivers to employment as such. For the other types of cancer for which increased risks were observed in the retrospective study (Soll-Johanning et al., 1998), no firm conclusion could be drawn based on the more recent case–control study, which investigated cases of lung and bladder cancer only.

Urban drivers – such as taxi drivers and short-haul lorry drivers – were reported to have a higher risk (RR: 2.0; 95% CI: 1.5–2.6) of lung cancer when they were compared with colleagues from rural areas of Sweden (Jakobsson et al., 1997). For the short-haul lorry drivers, the RR remained high, even after adjusting for smoking. The contribution of exposure to an increased risk of lung cancer was also indicated by a ten-year follow-up study on the mortality of lorry drivers (SMR: 160; 95% CI: 126–200) (Hansen, 1993), as well as by an exposure–response analysis, adjusted for smoking, among workers in the road-haulage industry (Steenland et al., 1998). The latter study was based on estimates of unknown past exposures and therefore should be characterized as rather exploratory. A recent major study on the incidence of cancer and mortality among lorry drivers exposed to diesel exhaust in Sweden (Jarvholm & Silverman, 2003) indicated an increased incidence of lung cancer (61 cases versus 47.3 expected) and prostate cancer (124 cases versus 99.7 expected). The higher mortality in the drivers was statistically significant for lung cancer only.

Many of the available epidemiological studies on the risk of cancer, particularly lung cancer, in professional drivers lack information on subjects' smoking habits; this complicates the interpretation of their results. The studies that could control for confounding by smoking (such as Damber & Larsson, 1985; Jakobsson et al., 1997; Brueske-Hohlfeld et al., 1999), however, indicate that the occupational exposure of drivers to exhaust fumes may affect the elevated risk of lung cancer.

Studies that investigated the links between transport-related air pollution and cancer included other professional groups besides drivers. Two major early studies investigated the risk of lung cancer from exposure to diesel exhaust among railroad workers (Garshick et al., 1987, 1988). Both indicate that such exposure may result in an elevated risk of lung cancer. A study of traffic police in Rome (Forastiere et al., 1994) suggested an increased risk of various cancer types: cancer of the colon, male breast, endocrine glands, bladder and kidney, and non-Hodgkin's lymphoma, but not lung cancer.

A study of another group, road construction workers, indicated a statistically significant increase in the risk of lung cancer (adjusted for smoking) in workers exposed to diesel engine exhaust (Brueske-Hohlfeld et al., 2000). Data from the Danish census in 1970 identified more than 4000 men (mostly petrol station attendants) as having indicated retail sale of oil and petrol as their employment. A significant excess of respiratory cancer (SMR: 158; 95% CI: 125–200) could be shown when compared with all men gainfully employed at the time of the census (Grandjean et al., 1991).

Occupational exposures and biomarkers

A few studies on the effects of exposure to air pollution have considered DNA damage as an end-point, particularly "bulky" DNA adducts, which are related to exposure to aromatic compounds, including PAHs. Studies in western Europe

have shown that the levels of white blood cell DNA adducts were higher among subjects heavily exposed to air pollutants. This has been observed in police officers (Peluso et al., 1998), newspaper vendors exposed to urban traffic (Pastorelli et al., 1996), residents in a highly industrialized area in the United Kingdom (Farmer et al., 1996) and bus drivers in Denmark (Nielson et al., 1996).

Based on the assumption that oxidative DNA damage might be involved in the increased risk of cancer associated with exposure to urban air pollution, a biomarker for oxidative DNA damage (CYP1A2) in bus drivers was determined (Loft et al., 1999). In a comparison between bus drivers from central Copenhagen and drivers from rural/suburban greater Copenhagen, the urban Copenhagen drivers excreted an increased amount of this biomarker, indicating that exposure to ambient air pollution may cause oxidative DNA damage.

A cross-sectional study among fuel system maintenance personnel showed that they had significantly higher counts of white blood cells, neutrophils and monocytes than a group with low exposure, after adjustment for relevant covariates (Rhodes et al., 2003). This indicative study, however, needs to be followed by results on whether a modulation of the immune system takes place in these people.

Experimental exposure studies

At sufficiently high levels of exposure, DEPs cause pathogenic effects, including cancer in rats (Mauderly et al., 1994; Nikula, 2000; Kato et al., 2000). It is plausible, however, that these effects are attributable to high levels of exposure and rat lung overload and therefore might not be generally applicable to relatively low levels of human exposure (ILSI Risk Science Institute Workshop Participants, 2000). Several studies have shown that filtering out the particles, but leaving the gases, abolished the carcinogenicity of diesel exhaust, clearly identifying a role for the particles (Nikula, 2000). Other studies, using carbon black as a surrogate for DEPs without their organic fraction, have found that the particles, not the organic fraction, are the main source of DEPs' carcinogenic (Nikula et al., 1995) and mutagenic (Bond et al., 1990) effects. DEPS with the organic fraction removed, however, caused fewer tumours than the parent DEPs in an instillation study (Dasenbrock et al., 1996).

