Chapter 17

Urban air pollution

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Summary

Current scientific evidence, derived largely from studies in North America and Western Europe (NAWE), indicates that urban air pollution,[1] which is derived largely from combustion sources, causes a spectrum of health effects ranging from eye irritation to death. Recent assessments suggest that the impacts on public health may be considerable. This evidence has increasingly been used by national and international agencies to inform environmental policies, and quantification of the impact of air pollution on public health has gradually become a critical component in policy discussions as governments weigh options for the control of pollution.

Quantifying the magnitude of these health impacts in cities worldwide, however, presents considerable challenges owing to the limited availability of information on both effects on health and on exposures to air pollution in many parts of the world. Man-made urban air pollution is a complex mixture with many toxic components. We have chosen to index this mixture in terms of particulate matter (PM), a component that has been linked consistently with serious health effects, and, importantly, levels of which can be estimated worldwide. Exposure to PM has been associated with a wide range of effects on health, but effects on mortality are arguably the most important, and are also most amenable to global assessment. Our estimates, therefore, consider only mortality. Currently, most epidemiological evidence and data on air quality that could be used for such estimates comes from developed countries. We have had, therefore, to make assumptions concerning factors such as the transferability of risk functions, exposure of the population and their underlying vulnerability to air pollution, while trying to ensure that these assumptions are transparent and that the uncertainty associated with them is assessed through appropriate sensitivity analyses.

In order to provide estimates for all 14 subregions,[2] models developed by the World Bank were used to estimate ambient concentrations of inhalable particles (particulate matter with an aerodynamic diameter of <10 μm, PM_{10}) for PM in 3211 national capitals and cities with populations of >100 000 using economic, meteorological and demographic data and the available measurements. To allow the most appropriate epidemiological studies to be used for the estimation of the burden of disease, the estimates for PM_{10} were converted to estimates of fine particles (particulate matter with an aerodynamic diameter of <2.5 μm, $PM_{2.5}$) using available information on geographic variation in the ratio of $PM_{2.5}$ to PM_{10}. Population-weighted subregional annual average concentrations of $PM_{2.5}$ and PM_{10} were obtained using the population of the cities in the year 2000.

Our estimates of the burden of disease were based on the contributions of three health outcomes: mortality from cardiopulmonary disease in adults, mortality from lung cancer, and mortality from acute respiratory infections (ARI) in children aged 0–4 years. Numbers of attributable deaths and years of life lost (YLL) for adults and children (aged 0–4 years) were estimated using risk coefficients from a large cohort study of adults in the United States of America (Pope et al. 2002) and a meta-analytical summary of five time-series studies of mortality in children, respectively. Base-case estimates were calculated assuming that the risk of death increases linearly over a range of annual average concentrations of $PM_{2.5}$, between a counterfactual (or referent) concentration of 7.5 μg/m^3 and a maximum of 50 μg/m^3.

The results indicate that the impact of urban air pollution on the burden of disease in the cities of the world is large, but this is likely to be an underestimate of the actual burden, on the basis of an assessment of sources of uncertainty. There is also considerable variation in our estimates among the 14 subregions, with the greatest burden occurring in the more polluted and rapidly growing cities of developing countries. We estimated that air pollution in urban areas worldwide, in terms of concentrations of PM, causes about 3% of mortality attributable to cardiopulmonary disease in adults, about 5% of mortality attributable to cancers of the trachea, bronchus and lung, and about 1% of mortality attributable to ARI in children. This amounts to about 0.80 million premature deaths (1.4% of the global total) and 6.4 million YLL (0.7% of the global total). This burden occurs predominantly in developing countries, with 39% of attributable YLL occurring in WPR-B and 20% in SEAR-D. The highest proportions of the total burden occurred in WPR-B and EUR-B, where urban air pollution caused 0.7–1.0% of the burden of disease.

We quantified the statistical uncertainty of our base-case estimates by estimating the joint uncertainty in the estimates of annual average concentration of PM and the estimates of the relative risks. Estimates worldwide and for most subregions vary by less than two-fold (50%

uncertainty interval). Model uncertainty due to assumptions about the shape of the concentration–response function, the choice of counterfactual level for PM, and other factors were assessed in sensitivity analyses. For the most part, the worldwide estimates in each sensitivity case are within the 50% uncertainty intervals for the base-case estimates. The sensitivity analyses indicate that our base-case estimates were most sensitive to our choice of concentration–response function and theoretical level of minimum exposure.

1. Introduction

The potential for serious consequences of exposure to high levels of ambient air pollution was made clear in the mid-20th century, when cities in Europe and the United States experienced episodes of air pollution, such as the infamous London Fog of 1952 and Donora Smog of 1948, that resulted in large numbers of excess deaths and hospital admissions. Subsequent clean air legislation and other regulatory actions led to the reduction of ambient air pollution in many regions of the world, and particularly in the wealthy developed countries of North America and Europe. New epidemiological studies, however, conducted over the last decade, using sensitive designs and methods of analysis, have identified adverse health effects caused by combustion-derived air pollution even at the low ambient concentrations that now generally prevail in cities in North America and western Europe (Health Effects Institute 2001). At the same time, the populations of the rapidly expanding mega-cities of Asia, Africa and Latin America are increasingly exposed to levels of ambient combustion-related pollution that rival and often exceed the levels experienced in developed countries in the first half of the 20th century. Current scientific evidence, derived largely from studies in North America and western Europe, indicates that urban air pollution causes a spectrum of effects on health, ranging from eye irritation to death (Anonymous 1996a, 1996b). Recent assessments suggest that the impacts on public health may be considerable (Brunekreef 1997; Cifuentes et al. 2001; COMEAP 2001; Künzli et al. 2000; Ostro and Chestnut 1998). This evidence has increasingly been used by national and international agencies to inform environmental policies, and quantification of the impact of air pollution on public health has gradually become a critical component in policy discussions as governments weigh options for the control of pollution.

Quantifying the magnitude of the impact of air pollution in cities worldwide, however, presents considerable challenges owing to the limited availability of information on both effects on health and on exposures to air pollution in many parts of the world. Measurements of urban air pollution, when available, are available largely for a non-representative sample of urban areas. Many areas of the world lack measurements of any kind, and these must then be estimated using

statistical models (see below). On the basis of these considerations, we defined the target population for this risk assessment exercise as the residents in the year 2000 of national capital cities and of cities worldwide with populations of >100 000.

Man-made urban air pollution, which is derived largely from combustion processes, is a complex mixture containing many toxic components. We indexed this mixture in terms of PM, a component that has been consistently linked with serious effects on health, and, importantly, the levels of which can be estimated worldwide. Exposure to PM has been associated with a wide range of effects on health, but its effects on mortality are arguably the most important, and are also most amenable to global assessment. Our estimates, therefore, consider only mortality. Currently, most epidemiological evidence and data on air quality that could be used for such estimates come from developed countries. We have had, therefore, to make assumptions concerning factors such as the transferability of risk functions, exposure of the population and their underlying vulnerability to air pollution, while trying to ensure that these assumptions are transparent and that the uncertainty associated with them is assessed through appropriate sensitivity analyses.

The general framework for estimating the global burden of disease attributable to specific risk factors is described in chapters 1 and 25. Briefly, the approach involves estimating an attributable fraction(s) for each risk factor in each of the 14 subregions of the world. Estimating the attributable fraction for urban air pollution requires several steps. First, the exposure to urban air pollution of the population of each subregion must be estimated. Second, a theoretical minimum level of exposure must be specified. The attributable fraction quantifies the impact of exposure above this theoretical minimum level. Finally, deriving the attributable fraction requires the estimation of the gradient of risk between the theoretical minimum level and the estimated subregional exposure. These risk functions are derived from epidemiological studies for the purposes of estimating the global burden of disease. As discussed below, epidemiological studies generally estimate exposure to air pollution in terms of ambient concentrations, thus, we use the term "concentration–response" (rather than "exposure–response") to describe the risk function.

This chapter describes our approach to estimating the attributable fraction and presents our estimates of the attributable burden of disease caused by urban air pollution. First, we briefly review background information on exposure to air pollution and then describe our choice of the theoretical minimum level and the approach to estimating the exposure to PM of the populations of the world's cities. Next, we review the current information on the effects of air pollution on health and describe our approach to deriving the concentration–response function(s). Finally, we present and discuss our estimates of the attributable burden and their uncertainties.

2. Exposure to urban air pollution from combustion sources

Combustion of fossil fuels for transportation, power generation, and other human activities produces a complex mixture of pollutants comprising literally thousands of chemical constituents (Derwent 1999; Holman 1999). Exposure to such mixtures is a ubiquitous feature of urban life. The precise characteristics of the mixture in a given locale depend on the relative contributions of the different sources of pollution, such as vehicular traffic and power generation, and on the effects of the local geoclimatic factors. The relative contribution of different combustion sources is a function of economic, social and technological factors, but all mixtures contain certain primary gaseous pollutants, such as sulfur dioxide (SO_2), nitrogen oxides (NO_X) and carbon monoxide (CO), that are emitted directly from combustion sources, as well as secondary pollutants, such as ozone (O_3), that are formed in the atmosphere from directly-emitted pollutants. The pollutant mixture also contains carcinogens such as benzo(α)pyrene, benzene and 1,3-butadiene. When petrol contains lead (Pb), as is still the case in many developing countries, this element is a common constituent of the pollution mix, assessed in a separate chapter in this volume (chapter 19).

All combustion processes produce particles, most of which are small enough to be inhaled into the lung either as primary emissions (such as diesel soot), or as secondary particles via atmospheric transformation (such as sulfate particles formed from the burning of fuel containing sulfur). Their concentrations (in micrograms per cubic metre, or $\mu g/m^3$) are generally measured as inhalable and fine particles, PM_{10} and $PM_{2.5}$, respectively.[3] However, the total suspended particle mass (TSP) is still the only particle measurement available in many developing countries (Krzyzanowski and Schwela 1999).

Pollution from the combustion of fossil fuels is largely emitted into the outdoor air, but human exposure occurs both indoors and outdoors (Ozkaynak 1999). An individual's exposure to ambient urban air pollution depends on the relative amounts of time spent indoors and outdoors, the proximity to sources of ambient air pollution, and on the indoor concentration of outdoor pollutants. The indoor concentrations depend on factors such as the circulation of the indoor air and the degree to which constituents of the outdoor combustion mixture penetrate and persist in the indoor environment. Studies conducted largely in Europe and North America have shown that the fine particles generated from combustion outdoors both effectively penetrate and persist in many indoor environments. Gases, such as sulfur dioxide and ozone, may penetrate the indoor environment, but generally do not persist because of their reactivity. In some rural areas of developing countries, indoor cooking on unvented coal- or biomass-burning stoves is the most significant exposure to pollution from combustion sources. The burden of disease caused by

such exposure is addressed in chapter 18. The actual dose delivered to the lung or other organs will further depend on the type of pollutant, the breathing pattern and physical characteristics of the individual that determine the extent and site of deposition.

Governments in many parts of the world monitor ambient concentrations of air pollution as part of regulatory programmes designed to protect public health and the environment (Grant et al. 1999). The most extensive monitoring systems are in the United States and western Europe, where regular monitoring of ambient air quality has been in place since the mid-1970s. The most frequently and routinely monitored air pollutants include sulfur dioxide (SO_2), nitrogen oxides (NO_X, including NO and NO_2), carbon monoxide (CO), ozone (O_3), lead (Pb), black smoke (BS) or soot, and PM. National monitoring systems also exist in other parts of the world, but access to the data collected by these systems and international standardization of the monitoring methods are limited. The World Health Organization (WHO) Air Management Information System (AMIS) (WHO 2001c) collects the available information, but the reporting from many regions is poor, and for some regions there are no data in the WHO database. The various designs of the networks, differences in monitoring objectives and limited availability of the collected data for the outside users limit access to the information on population exposure in the greater proportion of the world's cities. In some parts of the world (e.g. in most of the countries of the former Soviet Union), the monitoring systems exist but do not provide the data necessary for assessment of the impact on health (Krzyzanowski and Schwela 1999). More details about the data available for this analysis are provided in further sections of this chapter.

These monitoring systems currently provide much of the data on exposure to urban air pollution that have been used in epidemiological research, although some studies establish their own monitoring networks when routinely-collected data are either unavailable or of poor quality, or to measure specific air pollution constituents, such as specific known carcinogens. Typically, monitoring sites are located in the city centre or throughout a given metropolitan area, in order to more accurately reflect the average residential exposure of the population. The data from monitors sited so as to measure emissions from specific sources, such as a local industry or heavy vehicular traffic, are frequently excluded from the data sets, as they may significantly deviate from the average levels of exposure experienced by the population.

Exposure estimates that rely exclusively on data from one or more stationary monitoring sites may provide inaccurate estimates of the short- and/or long-term average personal exposures of study populations (Navidi and Lurmann 1995; Zeger et al. 2000). The direction and magnitude of the errors that will be induced in estimates of the relative risk attributable to exposure to air pollution depend on the precision of the air quality monitoring data (or models used to generate the estimates of

the concentration of pollution), the applicability of one estimate to the entire target population and the correlation of the errors with the health outcome. Generally, such errors will be smaller for pollutants that tend to be uniformly distributed over large urban areas, and that penetrate efficiently indoors, both of these features being the case for fine PM produced by combustion. If the errors in the estimates of exposure are uncorrelated with the risk of the health outcome, then the estimates of relative risk attributable to air pollution will, in most cases, be too low (i.e. biased to the null) (Navidi and Lurmann 1995).

2.1 Definition of the air pollution metric for exposure variable

We selected PM_{10} and $PM_{2.5}$ as the indicators of exposure to urban air pollution from combustion sources. As noted above, PM is a ubiquitous component of the mixtures emitted into, and formed in, the ambient environment by combustion processes, and indicates the presence of these mixtures in outdoor air. Most importantly, these measures of particulate air pollution have been used in many epidemiological studies from around the world, of both mortality and morbidity of air pollution, and so provide the best overall indicator of exposure for our purposes (see section 3). Although other components of ambient air pollution from combustion sources are associated with these and other effects on health (Anonymous 1996a, 1996b), particulate air pollution has been found to be consistently and independently related to the most serious effects of air pollution, including daily and longer-term average mortality (California Air Resources Board 2002; Health Effects Institute 2001; U.S. Environmental Protection Agency 2002; WHO 2000a, 2003). There is some evidence, although much less than that for PM, linking ozone to premature mortality, particularly during the summer months (Abbey et al. 1999; Health Effects Institute 2000b). However, despite recent progress in developing models to estimate tropospheric (ground-level) ozone on a global scale, it was not currently feasible to derive the subregional estimates that would have been required for this project. In many developing countries, exposure to lead in the ambient air may also be of great consequence, having effects on mortality perhaps via effects on blood pressure. The impacts of lead in outdoor air are dealt with in chapter 19.

PM has been linked to serious effects on health after both short-term exposure (days to weeks), and more prolonged exposure (years), although there remains some uncertainty as to the distribution of induction times with regard to mortality (see below). We chose the annual average concentration(s) of PM as the exposure metric(s) because it corresponds to the time-scales of *a priori* interest for estimates of attributable and avoidable burden in the Global Burden of Disease (GBD) project, and because it was used to estimate the effects of exposure to PM in the key epidemiological study that provides our estimates of the concentration–response function.

2.2 Estimation of annual average concentrations of particulate matter

Air pollution measurements used in estimating annual average concentrations

The availability of measurements of ambient concentrations of PM varies widely across the globe, making estimation of annual average concentrations a considerable challenge (Krzyzanowski and Schwela 1999). To estimate ambient PM concentrations for all 14 subregions, we used a model (Global Model of Ambient Particulates [GMAPS]) recently developed at the World Bank to estimate concentrations of PM_{10} in cities, on the basis of available measurements of PM at population-oriented monitoring sites (Pandey et al. forthcoming). The model incorporates information on factors such as fuel mix, level of economic development, demographics and weather, in order to predict ambient concentrations of PM_{10} in urban residential areas. These estimates of PM_{10} were converted to $PM_{2.5}$ using available information on geographic variation in the ratio of $PM_{2.5}$ to PM_{10}. For each PM metric, the population-weighted subregional annual average was derived using the population of each city within each subregion in the year 2000.

The GMAPS model developed at the World Bank can be used to generate estimates of concentrations of PM_{10} in all world cities with populations of >100 000, and in national capitals. The estimation model is based on available measurements of PM_{10} and TSP from population-oriented monitoring stations in cities worldwide for the period 1985 to 1999, retrieved in October 2001. In all cases, data from a monitoring site were included if and only if it was clearly identified as a residential or mixed residential site (see section 2.3 for definition). For instance, city averages reported for many Chinese cities (National Environmental Protection Agency of China 2000) were not included in the model estimation because the location of these sites could not be ascertained.

In principle, the monitoring data used for calculation of annual averages should be collected throughout the year, since seasonal patterns in the data are fairly common. More than 85% of cities in Europe and the United States collect measurements of PM throughout the year. The representativeness of the data for cities in other parts of the world could not be confirmed. In addition, in many countries where PM was measured throughout the year, it was only measured on every sixth day. The methods for measuring concentrations of PM also varied, both gravimetric and automatic methods (tapered element oscillating microbalance monitors [TEOMS] or beta gauge monitors) being included.

Most of the data on annual average ambient concentrations used in the model come from AMIS (WHO 2001c). This information is submitted to WHO by national environmental agencies and air quality control authorities, which perform these measurements using nationally

approved methods and standards of data quality. The data set contains the annual mean concentration of selected air pollutants, including PM, by monitoring site. Additional data, such as 95th percentiles of daily means, are also available for some sites. Although WHO requests that all Member States provide data for compilation in the AMIS database, the reported data are still limited because many countries do not have air quality monitoring networks. Additionally, some countries with monitoring networks may not report the data because of poor data quality or limited ability to process and report the data.

The data from AMIS were supplemented with other sources of data on TSP and PM_{10} from monitoring sites. These included data for European cities collected by WHO/European Centre for Environment and Health (ECEH) for the Health Impact Assessment of Air Pollution (HIAAP) project in 1999 from both national and local environmental agencies (WHO 2001a), data for Canadian cities provided by Environment Canada (www.ec.gc.ca) and statistics Canada (http://www.statcan.ca/english/ads/cansimII/index.htm), and data for cities in the United States from the U.S. Environmental Protection Agency AIRS database (Aerometric Information Retrieval System 2001). Data for Chinese cities were also obtained from the Environmental Quality Reports from China (National Environmental Protection Agency of China 2000), and Mexican cities from the Instituto Nacional de Ecología (INE), SEMARNAP, Mexico (Instituto Nacional de Ecología 2000). Additional data were also obtained from the World Bank URBAIR studies of air pollution in Jakarta and Kathmandu (Grønskei et al. 1997a, 1997b). To limit undue influence of the data from cities in the United States, data used from the United States AIRS database were limited to the years 1996–1999.[4]

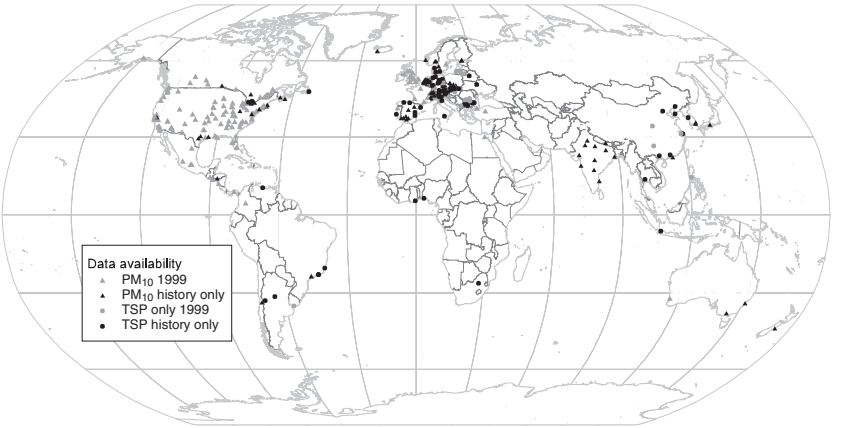
Measured annual average concentrations of PM_{10} and TSP data from monitoring sites were available for 512 unique locations in 304 cities in 55 countries over the period 1985–1999, and provided 1997 time–location data points. For some sites and years, data on both TSP and PM_{10} were available, yielding a total of 2344 individual observations.[5] The number of cities with measured data on PM from monitoring sites in each subregion and for each year by PM measure is shown in Table 17.1. A total of 304 cities reported either the annual average concentrations of PM_{10} or TSP for at least 1 year between 1985 and 1999. Of these, 51 cities reported both PM_{10} and TSP while 165 cities, mostly in North America and western Europe, reported PM_{10} only, and the remaining 88 cities reported data for TSP only.

