

Environmental Burden of Disease Series, No. 5

# Outdoor air pollution

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Assessing the environmental burden of disease  
at national and local levels

Bart Ostro

Series Editors

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A Microsoft Excel spreadsheet for calculating the estimates described in this document can be obtained from WHO/PHE.  
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## Preface

The disease burden of a population, and how that burden is distributed across different subpopulations (e.g. infants, women), are important pieces of information for defining strategies to improve population health. For policy-makers, disease burden estimates provide an indication of the health gains that could be achieved by targeted action against specific risk factors. The measures also allow policy-makers to prioritize actions and direct them to the population groups at highest risk. To help provide a reliable source of information for policy-makers, WHO recently analysed 26 risk factors worldwide, including outdoor air pollution, in the *World Health Report* (WHO, 2002).

The Environmental Burden of Disease (EBD) series continues this effort to generate reliable information, by presenting methods for assessing the environmental burden of outdoor air pollution at national and local levels. The methods in the series use the general framework for global assessments described in the *World Health Report* (WHO, 2002). The introductory volume in the series outlines the general method (Prüss-Üstün et al., 2003), while subsequent volumes address specific environmental risk factors. The guides on specific risk factors are organized similarly, first outlining the evidence linking the risk factor to health, and then describing a method for estimating the health impact of that risk factor on the population. All the guides take a practical, step-by-step approach and use numerical examples. The methods described in the guides can be adapted both to local and national levels, and can be tailored to suit data availability.

The methods used in this guide are generally consistent with those used for the global analysis of disease burden due to outdoor air pollution (WHO, 2002; Cohen et al., 2004), but do include some modifications and additional developments.

Calculation sheets and other resources are available from the WHO web site or by contacting WHO<sup>1</sup> to assist in the estimation of disease burden as outlined in this document.

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<sup>1</sup> By contacting EBDassessment@who.int

## Affiliations and acknowledgements

This document was prepared by Bart Ostro, and edited by Annette Prüss-Üstün, Diarmid Campbell-Lendrum, Alistair Woodward and Carlos Corvalán. Bart Ostro is from the Air Pollution Epidemiology Unit, Office of Environmental Health Hazard Assessment, California EPA, Oakland, CA, USA. Annette Prüss-Üstün, Diarmid Campbell-Lendrum and Carlos Corvalán are from the World Health Organization, and Alistair Woodward is from the School of Population Health, University of Auckland, New Zealand. Valuable input was provided by Michal Krzyzanowski, also from the World Health Organization.

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## Abbreviations

AF	Attributable fraction.
CI	Confidence interval.
DALYs	Disability-adjusted life years.
EBD	Environmental burden of disease.
GBD	Global burden of disease.
IF	Impact fraction.
OAP	Outdoor air pollution.
PM	Particulate matter.
PM10	Particulate matter less than 10 µm in diameter.
PM2.5	Particulate matter less than 2.5 µm in diameter.
RR	Relative risk.
TSP	Total suspended particles, or PM of any size.
YLL	Years of life lost.

## **Summary**

This guide outlines a method for estimating the disease burden associated with environmental exposure to outdoor air pollution. In a recent estimate of the global burden of disease (GBD), outdoor air pollution was estimated to account for approximately 1.4% of total mortality, 0.4% of all disability-adjusted life years (DALYs), and 2% of all cardiopulmonary disease. To obtain estimates of the impact of outdoor air pollution, population exposures are based on current concentrations of particulate matter (PM) measured as either PM<sub>10</sub> or PM<sub>2.5</sub> (i.e. PM less than 10 µm or 2.5 µm in diameter, respectively). PM is a mixture of liquid and solid particle sizes and chemicals that varies in composition both spatially and temporally. After multiplying the exposure concentrations by the numbers of people exposed, concentration–response functions from the epidemiological literature are applied. These functions relate ambient PM concentrations to cases of premature mortality, and enable the attributable risk to be calculated.

For the quantitative assessment of health effects, PM<sub>2.5</sub> and PM<sub>10</sub> are selected because these exposure metrics have been used in epidemiological studies throughout the world. In addition, over the past two decades, epidemiological studies spanning five continents have demonstrated an association between mortality and morbidity, and daily, multi-day or long-term (a period of more than a year) exposures to concentrations of pollutants, including PM. The estimated mortality impacts are likely to occur predominantly among elderly people with pre-existing cardiovascular and respiratory disease, and among infants. Morbidity outcomes include hospitalization and emergency room visits, asthma attacks, bronchitis, respiratory symptoms, and lost work and school days. However, this guide does not provide a method to quantify morbidity attributable to air pollution, since such calculations require an estimate of background disease rates in the absence of air pollution.

In most urban environments, PM is generated mainly from fuel combustion in both mobile (diesel and non-diesel cars, trucks and buses) and stationary (power plants, industrial boilers and local combustion) sources. PM can also be generated by mechanical grinding processes during industrial production, and by natural sources such as wind-blown dust. To select the most suitable interventions for reducing the disease burden associated with outdoor air pollution, an inventory of the principal local and regional sources would be useful. Typically, mobile sources contribute 50% or more of PM concentrations in urban areas. In certain cities and regions, however, other sources may predominate. In rural areas, biomass burning may be the largest source.

Estimates of the burden of disease attributed to outdoor air pollution can help set the priority for controlling air pollution, relative to other interventions that improve public health.



## 1. Background

The health impact of air pollution became apparent during smog episodes in cities in Europe and the United States of America (USA), such as the London fog episodes during the winters of 1952 and 1958. Subsequent analysis of data for the London winters of 1958–1971 demonstrated that mortality was associated with air pollution over the entire range of ambient concentrations, not just with episodes of high pollutant concentrations (Ostro, 1984). The ability to measure the environmental health effects of pollution has improved over the last several decades, owing to advances in pollution monitoring and in statistical techniques. Current methods often measure the effects of air pollution in terms of particulate matter (PM), and increases in both mortality and morbidity have been detected at existing ambient PM concentrations. Significant health impacts of pollution can therefore be expected in urban centres throughout the world, since exposure to PM is ubiquitous. The largest source of PM is often fuel combustion from both mobile (e.g. cars, trucks and buses) and stationary (e.g. power plants and boilers) sources, but other sources such as road dust, biomass burning, manufacturing processes and primary pollutants from diesel engines also contribute.

Most of the health evidence on PM has been derived from epidemiological studies of human populations in a variety of geographical (principally urban) locations. Epidemiological studies have provided “real world” evidence of associations between concentrations of PM and several adverse health outcomes including: mortality, hospital admissions for cardiovascular and respiratory disease, urgent care visits, asthma attacks, acute bronchitis, respiratory symptoms, and restrictions in activity. In a recent estimate of the global burden of disease (GBD), outdoor air pollution was found to account for approximately 1.4% of total mortality, 0.5% of all disability-adjusted life years (DALYs) and 2% of all cardiopulmonary disease (Ezzati et al., 2002; WHO, 2002, Cohen et al, 2004). These estimates of the total disease burden were based solely on the effects of PM on mortality in adults and children. Because the epidemiological studies suggested that mortality impacts were likely to occur primarily among the elderly, the WHO estimates indicated that 81% of the attributable deaths from outdoor air pollution and 49% of the attributable DALYs occurred in people aged 60 years and older. Children under 5 years of age accounted for 3% of the total attributable deaths from outdoor air pollution and 12% of the attributable DALYs (WHO, 2002).

The GBD estimates were based on average urban concentrations of PM<sub>10</sub> and PM<sub>2.5</sub> (particulate matter less than 10 µm and 2.5 µm in diameter, respectively) as markers for outdoor air pollution. Traditionally, monitors for PM have been established to determine the concentration of pollutants in regional and background population exposures. As such, the estimates incorporated some of the larger urban sources of pollution such as traffic, industrial boilers and incineration. On the other hand, because the monitors were fixed-site, the estimates did not take into account pollution “hot spots” that may have affected segments of the population, without affecting the overall urban average. In addition, the GBD estimates did not incorporate the effects of outdoor air pollution in cities with a population less than 100 000 or in rural populations, nor the effects of other pollutants such as ozone and toxic air contaminants not included in the mixture of PM<sub>10</sub>.

The burden of disease in major cities will vary due to factors such as the amount of fossil fuel used, weather, underlying disease rates, and population size and density. Burden of disease estimates will be higher in certain regions of the world, such as those heavily dependent on coal for fuel use, those with topographical and climatic conditions that limit the dispersion of pollution, and in mega-cities with significant concentrations of PM<sub>10</sub> or PM<sub>2.5</sub> from traffic congestion. PM<sub>2.5</sub> is believed to be a greater health threat than PM<sub>10</sub> since the smaller particles are more likely to be deposited deep into the lung. In addition, studies have shown that particles this small will penetrate into the indoor, home environment. However, the majority of studies have reported effects using PM<sub>10</sub>, since PM<sub>2.5</sub> has been monitored less frequently. Therefore, the GBD and our proposed methods for estimating the Environmental Burden of Disease (EBD) use both PM<sub>10</sub> and PM<sub>2.5</sub> as indicators of exposure to outdoor air pollution.

To estimate the EBD, we used a methodology similar to that used to estimate the GBD, with similar caveats and uncertainties. As with the GBD study, EBD estimates are provided for several health outcomes including: adult cardiovascular mortality and lung cancer associated with long-term exposure to PM<sub>2.5</sub>, all-cause mortality for all ages associated with short-term exposure to PM<sub>10</sub>, and infant and childhood mortality from respiratory diseases associated with PM<sub>10</sub> exposure. Quantification of these estimates on a national or city-specific level, especially if local studies were utilized, will help to determine priorities for air pollution control, among other potential measures for improving public health.

Prior to the EBD study, there were several estimates of the health benefits associated with reducing population exposures to PM. Ostro & Chestnut (1998) generated estimates of the health benefits associated with the United States Environmental Protection Agency's proposed standards for PM<sub>2.5</sub>, while Kunzli et al. (2000) estimated the health effects attributed to traffic-related PM in three European countries. Similarly, Deck et al. (2001) estimated the health benefits associated with attaining US PM<sub>2.5</sub> standards in two US cities. Estimates have been developed for 26 cities in 12 European countries (APHEIS, 2001), and applying dose-response information primarily from the industrialized nations, the World Bank estimated the benefits of air pollution control in Mexico City (World Bank, 2002). Additional guidance for estimating the health effects of air pollution has been provided by the World Health Organization (WHO, 2001) and by the National Research Council (NRC, 2002).

Aspects of the EBD approach for outdoor air pollution are discussed in the following Sections 2–7. A summary of the proposed method for estimating the EBD of outdoor air pollution is given in Section 2. Section 3 briefly reviews the scientific evidence for the effects of air pollution on both mortality and morbidity, and provides the relative risk estimates used for the quantitative assessment. Section 4 summarizes the steps used in calculating the disease burden. Section 5 provides a discussion of the exposure assessment methods that are currently available, while in Section 6 underlying uncertainties in the proposed assessment method are discussed. In Section 7, an illustration of how to apply the methodology is given, using a step-by-step numerical example for Bangkok, Thailand.

## 2. Summary of the method

For a given city or region, the quantitative assessment of the health impact of outdoor air pollution, using PM10 or PM2.5 measurements, is based on four components:

1. An assessment of the ambient exposure of the population to PM (either PM10 or PM2.5), based either on existing fixed-site monitors or on model-based estimates. In addition a background or “target” concentration is needed as a comparison, to determine the attributable disease or potential benefits of reducing the risk factor by a specified amount.
2. A determination of the size of the population groups exposed to PM10 and PM2.5, and the type of health effect of interest.
3. The incidence of the health effect being estimated (e.g. the underlying mortality rate in the population, in deaths per thousand people).
4. Concentration–response functions from the epidemiological literature that relate ambient concentrations of PM10 or PM2.5 to selected health effects, and provide the attributable fractions (AFs) that are then used to estimate the following:
  - the number of cases of premature mortality and DALYs (cardiopulmonary and lung cancer) attributed to long-term exposure to PM2.5, for people >30 years old.
  - the number of cases of premature mortality and DALYs from respiratory diseases attributed to the short-term exposure to PM10, for children younger than five years old.
  - the number of cases of premature mortality from all causes from short-term exposure to PM10 (Note that this estimate should not be added to those above since this would involve double-counting. However, calculation of this number may provide useful information and is based on a separate set of studies)

The outcomes, exposure metrics, and relative risk functions are summarized in Table 1.