Inflammatory cell-derived oxidants have been implicated in the mutagenic effects of particles (Driscoll et al., 1997). Hydroxyl radicals have been detected in the lungs of DEP-exposed rats (Han et al., 2001), and there is evidence of 8-OHdG adducts in chronically exposed rats (Jing et al, 1996; Tsurudome et al., 1999; Iwai et al., 2000). The source of the hydroxyl radical may be Fenton chemistry, as a consequence of metals associated with the particles or the accumulation of endogenous iron around the DEPs in the lung tissue (Ghio et al., 2000). Gallagher et al. (1993) found that diesel exhaust extracts, particularly nitrated PAHs and benzo[a]pyrene, formed DNA adducts in human lymphocytes. Cyclopenta-fused

PAHs, benz[l]aceanthrylene, benz[j]aceanthrylene and benzo[a]pyrene were metabolized in rat and rabbit primary lung cells, Clara cells, type-2 cells and macrophages, and formed DNA adducts in all these cell types from both species (Holme et al., 1993; Johnsen et al., 1997). Also, the lungs retain inhaled PAHs for a longer time when these compounds are associated with DEPs (Sun et al., 1984).

Air toxics are volatile toxic organic molecules found in air pollution in various circumstances. Those derived in greatest quantity from vehicle emissions are acetaldehyde, benzene, 1,3-butadiene and formaldehyde. All are considered to be carcinogenic in some animals and all are classified by the International Agency for Research on Cancer (IARC) as carcinogenic for human beings, with varying degrees of certainty. Considering ambient concentrations, however, the risk is rather low.

Conclusions

A wide range of studies indicates an increased risk of various types of cancer in people with prolonged exposure to higher levels of transport-related air pollution. Such effects have been measured or modelled mainly in subgroups that are susceptible or have higher levels of exposure than average, such as those with higher occupational exposure. A few studies estimating the general population's exposure to transport-related air pollution, however, suggest an increased incidence of lung cancer associated with increased exposure.

For certain occupational groups, such as professional drivers and railway workers, increased incidence of and mortality from lung cancer has been reported, and the increases are greater in people with long histories of exposure. Increased cancer rates were also observed in animals exposed to diesel exhaust.

Biomarker assessments in human beings and animals suggest that oxidative stress and DNA damage are linked to exposure to transport-related air pollution, which may play a role in the development of cancer. Both particles and semi-volatile compounds at current concentrations may affect the induction of cancer. For the volatile compounds, the ambient levels are in general so low that the risk of effects is low.

Pregnancy outcomes and male fertility

Pregnancy outcomes

Fetuses are considered to be highly susceptible to a variety of toxicants, because of their exposure pattern and physiologic immaturity (Perera et al., 1999; Šrám, 1999). Their developing organ systems can be more vulnerable to environmental toxicants during critical periods, due to higher rates of cell proliferation or changing metabolic capabilities.

Several studies have shown the adverse effects of ambient air pollution on pregnancy outcomes, including an increase in post-neonatal infant mortality

(WHO Regional Office for Europe, 2004b). Only a few studies, however, have sought more specific associations between these outcomes and transport-related air pollution.

Modelled exposure

In Seoul, the Republic of Korea, Ha et el. (2001) examined the associations between low birth weight and exposure to transport-related air pollution at the mother's residence during pregnancy. The adjusted RR of low birth weight was 1.08 (95% CI: 1.04–1.12) for interquartile increase in carbon monoxide concentration during the first trimester of pregnancy. The RRs were 1.07 (95% CI: 1.03–1.11) for nitrogen dioxide, 1.06 (95% CI: 1.02–1.10) for sulfur dioxide and 1.04 (95% CI: 1.00–1.08) for total suspended particles – all for interquartile increases in exposure. Also, several studies conducted by Ritz and colleagues in Los Angeles indicate that the risk of an adverse outcome at birth, such as premature birth and low birth weight, may be affected by exposure to transport-related air pollution, as indicated by the distance-weighted traffic density (Ritz & Yu, 1999; Ritz et al., 2000; Wilhelm & Ritz, 2003).

Experimental exposure studies

Diesel exhaust fosters abnormal delivery in pregnant mice and affects the growth of their young. Tsukue et al. (2002) reported that exposure of pregnant female mice to 0.3 mg/m^3, 1.0 mg/m^3 and 3.0 mg /m^3 DEPs resulted in 9.1%, 10.0% and 25.0% abnormal deliveries, respectively. The offspring of exposed females showed significantly lower body weights at the ages of 6 and 8 weeks, and delayed sexual maturation. Watanabe & Kurita (2001) exposed female rats to diesel engine exhaust from day 7 to day 20 of pregnancy and observed that the differentiation of the testis, ovary and thymus was delayed and disturbed. Maternal testosterone and progesterone levels were significantly higher in the exposed pregnant rats than in controls.