Coverage of cities and populations with data from monitoring sites varies significantly across different subregions (Figure 17.1). For instance, data from monitoring were available for fewer than two cities for six of the subregions, AFR-D, AFR-E, AMR-D, EMR-B, EMR-D and SEAR-B. In contrast, data from monitoring sites were available for 218 cities in NAWE, of which 174 report data on PM_{10}. The 304 world cities

Table 17.1 Number of cities for which data on particulate matter are available from monitoring sites, by subregion, year and type of particulate matter

	PM_{10} or TSP	PM_{10}	TSP
Subregion			
AFR-D	2	0	2
AFR-E	1	0	1
AMR-A	123	118	25
AMR-B	19	12	12
AMR-D	2	2	2
EMR-B	0	0	0
EMR-D	1	1	0
EUR-A	95	56	43
EUR-B	22	7	17
EUR-C	7	1	7
SEAR-B	2	0	2
SEAR-D	11	11	10
WPR-A	5	5	4
WPR-B	14	3	14
World	304	216	139
Year			
1985	28	7	28
1986	52	15	50
1987	53	9	52
1988	47	16	45
1989	53	17	51
1990	64	20	60
1991	63	30	60
1992	70	34	67
1993	73	41	68
1994	78	40	73
1995	73	42	68
1996	156	132	54
1997	144	127	40
1998	211	150	81
1999	166	143	40
1985–1998	267	187	127

Figure 17.1 Cities from which data on exposure to PM_{10} or TSP during 1985–1999 are available from monitoring cites



Source: K. D. Pandey, Personal Communication.

with data from monitoring account for 9% of the total number of cities with a population of >100 000 worldwide and have a combined population in the year 2000 of around 559 million, or about 28% of the global urban population (Table 17.2).

Global Model of Ambient Particulates (GMAPS)

The GMAPS model econometrically estimates a fixed-effect model of the concentrations of urban ambient PM using the latest available data from WHO and other sources, as outlined above. The estimating Equation 1 focuses on the anthropogenic sources of pollution and the capacity of the natural environment to generate, disperse and dissipate pollutants.[6] Its determinants include the scale and composition of economic activity, the energy mix, the strength of local regulation of pollution, and geographic and atmospheric conditions that affect the transport of pollutants.

$$\begin{aligned} C_{ijkt} = & \sum_{k=1}^{K} \beta_k Z_k + \sum_{f=1}^{F} \beta_{Ef} E_{fkt} + \sum_{g=1}^{G2} \beta_{Mg} M_{gik} + \beta_R R_{kt} + \beta_N N_{jkt} + \beta_D D_{jk} \\ & + \beta_{Scale} Scale_{jkt} + \beta_Y Y_{kt} + \beta_T Trend_{ijkt} + \beta_{YT} Y_{kt} Trend_{ijkt} \\ & + \theta_S S_{ijkt} + \theta_{Scale} S_{ijkt} Scale_{jkt} + \theta_Y S_{ijkt} Y_{kt} + \theta_T S_{ijkt} Trend_{ijkt} \\ & + \theta_{YT} S_{ijkt} Y_{kt} Trend_{ijkt} + \sum_{g=1}^{G1} \theta_{Mg} S_{ijkt} M_{gjk} + \varepsilon_{ijkt} \end{aligned} \quad (1)$$

Table 17.2 Cities for which measurements of particulate matter are available from monitoring sites, by subregion

	Number of cities			*Urban population (000s) in 2000*[a]		
Subregion	*Cities in subregion*	*Cities with monitoring sites*	*% with monitoring sites*	*Cities in subregion*	*Cities with monitoring sites*	*% with monitoring sites*
AFR-D	107	2	2	66 960	14 914	22
AFR-E	105	1	1	68 367	2 388	3
AMR-A	267	123	46	232 439	178 240	77
AMR-B	399	19	5	217 159	64 121	30
AMR-D	47	2	4	29 512	3 291	11
EMR-B	89	0	0	56 621	0	0
EMR-D	126	1	1	99 397	8 124	8
EUR-A	429	95	22	161 808	79 160	49
EUR-B	182	22	12	81 756	21 494	26
EUR-C	275	7	3	109 178	7 670	7
SEAR-B	68	2	3	53 708	18 793	35
SEAR-D	356	11	3	214 175	67 081	31
WPR-A	242	5	2	100 079	11 459	11
WPR-B	519	14	3	528 318	81 817	15
World	3 211	304	9	2 019 479	558 553	28

[a] The total urban population is for 3211 cities with populations >100 000 and national capitals.
The total urban population in 2000, including cities of all sizes, is 2.8 billion.

where

C_{ijkt} = log of concentration of PM in monitoring station i, city j, country k, at time t

Z_k = binary variable for country k

E_{fkt} = log of per capita energy consumption of energy source type f for country k at time t (f=1 . . . F)

M_{gjk} = log of meteorological/geographic factor g for city j, country k (factors g=1 . . . $G1$ affect PM_{10} concentration in a different way than TSP concentration. Factors g=$G1$+1 . . . $G2$ do not make a distinction between PM_{10} and TSP)

R_{kt} = log of population density of country k at time t

N_{jkt} = log of population of city j, country k, at time t

D_{jk} = log of local population density in the vicinity of city j in country k

$Scale_{jkt}$ = log of scale of economy (intensity of economic activity) for city j, country k at time t

Y_{kt} = log of income per capita (1-year lagged 3-year moving average) of country k at time t

$Trend_{ijkt}$ = time trend (1985=1, 1986=2, . . . 1999=15)

S_{ijkt} = binary variable for PM type measured at monitoring station i, city j, country k, at time t, (1=TSP, 0=PM_{10}), and

the β_S and θ_S are the parameters that are estimated by the model.

Equation 1 jointly determines the concentrations of total suspended particulate matter (TSP) and inhalable particulates (PM_{10}) in residential areas. Most cities in developing countries only monitor TSP and not PM_{10}. Adoption of the pooled specification permits use of all available data and provides better information about the concentrations of PM, especially for cities in developing countries. Limiting the estimation sample to PM_{10} observations is sensible only if knowledge of the concentration of TSP in a city makes no contribution to predicting PM_{10}. Since PM_{10} comprises the smaller size particles within TSP, this assumption is clearly unreasonable. The pooled specification allows for separate estimation of concentrations of PM_{10} and TSP for each city by setting the binary variable, S_{ijkt}, equal to zero or one, as shown in Equations 2 and 3.

$$\log[PM10_{ijkt}] = \sum_{k=1}^{K} \beta_k Z_k + \sum_{f=1}^{F} \beta_{Ef} E_{fkt} + \sum_{g=1}^{G2} \beta_{Mg} M_{gjk} + \beta_R R_{kt} + \beta_N N_{jkt} + \beta_D D_{jk} + \beta_{Scale} Scale_{jkt} + \beta_Y Y_{kt} + \beta_T Trend_{ijkt} + \beta_{YT} Y_{kt} Trend_{ijkt} \quad (2)$$

$$\begin{aligned}\log[TSP_{ijkt}] = &\sum_{k=1}^{K}\beta_k Z_k + \sum_{f=1}^{F}\beta_{Ef}E_{fkt} + \sum_{g=1}^{G2}\beta_{Mg}M_{gjk} + \beta_R R_{kt} + \beta_N N_{jkt} \\ &+ \beta_D D_{jk} + \beta_{Scale}Scale_{jkt} + \beta_Y Y_{kt} + \beta_T Trend_{ijkt} + \beta_{YT}Y_{kt}Trend_{ijkt} \\ &+ \theta_S + \theta_{Scale}Scale_{jkt} + \theta_Y Y_{kt} + \theta_T Trend_{ijkt} + \theta_{YT}Y_{kt}Trend_{ijkt} \\ &+ \sum_{g=1}^{G1}\theta_{Mg}M_{gjk}\end{aligned} \tag{3}$$

To reduce undue influence from extreme values, all of the continuous variables in the model were specified in log form and each exogenous variable in the estimation sample was truncated to the middle 98% range observed in the estimation sample.

The estimation Equation 1 includes country-specific binary variables, Z_k, to control for economic, social and natural factors that are not captured by the other explanatory variables. These include differences in the quality of the data on ambient concentration and in collection methods across countries, the degree of regulatory heterogeneity within a country, the relative importance of intercity transport, proximity of and pollution levels in neighbouring cities and the composition of economic activity. The country-specific binary variables measure the average concentration of PM in each country during the 15-year period 1986–1999, controlling for variations within the country caused by factors accounted for in the remainder of the estimating Equation 1. In contrast, the rest of the estimation model (1) explains the marginal contribution of the included factors to deviations in the ambient concentration in the city from this average.

The primary determinants of the observed variations in the ambient concentrations of PM within a country in the estimation model are:

Energy consumption. The model includes six separate per capita energy consumption categories—coal, oil, natural gas, nuclear, hydroelectric, combustible renewables and wastes—that account for all energy consumed in each country for which data are available from the International Energy Agency's (IEA) Annual Energy Balance database (International Energy Agency 2001a, 2001b). The separate inclusion of each type of energy source accounts for differences in emission factors, variations in economic activity and intensity of fuel use across countries. In addition, the model also includes per capita consumption of petrol and diesel used in the transportation sector, also available from IEA's database, to capture additional detail about one of the most significant contributors to ambient concentrations of PM.

Meteorological and geographic factors. The model includes 22 atmospheric and geographic factors for each city to account for both the dissipative/dispersive capacity of the natural environment and natural sources of particulates, such as desert dust storms, forest fires and sea spray. These include a suite of 18 climatic variables representing the long-

term average climatic conditions related to local atmospheric conditions and transport of PM, consisting of the annual average (average of the monthly data) and seasonal changes (measured as the standard deviation of the monthly data) for the following nine factors: mean temperature, diurnal temperature, mean precipitation, barometric pressure, wind speed, percentage cloud cover and frequency of wet, sunny and frosty days (New et al. 1999).[7] In addition, two meteorological variables related to energy demand (heating and cooling degree-days) are estimated for each city from the mean monthly temperature. Two topographical variables related to atmospheric transport—distance from the city centre to the nearest point on the coastline, calculated using the geographic information system (GIS), and elevation of the city, derived from a global digital elevation model (USGS 1996)—are also included in the model.

City and national population and national population density. These variables provide measures of the scale and intensity of the pollution problem in each city. The data on population comes from the *Demographic yearbook* published by the United Nations (UN 2001).

Local population density. The local population density in the vicinity of each city provides a measure of the intensity of pollution. It is estimated from the Gridded Population of the World (version 2), available from the Consortium for International Earth Science Information Network (CIESIN 2000). This data set provides the best available population data for about 120 000 administrative units, converted to a regular grid of population counts at a resolution of about 5 km. The local population density in the vicinity of each city is the average population density for all grid cells within a 20-km radius of the city centre.

Local intensity of economic activity. Most cities do not collect data on the amount or composition of economic activity. Instead, the local gross domestic product (GDP) per square kilometre computed as the product of the national per capita GDP and the local population density in the vicinity of each city is used as a proxy for the intensity of economic activity within each city (World Bank 2002).

National income per capita. This variable is used to capture the following national indicators: valuation of the quality of the environment, strength of environmental policy and regulation, the institutional capacity to enforce environmental policies, and the potential use of cleaner fuels along the fuel-use chain as countries develop. It is measured as a 1-year lag of the average of the previous 3 years (World Bank 2002).

Time trends. The model includes two time-trend variables (with 1985=1 . . . 1999=15) to allow for differential time trends for PM_{10} and TSP particulate pollution. Both of these variables are in turn interacted with lagged national per capita income to allow trends to vary across countries on the basis of differential valuation and improvements in environmental quality across countries as measured by the level of economic development. These trends measure changes in concentrations of PM

that are caused by factors not already captured in the model, such as technological changes, improvements in knowledge and structural shifts in the composition of economic activity. They do not represent the unconditional aggregate trends in concentrations of PM.

Binary variable to differentiate PM_{10} and TSP. The model includes a binary variable indicating whether PM is measured as TSP or PM_{10}. This binary variable is also allowed to interact in the model with other variables to allow for size class differences in the composition of particulates across cities and countries. It provides a better representation of intercity differences across the world, rather than assuming a uniform relationship across all cities. The log of the ratio of PM_{10} to TSP in each city can be estimated by subtracting Equation 3 from 2, as shown in Equation 4. The key determinants of this ratio are the scale of economic activity, differential trends across countries, level of economic development and strength of environmental policy, and the subset of meteorological variables that are directly related to particle size (annual mean and seasonal variations in wind speed, precipitation and frequency of wet days).

$$\log[PM10_{ijkt}] - \log[TSP_{ijkt}] = -\theta_S - \theta_{Scale} Scale_{jkt} - \theta_Y Y_{kt} - \theta_T Trend_{ijkt} - \theta_{YT} Y_{kt} Trend_{ijkt} - \sum_{g=1}^{G1} \theta_{Mg} M_{gjk} \quad (4)$$

In order to facilitate predictions for countries not included in the estimation, a secondary model shown in Equation 5 is estimated to explain the average level of ambient PM concentration in each country.

$$\hat{\beta}_k = \sum_{f=1}^{F} \gamma_{Ef} \overline{E}_{fk} + \gamma_R \overline{R}_k + \gamma_R \overline{Y}_k + u_k \quad (5)$$

where

$\hat{\beta}_k$ = country-specific binary variable coefficient estimated in Equation 1

$\overline{E}_{fk}$ = log of average per capita energy consumption of energy type f for country k during 1985–1999 ($f = 1 \ldots F$)

$\overline{R}_k$ = log of average population density of country k during 1985–1999

$\overline{Y}_k$ = log of average national per capita income of country k during 1985–1999 (1-year lagged average of previous 3 years)

This secondary model (5) explains the average level of pollution under reference conditions for a country, on the basis of the scale of the economy, the composition of economic activity as measured by the energy mix, and the strength of local pollution regulations and the institutional capacity for implementing these regulations.

2.3 Model outputs

The GMAPS model is designed to obtain the best city-level prediction of concentrations of PM for a wide range of cities on the basis of the limited amount of data from monitoring available, so it focuses on increasing the fit of the model. It is not designed to provide a causal model for ambient concentrations of air pollution. The estimation model (1) explains 88% of the variation in the observed data from monitoring, indicating a good fit (Pandey et al. forthcoming). The overall correlation between the measured and the predicted data is around 0.9 for both PM_{10} and TSP observations (see Table 17.3), and is >0.80 for all years and for both observations of PM_{10} and TSP, with the exception of PM_{10} in 1985. The correlation by subregion is smaller than that over time, ranging between 0.2 and 0.9 for subregions with more than 10 data points. The correlations for subregions with fewer data points are smaller than 0.2 and are less precisely estimated. A negative correlation for EUR-B is driven by a single erroneous observation for Bucharest, Romania, where the observed concentration of PM_{10} is higher than that of TSP. These results originated from two different monitoring locations; had the model been re-estimated without this particular PM_{10} observation, the correlation for the subregion would have been 0.32.

Subregion- and PM type-specific scatter plots of model predictions compared to actual data also show a clustering of points around the solid line drawn at a 45° angle, indicating that the actual values are close to the predicted values. As would be expected, the predicted values are less extreme than the actual values at both tails, owing to the truncation of all explanatory variables to the middle 98% range of the estimation sample. F-tests revealed that all of the eight aggregate factors in the model added significant explanatory power to the regression.

The secondary estimation model (5) explains 85% of the variation in the estimated average level of pollution in a country, indicating that this model provides a good fit. The explanatory power of the secondary model is not as robust to changes in the estimation sample owing to significant uncertainties in the estimated dependent variable.

Out-of-sample predictions were used to validate the model using both statistical and heuristic criteria. The model was re-estimated using subsamples of the data on the basis of different cut-off points for per capita income, to examine the appropriateness of extrapolating from a model primarily based on industrialized cities in North America and western Europe to cities in developing countries. The resulting estimates from the model were used to predict concentrations of PM_{10} in residential areas in the out-of-sample cities located in developing countries. A second set of estimates was also made comprising income-based subsamples using only the available data on PM_{10} from monitoring in residential sites. These validation estimates consistently showed that out-of-sample correlations were higher when data on TSP were included in the estimations. Furthermore, the out-of-sample correlations on aggregate ranged

Table 17.3 Correlation between observed concentrations of particulate matter at monitoring sites and predictions by subregion, year and type of particulate matter

	PM_{10} or TSP		*PM_{10}*		*TSP*	
	No. of observations	*Correlation*	*No. of observations*	*Correlation*	*No. of observations*	*Correlation*
Subregion						
AFR-D	6	0.86	0	NA	6	0.86
AFR-E	2	–1.00	0	NA	2	–1.00
AMR-A	1 273	0.79	938	0.59	335	0.67
AMR-B	361	0.80	215	0.52	146	0.75
AMR-D	34	0.88	18	0.31	16	0.72
EMR-D	1	NA	1	NA	0	NA
EUR-A	182	0.85	75	0.82	107	0.73
EUR-B	63	0.83	16	–0.29	47	0.78
EUR-C	54	0.84	1	NA	53	0.83
SEAR-B	9	0.14	0	NA	9	0.14
SEAR-D	158	0.81	65	0.69	93	0.80
WPR-A	69	0.85	36	0.86	33	0.20
WPR-B	132	0.92	21	0.49	111	0.89
World	2 344	0.94	1 386	0.89	958	0.92
Year						
1985	35	0.95	7	0.11	28	0.85
1986	68	0.93	17	0.81	51	0.92
1987	65	0.93	11	0.94	54	0.92
1988	70	0.93	20	0.91	50	0.92
1989	76	0.94	21	0.90	55	0.94
1990	91	0.94	24	0.90	67	0.94
1991	101	0.96	34	0.94	67	0.95
1992	116	0.94	38	0.95	78	0.93
1993	130	0.94	49	0.95	81	0.92
1994	138	0.94	46	0.94	92	0.93
1995	144	0.94	59	0.94	85	0.93
1996	330	0.92	259	0.88	71	0.90
1997	298	0.89	253	0.79	45	0.92
1998	377	0.88	289	0.84	88	0.84
1999	305	0.90	259	0.83	46	0.87
All years except 1999	2 039	0.94	1 127	0.90	912	0.92

NA Not applicable.

between 0.40 and 0.59, based on the income cut-off used, and lend support to the modelling approach.

Since cities with data from monitoring are not representative of all cities and account for a small fraction of urban residents in developing countries, the following heuristic criteria were also used to evaluate the predictions of the model.

- *Comparison of the relative variation of the predictions within countries and between countries relative to the actual data*: The model predictions exhibited significant variations both across countries and across cities within a country. The predicted variations within a country were about 60% of those between countries and were comparable to the corresponding variations in the actual data.
- *Number of cities for which predictions were outside the range of the estimation sample*: The predictions for PM_{10} were within the range observed in the actual data. They continued to be within bounds when the same fractions of values are removed from the tails of the estimated and measured data.
- *Magnitude of predictions outside the range observed in the estimation sample*: Of the 304 cities with data from monitoring, concentrations of PM_{10} exceeded 200 $\mu g/m^3$ in three cities and concentrations of TSP exceeded 400 $\mu g/m^3$ in 10 cities. The predicted concentrations of PM_{10} exceeded 200 $\mu g/m^3$ in only four out of 3226 cities.
- *Range of the PM_{10}:TSP ratio*: The PM_{10}:TSP ratio predicted by the model is between 0.24 and 0.98 and spans the middle 95% of the range observed in the actual data. The mean ratio predicted by the model is 0.49; the ratio for half of the cities lies between 0.39 and 0.56.
- *Comparison of the uncertainty in estimates for cities, relative to the amount of available information for neighbouring cities*: Bootstrap error estimates of the prediction error for the city showed that the confidence intervals were wider for cities with no data from monitoring and are largest in the countries with no data from monitoring.