**Table 1** Recommended health outcomes and risk functions used to calculate the EBD

Outcome and exposure metric	Source	Relative risk function <sup>a</sup>	Suggested $\beta$ coefficient (95% CI)	Subgroup
All-cause mortality and short-term exposure to PM10 <sup>b</sup>	Meta-analysis and expert judgment (see text)	$RR = \exp[\beta (X - X_o)]$	0.0008 (0.0006 - 0.0010) <sup>c</sup>	All ages
Respiratory mortality and short-term exposure to PM10 (all-cause mortality for upper bound where applicable)	Meta-analysis (Table 2)	$RR = \exp[\beta (X - X_o)]$	0.00166 (0.00034, 0.0030)	Age <5 years
Cardiopulmonary mortality and long-term exposure to PM2.5	Pope et al. (2002); R Burnett <sup>d</sup>	$RR = [(X+1)/(X_o+1)]^\beta$	0.15515 (0.0562, 0.2541)	Age >30 years
Lung cancer and long-term exposure to PM2.5	Pope et al. (2002); R Burnett <sup>d</sup>	$RR = [(X+1)/(X_o+1)]^\beta$	0.23218 (0.08563, 0.37873)	Age >30 years

<sup>a</sup> X = current pollutant concentration ( $\mu\text{g}/\text{m}^3$ ) and  $X_o$  = target or threshold concentration of pollutant ( $\mu\text{g}/\text{m}^3$ ).

<sup>b</sup> Not used in DALY calculations and should not be added to the other mortality estimates.

<sup>c</sup> Presentation of a range rather than a point estimate is preferred.

<sup>d</sup> Personal communication.

### 3. The evidence base

Over the past two decades, epidemiological studies carried out on five continents have demonstrated that there are associations between a range of adverse health outcomes and daily, multi-day or long-term (one year to several years) changes in the concentrations of common air pollutants, including PM. PM is a mixture of liquid and solid particles of different sizes and chemicals. In urban environments, PM is derived mainly from fuel combustion by mobile sources (cars, trucks and buses) and by stationary sources (power plants and industrial boilers). PM can also be generated by mechanical grinding processes during the production phase, and by natural sources such as sea salt and blowing dust. Various particulate matter metrics – including PM<sub>10</sub>, PM<sub>2.5</sub>, black smoke, and sulfates – appear to show the most consistent associations with mortality and morbidity, although some associations have also been reported for ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide. For the quantitative assessment of health effects, however, PM<sub>2.5</sub> and PM<sub>10</sub> have been selected because of the relative wealth of epidemiological evidence and the existence of monitors or model-based estimates of PM in many countries.

The health effects associated with PM in epidemiological studies include mortality, lung cancer, hospitalization for cardiovascular and respiratory disease, emergency room and physician office visits, asthma exacerbation, respiratory symptoms, loss of schooling, restrictions in activity, and acute and chronic bronchitis. In addition, more-specific cardiovascular outcomes, such as heart attacks, changes in blood composition, and changes in heart rate and heart rate variability, have been found to be associated with PM exposure.

As with the global estimates (WHO, 2002), the EBD estimates for outdoor air pollution are based on three different outcomes:

- adult mortality (cardiopulmonary and lung cancer) related to long-term exposure;
- respiratory mortality in infants and children related to short-term exposure;
- all-cause mortality associated with short-term exposure for the full population (this estimate should not be added to those above since this would involve double-counting. Usually, the estimates from short-term exposure will only capture a part of the total burden of outdoor air pollution. In addition, there cannot be attribution of DALYs for this endpoint since the number of life years lost per case is generally unknown.

The underlying scientific evidence for the three mortality outcomes is reviewed below. Although there is also fairly strong scientific evidence for several morbidity outcomes related to exposure to PM, quantitative estimates are not proposed for these outcomes at this time given the difficulty in determining appropriate baseline rates in many countries, in particular developing countries. Previous impact assessments have indicated that mortality tends to dominate the overall burden of disease and this outcome is fully reflected in the proposed methodology. Nevertheless, concentration-response functions

for some of the morbidity endpoints are provided in WHO (2004)<sup>2</sup>. A more complete review of the evidence is given in USEPA (1996) and WHO (2003).

### **3.1 Mortality related to short-term exposure**

Time-series studies examine daily changes in air pollution (typically based on 24-h average concentrations) in relation to daily counts of mortality. Studies of the acute effects of PM exposure typically involve daily observations over several months or years. The analysis involves multivariate regression models that control for potentially confounding factors that may vary over time and be associated with mortality. Studies of the effects of PM often examine whether daily counts of mortality or cause-specific hospitalizations are correlated with daily concentrations of PM, after controlling for the effects of other covariates and potential confounders. Such factors include temporal and meteorological variables (e.g. day of the week, extremes in temperature, humidity or dew point), co-pollutants, and longer-term trends represented by seasonal changes or population growth. Well designed time-series studies can have several methodological strengths, including:

- a large sample size (up to eight years of daily data), which increases the sensitivity of the statistical analysis for detecting effects;
- data are collected for a range of population demographics, baseline health characteristics and human behaviours, which makes the results more widely applicable;
- the exposures are “real-world” and avoid the need to extrapolate to lower concentrations, or across species.

Limitations of time-series studies include:

- the difficulty in determining actual pollutant concentrations to which people are exposed;
- the potential for misclassifying the exposure;
- there can be covariation among pollutants, which makes it difficult to attribute an effect to a single pollutant.

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<sup>2</sup> For cities or countries that have baseline data on health outcomes, also for other endpoints such as disease-specific hospitalisation, asthma exacerbation, and chronic bronchitis, the software AirQ2.2 from the WHO European Office, can assist in developing estimates including a life table analysis for determining life years lost from exposure to air pollution .  
([http://www.euro.who.int/eprise/main/WHO/Progs/AIQ/Activities/20040428\\_2](http://www.euro.who.int/eprise/main/WHO/Progs/AIQ/Activities/20040428_2)).

### 3.1.1 Short-term exposure and mortality: all ages

#### *Key studies from the literature*

Several multi-city studies and more than 100 single-city studies have been published on the association between daily exposure to PM and mortality. To synthesize the evidence, we reviewed all multi-city studies and checked for consistency with single-city studies. Most of the air pollution–mortality studies published over the last decade employ fairly standardized, statistical techniques that control for potentially confounding influences. In particular, recent, higher-quality studies are characterized by:

- the use of Poisson regression models, since mortality is a rare event and can be described by a Poisson distribution;
- three or more years of daily data in a given city or metropolitan area;
- an examination of the effects of day-of-the-week and daily changes in the weather;
- the use of general additive models with nonparametric smoothing, or general linear models with parametric splines to control for time, season and weather.

With increasing statistical sophistication, these studies have shown that either one-day or multi-day PM average concentrations are associated with both total mortality and cardiopulmonary mortality. Among the first of the multi-city studies on mortality, Schwartz et al. (1996) examined data from the Harvard Six Cities study. This database included monitors sited specifically to support ongoing epidemiological studies and to be representative of local population exposures. Consistent associations were reported between daily mortality and daily exposures to both PM<sub>10</sub> and PM<sub>2.5</sub>, with a 0.8% (95% confidence interval (CI) = 0.5–1.1) increase in daily total mortality per 10 µg/m<sup>3</sup> of PM<sub>10</sub>.

In a study of 10 USA cities, Schwartz (2000a) examined the daily effects of PM<sub>10</sub> and reported that a 10 µg/m<sup>3</sup> change in PM<sub>10</sub> (measured as a two-day average of lag 0 and lag 1) was associated with a 0.7% increase in daily mortality. In another multi-city study, Burnett et al. (2000) analyzed mortality data for 1986–1996 from the eight largest Canadian cities and found that both PM<sub>10</sub> and PM<sub>2.5</sub> were associated with daily mortality. For PM<sub>10</sub>, a 10 µg/m<sup>3</sup> increase was associated with a 0.7% (95% CI = 0.2–1.2) increase in daily mortality.

Another study involving 29 European cities measured PM<sub>10</sub> using a methodology similar to the USA studies cited above (although in some of the cities PM<sub>10</sub> was estimated from observations collected from a subset of days using co-located black smoke or total suspended particulate matter (TSP)). Again, an association between daily mortality and PM<sub>10</sub> was reported, with an overall effect estimated at 0.6% per 10 µg/m<sup>3</sup> (Katsouyanni et al., 2001).

Samet et al. (2000a) applied a range of statistical tools and sensitivity analyses to a database consisting of the 88 largest cities in the USA (NMMAPS), while a second study focused on the 20 largest cities (Samet et al., 2000b). The combined results for all of the cities indicated an association between mortality and PM of approximately 0.5% per 10

$\mu\text{g}/\text{m}^3$  of PM<sub>10</sub>, which was near the lower end of the range found in earlier studies. More recent studies used an alternative statistical model and found an association of about 0.27% per 10  $\mu\text{g}/\text{m}^3$  of PM<sub>10</sub> (Dominici et al., 2002). These effects may be at the lower end of the range because the studies only considered lags (or delayed effects) of zero, one and two days. Other studies have reported greater effects with longer lags or multi-day moving averages. Since many of the cities in the study collected PM<sub>10</sub> data on an every-sixth-day basis, cumulative averaging times could not be examined. Another possible reason for the lower effect estimates in the Dominici et al. (2002) study relates to the number of covariates used in the regression model. Besides PM<sub>10</sub>, day of week, and a smoothing of time using seven degrees of freedom (or cycles of about seven weeks), two variables were included for temperature and two for dew point (same day and an average of the three previous days). Thus, it is possible that these factors explain some of the variability in mortality that may be better attributed to air pollution. In addition, the authors found that measurement error would likely underestimate the effect of PM (Zeger et al., 2000), and that co-pollutants such as ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide did not significantly affect or confound the estimated effect of PM (Samet et al., 2000a).

Meta-analyses of earlier mortality studies suggest that, after converting the alternative measures of particulate matter used in the original studies to an equivalent PM<sub>10</sub> concentration, the effects on mortality are fairly consistent (Ostro, 1993; Dockery and Pope, 1994). Specifically, the mean estimated change in daily mortality associated with a one-day 10  $\mu\text{g}/\text{m}^3$  change in PM<sub>10</sub> implied by these studies is approximately 0.8 percent, with a range of 0.5 percent to 1.6 percent. More recent studies have also been summarized in meta-analyses. For example, a recent meta-analysis of European studies suggested a mean increase of the risk of 0.6% per 10  $\mu\text{g}/\text{m}^3$  PM<sub>10</sub> (WHO, 2004). In addition, a meta-analysis of Asian studies indicated a mean increase of the risk of 0.4% to 0.5% per 10  $\mu\text{g}/\text{m}^3$  PM<sub>10</sub> (HEI, 2004).

In addition to these multi-city investigations and meta analyses, studies examining the effect on mortality of short-term exposure to PM have been conducted in over 100 separate cities. Some of these studies have been conducted in cities outside of the western industrialized nations and in developing countries, and report effect estimates that are similar to those for North America and Europe. For example, the following effect estimates have been reported for total populations and a 10  $\mu\text{g}/\text{m}^3$  change in PM<sub>10</sub> (with 95% confidence intervals): 1.7% (1.1–2.3) Bangkok, Thailand (Ostro et al., 1999a); 1.83% (0.9–2.7) Mexico City (Castillejos et al., 2000); 1.1% (0.9–1.4) Santiago, Chile (Ostro et al., 1996); 0.8% (0.2–1.6) Incheon, South Korea (Hong et al., 1999); 1.6% (0.5–2.6) Brisbane, Australia (Simpson et al., 1997); and 0.95% (0.32–1.6) Sydney, Australia (Morgan et al., 1998). Mortality estimates associated with PM<sub>10</sub> or TSP have also been reported for Shenyang, China (Xu et al., 2000); seven cities in South Korea (Lee et al., 2000); and New Delhi, India (Cropper et al., 1997). It is reasonable to extrapolate these estimates to those areas where studies have not been undertaken, since the existing studies were conducted in cities that involve a range of underlying conditions (e.g. demographics, smoking status, climate, housing stock, occupational exposure, socioeconomic status) and PM concentrations. For example, studies in Mexico City, Bangkok and Santiago reported mean PM<sub>10</sub> concentrations of 45, 60 and 115  $\mu\text{g}/\text{m}^3$ , and



maximum PM<sub>10</sub> concentrations of 121, 227 and 360 µg/m<sup>3</sup>, respectively. However, in very polluted cities the concentration-response relationship will probably deviate from being linear. Therefore, it may be prudent to cap the range for the assumption of linearity (see the uncertainty section below).