Male fertility

Levels of transport-related air pollution may also affect male fertility, although the number of studies that address this hypothesis is rather small. De Rosa et al. (2003) compared male motorway toll-gate workers with men living in the same area. The results showed that sperm count and serum levels of follicle-stimulating hormone, leuteinizing hormone and testosterone were within the normal range in both groups, while total motility, forward progression, functional tests and sperm kinetics were significantly lower in the toll-gate workers than in controls. The finding that blood methaemoglobin and lead were inversely correlated with sperm parameters indicates that nitrogen oxide and lead may adversely affect semen quality.

In experimental studies, Fredricsson et al. (1993) exposed human spermatozoa to DEP extracts and found that the pollutant interfered with sperm motility in a

dose–response fashion. In male mice, daily sperm production per gram of testis decreased dose dependently when the animals were exposed to diesel exhaust for six months (Yoshida et al., 1999). A study by Watanabe & Oonuki (1999) indicates that spermatogenesis in growing rats was inhibited after exposure to diesel engine exhaust. In exposed animals, serum levels of testosterone and estradiol were significantly higher, and sperm production and activity of testicular hyaluronidase were significantly reduced.

Conclusion

There is evidence that implicates ambient air pollution in adverse effects on pregnancy, birth outcomes and male fertility. Modelled studies on exposure to traffic-related air pollutants suggest that they are a risk factor for adverse birth outcomes, but further studies are needed that estimate this exposure more precisely, in terms of both pollution components and timing.

Intervention studies

Intervention studies address the health benefits of improvements in air quality and provide useful information to decision-makers and air-quality managers. Unfortunately, these studies are very rare, particularly when restricted to the adverse effects on health related to transport. The few examples that exist can be classified as describing short-term and long-term interventions.

Short-term changes in air-pollution levels

The implementation of a modified transport strategy, to reduce traffic congestion during the 1996 Summer Olympic Games in Atlanta, Georgia, United States provided the opportunity to study the health impact of a short-term change in levels of transport-related air pollution (Friedman et al., 2001). For a total of more than 10 weeks (4 weeks before, 17 days during and 4 weeks after the Games), data were registered for: the number of medical emergency visits, the number of hospitalizations for asthma and non-asthma events, air quality, weather conditions, and traffic and public transportation. The air-quality data included measurements of PM10, nitrogen dioxide and ozone.

The results of the analysis show a significant decrease in the number (41.6%) and incidence of acute care events for asthma (RR: 0.48; 95% CI: 0.44–0.86) during the Olympic Games. In the same period, air quality improved, with significant reductions in ozone (from 163 $\mu g/m^3$ to 117 $\mu g/m^3$ mean of one-hour daily maximum), carbon monoxide (from 1.80 mg/m^3 to 1.47 mg/m^3, eight-hour means) and PM10 (from 36.7 $\mu g/m^3$ to 30.8 $\mu g/m^3$ daily mean) concentrations. The peak weekday morning traffic counts were reduced by 22.5% from the baseline period. The peak daily ozone concentration was significantly correlated with traffic counts (correlation coefficient $r = 0.36$).

These results suggest that reductions in car emissions and the associated ozone and PM levels, resulting from changes in city transportation systems can prevent disease and lead to a reduced number of asthma exacerbations that require medical attention. This study, however, has some noticeable weaknesses that should be considered. They include low statistical power (only one of the four sources of medical data taken into consideration provided statistical significance), a limited number of air-pollution monitoring sites and a non-optimal traffic counting system. Moreover, due to the high correlation between the levels of PM10 and ozone, it is impossible to determine which pollutant's reduction was responsible for the decrease in asthma events, although ozone levels were reduced further (28%) than that PM levels (16%). The study supports the general perception that the change in car emissions contributed to the improvement in air quality – not the weather conditions, which remained relatively constant during the observation period.

Despite the limitations of the study, the immediate implications are of major importance and indicate the need to introduce changes in traffic that improve air quality and reduce the morbidity associated with air pollution.

Long-term changes in air-pollution levels

Very few published intervention studies assess the health impact of long-term changes in traffic-related air pollution.

Regulation of traffic

Various long-term changes can be made to reduce the adverse effects on health of exposure to transport-related air pollution. One increasingly used option is the regulation of traffic by, for example, building tunnels, diverting traffic to different routes, constructing roundabouts and regulating speed. Environmental evaluation reports often document the impact of traffic regulation in terms of changes in air-pollution levels, living conditions and the well-being of local residents. The number of published epidemiological studies that assess the effect on health of traffic regulations is very small, however.