The robustness of the model was tested using alternative specifications of the model based on the goodness-of-fit of the model and the heuristic criteria outlined above. The alternative models that were considered were:

- *Linear model*. The linear model provides undue weight to the extreme values in the explanatory variables, resulting in predictions that are orders of magnitude larger than those for cities with data from monitoring.

- *Explanatory variable truncation.* The model was re-estimated with four different levels of truncation for the explanatory variables: no truncation, truncation to the actual range for the cities with data from monitoring, truncation to the middle 98% range of all explanatory variables for these cities, and truncation to the middle 90% range for these cities. Estimates based on the first two of these were sensitive to some of the extreme data points in the estimation sample, resulting in large variations in the predictions. Estimates from the last truncation were rejected because more than one quarter of the observations were truncated, leading to a poorer model fit.
- *Energy consumption variables.* The model was re-estimated with three alternative measures for the energy consumption variables: energy consumption per area, total per capita energy consumption and share of each energy type in the total energy mix, and the product of national per capita energy consumption by energy type and city population density. The specification per area resulted in predictions that were unstable and orders of magnitude larger than those observed in any city because of truncation of extreme values in countries with missing data on fuels. The second and third specifications resulted in poorer fits with over-predictions for >100 cities with values outside of the observed range of concentrations of PM_{10}.
- *Income.* The model was estimated using income-squared and income-cubed terms to measure the impact of national per capita income. Higher order terms were unstable and resulted in predictions that were orders of magnitude larger than those observed in any city.
- *PM_{10}:TSP ratio.* A number of different models were estimated from full interactivity of the binary variable S_{ijkt} with all of the continuous variables, to no interactivity with the continuous variables. The full interactivity model was rejected because it predicted physically implausible PM_{10}:TSP ratios of 2 for a significant number of cities. The limited model with no interactivity was rejected because it over-predicted the results for many cities in the Middle East and North Africa that contain a larger fraction of wind-blown coarser particles. Other models were estimated that incrementally added groups of variables, such as energy type and the other climate variables. These were all rejected using the heuristic criteria outlined above.
- *Location of monitoring sites.* The sensitivity of the model predictions to the inclusion of mixed residential sites was examined by re-estimating the model using only pure residential sites.[8] Although estimates for some individual cities change in significant ways, predictions at the subregional level, and for most countries, are not statistically different as compared to when mixed sites are included.

- *Inclusion of non-residential sites*. A more inclusive model, which jointly estimates concentrations of PM in residential and non-residential sites indicated that most model parameters were relatively stable and that the model predictions for subregional residential concentrations of PM_{10} were not significantly different for most subregions.
- *Additional monitoring data*. The sensitivity of the model was tested to the inclusion of additional monitoring data that became available between October 2001 and July 2002. The aggregate PM predictions were not statistically different for all subregions, except for EMR-B where concentrations of PM increase by nearly 50%. This is primarily owing to the inclusion of data for Kuwait City, which is the only city for which data from monitoring sites are available in this subregion.
- *Influential data point*. The sensitivity of the model predictions to influential data points was examined using bootstrap error techniques. Variations in the predictions based on different subsamples of the data were used to estimate the degree of uncertainty in the model estimates.

Estimating ambient concentrations of particulate matter in cities

The average subregional ambient concentrations used in this work are estimated from the city-level model predictions for 1999, the latest year for which all of the explanatory variables were available. The estimates of concentrations of PM_{10} in each city for 1999 were generated using a three-step approach. First, for all cities located in countries with at least one population-oriented monitoring site, the concentration of PM_{10} was estimated using the GMAPS model, as specified in Equation 1. The concentration of PM_{10} cannot be estimated using Equation 1 alone for cities in every country, because the average level of pollution in the country as measured by the country binary variable was not available for countries without monitors. Therefore, in the second step, the secondary Equation 5 was used to predict the country coefficient for countries without monitors. These predictions were combined with estimates from Equation 1 that explain variations around the average level to generate 1999 concentrations of PM_{10} for these cities in these countries.

Finally, for cities with actual data from monitoring, a best estimate of concentration of PM_{10} in the city in 1999 was generated by incorporating information on concentrations from previous years. Specifically, an average residual for each city was determined by comparing each year-specific predicted value generated by the estimation model (1) with the actual monitored value for that year and city. This served to adjust the model predictions for local factors that are known but unmeasured in the model, such as the composition of local economic activity. Given the

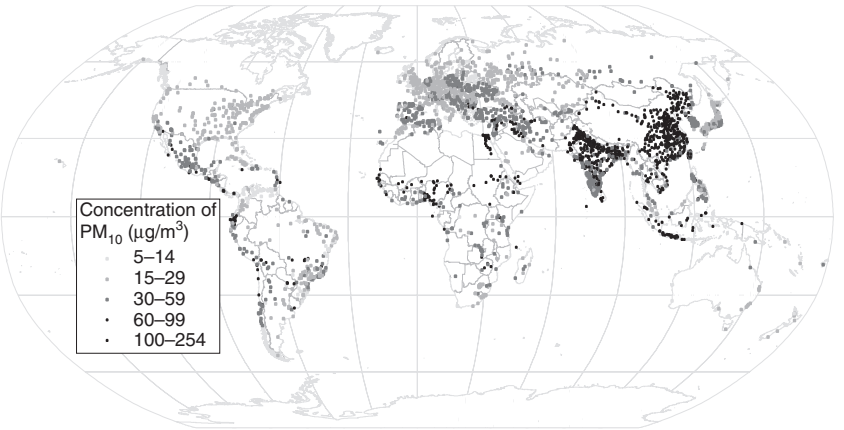
large year-to-year variations in the available measured data even at the same monitoring station, correcting for the average residual provides a better representation of long-term average factors affecting concentrations of PM in a city than using the actual monitored value for the last year of data from monitoring alone.

ESTIMATING SUBREGIONAL AMBIENT CONCENTRATIONS OF PARTICULATE MATTER

To avoid extrapolating outside the sample frame, all exogenous variables were truncated to the range used in the estimation sample. When necessary, missing explanatory variables for the country were filled in with the median values for economically similar countries located in the same geographic area. For most subregions, data were available for at least 95% of the cities, accounting for at least 95% of the population in each subregion. In contrast, data on either fuel, GDP or gross national product (GNP) were missing for 20–30% of the cities, accounting for 20–30% of the population for each of the four subregions AFR-D, AFR-E, EMR-B and EMR-D. In all, data on either fuel, GDP or GNP were completed in this way for 176 cities worldwide, accounting for 5% of the total world urban population.

The estimated annual average concentrations of PM_{10} in urban areas for world cities with populations of >100 000 and national capitals are shown in Figure 17.2. Each circle on the map represents a city and is shaded according to the estimated concentrations of PM_{10} in that city. Standards currently in place in North America and western Europe lie

Figure 17.2 Estimated annual average concentrations of PM_{10} in cities with populations of >100 000 and in national capitals



Source: Map provided by Kiran Dev Pandey, World Bank.

Table 17.4 Population-weighted predicted PM_{10} and TSP and percentiles of the distribution of estimated concentrations of PM_{10}

	Predicted point estimate ($\mu g/m^3$)			*Percentiles of the distribution of estimated PM_{10} ($\mu g/m^3$)*				
Subregion	*PM_{10}*	*TSP*	*PM_{10}/TSP*	*5%*	*25%*	*50%*	*75%*	*95%*
AFR-D	68	195	0.350	32	43	61	72	84
AFR-E	39	104	0.372	30	35	39	44	58
AMR-A	25	39	0.642	24	25	25	25	25
AMR-B	37	79	0.470	35	36	38	39	42
AMR-D	51	146	0.349	37	43	48	53	58
EMR-B	40	118	0.341	23	30	34	39	48
EMR-D	110	276	0.397	62	78	99	110	127
EUR-A	26	49	0.531	25	26	26	27	28
EUR-B	48	118	0.406	41	44	46	48	50
EUR-C	31	90	0.340	21	25	29	33	38
SEAR-B	108	245	0.439	39	86	105	129	151
SEAR-D	84	206	0.409	73	80	84	88	96
WPR-A	32	50	0.646	27	30	32	34	37
WPR-B	89	221	0.403	73	83	89	96	104
World	60	144	0.417	51	56	58	62	65

between 30–60 $\mu g/m^3$. Therefore, we defined a middle group with concentrations of PM_{10} in the range of 30–60 $\mu g/m^3$. Cities with values that fell outside this range were sorted into two groups of cities with higher concentrations and two groups with lower concentrations (thus forming a total of five groups). Worldwide, about 30% of the urban population live in the less polluted cities while 40% live in the more polluted cities. The remaining 30% of people live in cities with concentrations of PM_{10} in the middle range. However, there are significant regional differences. More than 70% of the people living in NAWE live in cities with concentrations of less than 30 $\mu g/m^3$, meeting the most stringent standards. In contrast, more than 70% of the populations in SEAR-D, WPR-B, EMR-D and SEAR-D live in cities where concentrations exceed even the most lenient standards.

This difference can also be seen in the estimated population-weighted mean concentrations of PM_{10} for each subregion, which are presented in Table 17.4. These are computed from 1999 estimates of concentrations of PM_{10} in cities, using the populations of each city in 2000 as weights. We have not directly used data for cities with data from monitoring in computing the subregional averages, to avoid incorporating short-term transitional variations into our exposure estimates.[9] The mean exposures in the most polluted subregions (EMR-D, SEAR-B, SEAR-D and

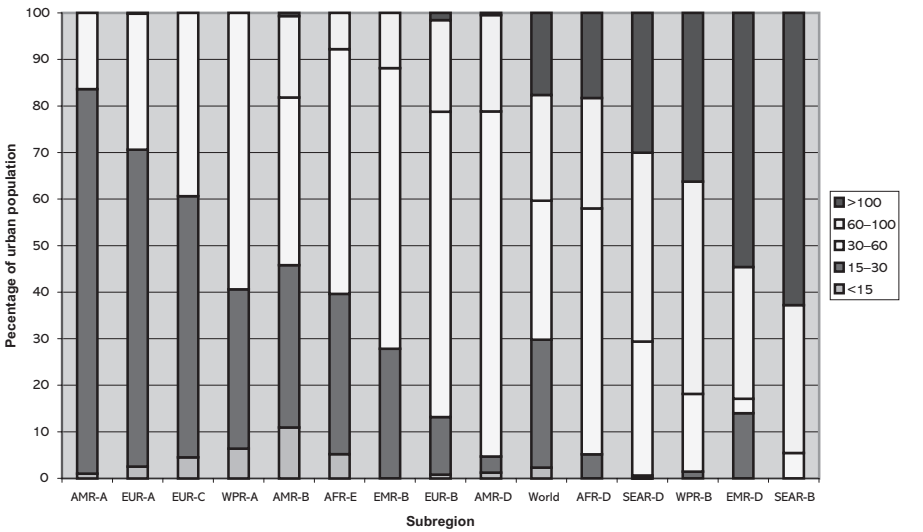
WPR-B) are about three times higher than those in the least polluted subregions (AMR-A and EUR-A). The table also shows predictions of population-weighted average concentrations of TSP and the size composition of PM for each subregion. Finer particles account for a larger share of PM in the highly industrialized countries of AMR-A and EUR-A compared to the other less industrialized subregions.

We quantified the uncertainty in our estimates of the subregion-specific mean concentrations of PM using a bootstrap technique. In this method, the model is re-estimated many times (200 trials) using a randomly repeated sample of the observations used in estimating the model. For each trial, city and population-weighted subregional predictions of PM are generated using the methods described above. The predictions from all trials are sorted from highest to lowest to obtain the percentile distribution of concentrations of PM_{10} for each subregion and are also shown in Table 17.4. The degree of certainty in the point estimates of concentration of PM_{10} for each subregion is directly related to the number of observations available from monitoring of PM. For example, the two subregions (AMR-A and EUR-A) with the most frequently monitored cities also have the smallest confidence intervals for PM_{10} values. In contrast, the five subregions with two or fewer cities that are monitored (AFR-E, AFR-D, EMR-B, EMR-D and SEAR-B) have larger confidence intervals than the other subregions. The width of the confidence intervals for these subregions depends on the geographic and climatic similarity of their cities with monitored data. For example, confidence intervals for AFR-E and EMR-B are about half of those for AFR-D and for EMR-D.

The estimates also show that substantial differences exist in the average concentration of PM within each subregion. The share of the urban population in cities with populations >100 000 and in national capitals according to estimated concentrations of PM_{10} is given in Figure 17.3. All cities in AMR-A, EUR-A, EUR-C and WPR-A are estimated to have concentrations of PM_{10} of $<60\,\mu g/m^3$. In contrast, 95% of the urban population in SEAR-B and about 82% of the urban population in WPR-B and EMR-D are exposed to $>60\,\mu g/m^3$ PM_{10}. We also estimate that a high proportion of the urban population in SEAR-D is exposed to high annual average concentrations of PM_{10}.

Since some of the health outcomes are based on $PM_{2.5}$, rather than PM_{10}, concentrations for this pollutant had to be estimated. City-specific concentration of $PM_{2.5}$ was estimated as a fixed proportion of PM_{10}. Available measurements indicate that the ratio of $PM_{2.5}$ to PM_{10} ranges from 0.5 to 0.8 in many urban areas in developed countries, (California Air Resources Board 2002; U.S. Environmental Protection Agency 2002). Limited evidence suggests that a similar ratio may exist in large cities in other subregions. For example, a recent study from China reports the $PM_{2.5}$:PM_{10} ratio to be in the range of 0.51 to 0.72 in four urban locations (Quian et al. 2001). However, in areas impacted

Figure 17.3 Distribution of the urban population according to estimated concentrations of PM_{10} in cities with populations of >100 and in national capitals, by subregion



by more crustal particles (e.g. arid areas or cities with a significant number of unpaved roads or windy days), the ratios are likely to be much lower. These areas will have a greater proportion of PM_{10} in the coarse size range of 2.5–10μm. For example, evidence from the Coachella Valley (i.e. the Palm Springs area), an arid range of southern California suggests that the $PM_{2.5}$:PM_{10} ratio is 0.35 (Ostro et al. 1999b). Therefore, we assumed a ratio of 0.5 for our base case and have examined the sensitivity of our results to this assumption. Specifically, for our sensitivity analysis, for cities in AMR-A, EUR-A, EUR-B, EUR-C and WPR-A (including the United States, Canada, all Europe, Japan, Singapore, Australia and New Zealand), a higher scaling factor of 0.65 was used, assuming relatively more combustion-related particles, while a lower scaling factor of 0.35 was used for cities in all other subregions.

Estimates of the annual average population-weighted concentration of $PM_{2.5}$ for each subregion were calculated in a similar manner to that for PM_{10}, using the estimated concentration of $PM_{2.5}$ for the city in 1999 and the population for each city in 2000.

2.4 Choice of the theoretical-minimum-risk exposure

Studies of mortality associated with both short- and long-term exposure to PM, discussed below, have been unable to detect a threshold below

which there is no effect of exposure. For most results presented below, we estimated the burden of disease with respect to a counterfactual concentration of 7.5 $\mu g/m^3$ $PM_{2.5}$ (or 15 $\mu g/m^3$ PM_{10}). This value is close to the lowest concentration observed in the epidemiological study (Pope et al. 2002) from which we derived the concentration–response functions used for the majority of our estimates. This choice avoids extrapolating the concentration–response function(s) below the concentrations actually observed in the epidemiologic studies from which they were derived, although health benefits may well accrue from reductions below those concentrations.

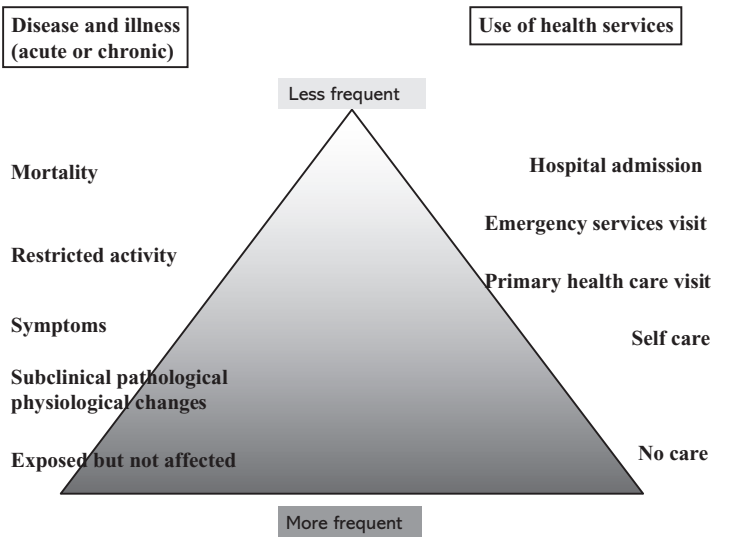
We were aware, however, that for some cities the estimated (and observed) concentrations of PM are lower, e.g. in AMR-A (United States and Canada), and that achieving such concentrations more widely would be not only desirable, but also feasible in some settings (U.S. Environmental Protection Agency 2002). Moreover, previous impact estimates have been sensitive to where this value was set (Künzli et al. 2000). Therefore, we also conducted sensitivity analyses in which the theoretical minimum concentration was halved and doubled (see below).

3. Health effects of exposure to urban air pollution

The past 10–15 years have seen a rapid increase in research on the health effects of air pollution, and it is now widely accepted that exposure to urban air pollution is associated with a broad range of acute and chronic health effects, ranging from minor physiological disturbances to death from respiratory and cardiovascular disease (Anonymous 1996a, 1996b; Figure 17.4). Recently, a committee of the American Thoracic Society identified effects on respiratory health associated with air pollution, which should be considered adverse, spanning outcomes from death from respiratory diseases to reduced quality of life, and included some irreversible changes in physiological function (American Thoracic Society 2000). In general, the frequency of occurrence of the health outcome is inversely related to its severity, with the consequence that assessing total health impact solely in terms of the most severe, but less common, outcomes, such as mortality, will underestimate the total health burden of air pollution (WHO 2001b).

A large body of epidemiological research, discussed in more detail below, provides evidence that exposure to air pollution is associated with increased mortality and morbidity. The respiratory and cardiovascular systems appear to be the most affected. A growing body of toxicological and clinical evidence currently offers some limited insight into the mechanisms through which exposure to air pollution may produce the effects on respiratory and cardiovascular outcomes observed in epidemiological studies (Anonymous 1996a, 1996b; Health Effects Institute 2002). These mechanisms may involve decrements in pulmonary func-

Figure 17.4 The relative frequencies of health events associated with exposure to air pollution



tion, effects on heart rate variability and inflammatory response. Long-term bioassays and other studies of toxicity provide evidence for the mutagenicity and/or carcinogenicity of some components of urban air pollution, such as emissions from diesel-powered vehicles (Cohen and Nikula 1999; Diesel Working Group 1995).