Taken together, these studies provide compelling evidence that PM significantly increases mortality rates. Although the relative risk per person is low, the large number of people exposed suggests that PM has a major impact on public health. Also, many of the above studies reported a stronger association between PM<sub>10</sub> exposure and mortality when the mortality measurements lagged exposure by one to four days, compared to same-day mortality measurements. In addition, cumulative exposures of three or five days often had stronger associations with mortality than single-day lags. For example, a regression model that allowed for air pollution effects in 10 USA cities to persist over several days suggested that the relative mortality risk doubled for people older than 65 years of age, to approximately 2% per 10 µg/m<sup>3</sup> of PM<sub>10</sub> (Schwartz, 2000b).

#### *Recommended relationships for quantifying disease*

It is important to note that estimation of the effects of short-term exposure would, to a certain extent, double-count those cases estimated to result from long-term exposure, and the burden specifically estimated for children under age 5. The details for quantification, therefore, are provided so analysts can generate additional information based on the time-series studies. These estimates, however, should not be added to those generated from the studies of long-term exposure, described below. The latter are preferred since they can be used to determine life years lost and DALYs. In contrast, no evidence is currently available regarding the amount of life shortening involved with each fatality associated with short-term exposure. Therefore, these calculations are used only to provide an estimate of the number of premature deaths per year, not years of life lost (YLL) or DALYs.

Based on available evidence, a reasonable estimate of the EBD for mortality due to short-term exposure is generally a 0.6% to 1% increase per 10 µg/m<sup>3</sup> PM (possibly more, depending on local conditions and mortality structure). This range reflects the evidence from a variety of cities and averaging times (including single and multi-day lags). If a central estimate is needed, 0.8% may be most appropriate and local studies may provide more specific results.

For quantifying this effect, the relative risk (RR) can be specified as follows (Figure 1):

$$RR = \exp[\beta(X - X_o)] \quad (\text{Equation 1})$$

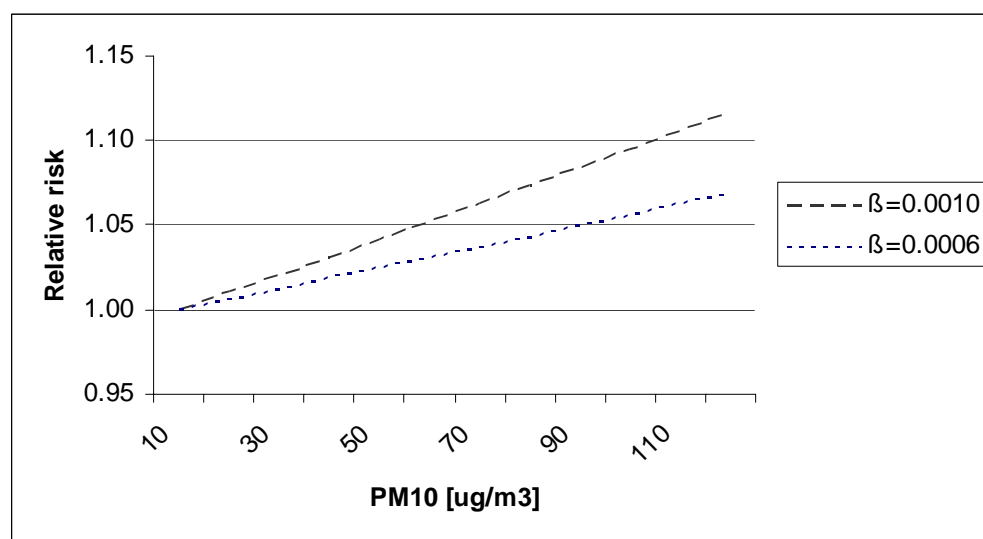
where:

- $\beta$  = range 0.0006 – 0.0010; (proposed best estimate = 0.0008).
- $X$  = current annual mean concentration of PM<sub>10</sub> (µg/m<sup>3</sup>).
- $X_o$  = baseline concentration of PM<sub>10</sub> (µg/m<sup>3</sup>).

Comparing current and background concentrations is one step in calculating the attributable burden (i.e. the total health impact of the risk factor). The current

concentration will be determined from existing monitoring data, model estimates, or best judgement. The baseline concentration is assumed to be the background concentration (i.e. the level that would exist without any man-made pollution, which is approximately  $10 \mu\text{g}/\text{m}^3$  PM10). If current pollution levels are compared with some regulatory target greater than background concentrations, as an alternative, the associated disease burden that would be avoided could also be calculated (see Section 5 for calculations). The relative risk estimate can be applied to the entire population (i.e. all ages) and over the full range of PM10 concentrations, since the relationship appears to be almost linear up to relatively high PM10 concentrations, typically 125 to  $150 \mu\text{g}/\text{m}^3$ .

**Figure 1** Relative risks for short-term mortality and OAP for all ages  
Based on a background concentration of  $10 \mu\text{g}/\text{m}^3$  PM10



An estimate of all-cause mortality associated with short-term exposure to PM10 was not included in the global estimate of disease burden from outdoor air pollution (WHO, 2002), since the number of life-years lost (and therefore DALYs) cannot be determined for each of the premature deaths. For the EBD calculation, however, estimates of premature mortality associated with short-term exposures can be used as an alternative to DALYs, and used as a basis for comparing short-term and long-term effects of pollutant exposure. Short-term estimates should not be added to long-term estimates or estimates for children, however, since that would involve some double counting of the mortality cases. A summary of the relative risk function and model parameters for all-cause mortality from short-term exposure is provided in Table 1.

One significant uncertainty associated with this outcome relates to differences in the distribution of mortality causes in different cities, countries or regions. Presumably, most of the “all-cause” mortality resulting from exposure to PM is associated with cardiovascular and pulmonary disease. Therefore, in an area with a relatively low proportion of cardiopulmonary mortality (e.g. in developing countries with relatively more mortality from malnutrition and diarrhoea), it is more likely that the short-term impact of air pollution will be overestimated. This is the result of applying the

percentage increase in mortality due to air pollution, to a mortality rate that includes relatively more non-cardiopulmonary disease. However, existing studies from developing countries suggest that an increase in mortality of about 1% per 10  $\mu\text{g}/\text{m}^3$  PM is a reasonable approximation, and that the likely effect lies within the range that has been proposed for calculating the attributable burden of disease.

#### *Uncertainty estimate*

Uncertainty in such estimates could arise from a number of causes (see Section 6). In this context, upper and lower estimates could be obtained by applying the upper and lower coefficients of the confidence intervals for estimating the relative risks. This would however only cover statistical uncertainty related to the risk estimates, while further uncertainty is added due to potential errors in measuring population exposure, differences in pollution mixtures and baseline health status, and in extrapolating existing results to very high concentrations only found in developing countries. For the latter case, it is likely that linear extrapolations of our estimates will overestimate the effect of PM on mortality for cities where PM<sub>10</sub> is greater than approximately 125  $\mu\text{g}/\text{m}^3$ , a value among the highest PM<sub>10</sub> concentrations typically reported in the epidemiologic studies in North America and Western Europe. For such cities, analysts should consider capping the highest relative risk at that found at 125  $\mu\text{g}/\text{m}^3$ .

### **3.1.2 Short-term exposure and mortality: children**

#### *Key studies in the literature*

The evidence that daily exposure to air pollution increases the mortality rate for all-ages includes data specific to children younger than five years of age. The mortality rate for such children should not be added to the total number of premature cases of mortality calculated by Equation 1, since the calculation already accounts for all ages. However, estimates for children can be used to calculate YLL and DALYs, since there is a significant loss of life involved, and the results added to those calculated for the effects of long-term exposure on the cohort aged 30 years and older.

While the elderly may dominate the potential population at risk, several recent cross-sectional, cohort and time-series studies have reported associations between ambient PM and neonatal or infant mortality, low birth weight or higher rates of prematurity (e.g. in Rio de Janeiro: Penna & Duchiade, 1991; in the Czech Republic: Bobek & Leon, 1998; and in the USA: Woodruff, Grillo & Schoendorf, 1997). Associations between PM and both low birth weight and premature delivery were also reported among a cohort of 98 000 neonates in Southern California between 1989–1993 (Ritz et al., 2000).

In both cross-sectional and cohort studies, it may be difficult to separate the effects of pollution from other factors such as poverty, exposure patterns (e.g. in higher pollution areas people may spend more time outside or live closer to highways), and other factors related to socioeconomic status, such as diet. However, daily time-series studies in several cities have also demonstrated associations between PM and mortality for those under five years old (or in one case, under one year old), and these studies provide a basis for our estimates of the effects of PM<sub>10</sub> on infant mortality. Three studies have been

conducted for different years in Sao Paulo, Brazil (Saldiva et al., 1994; Gouveia & Fletcher, 2000; Conceição et al., 2001). Studies have also been conducted in Mexico City (Loomis et al., 1999) and Bangkok (Ostro et al., 1998, 1999a). These five studies estimated the increase in daily mortality from acute respiratory infections, or from all-cause mortality, associated with short-term changes in ambient particulate air pollution. The statistical models used in these studies were similar to those used in the adult mortality studies of acute exposure: general additive Poisson models, controlling for time, season and weather. One study (Loomis et al., 1999) used PM<sub>2.5</sub>, which was converted to PM<sub>10</sub> assuming  $PM_{2.5} = 0.6 \times PM_{10}$ , based on locally available data. This study also focused on infants under one year old, and the data were extrapolated to all children under five years old. For Bangkok, we used the data of Ostro et al. (1998), rather than Ostro et al. (1999a), since the former study explored different lag structures. This is a slight departure from the method used in the global analysis of disease burden from outdoor air pollution (WHO, 2002; Cohen et al., 2004). These studies are summarized in Table 2.

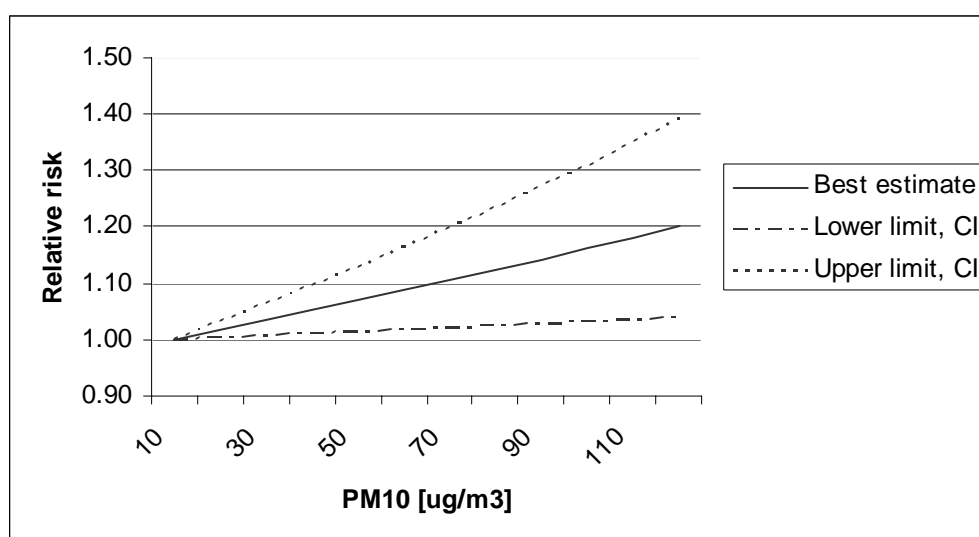
#### *Recommended relationships for quantifying disease*

Combining the estimates reviewed above, using a fixed-effects model that weights each estimate by the inverse of its standard error, we estimate that a 10  $\mu\text{g}/\text{m}^3$  increase in ambient PM<sub>10</sub> concentration results in a 1.66% (95% CI = 0.34–3.0) mean increase in daily mortality from acute respiratory infections in children 0–5 years of age. Although studies indicate that the 1.66% increase per 10  $\mu\text{g}/\text{m}^3$  increase in ambient PM<sub>10</sub> concentration are applicable to all-cause mortality, a generalization to other parts of the world would assume a similar structure in mortality patterns and similar levels of health care. As this cannot always be assumed, we suggest the application of this rate to respiratory diseases alone. The application of this rate to all-cause mortality could however represent an upper boundary of disease burden caused by outdoor air pollution, but may result in an overestimate when applied to certain regions. Thus, the linear exposure model (Equation 1, Figure 2) should be used to quantify the relative risks for the endpoint of respiratory diseases with  $\beta = 0.00166$  (95% CI of 0.00034–0.0030), and where applicable for all causes for the upper bound. As in the case for all-cause mortality for all ages reported above, analysts should cap the maximum risk estimates to those found when PM<sub>10</sub> is approximately 125  $\mu\text{g}/\text{m}^3$ .