A few studies have been published in connection with the construction of two tunnels in Norway. They were constructed to reduce the effect of traffic on the urban environment in Oslo. Bartonova et al. (1999) used a dispersion model to estimate the effect of these changes on the residents' levels of exposure. After both tunnels were in use, the average exposure to nitrogen dioxide decreased from 51 $\mu g/m^3$ to 40 $\mu g/m^3$ (Bartonova et al., 1999). Another study investigated the effect of the tunnels on the self-reporting of symptoms of reduced health and on the well-being of adults living in Oslo (Clench-Aas et al., 2000). The decrease in the levels of nitrogen dioxide reported by Bartonova and co-workers was related to a decrease of about a 5–10% in the risk of being bothered by fatigue (Clench-Aas et al., 2000). Finally, Klaeboe et al. (2000) studied the combined effect of

changes in air pollution and noise level on annoyance, caused by the construction of these tunnels. This study indicated that the higher the levels of road-traffic noise to which people are exposed, the more likely they are to be annoyed by the smell of traffic exhaust at a specified level of air pollution.

Changes in fuel composition

Improving technology, such as emission controls or changes in fuel composition, is another option that might benefit health. In 1990, a fuel restriction was introduced in Hong Kong, requiring that all power plants and road vehicles use fuel oil with a sulfur content of not more than 0.5% by weight. In the first year after introducing this intervention, the mean reduction in sulfur dioxide was 53%, and this reduction was sustained at 35–53% for 5 years (Hedley et al., 2002). No significant change was noted in mean PM10 and nitrogen dioxide concentrations, but a significance increase in ozone was noted over this period (Hedley et al., 2002).

Studies examined the effect of this intervention on the differences between two districts in changes in bronchial responsiveness (Wong et al., 1998) and in immediate and long-term health benefits (Hedley et al., 2002). A comparison of measurements made before the intervention with those made a year later showed that both the bronchial hyperreactivity slope and the bronchial reactivity slope declined from 29% to 16% and from 48% to 39%, respectively, in the polluted district, and from 21% to 10% and from 42% to 36%, respectively, in the less polluted district (Wong et al., 1998). Comparing measurements made in 1991 (a year after the intervention) with those in 1992, only the polluted district showed a significant decline, from 28% to 12% and from 46% to 35% for bronchial hyperreactivity and bronchial reactivity slopes, respectively (Wong et al., 1998). Also, an immediate reduction in cool-season deaths was reported, which suggests that, in the first year, many people survived who would have otherwise died (Hedley et al, 2002). The intervention led to significant declines in the average annual trend in deaths from all causes (2.1%; $P = 0.001$), respiratory causes (3.9%; $P = 0.0014$) and cardiovascular diseases (2.0%; $P = 0.0214$) (Hedley et al., 2002). Reductions in risks for overall mortality were greater in districts that had large reductions in sulfur dioxide than in those that did not (Hedley et al., 2002). Differences in age-specific death rates before and after the intervention suggest that it resulted in an average gain in life expectancy of 0.73 years for men aged 25–100 years, because of a 10-μg/m^3 reduction in exposure to sulfur dioxide for 15 years (Hedley et al., 2002).

Since the 1970s, a series of regulations on the lead content of petrol has been adopted. Modelled data from a recently published German study (von Storch et al., 2003) shows that atmospheric lead concentrations in Europe increased heavily until the 1970s and fell strongly in the following years, largely because of the reduced lead content of petrol. Many studies investigated the impact of

this fuel change on levels of lead in blood. Thomas et al. (1999) examined 17 of these studies, and found a strong linear correlation between lead concentrations in petrol and lead levels in blood. They also reported that lead levels in blood in the population decreased from the late 1970s to the early 1990s, and that average levels in the population of about 30 µg/l are widely achievable.

A more recently published study (Lou et al., 2003) provides additional support for the beneficial effects of this change in fuel composition on levels of lead in blood in China. In 1998, petrol stations in the city of Shantou were prohibited from selling leaded petrol. The effects of this intervention on the levels of lead in children's blood were investigated for three consecutive years, when the average levels declined from 104 µg/l in 1999 to 94 µg/l in 2000 and 79 µg/l in 2001. These decreases were all statistically significant. The current standard elevated level of lead in blood for children, set by the Centers for Disease Control and Prevention in the United States, is 100 µg/l. Lou et al. (2003) found that the percentage of children in Shantou with levels above this standard was reduced by 35.8% in 2000 and 23.0% in 2001. These numbers indicate that prohibiting the sale of leaded petrol in the city had beneficial effects. With regard to transit traffic, the implementation of similar regulations in other parts of China might reduce these numbers even more. Nevertheless, one should remember that elevated levels of lead in blood can have other sources, although automobile emissions from the combustion of leaded petrol have been recognized as one of the major sources of widespread environmental lead contamination.