Air pollution may elicit both acute and chronic biological responses. Acute responses to air pollution in otherwise healthy persons may be confined to reversible physiological adaptations resulting from natural defence mechanisms (e.g. watery eyes, cough or a transient fall in lung function). Acute responses may, however, also increase the severity or duration of an already established respiratory infection or of diseases such as asthma or chronic obstructive lung disease that have already placed the individual in a vulnerable position, and increase the risk of hospital admission or even death. If such vulnerability were temporary, for example, a severe infection of the lower respiratory tract, the individual might have recovered and lived for some time, had it not been for the added factor of exposure to air pollution at the time the individual was most vulnerable because of the infection. On the other hand, if the individual had a terminal chronic condition, such as severe chronic obstructive pulmonary disease or chronic congestive heart failure, exposure to air pollution might advance death by only a short time, this being imminent in any case. There is limited epidemiological evidence to suggest that ambient air pollution may contribute to the development

of diseases such as chronic obstructive pulmonary disease, for which smoking and, in developing countries, indoor air pollution, are also risk factors (Abbey et al. 1999; Pope and Dockery 1999; Tager et al. 1998). Distributions of short-and long-term vulnerability, reflecting the prevalence of acute and chronic cardiorespiratory disease, may well differ across populations worldwide. This will have implications for the transferability of risk functions from studies in populations in NAWE to populations where differences in genetic factors, diet, tobacco smoking, extent of urbanization, distribution of wealth and other factors related to social class, have resulted in different patterns of disease.

For example, recent analyses of two cohorts in the United States (Krewski et al. 2000) showed clearly that the effects of long-term exposure to air pollution on mortality depend on attained educational level, with the largest relative effects observed among the least educated. Recent studies in developing countries have also reported such gradients in the relative risks of mortality (O'Neill et al. 2003). It is not clear what factor(s) might be responsible for these observations (e.g. aspects of occupation or diet), but it is reasonable to expect that they might vary across the globe. Differences in vulnerability to air pollution introduce a source of uncertainty in our estimates that can currently be only partially quantified.

Epidemiological evidence about exposure–response relationships is most directly applicable to the risk assessment of air pollution, because it comes from the direct observation of human populations under relevant conditions (Samet 1999). Epidemiological studies have limitations that are largely a result of their observational nature. These relate to the accurate measurement of exposure, definition of outcomes and interpretation of associations that are observed. Assessing the causality of such associations requires a process of scientific reasoning that considers all evidence, including that from experimental studies (WHO 2000b). While there remain many gaps in our knowledge about the explanations for epidemiological associations, they can provide the best evidence to guide action to reduce the exposure of the population to air pollution, and to undertake health impact assessments, provided the uncertainties are recognized.

The epidemiology of air pollution takes advantage of the fact that concentrations of urban air pollution, and thus human exposure, vary in both time and space. For the most part, current epidemiological research has focused on either one or the other dimension, but infrequently on both within the same population(s). Short-term temporal variation in concentrations of air pollution over days and weeks has been used to estimate effects on daily mortality and morbidity. Spatial variation in long-term average concentrations of air pollution has provided the basis for cross-sectional and cohort studies of long-term exposure.

3.1 Studies of short-term exposure

The effects of short-term exposure to air pollution have been extensively studied in time-series studies in which daily rates of health events (e.g. deaths or hospital admissions) in one or more locales are analysed in relation to contemporaneous series of daily concentrations of air pollutants, and other risk factors (e.g. weather) that vary over time periods of months or years. Regression techniques are used to estimate a coefficient that represents the relationship between exposure to pollution and the outcome variable. The usual method of regression models the logarithm of the outcome, and thus arrives at an estimate of the relative risk, a proportional change in the outcome per increment of ambient concentration. There has been a rapid increase in the number of these studies as computing and statistical techniques have improved and as data on outcomes and air pollution have become more extensive and easily accessible from routine sources. It is a strength of these studies that individual cofactors, such as smoking, nutrition, behaviour, genetic factors, etc., are unlikely confounders because they are not generally associated, on a day-to-day basis, with the daily concentration of air pollution. Studies of time series have found associations between concentrations of PM in the air and a large range of outcomes. These have been reviewed by Pope and Dockery (1999) and include daily mortality (all causes, respiratory, cardiovascular), hospitalization for respiratory diseases (all causes, chronic obstructive pulmonary disease, asthma, pneumonia) and for cardiovascular diseases (acute myocardial infarction, congestive cardiac failure). Since this review, associations have also been reported for primary health care visits for disease of the lower respiratory tract, and diseases of the upper respiratory tract of both infective and allergic origin (Hajat et al. 2001, 2002). However, recent methodological studies and re-examination of earlier work indicate that the magnitude of the estimates of relative risk from time-series studies of daily mortality depends on the approach used to model both the temporal pattern of exposure (Braga et al. 2001) and potential confounders that vary with time, such as season and weather (Health Effects Institute 2003).

The acute effects of air pollution have also been studied longitudinally in panel studies, which can provide evidence of physiological effects at an individual level. Small groups, or panels, of individuals are followed over short time intervals, and health outcomes, exposure to air pollution and potential confounders are ascertained for each subject on one or more occasions. Panel studies have generally reported associations of exposure to urban air pollution with increased prevalence of symptoms involving the upper and lower respiratory tract, and increased rates of asthma attacks and medication use. Associations with short-term reduction in lung function and the prevalence of cough symptoms have been reported in studies in the United States (Pope and Dockery 1999), but are not consistently supported by studies in Europe (Roemer et al. 1999).

Time-series studies in adults across the world

Studies of time series concerning daily mortality and, to a lesser extent, daily hospital admissions, have been conducted in cities throughout the world. A recent meta-analysis summarized the evidence from >100 studies of daily mortality (Stieb et al. 2003). In addition, large studies have now been conducted using uniform methods for assembling and analysing data from multiple cities: APHEA 2 (Air Pollution and Health: A European Approach) (Katsouyanni et al. 1996, 2001) and NMMAPS (National Mortality and Morbidity Air Pollution Study) (Health Effects Institute 2000a, 2000b) in the United States. These multi-city studies have confirmed the findings of earlier studies of individual cities in finding positive associations between daily mortality and hospital admissions and concentrations of PM, and have also attempted to explain the heterogeneity among cities in the relative risks associated with exposure to air pollution. For example, in the APHEA 2 Study, it was found that the effects of PM on mortality were modified by mean concentrations of nitrogen dioxide (Katsouyanni et al. 2001), and in the NMMAPS Study, daily mortality was modified by the long-term average concentrations of PM_{10}. Levy et al. (2000) reported that the effects of PM_{10} were greater in cities where $PM_{2.5}$ comprised a higher proportion of PM_{10}.

Most studies of time series are from countries in NAWE, where air pollution is low and decreasing and populations are characterized by western lifestyles and patterns of disease. To examine the epidemiological evidence for other non-NAWE countries, we searched a database of studies of time series and panel studies compiled at St George's Hospital Medical School, for which researchers had systematically ascertained, reviewed, and abstracted results from studies published in the peer-reviewed scientific literature (WHO 2003). All studies meeting pre-specified quality criteria related to adequacy of confounder control, and which provided estimates of the concentration–response relationship and its statistical precision were included. We classified them by the subregion in which the study was performed and tabulated the results for six outcomes: all-cause mortality, respiratory mortality, cardiovascular mortality, infant and child mortality, reduction in peak expiratory flow rate and cough symptom.

The distribution of time-series and panel studies by outcome and subregion is shown in Tables 17.5(a) and (b). Up to mid-November 2001, the number of studies from AMR-A (71) and EUR-A (75) far exceeded the total for the remaining 12 subregions (42). The next largest contributor was AMR-B (Central and South America), with 18 studies. The table shows the numbers of panel studies presenting usable numerical estimates. Only 14 studies from non-NAWE subregions were identified, compared with 64 from the NAWE subregions. Some of the non-NAWE countries had lifestyles and patterns of disease similar to those in NAWE—those in Australasia, for example.

Table 17.5 Distribution of studies by outcome status and subregion

(a) Selected time-series studies

	Outcome status					
	Cause of mortality			*Hospital admissions/ emergency room visits*	*Other time-series studies*	*Total (from subregion)*
Subregion	*All causes*	*Respiratory disease*	*Cardiovascular disease*			
AFR-D	0	0	0	0	0	0
AFR-E	0	0	0	0	0	0
AMR-A	34	11	13	39	2	71
AMR-B	10	9	6	5	0	18
AMR-D	0	0	0	0	0	0
EMR-B	0	0	0	0	0	0
EMR-D	0	0	0	0	0	0
EUR-A	44	28	24	28	3	75
EUR-B	4	3	4	0	0	5
EUR-C	0	0	0	0	0	0
SEAR-B	1	1	1	0	0	1
SEAR-D	0	0	0	0	0	0
WPR-A	4	4	4	2	0	6
WPR-B	8	3	4	4	0	12
World (from outcome status)		113		78	5	187

(b) Selected panel studies

	Outcome status for valid studies		*Total for selected studies (from subregion)*
Subregion	*Lung function*	*Symptoms*	
AFR-D	0	0	0
AFR-E	0	0	0
AMR-A	19	17	28
AMR-B	3	3	3
AMR-D	0	0	0
EMR-B	0	0	0
EMR-D	0	0	0
EUR-A	32	31	38
EUR-B	3	2	3
EUR-C	2	1	2
SEAR-B	0	0	0
SEAR-D	0	1	1
WPR-A	4	1	4
WPR-B	0	1	1
World (from outcome status)	62	57	79

Figures 17.5 and 17.6 show the results for daily mortality and PM, by subregion, for adults and children, respectively. These estimates were scaled to PM_{10} ($PM_{2.5} = 0.6 \times PM_{10}$, $BS = 0.5 \times PM_{10}$, $TSP = 2 \times PM_{10}$). These scaling factors were decided after examining a number of co-located measures, but are likely to be variable across the individual cities. Estimates of random effects and fixed effects are shown because there is heterogeneity.

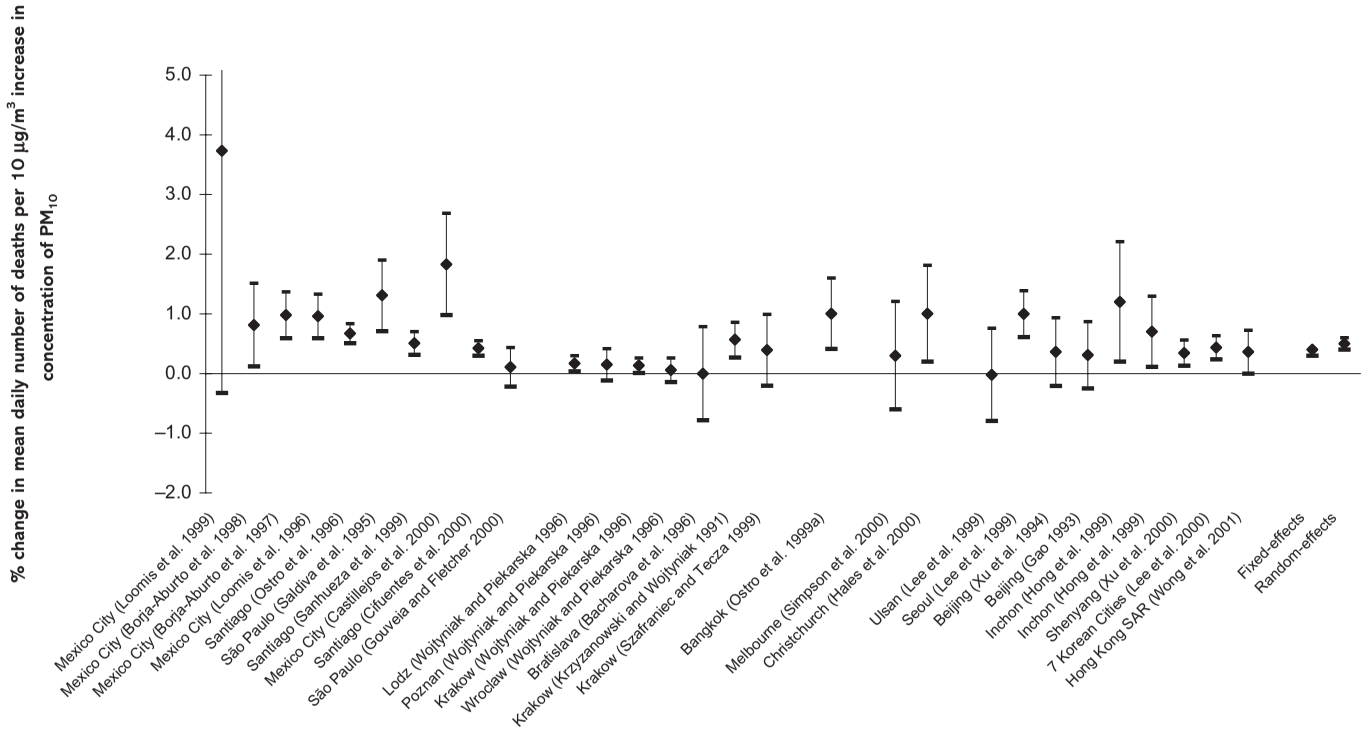
The pooled estimates are shown in Table 17.6. Most of the studies of mortality showed relative risks of >1.0 (i.e. a change of >0.0%), with lower 95% confidence intervals also >1.0. However, the studies showing the largest confidence intervals also tended to have the largest effects, this indicating the possibility of publication bias. There was considerable heterogeneity in the estimates. For this reason, the summary estimate for random effects is more appropriate because it takes into account the greater uncertainty. The estimate for random effects was increased and statistically significant for mortality from all causes, respiratory and cardiovascular diseases. It is remarkable that for daily mortality, the pooled estimate for the non-NAWE subregions of 0.5% increase in daily mortality per 10 $\mu g/m^3$ increase in PM was very similar to the estimates produced by the APHEA 2 and NMMAPS studies of 0.6 and 0.5, respectively. A recent meta-analysis of 109 published studies from around the world reports similar estimates (Stieb et al. 2003).

These results indicate that daily mortality is positively associated with short-term exposure to urban air pollution at time-scales in the order of days, in all subregions where this association has been measured. They also suggest that the relative effect of exposure may also be of similar magnitude in different parts of the world.

Air pollution and reproductive and child health

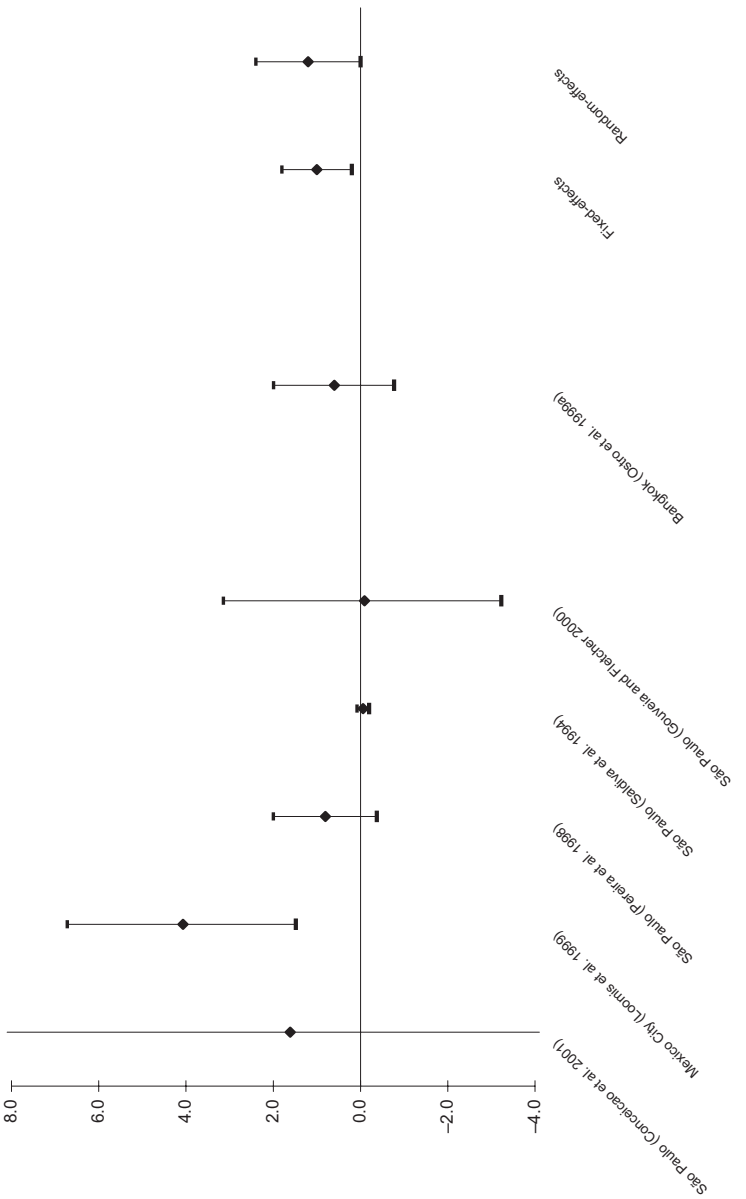
Six time-series studies of daily mortality report associations between particulate pollution and adverse effects in children, and all of them are from non-NAWE countries. Their estimates are shown in Figure 17.6. Four were from São Paulo (Conceiao et al. 2001; Gouveia and Fletcher 2000; Pereira et al. 1998; Saldiva et al. 1994), one from Mexico City (Loomis et al. 1999) and one from Bangkok (Ostro et al. 1999a). The Bangkok study was of PM_{10} and daily mortality from all causes in children aged <6 years. The study conducted in Mexico City evaluated the impact of daily changes in concentrations of $PM_{2.5}$ and total mortality in children aged <1 year. Three studies in São Paulo (Conceiao et al. 2001; Gouveia and Fletcher 2000; Saldiva et al. 1994), conducted during different periods of time, all reported an association between PM and mortality from respiratory disease in children aged <5 years. The study conducted in São Paulo by Pereira et al. (1998) investigated the association of exposure to urban air pollution with intrauterine mortality. Some of the relative risks reported in these studies were >1.0, but only the estimate from Mexico City was statistically significant at the 95% level.

Figure 17.5 Percentage change in mean daily number of non-accidental deaths, in adults, per 10 μg/m^3 increase in PM_{10}, by city



Note: Scaling factors used: $PM_{2.5} = 0.6 \times PM_{10}$, $BS = 0.5 \times PM_{10}$, $TSP = 2 \times PM_{10}$.

Figure 17.6 Percentage change in mean daily number of non-accidental deaths, in children, per 10μg/m^3 increase in PM_{10}, by study



Note: Scaling factors used: $PM_{2.5} = 0.6 \times PM_{10}$, $BS = 0.5 \times PM_{10}$, $TSP = 2 \times PM_{10}$.

Table 17.6 Pooled estimates of daily mortality from all causes and concentrations of PM_{10}, from time-series studies, excluding North America and western Europe

	All-cause mortality (% change)	*Mortality from respiratory disease (% change)*	*Mortality from cardiovascular disease (% change)*	*Infant and child mortality (% change)*	*Lung function (regression coefficients)*	*Symptoms (odds ratios)*
No. of studies	29	18	18	5[a]	11	10[b]
Heterogeneity	Q=121.62 on 28 *df* (P<0.001)	Q=68.86 on 17 *df* (P<0.001)	Q=51.40 on 17 *df* (P<0.001)	Q=7.72 on 4 *df* (P=0.102)	Q=16.16 on 10 *df* (P=0.095)	Q=26.68 on 9 *df* (P=0.002)
RE (95% CI)	0.5 (0.4–0.6)	0.6 (0.2–1.1)	0.3 (0.1–0.5)	1.0 (–0.9–3.1)	–0.024 (–0.146–0.098)	1.006 (0.990–1.022)
FE (95% CI)	0.4 (0.3–0.4)	0.4 (0.2–0.6)	0.1 (0.1–0.2)	1.0 (–0.1–2.1)	–0.007 (–0.095–0.081)	1.006 (0.998–1.013)
NMMAPS study 20 USA cities (Health Effects Institute 2000b)	0.46 (0.27–0.65)	—	—	—	—	—
APHEA 2 study 29 European cities (Katsouyanni et al. 2001)	0.62 (0.4–0.8)	—	—	—	—	—

Key: RE, random-effects estimate; FE, fixed-effects estimate; Q, χ^2 statistical test for heterogeneity; *df*, degrees of freedom.