**Table 2** Child and infant mortality related to PM10 exposure

Source	City	Country	Age group (years)	PM measure	Diagnosis	Change per 10 µg/m <sup>3</sup> increase (%)	95% CI
Conceição et al. (2001)	Sao Paulo	Brazil	0–4	PM10	All respiratory	1.61	-14.82, 21.22
Loomis et al. (1999)	Mexico City	Mexico	0–1	PM2.5 <sup>a</sup>	All cause	6.87	2.48, 11.45
Saldiva & Bohm (1995)	Sao Paulo	Brazil	<5	PM10	All respiratory	-1.98	-6.54, 2.57
Gouveia & Fletcher (2000)	Sao Paulo	Brazil	<5	PM10	All respiratory	-0.09	-3.23, 3.14
Ostro et al. (1998)	Bangkok	Thailand	<6	PM10	All cause	1.80	0.23, 3.37
Overall			<5	PM10	All respiratory (All cause for upper bound)	1.66	0.34, 3.00

<sup>a</sup> Converted to PM10 assuming PM2.5 = 0.5 x PM10 (Ostro et al., 1998).

**Figure 2** Relative risks for short-term mortality and OAP in children 0-4 years  
Based on a background concentration of 10 µg/m<sup>3</sup> PM10

### 3.1.3 Issues related to short-term exposure mortality studies

*Confounding factors.* The results of the time-series studies also indicate that the associations between PM and mortality are not significantly confounded by weather patterns, longer-term seasonality, or day of week. This evidence is provided by

modelling and by controlling for such factors, as well as by the heterogeneous nature of the cities examined in the studies. Specifically, consistent evidence for an effect of PM on mortality rates has been observed in areas and cities in both cold (Detroit and Montreal) and warm (Mexico City and Bangkok) climates; where PM peaks in the summer (Steubenville and Philadelphia), winter (Utah Valley) or spring (Helsinki); with substantial seasonal changes in mortality (Chicago); and with little seasonality (Coachella Valley, CA; Birmingham, UK, and Bangkok). Furthermore, factors such as smoking, exposure to second-hand smoke or occupational irritants, and personal characteristics are not confounders in these studies since they do not vary with air pollution on a daily basis.

*PM as an index.* A related issue is whether there is independent evidence for an effect of PM, or whether confounding by co-pollutants makes it impossible to implicate PM as a pollutant of concern. In many of the time-series mortality studies, including additional pollutants into the regression model did not alter the estimated impact of PM, suggesting the co-pollutants were not confounding factors. Samet et al. (2000a) also studied this issue using data from 90 USA cities, and found that there were minimal changes in the estimated PM10 coefficient after gaseous pollutants (ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide) were sequentially added to the regression model. Similar results have been reported in most studies that have examined PM10 and mortality (Schwartz 2000a). Katsouyanni et al. (2001) also found no effect modification or confounding associated with either ozone or sulfur dioxide. PM effects were greater in cities with higher concentrations of nitrogen dioxide, but the effects of PM were not attenuated. However, given that fuel combustion will generate multiple pollutants that are often correlated over time, PM still may be serving as a general proxy for the overall mixture. Thus, for the purposes of calculating the overall EBD, PM is useful as a proxy for combustion sources.

*Disease-specific effects.* Pre-existing cardiovascular and respiratory diseases are clearly risk factors for PM-related mortality, and many time-series studies have reported statistically significant associations between PM and cardiovascular-specific and respiratory-specific mortality (e.g. Schwartz, 1993; Fairley, 1999; Ostro et al., 1999a; Samet et al., 2000a). When compared with all-cause mortality, these disease-specific mortality analyses typically generate larger and more precise effect estimates for PM. In calculating the AF, however, the higher relative risk estimates will be offset by a lower baseline incidence level. Therefore, the total effect of using disease-specific estimates may be fairly similar to that obtained by using all-cause mortality. As data collection improves, analysts could use disease-specific relative risks and baseline mortality rates to generate estimates of attributable risk.

*Life shortening.* Although time-series studies to date have been unable to determine the amount of life shortening that is related to PM, there is indirect evidence that it is significant. Recent studies have reported associations between ambient PM and increased heart rate, decreased heart rate variability, and the incidence of arrhythmias (Liao et al., 1999; Pope et al., 1999; Gold et al., 2000; Peters et al., 2001). These outcomes are considered reliable predictors of the risk of death from heart disease (e.g. Nolan et al., 1998). More direct evidence for a nontrivial reduction in life expectancy has been provided by studies that statistically control for mortality displacement, where the

time of death might be delayed by only a few days. If all pollution-related deaths were associated with such mortality displacement, the total life shortening would likely be very small. However, using both frequency-domain and time-domain methods, it has been shown that most air pollution-associated mortality is not due to such displacement (Zeger, Domenici & Samet, 1999; Schwartz, 2000c). For cardiovascular deaths, mortality displacement does not appear to be a major factor, as the average life shortening appears to be greater than two or three months. In contrast, deaths from chronic obstructive pulmonary disease (COPD, which consists mainly of emphysema and chronic bronchitis) appeared to be more consistent with a mortality displacement hypothesis (Schwartz, 2000c, 2001).

Finally, evidence of a significant loss in life-years from air pollution has been provided by studies of infants and children (reviewed above). The studies indicated that infants and children, possibly those with pre-existing respiratory illness, may be especially sensitive to the effects of ambient PM pollution.

*Thresholds.* For short-term exposure to PM, two general methods are available to address the issue of a threshold (i.e. an ambient PM level below which there would be no risk of a significant adverse health outcome). The first method is indirect and uses data sets with very low mean ambient concentrations to examine whether there is a threshold. The second method is direct and uses statistical tests that carefully model the shape of the concentration–response function. Both approaches indicate there is no observable population threshold. For example, several studies have reported associations between PM and mortality in areas with low ambient concentrations of PM<sub>10</sub> including: Morgan et al. (1998) for Sydney, Australia (mean ambient PM<sub>10</sub> concentration = 18 µg/m<sup>3</sup>, based on conversion from co-located nephelometry data); Wordley, Walters & Ayres (1997) for Birmingham, UK (mean = 26 µg/m<sup>3</sup>); Schwartz, Dockery & Neas (1996) for the Harvard Six-Cities study (mean = 25 µg/m<sup>3</sup>); Burnett et al. (2000) for the eight largest Canadian cities (mean = 26 µg/m<sup>3</sup>); and Gwynn, Burnett & Thurston (2000) for Buffalo, NY and Rochester, NY (mean = 24 µg/m<sup>3</sup>).

Among the statistical approaches, Schwartz (2000a) examined the concentration–response relationship in 10 USA cities, restricting the data to days on which the PM<sub>10</sub> concentration was less than 50 µg/m<sup>3</sup>. The resulting risk estimates were statistically significant and greater than that for the entire data set. Using a different statistical approach in their analysis of 10 USA cities, Schwartz & Zanobetti (2000) also found no evidence for a threshold effect. Similarly, a study of the 20 largest cities in the USA found no evidence for a threshold (Daniels et al., 2000).

### ***3.1.4 Summary of findings on short-term exposure to particulate matter and mortality***

1. The associations between daily changes in PM<sub>10</sub> and mortality appear to be independent of weather factors, seasonality, time, and day of week – all of which were typically controlled for in the analyses. The studies included a range of environments, pollution–temperature conditions, population–age distributions, background health conditions, socioeconomic statuses, and health-care systems. The

range of the association is approximately a 0.5–1.6% increase in mortality per 10  $\mu\text{g}/\text{m}^3$  increment of PM10. However, when longer exposure averaging times are examined, using distributed lags of several days or cumulative exposures of up to several months, the estimated effects may be approximately a 2% increase in mortality per 10  $\mu\text{g}/\text{m}^3$  increment of PM10.

2. The effects of PM cannot be explained by exposure to other pollutants. As might be expected, examining several correlated pollutants in the same model often increases the variation of the estimated PM effect and attenuates the PM effect. However, the estimated PM impact is generally consistent regardless of the concentration of, or degree of co-variation with, other pollutants, which supports the idea that PM has an effect independent of other pollutants.
3. The elderly, those with chronic heart or lung disease, and infants appear to be at significantly greater risk of PM-associated mortality. Study results suggest that much of the mortality associated with acute exposure is not the result of just a few days of life shortening. Rather, for cardiovascular mortality, there is evidence that significant reductions in life expectancy may be involved. In addition, if the associations between PM and infant mortality represent causal relationships, large reductions of life expectancy could result as well. However, at this time, it is not possible to determine the number of life years lost using time-series studies. Therefore, only the number of premature deaths per year can be calculated, and not DALYs.
4. No threshold of response has been observed in PM–mortality studies. Several direct and indirect approaches have consistently found that non-threshold, linear models provided the best fit to the data. Evidence from the USA suggested that for PM10, the background concentration was 8–10  $\mu\text{g}/\text{m}^3$ , while the background for PM2.5 may be 3–5  $\mu\text{g}/\text{m}^3$ . These may be plausible lower bounds for the health effects calculated in the EBD, unless local data suggest different levels.
5. It is reasonable to apply the suggested relative risks to cities and regions throughout the world, since the studies have been replicated in many alternative physical and social environments and over a wide range of concentrations of PM10.

Rather than conducting a formal meta-analysis of the studies, we provide a reasonable range of estimates based on the available results. This range takes into account: the variability observed among the studies; the observation that multi-day averages significantly increase the size of the effect; and the larger effect sizes reported by some studies of developing countries. Therefore, we recommend a range of 0.6–1.5% increase in mortality per daily increase of 10  $\mu\text{g}/\text{m}^3$  in PM10. As a central estimate, we assume a 1% increase in mortality per 10  $\mu\text{g}/\text{m}^3$  increase in PM10. As new studies of cities in the developing world are published, the findings can be weighted together with the existing pool of studies, either informally or formally, using a Bayesian framework.

The estimate of mortality associated with short-term exposure to PM10 should not be added to mortality estimates associated with long-term exposure (described below). However, it is of interest to provide a quantitative estimate of the short-term mortality



effect so that policy-makers and other analysts can appreciate the implications of the short-term studies. Short of data to the contrary, a background concentration of  $10 \mu\text{g}/\text{m}^3$  PM<sub>10</sub> should be assumed. The form and coefficients of the recommended risk function for mortality associated with short-term exposure are summarized in Table 1.

### 3.2 Mortality related to long-term exposure

#### *Key studies from the literature*

Several air pollution studies used a prospective cohort design to examine the effects of long-term exposure to PM. In this type of study, a sample of individuals are selected and followed over time. For example, Dockery et al. (1993) followed approximately 8000 individuals in six cities in the eastern USA over a 15-year period (the Harvard Six Cities study); and Pope et al. (1995) followed mortality rates over a 7-year period in approximately 550 000 individuals in 151 cities in the USA. These studies used individual-level data so that other factors that affect mortality can be characterized and adjusted for in the analysis. Once the effects of individual-level factors were determined, the models examined whether longer-term citywide averages in PM (measured as PM<sub>10</sub>, PM<sub>2.5</sub> or sulfates) were associated with different risks of mortality and life expectancies. Several different cause-specific categories of mortality were examined, including lung cancer, cardiopulmonary, and “all other causes”.

These studies incorporated much, but not all, of the impact associated with short-term exposures (Kunzli et al., 2001). One effect that would be difficult to capture in the long-term studies is mortality displacement of a few days, since it would not alter the differences in overall life expectancy predicted by the longer-term studies. The greatest uncertainties in long-term studies involve the disease-relevant times, durations, and intensities of exposure. Both studies assigned citywide, multi-year averages that occurred when the study participants were young to middle-aged adults (approximately between the ages of 20–50 years). Thus, early childhood exposure was not estimated and no within-city differences in exposure were incorporated into the analyses, making it difficult to detect an effect of pollution and likely biasing the analyses towards the null hypothesis of no effect. Therefore, it is unlikely that bias or misclassification of exposure could explain the statistically significant associations between long-term exposure to PM and measures of mortality that were reported by the two studies.