Relocation to less polluted areas

A more invasive way to protect human health against transport-related air pollution is for people to move from more to less polluted areas. Within a large follow-up study in southern California, 110 children changed their place of residence, providing an opportunity to investigate whether changes in air quality due to relocation were associated with changes in growth rates of lung function (Avol et al., 2001). As a group, subjects who had moved to areas of lower PM10 showed an increased growth rate of lung function; those who moved to communities with higher PM10 showed a decreased growth rate. A stronger trend was found for subjects who had migrated at least 3 years before the follow-up visit than for those who had moved in the previous 1–2 years (Avol et al., 2001).

Conclusions and outlook

Intervention studies can provide valuable information that aids in understanding the epidemiology of respiratory disease associated with air pollution and in evaluating the observable health benefits of air-quality regulations and control measures, and the emission sources involved. As stated earlier, such studies are unfortunately rare, and more should be encouraged (for example, HEI, 2003). The only short-term intervention study performed so far prudently suggests that

a reduction in transport-related air pollution may have a direct health benefit in reducing acute asthma attacks and related medical care, as observed in children. The intervention studies of long-term changes reported several health benefits, such as a decline in bronchial hyperactivity, a decline in the average annual trends in deaths from all causes and from respiratory and cardiovascular diseases, and a gain in life expectancy. Owing to the limited amount of evidence, however, one should still be reluctant to draw firm conclusions about the health benefits of these particular changes.

Discussion

The available scientific evidence suggests that transport-related air pollution affects several health outcomes, including mortality and respiratory morbidity. This evidence is supported by both epidemiological studies using various designs and experimental studies (Table 4.1).

In the numerous epidemiological time-series studies conducted in urban areas, mortality and morbidity were associated with black smoke. Especially for diesel exhaust, black smoke is likely to be a good indicator of transport-related air pollution. In studies on the adverse effects on health of chronic exposures to transport-related air pollution, it was also a good predictor of mortality and morbidity. Black smoke, however, is not the sole indicator associated with effects on health. Others – such as nitrogen dioxide, carbon monoxide, sulfur dioxide and different indicators of PM – were also correlated with the adverse effects studied. The various pollutants examined are often highly correlated with each other. This is the case for black smoke, carbon monoxide and nitrogen dioxide, and, to a lesser extent, PM10 and PM2.5. The fraction of the air pollution mixture emitted by traffic that adversely affects health therefore remains unclear.

One strength of time-series studies of daily mortality and morbidity is that they use daily concentrations that are widely, consistently and, for the most part, completely recorded. Although these studies have been extremely helpful in assessing the role of air pollution in acute effects on health in urban populations, they are less helpful in quantifying the role of traffic, which they were seldom designed to do. Most of the studies measured air pollution at a single site and made no distinction between spatial variation in levels of traffic-related air pollution or exposure. Sometimes, however, these studies can help improve the understanding of the role of transport-related air pollution in acute effects on health. A good example is the finding in the APHEA2 study that the slope of the relationship between PM and health was higher in areas with relatively high nitrogen dioxide concentrations, providing some evidence of the enhanced toxicity of PM emitted by mobile sources.

A few panel studies conducted in urban and rural areas suggest that adverse effects on health (symptoms, reduced lung function, medication use) were associated with transport-related air pollution. Most of them made additional air pollution measurements, which better represent the real exposure of study

Table 4.1. Summary of health studies of transport-related air pollution

Health outcome	Population studies		Experimental studies		Comments
	Pollutant	Evidence	Pollutant	Evidence	
Mortality	Black smoke Ozone PM2.5	Some Some Some	None	None	No experimental studies
Respiratory diseases (non-allergic)	Black smoke Ozone Nitrogen dioxide VOCs	Some Some Some Some	CAPs Diesel Nitrogen dioxide Ozone	Strong Strong Strong Strong	Strong experimental evidence for inflammatory effects for several pollutants at relatively high concentrations
Respiratory diseases (allergic)	Ozone Nitrogen dioxide VOCs	Some Some Some	PM CAPs Diesel Nitrogen dioxide	Some Some Strong Some	Strong experimental evidence for allergy enhancement by DEPs; growing similar evidence for CAPs (experiments use relatively high concentrations of pollutants)
Cardiovascular diseases	Black smoke	Some	CAPs	Some	Some experimental evidence for cardiovascular effects
Cancer	Nitrogen dioxide Diesel exhaust	Some Some	Diesel exhaust VOCs	Some Equivocal	None
Reproductive outcomes	Nitrogen dioxide Carbon monoxide Sulfur dioxide Total suspended particles	Equivocal Equivocal Equivocal Equivocal	Diesel exhaust	Some	None

participants than do the data from existing measurement sites. Another advantage of this design, compared with time-series studies, is that the statistical analyses could compensate for the individual potential confounders.