— No data.

[a] 1 study (Pereira et al. 1998) not included in meta-analysis due to uncommon outcome.

[b] 1 study (Awasthi et al. 1996) not included in meta-analysis due to uncommon particle measurement.

Infant mortality from respiratory disease and other adverse perinatal events, such as low birth weight and malformations, have also been associated with more prolonged exposure to air pollution (Bobak and Leon 1999; Wilhelm and Ritz 2003; Woodruff et al. 1997). Woodruff et al. followed a large birth cohort in the United States for one year and estimated the relative risk of mortality associated with residential exposure to PM_{10} in the first two post-natal months, conditional on a variety of potential confounders, including maternal smoking. They reported an increase in total mortality of 4% per 10 $\mu g/m^3$, and 20% for mortality from respiratory causes. Two studies recently evaluated changes in infant mortality associated with reductions in industrial emissions caused by a recession and mandated reductions in pollution resulting from the United States Clean Air Act Amendments of 1970 (Chay and Greenstone 1999, 2001). Using county-level data, the authors estimated that 4–8 infant deaths per 100 000 live births were prevented for each 1 $\mu g/m^3$ reduction in TSP.

Studies of acute morbidity

Far fewer studies have been conducted of the association of exposure to urban air pollution with acute morbidity, especially in non-NAWE countries. There were insufficient studies in any one outcome group to allow formal meta-analysis of non-NAWE studies, but most reports suggested a positive association with urban air pollution, consistent with that observed in NAWE countries, especially for hospital admissions (Atkinson et al. 2001; Burnett et al. 1999; Health Effects Institute 2000b). A recent study compared directly the effects of air pollution on hospital admissions in China, Hong Kong Special Administrative Region (Hong Kong SAR) and London. Similar associations were observed for PM_{10} and gaseous pollutants and hospital admissions for ischaemic heart disease in both locations, and the associations were strongest during seasons of low humidity in both cities, but no association with admissions for cardiac disease was observed in Hong Kong SAR (Wong et al. 2002).

3.2 Studies of long-term exposure

Cohort studies of mortality from chronic respiratory and cardiovascular disease

Cohort studies take advantage of spatial heterogeneity in concentrations of air pollution to compare the incidence of disease and death in populations exposed in the long term to differing levels of pollution. By following large populations for a number of years, cohort studies provide estimates of both attributable numbers of deaths and, more importantly, average reductions in life span attributable to air pollution.

The evidence from cohort studies of populations in Europe and the United States indicates that long-term exposure to urban air pollution

is associated with an increase in total and cardiopulmonary mortality in adults (Dockery et al. 1993; Hoek et al. 2002; Lipfert et al. 2003; McDonnell et al. 2000; Pope et al. 2002). In each of these studies, the effects of potential confounders such as cigarette smoking, occupation and prior medical history were adjusted for in regression analyses. Most studies find the strongest and most consistent associations with exposure to PM, and $PM_{2.5}$ appears to be more closely associated with mortality than PM_{10} or TSP (Dockery et al. 1993; Pope et al. 2002). The recently published results of a study conducted in the Netherlands confirm the impacts of long-term exposure to air pollution, and in particular that related to road traffic, on mortality (Hoek et al. 2002).

Unfortunately, the cohort studies provide little information on when exposure to air pollution acts to increase the risk of mortality (i.e. the induction time for mortality attributable to exposure to long-term exposure to air pollution), making it difficult to estimate when the effects of reduction of air pollution might be observed.

Comparable cohort studies have not yet been carried out in developing countries. However, the imposition of restrictions on the sulfur content of fuel for power generation and transportation in Hong Kong SAR, instituted over short time intervals in 1990, provided opportunities for researchers to measure directly the impact of reducing air pollution on long-term average mortality (Hedley et al. 2002). Hedley et al. (2002) documented both changes in ambient air quality subsequent to the imposition of the restrictions, and declines in long-term average rates of mortality from cardiovascular and respiratory diseases associated with those changes. Comparison of changes in mortality in more and less polluted areas of Hong Kong SAR provided limited ability to account for secular changes in other risk factors for mortality that could have produced the observed decrease in mortality following the change in the sulfur content of fuel. A similar study was also published recently by Clancy et al. (2002), who measured decreased long-term average mortality in Dublin after the banning of the sale of bituminous coal in Dublin in 1990.

The American Cancer Society study

The American Cancer Society (ACS) study (Pope et al. 2002) in the United States is by far the largest cohort study of air pollution and long-term average mortality to date. The ACS study of air pollution and mortality is based in the ACS Cancer Prevention II Study, an on-going prospective cohort of approximately 1.2 million adults from all 50 states (Calle et al. 2002). Friends and neighbours recruited cohort members on behalf of the ACS. Participants were enrolled in 1982, when they were aged ≥30 years, and their mortality has been ascertained through to 1998. Data on a wide range of risk factors for cancer and other chronic diseases were obtained from each participant. The ACS study links the data for approximately 500 000 cohort members with data on air pol-

lution from metropolitan areas throughout the United States. The first study of air pollution and mortality in this cohort (Pope et al. 1995) was based on follow-up through to 1990. That study reported increases in mortality from cardiopulmonary disease for 19.9 $\mu g/m^3$ fine particulate sulfate (relative risk of 1.26, 95% CI 1.16–1.37), and from lung cancer (relative risk of 1.36, 95% CI 1.11–1.66). These findings were subsequently corroborated in an independent re-analysis (Krewski et al. 2000). A more recent analysis of this cohort extended follow-up through to 1998, and ascertained 40 706 deaths from cardiopulmonary disease, and 10 749 from lung cancer. Data were analysed using Cox proportional hazards regression models that incorporated both random effects and non-parametric spatial smoothing to adjust for unmeasured factors correlated spatially with air pollution and mortality across the United States. The models also adjusted for age, sex, race, education, marital status, body mass, diet, alcohol consumption, occupational exposures and the duration and intensity of cigarette smoking, all measured via questionnaire at enrolment.

Concentrations of ambient air pollution had, in general, declined across the United States between 1982 and 1998. Measurements of ambient concentrations of fine particulate air pollution ($PM_{2.5}$) in the cities where subjects resided at enrolment were available for periods both briefly preceding enrolment (1979–83) and immediately after follow-up (1999–2000). In separate regression analyses, cohort members were assigned estimates of exposure corresponding to their city-of-residence-specific value for each of those periods, as well as for the average value across the two periods. For a change of 10 $\mu g/m^3$ in the ambient concentration of $PM_{2.5}$, the smallest relative increases were observed for the mean concentration of the time period 1979–1983. This estimate was based on data from 61 cities, with a mean concentration of $PM_{2.5}$ of 21.1 $\mu g/m^3$, and a range of 10–30 $\mu g/m^3$. The relative risks for a 10 $\mu g/m^3$ change in the concentration of ambient $PM_{2.5}$ were larger when exposure was specified as the average of the ambient concentrations of the two time periods. This may be explained by the fact that the estimates from the earliest periods are more subject to random (and non-differential) error. However, it also suggests that more recent exposures may be exerting the strongest effects on mortality, an interpretation also offered in the recent re-analysis of the earlier follow-up of the ACS cohort (Krewski et al. 2000). Unfortunately, it was not possible to derive individual time-varying estimates of exposure from the available data (e.g. detailed residence histories were unavailable), precluding direct evaluation of the induction time for mortality attributable to exposure to air pollution.

Long-term exposure and the incidence of chronic disease

Little evidence is available concerning exposure to air pollution and the incidence of chronic cardiovascular or respiratory disease. One study in

the United States reported an association of long-term exposure to PM_{10} with the incidence of self-reported physician-diagnosed chronic bronchitis (Abbey et al. 1999). A recent case–control study reported an association between short-term exposure and the incidence of non-fatal myocardial infarction (Peters et al. 2001). Cross-sectional studies have found associations with reduced lung function and increased respiratory symptoms in both adults and children, which might in part represent chronic disease as the result of long-term exposure. Several recent cross-sectional studies in large Chinese cities have reported increased prevalence of respiratory symptoms in adults (Qian et al. 2001; Zhang et al. 1999) and elementary school children (Qian et al. 2000; Zhang et al. 2002) exposed to urban air pollution. A cross-sectional study in Delhi observed reductions in pulmonary function in residents of highly polluted areas, but little evidence of increased prevalence of symptoms (Chhabra et al. 2001).

Air pollution and lung cancer

Epidemiological studies over the last 40 years have observed that general ambient air pollution, chiefly composed of the by-products of the incomplete combustion of fossil fuels, is associated with small relative increases in the incidence of lung cancer. The evidence derives from studies of trends in the incidence of lung cancer, studies of occupational groups, comparisons of urban and rural populations, and case–control and cohort studies using diverse exposure metrics. Recent prospective cohort and case–control studies which have controlled for the effects of cigarette smoking, occupation and other risk factors have consistently observed small increases in the relative risk of lung cancer in relation to exposure to particulate air pollution (Abbey et al. 1999; Dockery et al. 1993; Krewski et al. 2000; Pope et al. 2002; Samet and Cohen 1999). A recent Swedish case–control study reported that excess lung cancer was related specifically to exposure to mobile sources of air pollution, with the largest effects observed for exposure occurring 20 years prior to diagnosis (Nyberg et al. 2000).

3.3 Choice of outcomes and hazards

Studies used for hazard estimates

The use of results from time series to estimate the disease burden attributable to urban air pollution is problematic for various reasons. First, data on rates of occurrence, such as hospital admissions, primary health care consultations or asthma exacerbation are not collected in many countries. Thus there is no baseline upon which to develop an estimate of health impact. Mortality is an exception in that data are available from death registration or indirect demographic methods in all subregions.

The application of the time-series concentration–response functions to the assessment of mortality impact, however, is limited. Specifically, it is not possible to use the results of studies of time series to estimate YLL in adults. This is because time-series studies of daily mortality do not in themselves allow the estimation of lost life time, but rather only allow estimation of the number of deaths that have been brought forward by an unspecified amount of time. Recent research has made clear that the time-series estimates reflect deaths that may have been brought forward by as much as several months, rather than simply advancing the time of death in frail people by a few days (Schwartz 2000; Zeger et al. 1999). The design of the time-series study of daily mortality, which requires the control of long-term variation in air pollution, precludes estimation of greater losses (Künzli et al. 2001; Leksell and Rabl 2001; McMichael et al. 1998). Thus, the time-series studies only provide an estimate of the daily number of deaths brought-forward.

Cohort studies include not only people whose deaths were advanced by recent exposure to air pollution, but also those who died from chronic disease caused by long-term exposure (COMEAP 1998; Künzli et al. 2001), thus they provide a more comprehensive estimate of the effects on mortality. Furthermore, because their relative risks can be applied to population life tables, the effects of air pollution on life span can be estimated (Brunekreef 1997; COMEAP 2001; Hurley et al. 2000; Sommer et al. 2000)

The situation may be different for children. In developing countries, the major causes of death, such as acute respiratory disease, are very likely to result from a severe acute infection, which represents a brief window of vulnerability. If the child survives, they might be expected to fully recover and enjoy a full life expectancy. If we assume that death was not otherwise imminent, then these deaths, on average, represent the loss of considerable life years. Under such an assumption, one could use time-series estimates to estimate YLL in children aged 0–4 years.

In making these estimates, several further considerations should be kept in mind. The first is that the effects of cumulative exposure over several weeks are several times greater than those obtained by using a single day lag and thus underestimate the impacts on health. The second is that other air pollutants in the mixture may be exerting additional effects, may interact with particles or may be confounding the associations of particles. Ozone, for example, is also toxic and while its effects tend to be independent of PM, it also seems to modify the effect of particles on number of hospital admissions (Atkinson et al. 2001).

Definition and specification of health outcomes

We estimated the burden of disease imposed by mortality from cardiopulmonary disease and lung cancer in adults, and from ARI in children aged 0–4 years. We made this choice despite the fact, discussed above, that other serious health effects of air pollution are well-

documented, and that still others appear from more limited evidence to be of potential concern. Our decision to focus on mortality outcomes was made on the basis of the following considerations.

- *Strength of evidence*. A large body of research from many parts of the world indicates that ambient air pollution causes increased daily mortality from cardiovascular and respiratory disease. There appear to be comparable effects in the cities of developed and developing countries, on the basis of the limited evidence available. Although no cohort studies of mortality have as yet been conducted in developing countries, the possibility that associations comparable to those observed in the United States would be observed is strengthened by the results of the studies of daily mortality, and the limited results from studies of morbidity.
- *Consistent definition of the end-point*. Mortality *per se* is a well-defined event that is registered in most countries. For this reason, epidemiologists have frequently measured the effect of air pollution on total mortality from all natural causes, ascertained from death certificates or other sources of vital statistics. Other outcomes, such as bronchitis and the symptom of wheeze, are subject to very large variations in severity, and without such qualification their impact on health is difficult to assess. The definitions of other possible health outcomes, such as restricted activity days, use of primary health care services, diagnoses and school absences, are likely to vary with national culture and among health care systems.

  The cause of death is more problematic because it is not certified medically in many countries, and there are considerable differences between and within countries in terms of diagnostic practice. Nevertheless, we propose to base our estimates primarily on cardiopulmonary, rather than total, mortality. There is strong evidence from both time-series and cohort studies that ambient air pollution specifically increases the risk of death from these causes. Moreover, variations in the relative contribution of non-cardiopulmonary mortality among countries could increase the error in the burden assessment, particularly in countries with lower cardiopulmonary death rates, potentially leading to overestimates of impact. Since considerable cross-coding is likely, we have chosen to use the combined cardiopulmonary group consisting of GBD infectious and chronic respiratory diseases and selected cardiovascular outcomes for adults. In children, death from cardiovascular diseases is rare and the pulmonary group is adequate.
- *Availability of baseline occurrence rates*. Data on age-specific mortality are collected or estimated using consistent methods for all subregions. This is not the case for some important potential measures of morbidity, such as the frequency of asthma attacks, or measures of

the utilization of health care services outside of Europe and North America.

- *Importance of the end-point in terms of health impact*. Although the impacts of air pollution on other health end-points must certainly contribute to the global burden of disease, mortality, quantified in terms of either numbers of deaths or reduced survival time, currently plays the most prominent role in impact assessments. We chose these three specific mortality outcomes (mortality from cardiopulmonary causes and lung cancer in adults aged ≥30 years, and mortality from ARI in children aged 0–4 years) because they allow us to estimate YLL, as discussed above.
- *Feasibility within the time constraints of the current work*. Given additional time and resources, future efforts might possibly consider, for example, using the evidence from the International Study of Asthma and Allergies in Childhood (ISSAC) (Anonymous 1998), which has data on prevalence from 60 countries as a baseline upon which to estimate the effect of particles on the exacerbation of asthma. Another possibility might be the effect on hospital admissions or primary health care visits.

3.4 Developing the concentration–response functions

We derived concentration–response functions for three end-points to produce the estimates of global burden of disease reported in this work: mortality from cardiopulmonary causes and lung cancer in adults aged ≥30 years, and mortality from ARI in children aged 0–4 years. As discussed earlier, we made no estimates of the impacts of PM on the incidence of disease, so the disability-adjusted life years (DALYs) quantify only YLL.

We assumed a log-linear risk model which leads to the following formula for the relative risk (RR) in a population whose exposure is estimated by an average concentration of pollution C relative to the reference level C_0:

$$RR = \exp[\beta(C - C_0)]; \tag{6}$$

where, C_0, the reference, or theoretical minimum level of exposure, is defined as above and β is the estimated effect of PM on the health outcome of interest. We calculated a subregion-specific relative risk for each of the 14 subregions using a population-weighted mean of concentrations for all cities in the subregion calculated as follows:

The subregion-specific relative risk for outcome i in subregion k related to $PM_{2.5}$, $RR_{2.5ik}$, is:

$$RR_{2.5ik} = \exp[\beta_{2.5i} \times (Ck_{2.5} - 7.5)] \tag{7}$$

where $C_{k2.5}$ is the subregion-specific population-weighted mean concentration of $PM_{2.5}$ (calculated from the estimated concentration of PM_{10} in Table 17.4, as described above), and $\beta_{2.5i}$ is the slope of the concentration–response function for $PM_{2.5}$ (Table 17.7).

The subregion-specific relative risk for outcomes quantified in terms of PM_{10}, RR_{10ik}, is:

Table 17.7 Estimates of relative risk of mortality, coefficients of concentration–response functions and study types

Health outcome	*Data source*	*PM exposure metric*	*Relative risk per 10 μg/m³ (95% CI), from data source*	*Concentration–response slope[a] per μg/m³ (standard error)*
Mortality from cardiopulmonary disease—adults	ACS study (Pope et al. 2002)	$PM_{2.5}$	1.059 (1.015–1.105)	Linear[b] 79–83 0.00575 (0.002160) Linear average[c] 0.008933 (0.002907) Log-linear[d] 79–83 0.11605 (0.044790) Log-linear average 0.155148 (0.050460)
Mortality from lung cancer	ACS study (Pope et al. 2002)	$PM_{2.5}$	1.082 (1.011–1.158)	Linear 79–83 0.00789 (0.003447) Linear average 0.012673 (0.00426) Log-linear 79–83 0.17114 (0.071968) Log-linear average 0.232179 (0.074770)
Mortality from acute respiratory infection—children aged 0–4 years	St George's Hospital meta-analysis of five time-series studies of daily mortality	PM_{10}	1.010 (0.991–1.031)	0.0010 (0.0010)
Deaths-brought-forward—all ages	St George's Hospital meta-analysis of 165 time-series studies of daily mortality	PM_{10}	1.006 (1.005–1.007)	0.0006 (0.00005)

[a] Slope of the concentration–response (CR) function for air pollution and mortality.

[b] Results form regression models in which annual average concentrations measured from 1979–1983 were used as estimates of exposure (Pope et al. 2002).

[c] Results form regression models in which the average of annual average concentrations measured from 1979–1983 and 1999–2000 were used as estimates of exposure (Pope et al. 2002).

[d] Results from regression models where exposure (i.e. annual average $PM_{2.5}$) is specified on the log scale.

$$RR_{10ik} = \exp[\beta_{10i} \times (C_{k10} - 15)] \quad (8)$$

where C_{k10} is the subregion-specific population-weighted mean concentration of PM_{10} (Table 17.4), and β_{10i} is the slope of the concentration–response function for PM_{10}. The city-specific concentrations of PM_{10} were truncated at 15 and 100 μg/m^3 for calculation of subregion-specific population-weighted mean concentrations of PM_{10}.

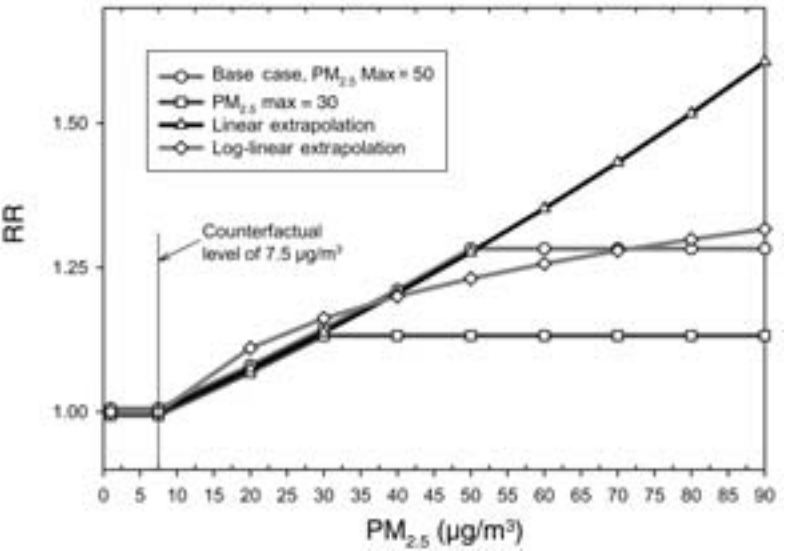
Mortality from cardiopulmonary disease and lung cancer in adults

We used the results of the ACS study of urban air pollution and mortality (Pope et al. 2002) to estimate attributable deaths and YLL from cardiopulmonary diseases and lung cancer in adults aged ≥30 years. In our base-case analyses we used the estimates of the concentration–response functions based on the ambient concentrations in 1979–1983, which correspond to increases of 5.9% and 8.2% in mortality from cardiopulmonary disease and lung cancer, respectively, for each 10 μg/m^3 change in the ambient concentration of $PM_{2.5}$ (Table 17.7).