Specifically, Dockery et al. (1993) reported associations between total and cardiovascular mortality, and PM<sub>10</sub>, PM<sub>2.5</sub> and sulfates. In this study, PM<sub>2.5</sub> concentrations ranged from 11 to  $29.6 \mu\text{g}/\text{m}^3$  and PM<sub>10</sub> ranged from 18 to  $46.5 \mu\text{g}/\text{m}^3$ . Similarly, Pope et al. (1995) reported associations between fine particles and sulfates with both “all-cause” mortality and cardiopulmonary mortality. Across the 50 cities with PM<sub>2.5</sub> data, PM<sub>2.5</sub> ranged from 7 to  $30 \mu\text{g}/\text{m}^3$ . The relative risk estimates for this study were smaller than those reported by Dockery et al. (1993), but the confidence intervals around the relative risk estimates overlapped. The estimated mortality effects of long-term exposure to PM<sub>10</sub> (approximately 4–7% per  $10 \mu\text{g}/\text{m}^3$  of PM<sub>10</sub>) are much larger than those associated with daily exposure (approximately 1% per  $10 \mu\text{g}/\text{m}^3$  of PM<sub>10</sub>).

These studies also provide a basis for calculating reductions in life expectancy associated with PM exposure. The results suggest that the 24  $\mu\text{g}/\text{m}^3$  difference in PM<sub>2.5</sub> between the cleanest and dirtiest cities is associated with an almost 1.5-year difference in life expectancy (Pope, 2000). Brunekreef (1997) used a life-table for men in the Netherlands and estimated an overall difference of 1.1 years in life expectancy between the two extreme cities in the Pope et al. (1995) study. The difference for people who actually died from diseases associated with air pollution was estimated to be about 10 years. This is because air pollution-related deaths make up only a small fraction of the total deaths in a city.

Krewski et al. (2000) completed an independent validation and re-analysis of both the Dockery et al. (1993) and Pope et al. (1995) studies. The first task was to recreate the data sets and validate the original results. Krewski et al. (2000) reported few errors in the coding and data merging in the original studies and basically replicated the results of both studies. The second task was to conduct an exhaustive sensitivity analysis of the original studies to determine whether the results were robust. Specifically, the authors examined the effects of:

- alternative statistical models;
- potential city-wide confounders, such as population growth, income, weather, number of hospital beds and water hardness;
- population subgroups;
- co-pollutants, including ozone, sulfur dioxide and nitrogen dioxide;
- alternative PM exposure estimates, including different years and particle sizes;
- spatial correlation between cities.

In general, the re-analysis confirmed the original results, that there was an association between mortality and long-term exposure to PM. Among the more important new findings were:

- education appears to be a significant effect modifier (possibly because it serves as a marker for socioeconomic status, health care, residential mobility or lifestyle factors);
- the results were not confounded by either individual-level or city-wide (ecological) covariates;
- the associations between exposures to sulfate and PM<sub>2.5</sub>, and all-cause and cardiopulmonary mortality were nearly linear within the relevant ranges, with no apparent thresholds.

One finding from the long-term exposure studies may be particularly relevant for extrapolating the data to other countries, especially those that are economically less developed. The studies found a consistent effect modification by education, since the effect estimates varied significantly when the analyses were stratified by educational attainment. Both Dockery et al. (1993) and Krewski et al. (2000) reported that the

relative risks associated with PM are significantly higher for those with less than a high school education. The more modest association with PM among more-educated individuals suggests that better nutrition and access to health care (or some other variables correlated with educational attainment) may be important co-factors in mortality associated with air pollution. Among individuals with lower educational attainment, poverty, poor nutrition, and less access to medical resources are all more common. Lower socioeconomic status is also likely to be associated with residences closer to mobile and stationary sources of pollution. Therefore, it is possible that socioeconomic status is simply associated with higher exposure to existing sources, rather than serving as an effect modifier. At this time the precise factor(s) driving this effect modification is not well characterized, and we do not recommend adjusting the estimate coefficient to take into account local educational or economic status. Hopefully future research will elucidate the role that socioeconomic status (or some factor associated with it) has on the health effects from air pollution exposure.

Recently, Pope et al. (2002) extended their analysis using 16 years of follow-up data (through 1998). Besides the ambient data from 1979 to 1983 that were used in the original study, more recent data on PM<sub>2.5</sub> for 1999 and 2000 were used to estimate exposure, based on city of residence of the study participants. Several alternative exposure scenarios were tested using Cox proportional hazard regression models that controlled for many individual-level risk factors. Specifically, the model was estimated using the data from 1979 to 1983, the data for 1999 and 2000, and the average of all of the available data. Associations were reported between all three of these alternative exposure assessments, and total mortality, cardiovascular mortality, and lung cancer mortality. Estimates of relative risk were larger for the 1999 and 2000 data, and largest when using the mean of all of the available exposure data. This may be owing to reduced measurement error for the more recent data, or because more recent exposures may be more relevant to pollution-related mortality. The latter finding was also reported in the re-analysis of Krewski et al. (2000). In addition, the lowest values of PM<sub>2.5</sub> in the 1999-2000 data are around 5  $\mu\text{g}/\text{m}^3$  which is close to background levels. This suggests that it is reasonable to calculate effects down to background concentrations.

There are several options for assigning risk related to long-term exposure. Therefore, the sensitivity of the estimates is demonstrated quantitatively below. Different studies could be used or combined, and the concentration–response functions could be linear or non-linear. Also, estimates are provided for many different models (Krewski et al., 2000) and for different years of exposure. Our primary risk estimates are based on the Pope et al. (2002) study since the number of cities and the sample size were much larger than in other studies, and the risk estimates of this study overlapped with those of Dockery et al. (1993). The published estimates of Pope et al. (1995) utilized a linear function of exposure. However, it is inappropriate to extrapolate this linear function to the higher levels observed in some of the mega-cities throughout the world – the resulting effects would be implausible.

To obtain a more realistic model, the concentration–response function was re-estimated using a log–linear function for exposure (i.e. a function whose slope flattens at higher concentrations). For the data in the Pope et al. (1995) study, linear and log–linear

exposure models fit equally well for PM<sub>2.5</sub> concentrations of 10–30 µg/m<sup>3</sup> (R. Burnett, personal communication). Empirically, the log–linear model generated slightly higher relative risks within the 10–30 µg/m<sup>3</sup> range, and lower relative risks below and above this range. To estimate the disease burden caused by outdoor air pollution, we propose that the log–linear model of exposure and the average of all years of available exposure be used, since the resulting estimate of the disease burden is likely to have the minimum measurement error.

### *Recommended relationships for quantifying disease*

Given the studies that are available to date, we recommend that the following log–linear exposure formula for relative risk be used when measuring cardiopulmonary mortality (Pope et al., 2002):

$$RR = \exp(\alpha + \beta \ln(X + 1)) / \exp(\alpha + \beta \ln(X_o + 1)) \quad (\text{Equation 2})$$

where:

$$\beta = 0.1551 \text{ (95\% CI = 0.05624 - 0.2541)}.$$

X refers to the annual mean concentration of PM<sub>2.5</sub>. A value of 1 was added to the X terms in the formula to ensure that the log function is defined at X = 0. The value of X<sub>o</sub> is usually assumed to be either: the background concentration<sup>3</sup> of PM<sub>2.5</sub> in the city or country (e.g. 3 µg/m<sup>3</sup>) or the lowest observed concentration in the original study (7.5 µg/m<sup>3</sup> in the Pope et al. (1995) study)<sup>4</sup>. This formula can be simplified as the ratio of the relative concentrations (of the current versus the target) raised to the power of β (which equals 0.1551 for cardiopulmonary mortality):

$$RR = [(X + 1) / (X_o + 1)]^\beta \quad (\text{Equation 3})$$

For lung cancer-related mortality, the β coefficient would be 0.232179 (standard error = 0.07477; 95% CI = 0.08563–0.37873) (Pope et al., 2002). The β-coefficient can then be substituted into Equation 3.

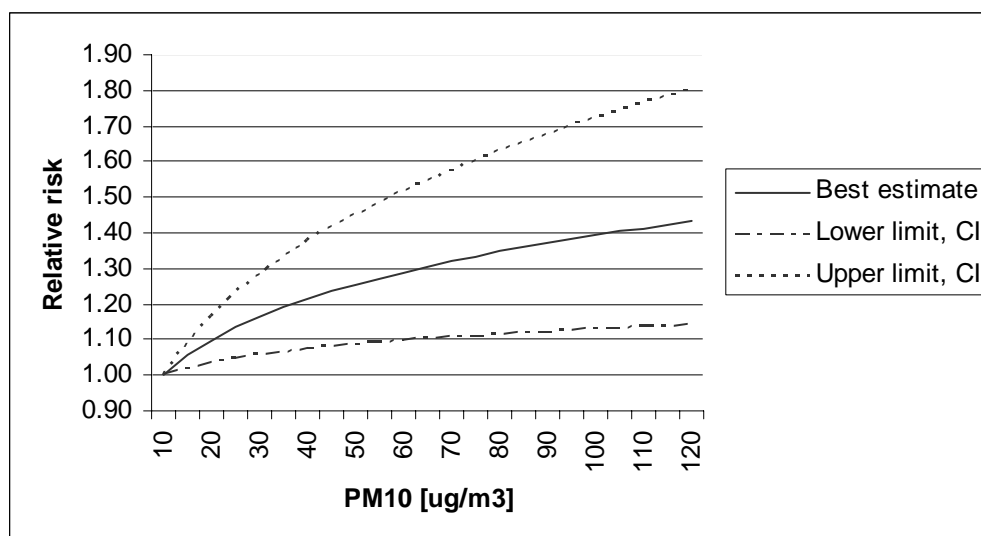
As the exposure-response relationships are expressed as PM<sub>2.5</sub>, but the mean air quality is generally measured as PM<sub>10</sub>, we need a PM<sub>2.5</sub>:PM<sub>10</sub> ratio to apply these relationships. Where available, it is preferred that such a ratio is measured locally. Alternatively, values of 0.65 could be used for developed countries (0.73 in Europe), and 0.5 for developing countries. Section 4.3 addresses this issue in more detail.

<sup>3</sup> The background level is the non-anthropomorphic concentration – that is, what would exist without any man-made air pollution. Typically, this level could be determined by examining monitors at very rural or coastal locations that are not impacted or minimally impacted by pollution sources.

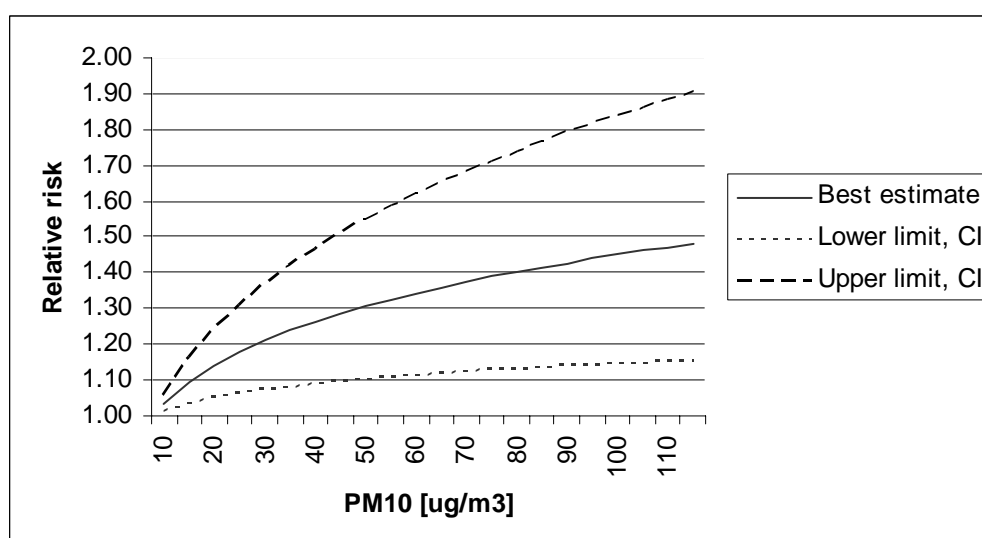
<sup>4</sup> As for equation 1, this formula could also be used to compare current concentrations with target concentrations such as regulatory targets. The excess risk could be calculated accordingly, provided only one exposure level is used. However, for the purpose of this guide, the calculated relative risks will refer to baseline concentrations.