A substantial part of current knowledge about the health effects of transport-related air pollution comes from cross-sectional or cohort studies, in which exposure to indicators of transport-related air pollution were measured, modelled or assessed by questionnaire, or indirect indicators of transport-related air pollution (such as distance to nearest street or traffic count) were used. Because long-term epidemiological studies were often started for reasons other than assessing the effects of air pollution or its transport-related portion, they are not designed optimally for contrasts in exposure and for end-point and confounder information.

Some studies were designed to achieve a contrast in exposure to transport-related air pollution. It is therefore likely that differences in health between study subjects can be attributed to differences in this exposure. Although such studies address the issue of the role of road traffic better than time-series and panel studies, they have other limitations.

Cross-sectional studies that used subjective, self-reported exposures or measures of health effects may suffer from responder bias, resulting in positive associations between exposure and adverse effects on health. Studies that use measured or modelled exposures and objective measures of adverse effects are less influenced by responder bias. Another limitation of cross-sectional studies is the absence of historical data on exposure, which limits the possibility of establishing the temporal order of cause and effect.

Important information on the association between disease and exposure to the products of fossil-fuel combustion can be extracted from epidemiological studies on groups with occupational exposure. Though other factors may also contribute to the increased risk in these groups, the studies consider air pollution from motor-vehicle emissions an important risk factor for coronary and respiratory diseases, as well as for several types of cancer.

Toxicological studies provide supportive arguments for the peril from transport- or traffic-related air pollution. They report the expression of many biological and pathological responses and, in some cases, the onset or exacerbation of disease. The lowest concentrations studied, however, are still in the range of episodes of high concentrations of pollutants. Also, the human subjects in the studies do not represent the most susceptible subpopulations, and extrapolations from animal and cell studies are difficult. Thus, the studies do not provide exposure–response functions, and it is unclear whether more susceptible individuals may experience effects at average ambient concentrations. Modified animal and cell-culture models could be used to a greater extent to investigate the effects of lower concentrations and the influence of pre-existing disease.

Overall, the toxicological studies lend support to the epidemiological findings. Several pollutants that show associations in epidemiological studies on adverse

effects on health also provoke biological or pathological reactions, or both, in experimental settings. These reactions are involved in the development or exacerbation of diseases investigated in epidemiological studies. Inflammation is an important response, often investigated in toxicological studies of the effects of air pollution. Though inflammation is a useful process for eliminating intruding pathogens, it may also cause damage to surrounding tissue and thus exacerbate pre-existing disease, if not resolved by a defensive process of the tissue. In particular, subjects with chronic lung disease would be expected to experience a negative effect from increased inflammation. Also, although the epidemiological evidence is weak in suggesting the involvement of air pollution in the development and exacerbation of respiratory allergy and allergic asthma, the toxicological results suggest such an involvement.

Toxicological studies have also observed cardiovascular effects, but the evidence here is just accumulating. The relative importance of possible pathways of adverse effects on health has not been clarified. Whether the cardiovascular effects are mediated through inflammation of the lungs, stimulation of the nervous system or a direct effect (for example, of ultrafine particles on the blood or heart) still remains to be elucidated. Toxicological studies of cancer have indicated the importance of diesel exhaust and PAHs at high concentrations and thus support epidemiological studies. Only the latter studies, using indicators, however, have found associations between relatively low levels of ambient pollution and cancer.

Attempts have been made to clarify which component of PM plays the most important role in eliciting adverse effects on health. The studies on this problem have been unable to single out specific components responsible for specific responses, except for the ability of PAHs to cause DNA damage and cancer. The diverse results show, depending on the particles sampled, the involvement of different components; this may indicate that a particular component, common in all or many types of particles, will not be found. The production of ROS seems to be important in the responses observed; in connection with this, the antioxidant status of the lung seems to be one crucial factor of susceptibility to effects in this organ. Other important factors may be lung SPs. Further, allergic asthmatics and people with a pre-existing cardiovascular disease may be at higher risk than healthy people. The data on likely or possible susceptibility traits are still far from sufficient, however, and there is still insufficient data on the importance of age.

Are the pollutants from traffic unique in exerting their effects or are their effects in keeping with those of pollutants from other sources? A number of studies on pollution from other sources have been carried out, including those on quartz and asbestos in occupational settings, residual oil fly ash, pollution from several industrial areas and pollution from domestic heating. Several sources produce the same components of pollution as those produced by traffic, but with quantitative variations. The greatest differences seem to be found in the composition of

particle fractions from different sources. Though there are many differences in the effects of particles from these sources, some principles seem to prevail between all of them. The formation of ROS seems to be an important factor, and has been tied to the activity of some surface or soluble element. It is linked to increased inflammation, which again is associated with disease. Though composition may vary considerably, the effect of PM pollution seems to change relatively little across many studies, even though some differences in estimates of effects between different cities have been reported, for example, in the APHEA studies. Also, the health benefits observed from reducing pollution from non-traffic sources – shown for example in studies in Utah Valley, United States and Dublin, Ireland (Pope et al., 1996; Clancy et al., 2002) – have to be considered.