Deaths from cardiopulmonary disease and lung cancer in the ACS cohort were defined as persons whose underlying cause of death was coded according to the International Statistical Classification of Diseases, ninth revision, on their death certificates as ICD-9 401–440 and 460–519, and 162, respectively. When calculating the attributable fraction, ACS concentration–response functions for cardiopulmonary disease defined in this way were applied to baseline cause-specific rates of mortality in the GBD project. For lung cancer, this corresponded exactly to the definition used in the ACS study. For cardiopulmonary deaths, the GBD groupings of cause of death (39, 40, 106–109, 111) did not include several ICD codes (406–409, 415–417, 423–424, 426–429, 440) that were included in the ACS definition. These codes represent diverse cardiac diseases, including conduction disorders, cardiac dysrhythmias, heart failure and ill-defined cardiac causes. Together they comprise approximately 18% of all cardiopulmonary deaths in the ACS study (R. Burnett, personal communication).

The ACS study estimated concentration–response functions for $PM_{2.5}$ over a range that extends from annual average concentrations of $PM_{2.5}$ of about 5–30 μg/m^3 (Pope et al. 2002). The shape of the concentration–response function for fine particulate air pollution outside that range is currently unknown, as noted above, and estimated annual average concentrations of $PM_{2.5}$ in some subregions are outside that range (Table 17.1). In our base-case analyses we limited the risk of mortality in any city to be no greater than that attained at a concentration of $PM_{2.5}$ of 50 μg/m^3 (Figure 17.7). Thus, for cities with estimated annual average concentrations of >50 μg/m^3, we assigned a maximum concentration, or C_m, equal to 50 μg/m^3, regardless of their actual estimated

Figure 17.7 Alternative concentration–response curves for mortality from cardiopulmonary disease, using different scenarios



concentration. This means that the excess risk is constrained to be no greater than that associated with an annual average concentration of 50 µg/m^3, regardless of the actual estimated annual average concentration. The counterfactual or theoretical minimum concentration was set at 7.5 µg/m^3, as discussed above.

We set the maximum city-specific concentration of $PM_{2.5}$, C_m, at 50 µg/m^3 to avoid producing unrealistically large estimates of mortality in the most extremely polluted subregions under a linear exposure model. With $C_m = 50$, the subregion-specific attributable fraction was restricted to no more than approximately 25% of the burden of a given health outcome, while not greatly exceeding the maximum observed annual average concentration in the ACS study. We also examined alternative values for the shape of the concentration–response function for $PM_{2.5}$ and mortality from cardiopulmonary disease and lung cancer in sensitivity analyses, as described below.

Mortality from acute respiratory infections in children aged 0–4 years

In view of the importance of mortality from ARI among children in developing countries and the suggestion from available evidence of an association with air pollution (Romieu et al. 2002), we decided to make

a summary estimate on the basis of these studies, in spite of their heterogeneity in outcomes and age groups.

To estimate the relationship between exposure to PM and mortality from ARI among children aged 0–4 years, we computed a summary estimate from the five published time-series studies discussed above (Table 17.6 and Figure 17.6). One study (Pereira et al. 1998) was excluded because the outcome, intrauterine mortality, was clearly unrelated to ARI. The five remaining studies were summarized as a weighted average of the estimates from individual studies (scaled to PM_{10}, as discussed above) with the weights determined by the inverse of the reported variance in the concentration–response function. We estimate that a 10 $\mu g/m^3$ increase in ambient concentrations of PM_{10} results in a 1.0% (95% CI—0.9%–3.1%) increase in daily mortality from ARI in children aged 0–4 years (Table 17.7).

When calculating the attributable fraction, this concentration–response function was applied to GBD baseline cause-specific rates of mortality from acute respiratory infection (GBD code 38) that includes ICD-9 codes 460–466, 480–487 and 381–382.

Numbers of deaths from all natural causes caused by short-term exposure to urban air pollution in adults

We also calculated an estimate of the numbers of deaths from all natural (non-injury) causes attributable to short-term exposure to urban air pollution using an estimate of concentration–response derived from international literature on air pollution and daily mortality. This estimate was not included in the total attributable deaths and disease burden because of the conceptual issue in quantifying the effects of short-term exposure discussed above.

Using the St George's Hospital Medical School database, described above, we identified 165 time-series studies of PM_{10} and daily mortality from all causes at all ages in all languages and countries, up to the end of July 2001. As we were concerned about the possibility of publication bias, we compared the summary estimates from 54 individual studies, a subset of the literature which would be expected to be susceptible to publication bias, with those of the two multi-city studies (Health Effects Institute 2000b; Katsouyanni et al. 2001), which selected cities from a pre-specified sampling frame, used uniform methods of analysis, and published all results. The pooled estimate for the cities of the combined APHEA and NMMAPS studies (n=111) was 1.005 (95% CI 1.004–1.006) with no evidence of publication bias in the funnel plot or on statistical testing. For the 54 studies of individual cities, graphical analysis showed some evidence of publication bias but when formally tested, this was weak (P=0.12). The pooled estimate was 1.007 (95% CI 1.006–1.008) but when adjusted for publication bias using Trim and Fill analysis, it was reduced to 1.006 (95% CI 1.004–1.007). We then examined the results for all 165 studies with results for $PM_{10.}$ There was

no evidence of publication bias on inspection of the funnel plot or on formal testing with Begg's or Eggar's tests (Begg and Mazumdar 1994). We calculated pooled estimates weighted according to the inverse of the variance of the individual study. Random effects models were used, as all showed significant heterogeneity. The pooled estimate was 1.006 (95% CI 1.005–1.007) (Table 17.7). This concentration–response function was applied to all GBD baseline cause-specific rates of mortality except GBD code 148 (injuries).[10]

4. Uncertainty estimates: statistical variability and sensitivity analyses

The total uncertainty in our estimates of the burden derives from both the statistical (sampling) variability of the parameter estimates in the models we chose to quantify disease burden, and our uncertainty with regard to those choices vs plausible alternatives, i.e. the form of our models (Morgan and Henrion 1998). We therefore quantified the statistical uncertainty of our estimates in terms of a combined, or propagated, uncertainty estimate, and used sensitivity analyses to quantify model uncertainty.

4.1 Statistical (sampling) variability

Our estimate of statistical uncertainty combined the sampling errors from two sources to derive an uncertainty distribution:

- sampling variability in the original concentration–response estimates from the ACS and time-series studies quantified in terms of their standard errors (Table 17.5); and
- sampling variability in the estimates of subregional concentration of PM from the exposure estimation model in terms of estimates of bootstrapped standard error described above (Table 17.4).

When presenting our results we show either the complete uncertainty distribution or the intervals between the 25th and 75th and/or 2.5th and 97.5th percentiles of that distribution, i.e. 50% and 95% uncertainty intervals.

4.2 Sensitivity analyses

We used sensitivity analyses, described below, to quantify the uncertainty in our base-case estimates, in which the burden of disease was estimated by applying the ACS concentration–response function over the range of 7.5 to 50 $\mu g/m^3$, as discussed above.

- *Cases 2–4: Shape of the concentration–response function for $PM_{2.5}$ and mortality attributable to cardiopulmonary disease and lung cancer.* We explored three alternatives to the base-case scenario for extrapolating the ACS concentration–response function beyond

30 μg/m^3, the highest annual average concentration observed in the ACS study (Figure 17.7).

- *Case 2: No incremental increase in excess mortality above 30 μg/m^3 $PM_{2.5}$.* Under this scenario, when calculating the population-weighted subregional average concentration of $PM_{2.5}$ we give the city-specific C_m a value of 30 μg/m^3, regardless of the estimated concentration, rather than the base-case concentration of 50 μg/m^3. This means that the excess risk is constrained to be no greater than that associated with an annual average concentration of 30 μg/m^3, regardless of the actual estimated annual average concentration. The counterfactual or theoretical minimum concentration was set at 7.5 μg/m^3. We considered this the estimator that would produce the smallest (i.e. most scientifically conservative) estimate of the impact of mortality consistent with the use of the ACS concentration–response function.
- *Case 3: Excess mortality increases linearly above 30 μg/m^3 $PM_{2.5}$.* Under this scenario, the city-specific concentration of $PM_{2.5}$ takes its actual estimated value when calculating the population-weighted subregional averages, i.e. in contrast to the base-case and case 2 scenarios. The counterfactual or theoretical minimum concentration was set at 7.5 μg/m^3. We considered this the estimator that would produce the largest estimate of mortality impact consistent with the use of the ACS concentration–response function.
- *Case 4: Excess mortality increases with the log of concentration of $PM_{2.5}$ across the entire range.* Under this scenario, the city-specific concentration of $PM_{2.5}$ takes the log of its actual estimated value when calculating the population-weighted subregional averages. Therefore, in contrast to case 3, the slope of the concentration–response function is constrained to decrease at higher concentrations. The counterfactual or theoretical minimum concentration was set at 7.5 μg/m^3.

  We included this estimator, proposed by an external reviewer after we had made our initial estimates (R. Burnett, personal communication), because it seemed a reasonable way to characterize an excess risk that we believed may: (i) increase directly with ambient levels over the entire range of annual average concentrations that we estimated for the world's cities, but (ii) be smaller at higher ambient concentrations, as has been observed for daily mortality in time-series studies (Daniels et al. 2000; Schwartz et al. 2002).
- *Cases 5 and 6: Choice of ACS concentration–response function.* In the base-case analyses, we used the ACS coefficients that were based on exposure of the cohort in 1979–1983. These arguably best represented the effects of long-term past exposure that some researchers assume are responsible for the increased mortality attributable to air pollution in that cohort through to 1998. There is, however, consid-

erable uncertainty regarding the timing of exposure with regard to risk of mortality (Krewski et al. 2000), so we calculated alternative estimates using the reported ACS coefficients based on the average of past (1979–1983) and recent (1999–2000) annual average concentrations using both a linear (case 5) and log-linear (case 6) extrapolation (Table 17.7).

- *Case 7: Change $PM_{2.5}$:PM_{10} ratio*. In the base-case analyses, we assumed a $PM_{2.5}$:PM_{10} ratio of 0.50, although higher and lower ratios have been observed in a number of locations, as discussed above. We examined the sensitivity of our base-case analyses by assigning cities in AMR-A, EUR-A, EUR-B, EUR-C and WPR-A a higher scaling factor of 0.65, while assigning a lower scaling factor of 0.35 to cities in all other subregions.
- *Cases 8 and 9: Choice of counterfactual or theoretical minimum concentration*. We evaluated the sensitivity of the base-case estimates to two different choices of counterfactual $PM_{2.5}$ concentration: 3 μg/m^3 (case 8) and 15 μg/m^3 (case 9). The former is close to the minimum background level of $PM_{2.5}$ observed in the United States, and the latter is the annual concentration of $PM_{2.5}$ proposed by the United States National Ambient Air Quality Standard (NAAQS) (U.S. Environmental Protection Agency 2002). Each value was substituted for the base-case concentration of 7.5 μg/m^3 in Equation 7 above, when calculating population-weighted subregional relative risks.

5. Results

5.1 Base-case estimates

We estimated that exposure to particulate air pollution caused approximately 800 000 excess deaths and 6.4 million DALYs (consisting only of years of life lost to premature mortality) in the year 2000 worldwide as a result of cardiopulmonary disease, lung cancer and ARI in children aged 0–4 years, combined (Table 17.8).

The worldwide estimates of attributable deaths and YLL from cardiopulmonary and lung cancer are subject to uncertainty contributed by the estimation of both the relative risks and the ambient concentrations of PM (Figure 17.8).

Cardiopulmonary disease in adults aged ≥30 years contributed 89% (712 000) and 78% (4.97 million) of attributable deaths and burden, respectively. Lung cancer contributed 8% and 9% and ARI in children, contributed 3% and 7% of deaths and YLL, respectively, to the total burden (Tables 17.9[a]–17.9[c]). The number of attributable deaths from all natural causes estimated from the daily time-series studies was roughly half the total attributable deaths, 378 000 vs 799 000 (Table

Table 17.8 Attributable deaths and DALYs in 2000, by subregion (50% and 95% confidence intervals)

Subregion	Deaths (000s)	50% CI	95% CI	DALYs (000s)	50% CI	95% CI
AFR-D	22	11.1–23.7	4.3–34.5	285	155.1–361.3	28.2–557.5
AFR-E	10	7.5–14.4	3.9–22.2	147	107.7–239.9	24.7–364.7
AMR-A	28	22.1–33.7	12.7–44.0	152	158.6–239.8	94.6–314.8
AMR-B	30	23.1–37.4	12.0–50.1	232	241.8–383.6	142.9–517.7
AMR-D	5	3.2–5.3	1.6–7.6	44	34.2–62.6	14.2–87.3
EMR-B	8	4.0–8.4	2.1–13.5	77	45.6–93.4	25.0–149.2
EMR-D	51	31.2–56.2	17.0–73.0	558	384.5–737.5	163.1–970.4
EUR-A	23	19.4–29.7	9.9–42.8	117	125.7–187.4	65.8–265.4
EUR-B	38	26.7–44.4	14.8–58.5	288	241.3–386.6	138.5–507.0
EUR-C	46	28.1–53.4	10.1–83.3	320	229.6–432.1	81.9–676.0
SEAR-B	32	19.2–37.5	5.5–51.5	282	191.0–388.9	67.0–532.6
SEAR-D	132	98.3–162.1	54.1–212.3	1 312	1 185.1–1 890.5	575.2–2 409.8
WPR-A	18	13.2–21.4	6.7–28.5	84	84.3–137.0	42.9–182.0
WPR-B	355	260.8–424.8	142.8–555.1	2 504	2 447.4–3 848.7	1 431.2–5 014.1
World	799	574.8–942.5	318.2–1 196.9	6 404	5 955.6–9 288.2	3 199.9–11 472.4

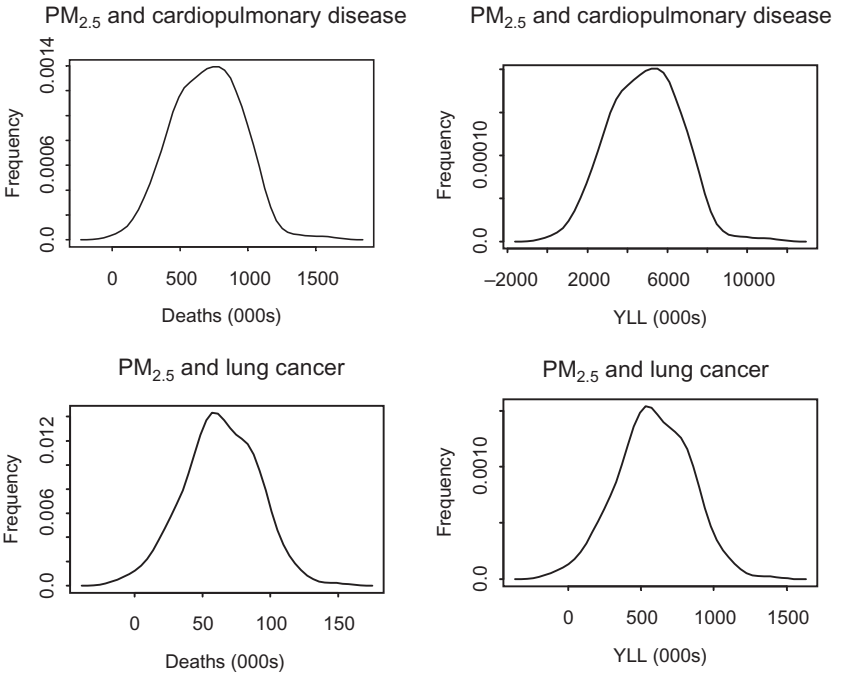
17.9[d]). The overall and cause-specific burden of disease varies across the 14 subregions, with the preponderance of the burden of air pollution contributed by cities in WPR-B, which includes China, and SEAR-D, which includes India. The variation in attributable deaths and YLL among the 14 subregions seen in Tables 17.9(a)–(d) reflects a subregional variation in the attributable fraction of approximately six-fold. For example, for all mortality end-points, EUR-A and WPR-B lie at the low and high ends, respectively, of the subregional distribution of attributable fractions. This largely reflects differences in the estimated population-weighted subregional ambient concentrations of PM_{10} and $PM_{2.5}$ (89 vs 26 $\mu g/m^3$ PM_{10} (see Table 17.4), rather than the proportion of the population that resides in cities. The proportion of the population of WPR-B that lives in cities is, in fact, lower than that in EUR-A (34% vs 39%).

5.2 Sensitivity analyses

Cases 2–4: Shape of the concentration–response function for $PM_{2.5}$

The estimates of attributable deaths from cardiopulmonary disease and lung cancer and YLL under the base-case and three alternative scenarios for the shape of the $PM_{2.5}$ concentration–response function are presented in Table 17.10. When the city-specific estimated concentrations

Figure 17.8 Uncertainty distributions for deaths and YLL from cardiopulmonary disease and lung cancer worldwide



of $PM_{2.5}$ are constrained to never exceed the concentrations observed in the most polluted city in the ACS study (annual average concentration of $PM_{2.5}$ of 30 μg/m^3), case 2, worldwide estimates of the number of deaths from cardiopulmonary disease and lung cancer are reduced by 29% and 27%, respectively. Extrapolation of the ACS coefficients to the highest estimated city-specific concentrations of $PM_{2.5}$ on the linear and logarithmic scales, cases 3 and 4, respectively, results in increases of 10% and 12% in the estimated number of attributable deaths from cardiopulmonary disease, and 8–24% increases in the estimated numbers of attributable deaths from lung cancer, relative to the base-case estimates.