The relative risks are graphically represented for a range of mean annual PM10 levels, and for a background level of PM2.5 = 5  $\mu\text{g}/\text{m}^3$  in Figures 3 to 6, for developed and developing countries, applying different PM2.5:PM10 ratios.

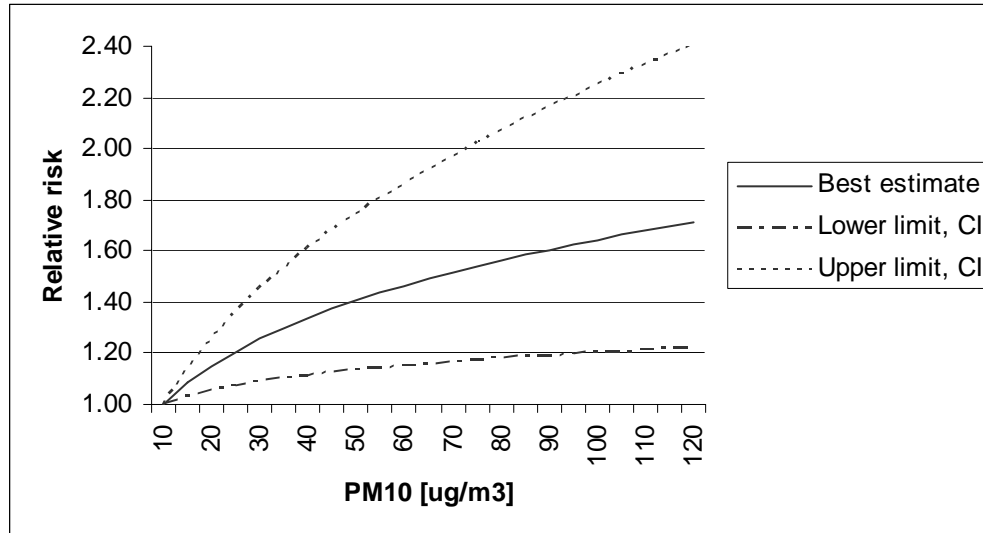
**Figure 3** Recommended relative risks for cardiopulmonary mortality and OAP in adults >30 years, with a PM2.5:PM10 ratio of 0.5 (default for developing countries)  
Based on a background concentration of 5  $\mu\text{g}/\text{m}^3$  PM2.5



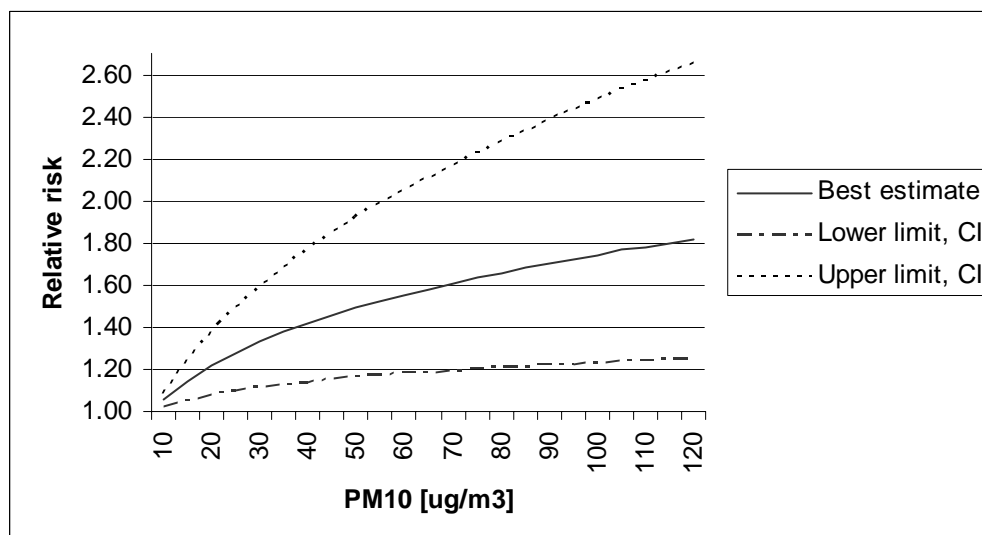
**Figure 4** Recommended relative risks for cardiopulmonary mortality and OAP in adults >30 years, with a PM2.5:PM10 ratio of 0.65 (default for developed countries)  
Based on a background concentration of 5  $\mu\text{g}/\text{m}^3$  PM2.5



**Figure 5** Recommended relative risks for lung cancer related mortality and OAP in adults >30 years, with a PM<sub>2.5</sub>:PM<sub>10</sub> ratio of 0.5 (default for developing countries)  
Based on a background concentration of 5 µg/m<sup>3</sup> PM<sub>2.5</sub>



**Figure 6** Recommended relative risks for lung cancer related mortality and OAP in adults >30 years, with a PM<sub>2.5</sub>:PM<sub>10</sub> ratio of 0.65 (default for developed countries)  
Based on a background concentration of 5 µg/m<sup>3</sup> PM<sub>2.5</sub>



### Alternative relationships

To illustrate the effects of making a different assumption about the shape of the concentration–response function, assume that mortality is linearly related to concentration (rather than a log–linear function). The RR would then be:

$$\begin{aligned} \text{RR} &= \exp(\alpha + \beta X) / \exp(\alpha + \beta X_o) \\ &= \exp[\beta(X - X_o)] \end{aligned} \quad (\text{Equation 4})$$

For the linear model,  $\beta = 0.008933$  (standard error = 0.002907) for cardiopulmonary mortality from long-term PM<sub>2.5</sub> exposure. For lung cancer mortality  $\beta = 0.012673$  (standard error = 0.00426). The form and coefficients of the risk functions for mortality associated with long-term PM exposure are summarized in Table 3.

**Table 3** Recommended and alternative models for estimating relative risk associated with long-term exposure to PM<sub>2.5</sub>

Outcome	Relative risk function <sup>a</sup>	$\beta$ coefficient (lower and upper bound)
Cardiopulmonary mortality (log–linear exposure)*	$\text{RR} = [(X + 1) / (X_o + 1)]^\beta$	0.15515 (0.0562, 0.2541)
Cardiopulmonary mortality (linear exposure)	$\text{RR} = \exp[\beta (X - X_o)]$	0.00893 (0.00322, 0.01464)
Lung cancer mortality (log–linear exposure)*	$\text{RR} = [(X + 1)/(X_o + 1)]^\beta$	0.23218 (0.08563, 0.37873)
Lung cancer mortality (linear exposure)	$\text{RR} = \exp[\beta (X - X_o)]$	0.01267 (0.00432, 0.02102)

<sup>a</sup>X = current PM<sub>2.5</sub> concentration ( $\mu\text{g}/\text{m}^3$ ) and  $X_o$  = target or threshold concentration of PM<sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )

\* recommended relationships

Because of the uncertainty in extrapolating the concentration–response function from the Pope et al. (2002) study to global estimates of the disease burden caused by outdoor air pollution (WHO, 2002), several alternative applications have been analyzed to determine the sensitivity of the estimates (Cohen et al., 2004). In providing estimates of the EBD, it is important to note the potential range of uncertainty. The different assumptions that were considered are detailed below, and vary by:

- the shape of the concentration–response function;
- the assumed background or lowest effect level;
- the assumed highest concentration (“upper truncation”) and relative risk that can be used in the extrapolation (i.e. the highest applicable relative risk);
- the exposure data used in the original estimates.

The concentration–response function can be either log–linear or linear (Equations 2 & 4, respectively). The background level can equal the lowest level in the original study ( $7.5 \mu\text{g}/\text{m}^3$ ), the background concentration of PM<sub>2.5</sub> in a “typical” urban area ( $3 \mu\text{g}/\text{m}^3$ ), or some presumed threshold level or target ambient concentration. Short of data to the contrary, a background concentration of  $3 \mu\text{g}/\text{m}^3$  PM<sub>2.5</sub> should be assumed. The highest applicable relative risk for extrapolation can be equal to the highest concentration in the original data ( $30 \mu\text{g}/\text{m}^3$ ), some arbitrary upper bound ( $50 \mu\text{g}/\text{m}^3$ ), or it can be without bounds. As described above, the exposure data reported in Pope et al. (2002) included either data between 1979–1983, data for 1999 and 2000, or all available years combined. The assumptions used in the estimates of worldwide cardiopulmonary mortality and the resultant sensitivity of the estimates are summarized in Table 4.

**Table 4** Effects of alternative assumptions on estimates for worldwide cardiopulmonary mortality associated with long-term exposure to PM<sub>2.5</sub><sup>a</sup>

Case	Shape of exposure function	Threshold ( $\mu\text{g}/\text{m}^3$ PM <sub>2.5</sub> )	Upper concentration truncation ( $\mu\text{g}/\text{m}^3$ of PM <sub>2.5</sub> )	Exposure data used (years)	Mortality estimates (in thousands)
1*	Log–linear	7.5	none	All	1069
2	Linear	7.5	50	1979–1983	712
3	Linear	7.5	none	1979–1983	783
4	Linear	7.5	30	1979–1983	506
5	Log–linear	7.5	none	1979–1983	794
6	Linear	3.0	50	1979–1983	882
7	Linear	15.0	50	1979–1983	474
8	Linear	7.5	50	All	1132
9	Linear <sup>b</sup>	7.5	50	1979–1983	609

<sup>a</sup> Adapted from WHO (2002) and Cohen et al. (2004).

<sup>b</sup> In this case, the assumed ratio between PM<sub>2.5</sub> and PM<sub>10</sub> is 0.65 for North America, Europe and China, and 0.35 elsewhere. All other cases assume that the ratio is 0.5 throughout the world. Other alternatives to the concentration–response function could include a weighted average (Dockery et al., 1995; Pope et al., 2002), or results from specific sensitivity analyses (Krewski et al., 2000).

\*recommended relationship

#### *Recommended sensitivity analysis for quantifying disease*

Given that available evidence at higher concentrations than  $30 \mu\text{g}/\text{m}^3$  of PM<sub>2.5</sub> is still limited, analysts are encouraged to examine sensitivity of the results using other forms and assumptions. Sensitivity analysis can be conducted by truncating the risks above certain concentrations (e.g.  $50 \mu\text{g}/\text{m}^3$ ), varying the background concentrations, and varying the ratio of PM<sub>2.5</sub> to PM<sub>10</sub>.



### 3.3 Morbidity

Several dozen epidemiological studies have reported associations between various measures of PM and a range of morbidity outcomes, including:

- hospitalization for cardiovascular or respiratory disease
- emergency room and urgent care visits
- asthma exacerbation
- acute and chronic bronchitis
- restrictions in activity
- work loss
- school absenteeism
- respiratory symptoms
- decreased lung function.

Most of these studies have been conducted in cities in North America, with a few conducted in Europe and very few outside of these two areas. Therefore, compared to the mortality findings, there is a greater degree of uncertainty when morbidity findings are extrapolated to developing countries, since estimation requires both a concentration–response function and a baseline incidence rate. As additional evidence is compiled on these effects for regions outside of North American and Western Europe, we can become more confident about applying these estimates to other economically developing regions. Below, we provide a brief review of some of the effects, focusing on those that have been reported in developing countries. The studies support the idea that PM exposure affects mortality, and that the data can be extrapolated to other regions of the world.

Typically, these studies have used one of two analytical methods. The first method, time-series analysis of daily count data, is similar to that used to determine mortality related to short-term PM exposure. Specifically, daily counts of an endpoint, such as hospitalization for cardiovascular disease, are examined in response to single-day and multi-day average concentrations of PM. As in the case of mortality, these models also control for potential confounders, such as season, meteorology, day of week, and time trends. The second approach involves the use of panel data, in which a cohort of subjects (e.g. asthmatic children) is followed prospectively over a period of several months or years, while daily health outcomes and pollution measures are recorded and compared. Below, we review some representative studies for some of these health endpoints.