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5. Health risk assessment of transport-related air pollution

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Identified health effects

The evidence from accumulated research has enabled the assessment of the health effects of ambient air pollution, including quantitative estimates of the burden of the pollution on a global scale (WHO, 2002) and on individual communities (Medina et al., 2002). These estimates indicate that about 100 000 deaths a year could be linked to ambient air pollution in cities in the WHO European Region, shortening life expectancy by an average of a year. The number of casualties attributed to air pollution is comparable to the number of fatalities from traffic accidents, and its imprint is observed in all age groups, including children (Valent et al., 2004).

A large part of the disease burden caused by non-fatal diseases attributable to air pollution is more difficult to quantify, but these diseases probably add substantially to the total burden of pollution-related ill health, especially in children. These analyses of adverse effects on health used exposure to PM10 or PM2.5, measured by monitoring networks or estimated by models, and attributed the effects to the total pollution load generated by all possible sources. Ozone, created in the atmosphere from gases produced largely by transport, also affects mortality, morbidity and the development of the lungs.

Traffic contributes substantially to PM and ozone pollution and to population exposure, but precisely quantifying transport's contribution to total exposure and its adverse effects are still difficult tasks. The review presented in this book clearly identifies the hazardous nature of transport-related air pollution, but also presents a variety of factors that may affect exposure and the attribution of the observed adverse health effects to pollution from traffic sources.

The effects of transport-related air pollution on health include an increased mortality risk, due particularly to cardiopulmonary causes, and an elevated risk of respiratory morbidity. Further, elevated incidence of non-allergic respiratory symptoms and diseases due to traffic, accompanied by extended non-allergic inflammation in the respiratory tract, has been reported. Exposure can exacerbate

allergic reactions in asthmatics. As to cardiovascular morbidity, exposure has been shown to increase the risk of myocardial infarction. In addition, evidence implicates air pollution in adverse outcomes of pregnancy, such as premature birth and low birth weight. There is insufficient evidence to draw firm conclusions about the association of transport-related air pollution with elevated incidence of cancer, although certain occupational groups with higher- and longer-than-average levels of exposure (such as professional drivers and railway workers) show an increased incidence of and mortality from lung cancer. The risks are not equally distributed in the population. Children and elderly people and people with pre-existing chronic diseases show increased susceptibility to the adverse effects of air pollutants.

Needs for further research

All this information identifies the hazard sufficiently, but is still too inconsistent to derive a well-based exposure–response function, which is needed to quantify precisely the adverse effects of transport-related air pollution. The epidemiological studies available used different exposure and outcome indicators, which limits the possibility of comparing their results quantitatively and estimating a common risk function, as has been done for studies of ambient air pollution (Anderson et al., 2004).

Also, it is still difficult to estimate the population's exposure to transport-related air pollution, which limits the precision with which the effects can be quantified. Estimating this exposure as correctly as possible requires knowledge of where people spend their time and what pollution levels prevail in these microenvironments. The quantity of passing traffic, the distance from a road to a residence, weather conditions and time spent in different traffic modes all contribute to the overall level of exposure. Elevated health risks are expected for people living and working near busy roads or travelling/commuting in heavy traffic, or both. Also, the intake of pollutants varies among such road users as drivers, bicyclists and pedestrians. Most commonly used estimates of levels of population exposure are based on fixed monitoring sites and do not reflect the spatial and temporal variability of personal exposures.

Despite the overall improvements in urban air quality in recent ecades, as demonstrated by the results of air-quality monitoring in many urban areas, the trends in population exposure, and particularly exposure to transport-related air pollution, are less clear. The steady increase in the number of vehicles may counterbalance the decrease in emissions per vehicle witnessed in Europe, where conventional diesel and petrol engines will continue to dominate transport patterns in coming decades. Other factors are expected to contribute to transport-related air pollution, such as the large number of short road trips, the increasing share of commuting by passenger vehicles and the increasing amount of time spent on high-volume roads due to increased traffic congestion. Owing to continuing

urbanization and expansion of urban areas, an increasing share of the population is likely to be exposed to transport-related air pollutants. The current trend towards the growth of road transport, observed throught the WHO European Region, runs contrary to the intended reduction of pollution levels, mainly because a substantial share of this growth is due to the shift of freight movement away from railways and towards lorries.