These changes in worldwide estimates reflect underlying differences in the subregion-specific estimates (Table 17.11). Truncating the city-specific annual average concentrations at a given level leaves the burden unchanged in subregions with cities with estimated concentrations of PM that are lower than the truncation point, while reducing the burden in subregions with cities with estimated concentrations of PM that are above that point. Most cities in Europe and North America have

Table 17.9(a) Attributable deaths and YLL: base-case scenario for cardiopulmonary disease (50% and 95% confidence intervals)

Subregion	*Relative risk*	*Attributable fraction (%)*	*Deaths (000s)*	*50% CI*	*95% CI*	*YLL (000s)*	*50% CI*	*95% CI*
AFR-D	1.148	2	18	8.7–20.4	4–31	162	80.2–188.3	36–283
AFR-E	1.071	1	9	6.1–11.7	3–22	84	59.0–113.8	25–210
AMR-A	1.029	2	23	17.5–29.0	8–38	116	87.6–144.7	41–190
AMR-B	1.066	3	27	21.3–35.5	10–47	201	157.4–262.7	77–347
AMR-D	1.108	3	4	2.7–5.0	1–8	31	20.1–36.2	9–60
EMR-B	1.075	3	8	3.7–8.3	1–13	65	32.0–70.9	11–111
EMR-D	1.214	4	45	27.7–51.0	12–72	386	237.2–437.8	106–618
EUR-A	1.032	1	20	15.7–26.5	8–36	90	69.2–116.7	34–156
EUR-B	1.098	3	34	24.3–41.4	11–53	238	167.9–286.4	78–370
EUR-C	1.046	2	43	26.4–52.4	12–83	291	180.0–356.9	83–565
SEAR-B	1.250	4	30	17.1–33.8	5–50	240	136.9–271.2	38–400
SEAR-D	1.190	3	119	88.0–152.7	44–198	1 006	744.7–1 293.0	373–1 677
WPR-A	1.051	3	15	10.7–18.8	5–26	65	46.9–81.9	24–115
WPR-B	1.216	6	317	235.2–402.6	105–524	1 992	1 477.0–2 528.5	658–3 290
World		3	712	507.0–874.7	245–1 107	4 966	3 537.2–6 083.2	1 695–7 700

Table 17.9(b) Attributable deaths and YLL: base-case scenario for lung cancer (50% and 95% confidence intervals)

Subregion	*Relative risk*	*Attributable fraction (%)*	*Deaths (000s)*	*50% CI*	*95% CI*	*YLL (000s)*	*50% CI*	*95% CI*
AFR-D	1.210	5	0.41	0.2–0.5	0.06–0.78	4.2	1.9–5.0	0.6–8.0
AFR-E	1.100	2	0.27	0.2–0.4	0.05–0.69	2.9	2.1–4.2	0.5–7.4
AMR-A	1.040	3	4.85	3.8–6.7	0.75–8.48	36.9	28.9–50.8	5.7–64.7
AMR-B	1.093	4	2.10	1.7–2.9	0.39–4.25	19.9	16.0–27.5	3.7–40.3
AMR-D	1.153	6	0.15	0.1–0.2	0.02–0.27	1.5	1.0–1.8	0.2–2.7
EMR-B	1.105	4	0.45	0.2–0.5	0.05–0.86	4.7	2.1–5.3	0.5–8.8
EMR-D	1.309	8	1.55	1.0–1.9	0.18–2.70	16.8	10.4–20.6	2.0–29.3
EUR-A	1.044	2	3.52	2.8–5.0	0.58–6.61	27.4	22.1–38.9	4.5–51.5
EUR-B	1.138	5	3.00	2.1–3.9	0.41–5.43	30.2	21.5–38.9	4.1–54.7
EUR-C	1.065	3	2.82	1.6–3.6	0.52–5.63	27.4	15.7–34.8	5.1–54.8
SEAR-B	1.363	6	2.17	1.1–2.7	0.28–4.04	21.8	11.4–26.8	2.8–40.6
SEAR-D	1.272	4	5.63	4.3–7.8	0.90–11.60	55.9	42.4–77.2	8.9–115.2
WPR-A	1.071	4	2.71	2.0–3.7	0.45–4.94	17.6	13.3–23.9	2.9–32.1
WPR-B	1.311	10	32.37	24.5–44.0	5.15–59.01	308.5	233.2–419.1	49.1–562.4
World		5	62	47.0–83.1	9.95–114.32	576	436.9–767.5	92–1 063

Table 17.9(c) Attributable deaths and YLL: base-case scenario for ARI in children aged 0–4 years (50% and 95% confidence intervals)

Subregion	*Relative risk*	*Attributable fraction (%)*	*Deaths (000s)*	*50% CI*	*95% CI*	*YLL (000s)*	*50% CI*	*95% CI*
AFR-D	1.050	0.8	3.5	0.8–4.9	–2.9–9.0	119	26.8–165.6	–96.0–301.2
AFR-E	1.024	0.3	1.8	0.7–3.4	–2.0–5.8	61	22.6–113.5	–65.8–193.3
AMR-A	1.010	0.0	0.0	0.0–0.0	0.0–0.0	0	0.1–0.3	–0.2–0.5
AMR-B	1.023	0.3	0.3	0.1–0.6	–0.3–0.8	11	3.9–19.1	–11.3–27.4
AMR-D	1.037	0.9	0.3	0.1–0.5	–0.3–0.9	11	3.2–18.2	–10.9–30.3
EMR-B	1.026	0.6	0.2	0.1–0.3	–0.2–0.6	7	1.8–9.6	–6.3–20.7
EMR-D	1.070	1.4	4.6	1.5–7.0	–4.1–11.5	155	51.5–237.9	–137.6–389.2
EUR-A	1.011	0.0	0.0	0.0–0.0	0.0–0.0	0	0.0–0.1	–0.1–0.2
EUR-B	1.033	0.7	0.6	0.2–1.0	–0.6–1.5	20	7.0–33.1	–20.1–51.1
EUR-C	1.016	0.1	0.1	0.0–0.1	–0.1–0.2	2	0.6–3.5	–1.9–5.9
SEAR-B	1.082	0.5	0.6	0.2–1.0	–0.6–1.7	21	5.4–35.3	–19.7–58.7
SEAR-D	1.063	0.6	7.4	2.6–13.3	–7.2–20.1	250	86.6–448.4	–243.8–678.5
WPR-A	1.018	0.0	0.0	0.0–0.0	0.0–0.0	0	0.1–0.2	–0.1–0.4
WPR-B	1.071	1.2	6.1	2.0–10.7	–6.1–16.5	204	68.8–358.8	–203.3–555.0
World		0.7	25.6	8.2–43.9	–23.7–66.1	862	277.7–1 480.7	–798.6–2 228.0

Table 17.9(d) Attributable deaths: base-case scenario for mortality from all natural causes

Subregion	*Relative risk*	*Attributable fraction (%)*	*Deaths (000s)*
AFR-D	1.029	0.67	26
AFR-E	1.015	0.29	16
AMR-A	1.006	0.42	11
AMR-B	1.014	0.68	15
AMR-D	1.022	0.90	4
EMR-B	1.015	0.62	4
EMR-D	1.042	1.20	37
EUR-A	1.007	0.26	10
EUR-B	1.019	0.74	14
EUR-C	1.009	0.43	13
SEAR-B	1.048	0.87	17
SEAR-D	1.037	0.64	70
WPR-A	1.011	0.68	7
WPR-B	1.042	1.43	133
World		0.75	378

estimated annual average concentrations of $PM_{2.5}$ of $<30\,\mu g/m^3$, while estimated concentrations in the cities of developing countries are frequently much greater. More than 95% of the decrease in the worldwide burden in case 2 and the increase in case 3 occurs in four subregions: WPR-B, EMR-D, SEAR-B and SEAR-D.

Log-linear specification of the concentration–response function, as in case 4, allows for a more gradual increase in the relative risk at concentrations of PM of $>30\,\mu g/m^3$ than does the linear extrapolation model of case 3. This specification also means that the relative risk increases more steeply at concentrations of $<30\,\mu g/m^3$. Since the estimates for burden in both the log-linear case and the base case are measured with reference to a counterfactual annual average concentration of $PM_{2.5}$ of $7.5\,\mu g/m^3$, the burden under the log-linear specification is higher than that under the base case at low levels of exposure, but lower than the base case at high levels of exposure. Differences in the subregion-specific estimates for burden under the log-linear specification relative to the base case depend on the subregion-specific distributions of the city-specific concentrations of PM. The burden of disease in subregions where exposure is relatively low (AMR-A, EUR-A, EUR-C and WPR-A) increases by 63%, relative to the base case, while the burden in subregions where exposure is high remains unchanged or is slightly reduced.

Table 17.10 Sensitivity analyses of base-case estimates of attributable deaths and YLL, by cause

		Cardiopulmonary disease		*Lung cancer*		*Acute respiratory infections*[a]	
Case	*Conditions*	*Attributable deaths (000s) (% change)*	*YLL (000s) (% change)*	*Attributable deaths (000s) (% change)*	*YLL (000s) (% change)*	*Attributable deaths (000s) (% change)*	*YLL (000s) (% change)*
Base-case	Maximum concentration of $PM_{2.5} = 50\ \mu g/m^3$	712	4 966	62	576	26	862
Case 2	Maximum concentration of $PM_{2.5} = 30\ \mu g/m^3$	506 (–29)	3 498 (–30)	45 (–27)	414 (–28)	NA	NA
Case 3	Linear extrapolation	783 (10)	5 507 (11)	67 (8)	623 (8)	NA	NA
Case 4	Log-linear extrapolation	794 (12)	5 476 (10)	77 (24)	698 (21)	NA	NA
Case 5	Change ACS coefficient/linear extrapolation	1 132 (59)	7 908 (59)	101 (63)	939 (63)	NA	NA
Case 6	Change ACS coefficient/log-linear extrapolation	1 069 (50)	7 385 (49)	105 (69)	955 (66)	NA	NA
Case 7	Change $PM_{2.5}$: PM_{10} ratio	609 (–15)	4 109 (–17)	58 (–7)	521 (–10)	NA	NA
Case 8	Theoretical minimum concentration of $PM = 3\ \mu g/m^3$	882 (24)	6 081 (23)	80 (29)	731 (27)	30 (–15)	1 012 (–17)
Case 9	Theoretical minimum concentration of $PM = 15\ \mu g/m^3$	474 (–33)	3 365 (–32)	39 (–37)	369 (–36)	19 (27)	627 (27)

NA Not applicable.

[a] In children aged –4 years.

Table 17.11 Subregion-specific estimates for number of deaths and YLL from cardiopulmonary disease, lung cancer and ARI[a] for base-case and alternative scenarios

	Cardiopulmonary deaths (000s) and YLL (000s)																	
	Base-case		*Case 2*		*Case 3*		*Case 4*		*Case 5*		*Case 6*		*Case 7*		*Case 8*		*Case 9*	
Subregion	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*
AFR-D	18	162	13	123	19	179	19	180	28	259	26	243	12	106	21	194	12	112
AFR-E	9	84	8	79	9	84	12	113	14	132	16	152	5	44	12	115	4	41
AMR-A	23	116	23	116	23	116	41	205	36	181	55	273	40	202	44	220	3	13
AMR-B	27	201	25	187	27	203	35	261	43	315	47	349	15	109	37	277	14	106
AMR-D	4	31	4	28	4	31	5	38	7	49	7	51	2	18	5	39	3	18
EMR-B	8	65	7	62	8	65	10	89	12	102	14	120	4	34	10	88	4	30
EMR-D	45	386	26	219	64	545	46	397	72	617	63	537	37	317	51	440	36	306
EUR-A	20	90	20	90	20	90	34	151	32	141	46	202	35	154	37	164	4	18
EUR-B	34	238	33	229	34	238	44	304	54	375	59	408	49	339	44	304	20	136
EUR-C	43	291	43	291	43	291	68	460	67	457	91	616	67	458	66	449	11	74
SEAR-B	30	240	17	132	36	287	28	226	49	390	39	309	22	174	34	269	24	191
SEAR-D	119	1 006	79	667	136	1 150	123	1 037	192	1 625	167	1 413	82	697	137	1 164	88	749
WPR-A	15	65	15	65	15	65	23	99	23	102	30	132	23	101	23	98	5	21
WPR-B	317	1 992	192	1 209	344	2 163	305	1 915	504	3 163	411	2 580	216	1 357	360	2 261	247	1 550
World	712	4 966	506	3 498	783	5 507	794	5 476	1 132	7 908	1 069	7 385	609	4 109	882	6 081	474	3 365

continued

Table 17.11 Subregion-specific estimates for number of deaths and YLL from cardiopulmonary disease, lung cancer and ARI[a] for base-case and alternative scenarios (*continued*)

	Lung cancer deaths (000s) and YLL (000s)																	
	Base-case		*Case 2*		*Case 3*		*Case 4*		*Case 5*		*Case 6*		*Case 7*		*Case 8*		*Case 9*	
Subregion	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*	*Deaths*	*YLL*
AFR-D	0.4	4.2	0.3	3.2	0.5	4.7	0.5	5.0	1	7	1	7	0.3	2.8	0	5	0	3
AFR-E	0.3	2.9	0.3	2.7	0.3	2.9	0.4	4.1	0	5	1	6	0.1	1.5	0	4	0	1
AMR-A	4.8	36.9	4.8	36.9	4.8	36.9	9.1	69.5	8	59	12	94	8.4	64.4	9	70	1	4
AMR-B	2.1	19.9	2.0	18.6	2.1	20.1	2.9	27.5	3	32	4	37	1.1	10.8	3	27	1	11
AMR-D	0.1	1.5	0.1	1.4	0.1	1.5	0.2	2.0	0	2	0	3	0.1	0.9	0	2	0	1
EMR-B	0.5	4.7	0.4	4.5	0.5	4.7	0.7	6.8	1	7	1	9	0.2	2.4	1	6	0	2
EMR-D	1.6	16.8	0.9	9.5	2.2	23.8	1.7	18.4	3	28	2	25	1.3	13.8	2	19	1	13
EUR-A	3.5	27.4	3.5	27.4	3.5	27.4	6.3	49.2	6	44	9	67	6.0	46.9	6	50	1	6
EUR-B	3.0	30.2	2.9	29.0	3.0	30.2	4.1	41.0	5	49	6	56	4.3	43.0	4	39	2	17
EUR-C	2.8	27.4	2.8	27.4	2.8	27.4	4.8	46.2	5	44	6	63	4.4	43.1	4	42	1	7
SEAR-B	2.2	21.8	1.2	11.9	2.6	26.2	2.2	22.0	4	37	3	31	1.6	15.7	2	25	2	17
SEAR-D	5.6	55.9	3.7	36.8	6.4	64.0	6.2	61.6	9	94	9	86	3.9	38.5	7	65	4	41
WPR-A	2.7	17.6	2.7	17.6	2.7	17.6	4.4	28.3	4	28	6	38	4.2	27.1	4	26	1	6
WPR-B	32.4	308.5	19.6	186.7	35.2	335.2	33.2	316.1	53	503	45	433	22.0	209.6	37	350	25	240
World	62.0	576	45	414	67	623	77	698	101	939	105	955	58	521	80	731	39	369

	Deaths (000s) and YLL (000s) from acute respiratory infections					
	Base-case		Case 8		Case 9	
Subregion	Attributable deaths	YLL	Attributable deaths	YLL	Attributable deaths	YLL
AFR-D	3.5	118.8	4	141	2	82
AFR-E	1.8	61.2	3	84	1	30
AMR-A	0.0	0.2	0	0	0	0
AMR-B	0.3	10.6	0	15	0	6
AMR-D	0.3	11.2	0	14	0	7
EMR-B	0.2	7.3	0	10	0	3
EMR-D	4.6	154.8	5	176	4	123
EUR-A	0.0	0.1	0	0	0	0
EUR-B	0.6	20.2	1	26	0	12
EUR-C	0.1	2.0	0	3	0	1
SEAR-B	0.6	21.3	1	24	1	17
SEAR-D	7.4	249.9	9	288	6	188
WPR-A	0.0	0.1	0	0	0	0
WPR-B	6.1	204.3	7	231	5	159
World	25.6	862.1	30	1012	19	627

[a] In children aged 0–4 years.

Cases 5 and 6: choice of ACS coefficient

Linear extrapolation beyond concentrations of PM of 30 μg/m^3 of larger alternative coefficients from the ACS study on the basis of the average of ambient concentrations measured in 1979–1983 and 1999–2000 resulted in increases of 59% and 63% in deaths attributable to cardiopulmonary disease and lung cancer, respectively, relative to the base-case estimates. Log-linear extrapolation of the larger coefficients produced increases of 50% and 69% in the number of deaths attributable to cardiopulmonary disease and lung cancer, respectively (Table 17.10).

Attributable burdens increased in all subregions (Table 17.11). The differences between the linear and log-linear estimates followed the same subregional patterns as in cases 3 and 4, discussed above.

Case 7: Choice of $PM_{2.5}$:PM_{10} ratio

Allowing limited subregional variation in the ratio of $PM_{2.5}$ to PM_{10} produced reductions of 15% and 7% in the worldwide estimates of numbers of deaths attributable to cardiopulmonary disease and lung cancer, respectively, relative to the base-case scenario in which this ratio was fixed at 0.50 (Table 17.10).

As one might expect, the burden of disease increases by 57% in those subregions assigned a ratio of 0.65, that is, AMR-A, all of Europe, and WPR-B. This increase is more than offset by the rest of the world, assigned a ratio of 0.35, where the burden of disease falls by 31% (Table 17.11).

Cases 8 and 9: Choice of theoretical minimum level of exposure

Halving and doubling the base-case theoretical minimum concentration of $PM_{2.5}$ of 7.5 μg/m^3 resulted in a 24% increase and a 33% decrease in the number of deaths attributable to cardiopulmonary disease, and a 29% increase and a 37% decrease in deaths attributable to lung cancer, but only minor variations in mortality from ARI (Table 17.10).

All subregions experienced increases in attributable burden when the theoretical minimum concentration was halved, with the largest proportional increases in less polluted subregions (AMR-A, EUR-A and WPR-A). These subregions also experienced the largest reductions in burden when the theoretical minimum concentration was doubled. Highly polluted subregions (WPR-B and SEAR-D) also experienced marked reductions in the estimated burden when the theoretical minimum concentration was doubled (Table 17.11).

6. Discussion

Previously, most large-scale estimates of the health impacts of urban air pollution were conducted for countries or regions where data on expo-

sure and estimates of effect required for impact estimation were available (e.g. Brunekreef 1997; COMEAP 1998; Künzli et al. 2000; Ostro and Chestnut 1998). In the few previous global estimates, systematic methods were not applied to extrapolate exposures and exposure–response functions to other parts of the world (Hong 1995; WHO 1997; Working Group on Public Health and Fossil Fuel Combustion 1997). Although our estimates exceed those reported earlier, the differences are not large, given the variation in the approaches that were taken (Smith and Mehta 2003). The global scope of the present analysis required new approaches for estimating exposure, absent measurements of air pollution in many developing countries, extrapolating the results of epidemiological studies more widely than had previously been attempted, and describing and attempting to quantify, the many uncertainties this entailed. The results indicate that the impact of urban air pollution on the burden of disease in the cities of the world is large and, for a variety of reasons discussed below, have probably underestimated the burden. There is also considerable variation in our estimates among the 14 subregions, with the greatest burden occurring in the more polluted and rapidly growing cities of developing countries.

The availability of actual measurements of outdoor concentrations of PM varied widely across the globe. In order to have estimates for all 14 subregions, models developed by the World Bank were used to estimate concentrations of inhalable particles (PM_{10}) using economic, meteorological and demographic data and the available measurements of PM for 3211 cities with populations of >100 000, and also capital cities. To allow the most appropriate epidemiological studies to be used for the estimation of the burden of disease, the PM_{10} estimates were converted to estimates of fine particles ($PM_{2.5}$) using information on geographic variation in the ratio of $PM_{2.5}$ to PM_{10}. Population-weighted subregional annual average exposure estimates for $PM_{2.5}$ and for PM_{10} were obtained using the population of the city in the year 2000.

The estimates of the burden of disease were based on three health outcomes: mortality from cardiopulmonary causes in adults, mortality from lung cancer and mortality from ARI in children aged 0–4 years. Attributable numbers of deaths and YLL for adults and children (aged 0–4 years) were estimated using risk coefficients from a large cohort study of adults in the United States (Pope et al. 2002) and a meta-analytic summary of five time-series studies of mortality in children, respectively. Base-case estimates were calculated assuming that the risk of death increases linearly over a range of annual average concentrations of $PM_{2.5}$ between a counterfactual (or referent) concentration of 7.5 and a maximum of 50 $\mu g/m^3$. For comparison, an additional estimate of attributable deaths was calculated from time-series studies of daily mortality, on the basis of results of a meta-analysis of the world literature, but was not used in the final calculations. Worldwide and subregion-

specific estimates of attributable deaths and burden of disease in terms of YLL were calculated based on the standard methodology developed for this project (see chapters 1 and 25).

We estimated that urban air pollution, as measured by PM, is responsible for about 3% of mortality caused by cardiopulmonary disease in adults, about 5% of mortality caused by cancers of the trachea, bronchus and lung, and about 1% of mortality caused by ARI in children worldwide in the year 2000. The total burden was about 0.80 million (1.2% of total) premature deaths and 6.4 million (0.5% of total) DALYs. This burden occurred predominantly in developing countries, with 30% of attributable disease burden occurring in WPR-B and 19% in SEAR-D. The greatest contributions to the total burden of disease occurred in WPR-B, EUR-B and EUR-C, where urban air pollution caused 0.7–0.9% of the total burden of disease.