#### 3.3.1 Hospital admissions

Associations between daily concentrations of PM<sub>10</sub> and daily hospital admissions for cardiovascular and respiratory disease have been reported for close to 100 cities in the USA, Canada and Europe (USEPA, 1996). Several multi-city studies have also examined the effects of short-term exposure to PM<sub>10</sub> on hospital counts (Schwartz, 1999; Samet et al., 2000a; Zanobetti, Schwartz & Dockery, 2000). In eight USA cities between 1988–1990, for example, statistically significant associations were found between PM<sub>10</sub>

and daily hospital admissions for cardiovascular disease (ICD9 codes 390–429) in individuals older than 65 years (Schwartz, 1999). The associations were significant for five of the cities individually, and for the effect estimate pooled across all eight cities. Across all the cities, a  $10 \mu\text{g}/\text{m}^3$  change in PM<sub>10</sub> was associated with about a 1% change in hospitalization for cardiovascular disease. Associations between PM and total cardiovascular disease, or subsets of cardiovascular disease (e.g. heart failure or ischemic heart disease), have been reported for a range of cities including: Chicago (Morris & Naumova, 1998), Edinburgh (Prescott et al., 1998), Hong Kong (Wong et al., 1999), London (Atkinson et al., 1999), Sydney (Morgan et al., 1998) and Toronto (Burnett et al., 1997). In Bangkok, Thailand, in one of the few hospital studies conducted in a developing country, Chestnut et al. (1998) reported an effect for cardiovascular hospital admissions for all ages and for those older than 50 years. This effect is slightly higher than, but generally consistent with, the results of many studies in North American and Europe. Besides the studies on hospitalization, epidemiological studies also support the idea that PM is associated with cardiovascular endpoints, such as changes in heart rate, heart rate variability, arrhythmia, heart attacks, and blood viscosity.

Time-series analyses also provide evidence of associations between daily PM and hospitalization for respiratory diseases. Associations have also been reported between PM<sub>10</sub> and visits to the emergency department or urgent care, that may or may not result in hospital admissions. In Santa Clara County, California, for example, Lipsett, Hurley & Ostro (1997) reported associations between PM<sub>10</sub> and emergency room visits for asthma during the winters, particularly on colder days. Several other studies have also reported effects among children. In Atlanta, Georgia during the summers of 1993–1995, PM<sub>10</sub> levels were associated with an increase in paediatric emergency room visits for asthma (Tolbert et al., 2000). Similarly, doctors' house calls for children's asthma in Paris, France between 1991–1995 were associated with levels of black smoke, another measure of PM (Medina et al., 1997); and in Santiago, Chile, associations between PM<sub>10</sub> and urgent care visits for lower respiratory symptoms were reported for children under age 2 years and for children aged 2–14 years (Ostro et al., 1999b).

Although there is adequate evidence for an effect of PM on hospital admissions, for several reasons it is difficult to develop estimates of an attributable risk, including: the dearth of evidence for non-industrialized nations; the difficulty in determining the baseline level of hospital admissions to use in the calculations; and the difficulty in relating hospital admissions to the ultimate disease burden. Therefore, a concentration–response function for this endpoint is not provided.

### ***3.3.2 Exacerbation of asthma***

In general, the study of air pollution and asthma is analytically challenging because the disease and its triggers are complex. Many studies have examined the effects of PM exposure on symptoms and lung function changes in asthmatics, typically by following a panel of subjects who record daily health outcomes over several months. The measured outcomes included specific symptoms (e.g. cough, shortness of breath, wheeze, chest tightness, phlegm), medication use, and lung function changes such as peak expiratory flow rate and forced expiratory volume. Concurrent air pollution was recorded, as well

as potential confounders that changed on a daily basis and that might be associated with the health outcome. The potential confounders included weather factors, exposure to environmental tobacco smoke or wood smoke, activity patterns, time spent outdoors, use of air conditioning and gas stoves, and day of week. Even though some of the studies combined individuals with different levels of asthma severity and medication use, or combined asthmatics and non-asthmatics, evidence for an effect of PM on asthma has emerged over the last several years.

In Los Angeles, California, PM<sub>10</sub> exposure was associated with daily reporting of cough, shortness of breath and wheeze among African-American children with current, physician-diagnosed asthma (Ostro et al., 2001). Also in southern California, PM<sub>10</sub> exposure from August to October 1995 was associated with asthma symptoms (cough, wheeze, sputum production, shortness of breath, or chest tightness) in a panel of 24 asthmatics aged 9–17 years (Delfino et al., 1998). The largest effects of PM<sub>10</sub> were in children not on anti-inflammatory medication. In a study of 75 physician-diagnosed asthmatic children, 6–13 years old, in Port Alberni, British Columbia, PM<sub>10</sub> exposure was associated with increases in both cough and phlegm, and decreased peak flow (Vedal et al., 1998). Stratified analysis indicated effects only among asthmatic children. Outside of the USA and Canada, studies have reported associations between PM and asthma symptoms in Amsterdam (Gielen et al., 1997), Erfurt (Peters et al., 1997) and Mexico City (Romieu et al., 1996).

Overall, the exacerbating effects of PM on asthma were not as consistent as those found with hospitalizations for cardiovascular or respiratory disease. This is likely due to the difficulty of estimating the impact of air pollution on a complex and multidimensional disease such as asthma. Nevertheless, several well-conducted prospective cohort studies, often involving over 100 children with asthma, have found associations between PM<sub>10</sub> and a range of asthma symptoms or medication use. Most of the studies have controlled for potentially confounding factors such as weather and pollutants (e.g. ozone). As with hospital admissions, although there is good evidence of an association between PM exposure and asthma exacerbation, it is difficult to estimate the ultimate burden of disease related to this endpoint. Asthma definitions and baseline rates vary widely among countries and regions, and there have been only a few studies conducted outside of the industrialized nations. Therefore, a concentration–response function is not provided for this endpoint.

### **3.3.3 Respiratory symptoms**

The effects of air pollution on the general population have been examined using panel studies and other analytical study designs. The outcomes in these studies included: upper and lower respiratory symptoms (in aggregate form and separated out by specific symptoms); changes in lung function; restrictions in activity owing to respiratory illness; school absenteeism; and work loss. Although these outcomes are not as serious as mortality and hospitalization, they may have an important effect on public health since they impact a greater proportion of the population.

A study of the daily effects of air pollution on 321 nonsmoking adults in three cities in Southern California reported associations between lower respiratory symptoms and PM (Ostro et al., 1993). It is reasonable to assume that the same effects would occur in children. Indeed, a study of elementary school children in six eastern USA cities between April and August found associations between both PM<sub>10</sub> and PM<sub>2.5</sub>, and the incidences of lower respiratory symptoms, cough and, to a lesser extent, upper respiratory symptoms (Schwartz et al., 1994). Schwartz & Neas (2000) also reported associations between PM and lower respiratory symptoms (any day with at least two of: cough, phlegm, chest pain or wheeze) in a panel of children in six USA cities. Similarly, a study in the Netherlands of children with or without asthma, chronic cough or wheeze found associations between PM<sub>10</sub> and lower respiratory symptoms among children with prior chronic disease (Van der Zee et al., 1999). The associations with chronic respiratory disease were seen for children in both urban and rural areas, but the effects were stronger for those in urban areas. In the urban areas, PM<sub>10</sub> averaged 48, 37 and 29  $\mu\text{g}/\text{m}^3$  during the three winters that were studied, versus 35, 35 and 24  $\mu\text{g}/\text{m}^3$  in the rural areas. Among children without chronic respiratory disease, in contrast, no association was found between PM<sub>10</sub> and symptoms. In one of the few panel studies conducted in a developing country, associations were reported between PM<sub>10</sub> and both upper and lower respiratory symptoms in school nurses, schoolchildren and adults in Bangkok, Thailand (Vichit-Vadakan et al., 2001).

#### **3.3.4 Other health outcomes**

Besides respiratory symptoms and changes in lung function, other minor health outcomes have been reported for the general population. An analysis of data from six years of the annual Health Interview Survey conducted by the National Center for Health Statistics, and which included 49 metropolitan areas, found that increased levels of PM<sub>2.5</sub> (estimated from airport visibility) were associated with restricted activity in adults, including days spent in bed, days missed from work, or days partially restricted due to illness (Ostro, 1987). Using the same data, Ostro & Rothschild (1989) also found an association between PM<sub>2.5</sub> levels and both respiratory-related restrictions in activity and minor restrictions (days when activity was restricted but did not result in work loss).

## 4. Exposure assessment

### 4.1 Using fixed-site monitors

The health effects of PM<sub>10</sub> and PM<sub>2.5</sub> exposure should ideally be quantified using several years of data for annual concentrations of PM<sub>10</sub> and PM<sub>2.5</sub>. Since the concentration–response functions derived from epidemiological studies used existing fixed-site, population-oriented monitors located throughout the metropolitan area of the study population, these monitors should be the basis of the extrapolation as well. In principle, the monitoring data used to calculate the average annual PM concentrations should be collected throughout the year, for several years, to reduce bias owing to seasonal fluctuations or to a non-representative year. High-quality measurements of PM concentrations from all of the monitors in the metropolitan area can be averaged to develop a single estimate. Care should be taken that the monitors used are not unduly influenced by a single large source of pollution (i.e. a power plant, factory or highway); rather, the monitors should reflect exposures over a wide area. Also, it is likely that PM data will be available only for larger cities, and health effects will therefore be calculated only for the urban population, though residents of rural areas are also exposed to PM from biomass fuels, open burning and regional haze. One solution is to assume that PM<sub>2.5</sub> concentrations are low in non-monitored areas (but above background levels of approximately 3 µg/m<sup>3</sup>) and vary between 5–10 µg/m<sup>3</sup>, depending on sources of pollution such as traffic, open burning, and local stationary sources.

### 4.2 Using model-based estimates to estimate burden of disease

If no data are available for the cities or countries under study, it is possible to use estimates of annual PM<sub>10</sub> and PM<sub>2.5</sub> concentrations recently developed by the World Bank and used in the global estimates (Pandey et al., 2004). Our description of the model borrows heavily from that document. To provide ambient PM concentrations for all 14 WHO subregions, we used an econometric model (AMPM CON Model) that incorporated available PM measurements at population-oriented monitors. The model predicts PM<sub>10</sub> concentrations in urban residential areas as a function of factors such as fuel mix, level of economic development, demographics, and geographical and meteorological variables that might impact pollution transport. The primary determinants of city-specific ambient PM concentrations in the model are shown in Box 1.

**Box 1** Model inputs for determining city-specific PM10 concentrations<sup>a</sup>

1. **Energy consumption.** The model separately includes per capita consumption of six categories of energy (coal, oil, natural gas, nuclear energy, hydroelectric energy, and combustible renewables and wastes). These categories account for all energy consumed in each country. In addition, the model includes data on per capita gasoline and diesel used in the transportation sector. The separate inclusion of each energy type accounts for variations in emission factors, economic activity, the intensity of fuel use, and traffic-related emissions in different countries.
2. **Atmospheric and geographical factors.** The model includes atmospheric and geographical factors for each city to account for the dispersion and transport capacity of polluting emissions. The model includes measures of the annual average 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 the frequency of wet days, sunny days and frosty days. 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 are also included in the model: the distance from the city centre to the nearest point on the coastline (calculated using a geographical information system) and the elevation of the city (derived from a global digital elevation model).
3. **City and national population density.** These variables provide measures of the scale and intensity of the pollution problem in each city.
4. **Local urban population density.** The local population density in the vicinity of each city provides a measure of the intensity of pollution and density of pollution sources.
5. **Local intensity of economic activity.** A proxy was created for the intensity of economic activity within each city (local gross domestic product per square kilometre), from the product of the national per capita gross domestic product and the local population density in the vicinity of each city.
6. **National income per capita.** This variable is used to capture the following country-level indicators: valuation of environmental quality, 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 one-year lag of the average of the previous three years (World Bank, 2002).
7. **Time trends.** The model includes time trend variables to allow for trends to vary across countries.
8. **Binary variable for each country.** The estimation equation includes country-specific binary variables to control for economic, social and the other county-specific factors that are not captured by the other explanatory variables.

<sup>a</sup> Adapted from Pandey et al. (2004).

The model is based on all reliable PM10 and TSP measurements from population-oriented monitoring stations at 512 unique locations in 304 cities and 55 countries for the period 1985–1999. In all, 1997 time-location data points were available from the cities worldwide. The fit of the multiple regression, ordinary least squares model was about  $R^2 = 0.88$ , indicating a good fit. F-tests revealed that all of the eight major factors outlined above added significant explanatory power to the regression. Other diagnostic techniques and heuristic criteria were employed to ensure the goodness of fit of the model, and that the predictions of the model for all 3211 cities in the world with populations over 100 000 were reasonable.

Initially, the AMPM CON model was used to generate estimates of PM10 concentrations in all world cities with populations greater than 100 000, as well as in national capitals. The range of uncertainty of the subregion-specific means was estimated using a bootstrap technique, in which the model is re-estimated many times (200 trials) using a random,

repeated sample of the observations used to estimate the model. Further details about the model and estimation results are available online at the World Bank website.