Although epidemiological evidence for the adverse health effects of exposure to transport-related air pollution is increasing, the associations still need to be adequately quantified. Methods of quantifying the adverse effects on health are available (Box 5.1), but they need to be applied more broadly. More studies, showing consistent results in different study locations and populations, are needed before a comprehensive risk assessment of the health effects of transport-related air pollution can be made. To evaluate the long-term adverse effects, study programmes on repeated exposure – which also focus on in-traffic exposures and emphasize the collection of personal exposure data along with population-level

Box 5.1. Examples of quantification of the health effects of transport-related air pollution

Using different methods based on epidemiology, an attempt was made to quantify the adverse effects on public health of current levels of transport-related air pollution. To estimate the effects of ambient air pollution and transport-related air pollution on public health in Austria, France and Switzerland, Künzli et al. (2000) used exposure–response functions for a 10-μg/m^3 increase in PM10. These functions modelled the exposure of a population to PM10 for each square kilometre and estimated the transport-related fraction, based on PM10 emission inventories. The effect of air pollution on public health was estimated to be 6% of total mortality in adults 30 years of age or older, accounting for more than 40 000 deaths a year. About half of all mortality caused by air pollution was attributed to motorized traffic, which also accounted for more than 25 000 new cases of chronic bronchitis in adults, more than 290 000 episodes of bronchitis in children, more than 500 000 asthma attacks and more than 16 million person-days of restricted activity.

Using another method, Forsberg et al. (2003) calculated the effect of a system of congestion pricing planned for Stockholm, Sweden, where relatively higher prices would be charged for travel during peak hours. Through the use of traffic models, a dynamic emission database and an air-quality dispersion model, the effect of congestion pricing on levels of different air pollutants was estimated and combined with a spatial distribution of the population, to obtain population-weighted means and extreme values. For about 330 000 inner-city residents, the reduction in long-term exposure was estimated to correspond to 47 preventable deaths when nitrogen dioxide is used as the indicator of exposure and 17 preventable deaths when PM10 is used as the indicator per year. The reduction of the population-weighted annual mean pollution level was estimated to be 1.2 μg/m^3 for both nitrogen dioxide and PM10.

estimates – have to be implemented. Further, exposure to pollution that comes specifically from traffic should be measured, modelled or both (WHO Regional Office for Europe, 2003). Epidemiological studies must take account of human mobility, to be able appropriately to assess health risks. Moreover, emission and exposure hot spots must be thoroughly studied, especially in urban settings, and exposure modelling needs to be linked to emission levels.

It is still unclear which constituents of traffic emissions are responsible for the observed adverse effects on people's health. Knowledge of such indicators would be very useful in implementing mechanisms that control air pollutants. Most epidemiological studies have concentrated on the classical air pollutants, such as black smoke, nitrogen dioxide or PM. A few studies have investigated the role of ultrafine particles. The choice of the indicator depends on the application (such as source apportionment, health risk assessment or transportation-flow management). Possible indicators of exposure to PM from traffic in urban areas might include black smoke and ultrafine particles.

In addition, studies with new designs are needed adequately to address the role of emissions from diesel-powered vehicles. Some suggest that emissions from vehicles with heavy-duty diesel engines are more relevant to adverse health effects than those from cars with light-duty engines. Specifically differentiating between light-duty and heavy-duty vehicles requires study populations whose exposures differ according to proximity to various diesel sources. Other studies are needed to understand the effects of new pollution constituents, such as trace elements from automobile catalytic converters (specifically, platinum, palladium and rhodium), the emissions of which are rapidly increasing and need to be monitored. There is also an urgent need to assess the benefits to public health of various measures to improve air quality, particularly through interventions that address transport-related air pollution (HEI Accountability Working Group, 2003; National Research Council Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Quality Regulations, 2002).

Justified action

Despite the remaining need for quantitative and qualitative studies, short-term measures to reduce exposure to transport-related air pollution are still well justified. Traffic management is one of the effective instruments for significantly reducing the exposure of residents of urban areas, and the nested adverse effects of the spread of transport-related air pollution to larger areas should be considered. Also, improvements are needed in integrating environmental and health considerations into urban planning, for example, by zoning offices, green areas and non-residential functions around urban highways and separating pedestrians and bicyclists from road traffic. In particular, urban planning may aim for integrative measures that lower emission rates; such measures include the promotion of highly efficient, service-oriented and clean public transport, and the promotion

of improved traffic flow. Revitalizing railways for freight transport, for example, can reduce road travel and the risk of increased air pollution from the expansion of urban areas.

Several technologies show promise in lowering the emission levels of conventional vehicles, including particle traps, systems to reduce emissions of nitrogen oxides, preheated catalytic converters and electronic vehicle controls. Their development should be promoted. Effective control mechanisms, such as mandatory car inspections, to eliminate gross polluters and badly maintained vehicles, should be more widely used. Further, alternative vehicle technologies (such as fuel cells, electric drives and hybrid engines) and substitute fuels (such as biofuels, natural gas and hydrogen) have the potential to reduce emission levels of hazardous air pollutants substantially in the future, and should they be further explored and developed.

Both research and action should form part of transport policies in the European Region that maximize the benefits to health.

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