6.1 Identifying and quantifying uncertainty in the estimates

These estimates are subject to considerable uncertainty given the need to estimate exposures and to extrapolate concentration–response relationships. This is almost invariably the case in quantitative risk assessment of complex environmental exposures; and is certainly to be expected in this particular exercise, for reasons discussed above.

We quantified the statistical uncertainty of our base-case estimates by estimating the joint uncertainty in the estimates of annual average concentration and the estimates of the relative risks. Worldwide and most subregional estimates vary by less than two-fold (50% uncertainty intervals (Tables 17.8 and 17.9[a]–[c]). Uncertainty of the model owing to assumptions about the shape of the concentration–response function, the magnitude of the relative risk of disease attributable to urban air pollution, the choice of counterfactual level for PM, and the ratio of $PM_{2.5}$ to PM_{10} was assessed in sensitivity analyses. For the most part, the estimated worldwide burdens in the various sensitivity analyses are within the 50% uncertainty interval for the base-case estimate of worldwide burden. The sensitivity analyses indicate that base-case estimates were most sensitive to choice of coefficient from the ACS study and theoretical minimum concentration.

Although some sources of uncertainty could be quantified, others that were no less important or were perhaps more important, could not. These additional sources of uncertainty arise from the methods we used to estimate annual average exposure of the population and our choice of health end-points and concentration–response functions.

Estimates of exposure to urban air pollution

There are four key uncertainties related to exposure that have not been quantified, and that could affect the estimates of burden of disease.

First, we used PM as the sole indicator of exposure to urban air pollution, although urban air pollution is a complex mixture, as noted above. Other frequently measured pollutants, notably ozone, carbon monoxide, oxides of sulfur and nitrogen, and lead are associated with mortality and morbidity, albeit not as consistently as PM, although the effects of a number of these pollutants may be at least partially captured via the use of a PM metric (Sarnat et al. 2001). Estimating the health impacts of specific components poses challenges for both scientific research and risk assessment, including how to avoid attributing the same burdens to multiple pollutants (i.e. double counting), and how to quantify the effects of possible interactions (i.e. synergistic effects) among pollutants. Nonetheless, there is evidence for an effect of ozone on daily mortality that is independent of PM (e.g. Health Effects Institute 2000b). Future estimates of the burden of disease should include the health impacts of ozone. Unfortunately, lead in petrol remains an important toxic component of air pollution in some cities of the developing world and its contribution to the burden of disease has been estimated elsewhere in this book (see chapter 19). Some combination of the GBD estimates for urban air pollution and lead probably provide the best overall estimate of the burden of disease attributable to urban air pollution.

Second, use of estimated levels of exposure introduces some uncertainties and biases in the predicted levels of exposure that could not be addressed, owing to lack of data. The most important of these is the lack of city-specific data on the structure of economic activity and on fuel consumption. The exposure model uses national average data for these variables as a reasonable proxy, which can lead to bias in unknown directions with regard to city-specific estimates. The net bias in estimates of the aggregate burden at the subregional level is unclear. The use of long-run average climatic conditions instead of time-varying local data may result in biased estimates for specific years, but may not pose a serious problem as we are interested mainly in the long-term average health effects of air pollution. We have also explicitly examined our uncertainty regarding spatial variations in the size composition of PM through sensitivity analysis. The model for the estimation of exposure clearly suggests that coarser particles account for a larger fraction of the TSP in developing countries than in developed countries, all other things being equal. The limited data from monitoring available on $PM_{2.5}$ also indicate that spatial variations may also exist in the sizes of finer particles. Consequently, we have used conservative estimates for the fraction of PM_{10} accounted for by finer particles in our overall estimates and further tested the implications of using an even more conservative estimate. The burden estimates should be relatively insensitive to PM size fraction.

Third, misclassification of exposure may have led to underestimation of the burden of disease. Like the epidemiological studies used to quantify the estimates of health impact, we used the annual average ambient

concentration measured from a few stationary sources in each city to estimate average personal levels of exposure. Differences between personal levels of exposure and concentrations measured at fixed points depend on how well the pollutant mixes in the environment and the efficiency with which the pollutant penetrates indoors. The exposure estimates are based on a model developed from population-oriented monitors. Measurements of PM from these sites in well-designed monitoring networks would provide representative city-wide levels of exposure for a pollutant that mixes uniformly in the environment. They would underestimate the actual level of exposure of people living near pollution hotspots, such as busy roads or local sources of pollutant, which can contribute to spatial heterogeneity of exposure within cities (Hoek et al. 2002; Jerrett et al. 2001). This underestimate would probably be more pronounced for cities in developing countries, where nearly one third of the population resides in slums, which are often in heavily-polluted parts of cities, and even larger populations work near pollution hotspots.

Exposure misclassification from using outdoor concentrations to represent personal exposure to urban air pollution also results from differences in the efficiency with which PM penetrates indoors. Use of ambient concentrations as surrogates for exposure tends to underestimate the risk per unit exposure because the penetration of particles indoors, where most exposure occurs, is less than 100%. If average penetration is 66%, for example, actual exposure–response per $1\,\mu g/m^3$ would be 1.5-fold that indicated by outdoor concentrations of pollution. However, because of climate and housing, the rates of penetration of pollution in most, but not all, cities in developing countries can be expected to be somewhat greater than those in the average city in the United States where the epidemiology used here has been undertaken. Not being able to consider this factor because of lack of data on penetration of the pollutant would bias estimates of burden downward if actual changes in exposures in developing countries are better indicated by changes in outdoor concentrations than in developed countries.

An additional source of misclassification concerns the time referent of our exposure estimate. The current burden is related to past exposure, but our model estimates current (i.e. 1999) levels only. However, even if we had been able to retrospectively estimate a time series of annual average concentrations for each subregion, the ACS study provides little information as to how the concentration–response function varies over time (Krewski et al. 2000). It is not clear how this source of misclassification would affect our estimates.

Fourth, our estimates do not include the attributable burden of disease among the 800 million additional urban residents living either in suburban areas of some of the cities or in cities with populations of <100 000 and in the >3 billion residents of rural areas. Although lower levels of emissions per area combined with the differences in the built-up envi-

ronment in rural areas probably results in a small average exposure to ambient pollution and a modest increase in the global burden of disease from such pollution in rural areas, the same is not true for the urban residents that were not included in the target population. The magnitude of the missing burden depends on the actual exposures of those living in smaller cities. The target population was identified for the study on the basis of data available from the United Nations, which compiles the data reported by Member States from national censuses and makes projections from them on the basis of expected changes in demographics. In compiling the population statistics, Member States, hence the United Nations, do not use uniform definitions either for city area (the characteristic such as city size that defines an urban area) or the population included for each identified city (whether political boundaries are used or agglomerations of contiguous urban areas are used). For the target population, we have used the population of the city agglomeration when this choice was available. If all of the remaining 800 million residents lived in suburban areas next to a targeted city, exposures and hence, estimates of burden, for these residents could be expected to be similar to those in the identified city resulting in an aggregate underestimate of the attributable fraction of the population of about 28%. The exposure model suggests, however, that concentrations gradually decrease as the local population density decreases, suggesting that levels of exposure and hence estimates of burden are lower for these residents compared to those living in larger cities. The net result is that our focus on residents in cities with >100 000 inhabitants may underestimate the aggregate burden by between 0% and 28%.

Choice of health end-points and concentration–response functions

Our base-case estimates of burden in terms of disease burden considered only the impact of air pollution on mortality. This approach is likely to have underestimated the true attributable burden, since there is evidence from studies of both epidemiology and toxicology, to suggest that air pollution may play a role in the incidence of cardiopulmonary disease, and thus contribute to years lived with disability (YLD). Lacking estimates of the concentration–response function for air pollution and the incidence of cardiopulmonary disease, lung cancer, and ARI in children, we calculated disease burden under the assumption that air pollution multiplies both incidence and mortality to the same extent, i.e. the relative risk of unobserved morbidity equals the observed relative risk of mortality (Table 17.12), an approach taken to estimating the attributable burden caused by other factors other than urban air pollution. The total disease burden, including YLD for cardiopulmonary disease, exceeds the YLL by 23% worldwide in the base-case analyses. The effect on the estimated burden for lung cancer and ARI in children is negligible.

Table 17.12 Attributable YLL and DALYs for cardiopulmonary disease, lung cancer, ARI[a] and total mortality

	Cardiopulmonary disease		*Lung cancer*		*Acute respiratory infections*		*Total*		
	YLL	*DALYs*	*YLL*	*DALYs*	*YLL*	*DALYs*	*YLL*	*DALYs*	
Subregion	*(000s)*		*(000s)*		*(000s)*		*(000s)*		*% change*
AFR-D	162	193	4	4	119	121	285	319	12
AFR-E	84	100	3	3	61	62	147	166	13
AMR-A	116	161	37	38	0	0	152	200	32
AMR-B	201	273	20	20	11	14	232	307	32
AMR-D	31	39	1	2	11	12	44	53	21
EMR-B	65	77	5	5	7	9	77	91	18
EMR-D	386	457	17	17	155	162	558	636	14
EUR-A	90	122	27	28	0	0	117	151	29
EUR-B	238	286	30	31	20	21	288	338	17
EUR-C	291	340	27	28	2	2	320	360	13
SEAR-B	240	291	22	22	21	25	282	339	20
SEAR-D	1 006	1 195	56	57	250	261	1 312	1 513	15
WPR-A	65	95	18	18	0	0	84	114	36
WPR-B	1 992	2 732	304	317	204	224	2 504	3 272	31
World	4 966	6 360	572	591	862	913	6 404	7 865	23

[a] In children aged 0–4 years.

The estimates of the attributable burden caused by cardiopulmonary disease and lung cancer were derived from a single cohort study in the United States (the largest and most extensively reviewed study suitable for the estimation of the burden of disease). This raises questions concerning whether these results can be generalized to other populations, especially those in developing countries, owing to differences in susceptibility to the effects of air pollution and the nature of the mixture of air pollutants. The apparent qualitative and quantitative similarity of the relative risks of daily mortality in developed and developing countries, discussed above, provides some evidence that these results are generally applicable. In addition, trends in known risk factors for chronic cardiovascular and respiratory disease, such as diet and cigarette smoking, suggest that the populations of cities in developing countries may now be more comparable to populations of cities in Europe and North America with regard to susceptibility to air pollution conferred by preexisting cardiovascular and respiratory morbidity (Reddy and Yusuf 1998). The increasing contribution of mobile sources to urban air pollution in developing countries also increases the similarity with cities in North America and Europe.

Other sources of uncertainty in our estimates cannot be readily quantified for the following reasons:

- *Lack of knowledge concerning differences between developed and developing countries in the physicochemical nature of PM produced by different sources*. The relative toxicity of PM may well vary according to the type of fuel burned and the type technology used to burn it. Increased burning of refuse outdoors and the prevalence of motor vehicles without emissions controls (e.g. vehicles powered by two-stroke engines) are two examples.

  Inhalable particles that are not the direct or indirect product of combustion sources may also be important. These particles are mainly of crustal origin and may be important, for example, in desert areas, or where there is disturbance of surface material owing to construction, use of unsurfaced roads, etc. They are largely found in the coarse fraction of inhalable particles, whereas combustion-derived particles tend to be found in the fine and ultra-fine fractions. The evidence concerning the toxicity of this fraction is mixed (Anderson 2000). Data on worldwide variation in the ratio of fine to coarse particles is limited, as discussed above, and our sensitivity analyses, which suggest relatively minor differences with our base-case estimates, may understate the uncertainty.

- *Lack of knowledge concerning differences in the susceptibility of the population*. Despite the trends discussed above, differences in demography and in the patterns of the incidence and prevalence of disease may be associated with differences in short-term and long-term vulnerability to air pollution. There exists the possibility of effect-modification factors related to health status, and behavioural factors, such as smoking and diet (Katsouyanni et al. 2001). The effects of previous or concurrent exposure to high levels of indoor air pollution may also play a role in determining susceptibility to urban air pollution. Poverty, which is a determinant of the factors just discussed, may also determine susceptibility in other ways. If the effects of air pollution are more severe among the poor, who comprise a large part of the world's population, then the magnitude of the burden would likely be greater than that which we estimated (Krewski et al. 2000; O'Neill et al. 2003).

- *The shape of the exposure–response relationship may differ between developing and developed countries in ways that were not captured in the sensitivity analyses*. For example, a recent time-series study of daily mortality in Mexico City did not observe a flattening of the PM_{10} concentration–response curve until 175 $\mu g/m^3$ (the daily mean) (Tellez-Rojo et al. 2000). These concentrations are measured in many mega-cities in developing countries.

We did not know which form of the concentration–response relationship should be used in extrapolating the results of the ACS study to the much higher concentrations observed in cities in India and China, for example. For this reason, we examined the sensitivity of the estimates to a range of scenarios, presenting a "base case", which we thought was a reasonable compromise between the conditions of the ACS and those of the rest of the world. Cohort evidence has recently been reported from Europe, although unfortunately it was unable to estimate concentrations of PM (Hoek et al. 2002). This study provides evidence that long-term exposure to urban pollution is associated with health effects elsewhere in developed countries, but we still lack cohort evidence from developing countries.

Mortality from acute respiratory infections in children aged 0–4 years

Despite limited evidence, discussed above, linking mortality from ARI to exposure to urban air pollution, we used the results from the small number of time-series studies in developing countries to estimate attributable deaths and YLL in children aged 0–4 years. In our view, most of these deaths are likely to be among children with temporary vulnerability owing to chest infections which would resolve eventually, and therefore represent, on average, the loss of many potential years of life, but this view is largely speculative.

Several studies that we used to derive the concentration–response function for ARI mortality actually reported results for all causes mortality in the 0–4 years age group (Ostro et al. 1999a), or total mortality in the first year of life (Loomis et al. 1999). We have assumed that the relationship between PM_{10} and mortality from ARI in children aged 0–4 years is similar to that for all-cause mortality. To some extent this is justified by the knowledge that mortality from ARI is an important component of all-cause mortality in developing countries.

6.2 Generalizability of our results

As a consequence of the uncertainties in this global assessment, its quantitative results cannot be confidently extrapolated to smaller geographic areas, such as specific countries or cities. The methods for estimation of exposure and extrapolation of concentration–response functions were developed specifically for estimating burdens for large geographic regions, often in the absence of essential data on exposure and response. Where better data exist, as they currently do in some parts of the world, they can, of course, be used.

Differences between our estimates and those of other groups may reflect other differences in methodology. For example, a tri-national European assessment recently estimated that some 40 000 deaths per year were attributable to exposure to ambient air pollution in a population of approximately 72 million, whereas the burden in EUR-A, in an urban

population of 80 million, was estimated to be 23 000 deaths per year, despite similar estimates of the concentration of ambient pollution (Künzli et al. 2000). The difference is largely owing to the different assumptions regarding the exposure reference level of 15 $\mu g/m^3$ PM_{10} in this work vs 7.5 $\mu g/m^3$ in the European study. In addition, the concentration–response functions were slightly higher in the tri-national project, which used estimates of total mortality from both the first ACS publication and the Harvard Six City estimates (Dockery et al. 1993; Pope et al. 1995).

6.3 Avoidable disease burden

We did not attempt to estimate the avoidable burden of disease, despite this being a specific objective of the project. Estimating the avoidable burden would have required making projections of concentrations of ambient air pollution and providing a model for the exposure time–response function for PM and mortality. Time constraints did not allow us to undertake the former task, although it is feasible. The latter information is not currently available from the existing cohort studies, although there is limited evidence that the induction time for mortality from lung cancer attributable to exposure to urban air pollution is in the order of decades (Nyberg et al. 2000), and that it is perhaps in the order of years for mortality from cardiovascular disease (Krewski et al. 2000). Evaluations of both "natural experiments" (Heinrich et al. 2000; Pope 1989), and regulatory interventions (Clancy et al. 2002; Hedley et al. 2002) provide further support for relatively rapid improvements in cardiovascular and respiratory outcomes. The latter studies also suggest that although rates of mortality may decrease after the successful implementation of air pollution reductions, the long-term benefits may extend well beyond that observed during the first years after the intervention is implemented.

6.4 How could a future risk assessment exercise provide better estimates?

There is a critical need for more information on the health effects of air pollution in developing counties. Research on exposure should aim to provide better estimates not only of ambient concentrations of pollutants, but also the characteristics of urban air pollution, including the contribution of various sources and the size distribution of PM. Epidemiological studies of mortality should be designed to provide age- and disease-specific estimates of the effects of air pollution, as well as identifying factors that confer susceptibility to air pollution. There is an obvious need for epidemiological studies of the effect of air pollution on the incidence of chronic cardiovascular and respiratory disease, and on the growth and development of children. Future estimates of the burden of disease attributable to urban air pollution should include morbidity outcomes, such as asthma exacerbation, which most certainly contribute to morbidity.

Estimates of uncertainty distributions should more fully incorporate model uncertainties, such as those related to the choice of concentration–response function. This could be accomplished via the elicitation and weighting of expert opinions in the context of a Bayesian approach to quantifying model uncertainty (Morgan and Henrion 1998; National Research Council 2002).

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Disclaimer

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Notes

1 Throughout the chapter we refer to urban air pollution using the terms "ambient air pollution" or "urban air pollution". For our current purposes, these terms are fully interchangeable.

2 See Preface for an explanation of this term.

3 Ambient particles fall into a trimodal size distribution, according to their aerodynamic diameter: coarse particles (>1 μm), fine particles (0.1–1 μm), and ultrafine particles (<0.1 μm). Ultrafine particles constitute a small percentage of the total mass of PM, but are present in very high numbers. Because of health concerns, the ambient concentrations (mass) of both coarse and fine PM are regulated by the United States Environmental Protection Agency (EPA) through the National Ambient Air Quality Standards for PM_{10} (PM <10 μm) and $PM_{2.5}$ (PM <2.5 μm) (USEPA 1997), and by the European Union through limit values for PM_{10}. $PM_{2.5}$, which includes only fine and ultrafine particles, is dominated by emissions from combustion processes; PM_{10}, which includes coarse as well as fine and ultrafine particles, has a much higher proportion of particles generated by mechanical processes from a variety of non-combustion sources. It is currently not clear how much particles of different sizes and composition differ in the effects on health that they cause.

4 Cities in the United States account for about 40% of the observations in the estimation model, even after this exclusion.

5 Data from monitoring were available for one additional city/country, Skopje in The former Yugoslav Republic of Macedonia, but were not used in the estimation model because of missing explanatory variables. In addition, 150 observations primarily from Germany (94), Lithuania (30) and other eastern European states (26) made during the early 1990s were excluded because of uncertainties in defining appropriate explanatory variables.

6 The model presented here is one of several versions of the GMAPS model developed at the World Bank. An alternative model jointly estimates concentrations of PM_{10} and TSP at residential and non-residential sites.

7 The climatic variables have been constructed from a global mean monthly climatology map with a resolution of 0.5° latitude × 0.5° longitude developed by researchers at the Climate Research Unit of the University of East Anglia. These data are available at http://ipcc-ddc.cru.uea.ac.uk/cru_data/examine/have_index.html. All of the climate variables are based on the conditions for the city centre.

8 Residential monitoring sites are located in residential areas but do not include pollution hotspots, such as locations that are immediately adjacent to industrial and commercial pollution sources or high traffic corridors. In contrast, mixed residential sites are characterized by both high population densities and the presence of some pollution sources that may result in elevated concentrations of PM in the immediate vicinity of the pollution source. Neither site includes areas of high pollution activity located in sparsely populated areas.

9 Had we instead included data from monitoring for cities with measured data for 1999, there would be an insignificant difference in the subregional average concentration, because a small fraction of the population in each subregion lives in monitored cities and most of the monitored cities are located in North America and western Europe, where the estimates of PM are more precise.

10 After these estimates had been made, investigators in the United States and Canada discovered several problems with the statistical software that had been used to estimate the relative risks associated with air pollution in the time-series studies (Health Effects Institute 2003). Correcting these problems reduced the magnitude of estimated relative risks and increased their standard errors.

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