The major assumptions involved in the model-based estimates of exposure include:

- the measured PM<sub>10</sub> levels at the population-oriented monitor sites are generally representative of exposure;
- within a given country, certain national estimates (i.e. fuel use, gross domestic product and gross national product) serve as reasonable proxies for city-specific estimates of the corresponding variables.

### 4.3 The PM<sub>2.5</sub>/PM<sub>10</sub> ratio

#### *Values from the literature*

Since some of the health outcomes are based on PM<sub>2.5</sub>, rather than PM<sub>10</sub>, the model PM<sub>10</sub> estimates can be converted to estimates of PM<sub>2.5</sub> using the available information on the ratio of PM<sub>2.5</sub> to PM<sub>10</sub>. For many urban areas in industrialized nations, PM<sub>2.5</sub> is 0.50–0.65 of PM<sub>10</sub> (USEPA, 1996). Evidence from data outside of the industrialized nations suggests a similar range for the ratio. For example, a recent study from China reported ratios in the range of 0.51–0.72 for four urban locations (Quian et al., 2001). However, in areas impacted 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, since these areas will have a greater proportion of the PM<sub>10</sub> in the coarse size range of 2.5–10 µm. For example, in the Coachella Valley, an arid, desert region of southern California, a PM<sub>2.5</sub> to PM<sub>10</sub> ratio of 0.35 was reported (Ostro et al., 1999). Therefore, for cities, countries or regions likely to have a high proportion of crustal material, a lower ratio should be assumed. For the global analysis of disease burden due to OAP (WHO, 2002), a ratio of 0.5 was assumed for the base case, but the sensitivity of the results to this assumption was explored. Specifically, a higher scaling factor of 0.65 was used for cities in subregions AMR A, EUR A, EUR B, EUR C, and WPR A (which included the USA, Canada, all of Europe, Japan, Singapore, Australia and New Zealand), since it was assumed there were relatively more combustion-related particles. A scaling factor of 0.35 was used for cities in all other subregions.

#### *Recommended ratios for national or local studies*

The best estimation of the PM<sub>2.5</sub>/PM<sub>10</sub> ratio would come from a local study, as it would capture all the local conditions and sources of pollution that condition that ratio. In the absence of a local measurement of the ratio, a value of 0.65 could be assumed for developed countries, and 0.5 for developing countries. For Europe, a mean of 0.73 has been reported and may be more adequate (Second Position Paper on Particulate Matter. Final Draft. April 2004)<sup>5</sup>. Alternative values could be explored as a sensitivity analysis (see Section 6).

<sup>5</sup> [http://europa.eu.int/comm/environment/air/cafe/pdf/working\\_groups/2nd\\_position\\_paper\\_pm.pdf](http://europa.eu.int/comm/environment/air/cafe/pdf/working_groups/2nd_position_paper_pm.pdf)

## 5. Calculating the disease burden

The relative risk for each included health outcome can be calculated using the risk functions in Table 1. Once the relative risks have all been determined, the AF (or impact fraction, IF) of health effects from air pollution for the exposed population can be calculated by:

$$AF = \frac{\sum P_i RR_i - 1}{\sum P_i RR_i} \quad (Equation 5)$$

where:

$P_i$  = the proportion of the population at exposure category “i”, including the unexposed (i.e.  $\sum P_i RR_i$  becomes  $(P_1 RR_1 + P_2 RR_2 + \dots + P_{unexposed} \times 1)$ ).

$RR_i$  = the relative risk at exposure category “i”, compared to the reference level.

Equation 5 takes into account various population groups exposed at different levels of pollutants, for example of the population of various cities in a country. In the case of the population of only one city with only one exposure level, this formula simplifies to Equation 6 (i.e.  $P_i$  becomes 1, as the total population is exposed, and only one relative risk value would apply):

$$AF = \frac{RR - 1}{RR} \quad (Equation 6)$$

To estimate the impact of changing the exposure from one distribution to another, for example through a public-health intervention, a more general formula than Equation 5 could be used (Equation 7). This formula can be used to estimate the fraction of the disease burden attributable to the risk factor, as compared to some “alternative” or counterfactual level, which might be the minimum that can be feasibly achieved in a given time frame. For example, the target concentration could be some regulatory target such as 20 µg/m<sup>3</sup> PM<sub>10</sub>, the proposed European Union Target Limit Value for 2010. For further details on the various formulas see Chapter 4 of Volume 1 "Introduction and methods" of this series (Prüss-Üstün et al., 2003).

$$IF = \frac{\sum P_i RR_i - \sum P'_i RR_i}{\sum P_i RR_i} \quad (Equation 7)$$

where:

$P_i$  = the proportion of the population at exposure category “i”.

$P'_i$  = the proportion of the population in exposure category “i” after an intervention or other change.

$RR_i$  = the relative risk at exposure category “i” compared to the reference level.



To calculate the expected number of mortality cases due to air pollution (E), the AF is applied to the total number of deaths:

$$E = AF \times B \times P \quad (\text{Equation 8})$$

where:

- E = the expected number of deaths due to outdoor air pollution.
- B = the population incidence of the given health effect (i.e. deaths per 1000 people).
- P = the relevant exposed population for the health effect.

The AF is based on relative risks derived from epidemiological studies and from the change in PM being evaluated. B is obtained or approximated from available health statistics, and P is obtained from census or other data for the area under study. Possible sources for health statistics are also addressed in Prüss-Üstün et al. (2003).

We have now seen how to estimate the number of expected (or attributable) cases or deaths caused by outdoor air pollution. As discussed previously, not all of the listed health outcomes can be reliably converted into DALY estimates. In case information in DALYs is available, the same procedure can followed for estimating the AF of each health outcome, which is then applied to the total disease burden of each outcome, measured in DALYs. Information on disease burdens can be obtained from several sources: a national burden of disease study; WHO “prior estimates” of specific disease burdens at the national level, obtainable on request from a relevant national organization (e.g. a ministry of health); your own calculations (for example, if your study area is a city or region, rather than a country). To make this conversion, you will need the age distribution of deaths to estimate YLL, and the age distribution of onset of disease, duration of disease and severity weight to estimate YLD. DALYs are then calculated as the sum of YLL and YLD. This can be done using a DALY calculation template (available at [www.who.int/evidence/](http://www.who.int/evidence/) under “Burden of Disease project”, “National Burden of Disease Manual” and “other files”). Additional information can be found in the introductory chapters to this EBD series (Prüss-Üstün et al., 2003).

## **6. Uncertainties**

Clearly, there are many uncertainties involved in estimating the health effects associated with outdoor air pollution. Over time, some of these will be reduced as new research is conducted. However, some uncertainty will be inherent in any estimate. For many of these issues, the bias could be either positive or negative. Below, we briefly discuss some of the major uncertainties.

A primary uncertainty is the choice of the specific studies and concentration–response functions used in this risk assessment, and their applicability to other regions of the world. Fortunately, studies of short-term exposure–mortality estimates have been replicated in many cities throughout the world, including several cities in countries with developing economies. Therefore, the application of mortality effects is reasonable. However, uncertainty remains about the actual magnitude of the effect and the appropriate confidence interval. Based on available information, we have attempted to provide a range for these estimates that will likely incorporate the “true” value. For long-term exposure effects on mortality, we have demonstrated the sensitivity of the estimates to the underlying assumptions.

A second major uncertainty relates to the general shape of the concentration–response function and whether there is a threshold concentration. This was discussed in detail earlier, with the conclusion that there was no evidence for a threshold in the studies that have explicitly examined the issue. In addition, studies have demonstrated health effects at very low concentrations of PM. Most studies also support the idea that there is a linear relationship between relative risk and ambient concentration, within the range of exposures examined. However, for long-term exposure-related mortality, a log–linear function is more plausible and should be used to generate effect estimates for cities with very high concentrations of pollution. For both short-term and long-term exposures, one can apply a threshold to determine the impact on the overall estimates.

A third uncertainty involves co-pollutants. Specifically, it is likely that some of the estimated health effects include the effects of both PM and other correlated pollutants. Since many of the pollutants come from a common source (e.g. fuel combustion), the use of PM as an index for the mix of pollutants is reasonable but conservative. Exposure to other pollutants, not related spatially or temporally with PM, has demonstrable health effects that are not included in our estimates.

A fourth source of uncertainty derives from the fact that estimates are provided for only a subset of adverse outcomes. For example, estimates of the effects of PM on asthma and hospitalizations have not been generated, though there is substantial evidence for such effects.

Fifth, there is also uncertainty concerning the baseline rates of the considered health outcome in the studied population. Often, one will have to assume a baseline incidence level for the city or country of interest. In addition, the incidence will change over time as health habits, income and other factors change.

A sixth uncertainty concerns the exposure assessment, specifically whether the existing monitoring network and model-based estimates of PM<sub>10</sub> and PM<sub>2.5</sub> were representative of the general population. In addition, we have proposed a given ratio of PM<sub>2.5</sub> to PM<sub>10</sub>. Clearly, variations in the measurement of current concentrations or the share of fine particles will have an impact on city-specific and region-specific estimates.

## **7. An example application of the methodology**

To demonstrate the methodology for estimating the effects of outdoor air pollution, we use data from Bangkok, Thailand. Bangkok, a warm and humid city, is situated on a relatively flat plain and has a population of approximately six million (with up to 10 million in the metropolitan area). Because of the relatively low proportion of roads to surface area, the city has difficulty supporting the large number of automobiles (approximately four million) and motorcycles used in the city, many with minimal pollution control devices. The ubiquitous traffic jams contribute a large share of the PM<sub>10</sub> coming from incomplete combustion of fossil fuels used by the transportation sector.

Our estimates for the annual average PM<sub>10</sub> were derived from continuous measurements taken between 1996–2001 from seven monitors throughout the metropolitan area. An eighth monitor, located at Dindang, was not included since it is a roadside monitor and was a clear outlier from the others. The annual average for PM<sub>10</sub> for the six years of data was 70.28  $\mu\text{g}/\text{m}^3$ . As suggested above, as a default we assumed that 50% of the PM<sub>10</sub> consists of fine particles or PM<sub>2.5</sub>. However, based on sparse data from three months of monitoring of both PM<sub>2.5</sub> and PM<sub>10</sub> in Bangkok, the ratio may be 0.6 or higher (Vichit-Vadakan et al., 2001). Given the large role of transportation as a source of the PM mix in Bangkok, it is likely that the ratio may be closer to the higher ratio of 0.65 reported in the USA and elsewhere. Thus, this ratio is used in a sensitivity analysis of the estimated effects.

Mortality data from the Thailand Ministry of Health for 1998–2001 were used to determine existing mortality rates. We assumed a total population of six million, based on data from the Thailand Ministry of Health. In the absence of locally specific data, we used a regional value for the fraction of acute respiratory disease mortality versus all-cause mortality in children under 5 years of 7.7% (WHO 2003). These data were used to develop estimates for the outcomes all-cause mortality (all ages), mortality from acute respiratory infections (children under five years), and long-term exposure and cardiopulmonary mortality and lung cancer for those older than 30 years. The baseline mortality rates used in the application, as well as the results for Bangkok, are given in Table 5. The  $\beta$ -coefficients used in the models are detailed in Table 1.

**Table 5** Annual number of deaths from outdoor air pollution for Bangkok according to the proposed method<sup>a</sup>

Outcome and Exposure metric	Subgroup	Mortality rate of disease group [deaths/person/year]	Population	Deaths from exposure to outdoor air pollution [deaths/year]	
				Best estimate	95% CI
All-cause mortality and short-term exposure to PM10 <sup>b</sup>	All ages	0.00558	6 000 000	1580	1 200–1 960
Cardiopulmonary mortality and long-term exposure to PM2.5 <sup>c</sup>	Age >30 years	0.0023	3 000 000	2 000	800–3 000
Lung cancer and long-term exposure to PM2.5 <sup>c</sup>	Age >30 years	0.00014	3 000 000	120	50–180
Respiratory mortality and short-term exposure to PM10 <sup>b</sup>	Age <5 years	0.00334	350 000	8 - 106	8 - 106

<sup>a</sup> Population and health data source: Thai Ministry of Health.

<sup>b</sup> Assumes background concentration of PM10 = 10 µg/m<sup>3</sup>.

<sup>c</sup> Assumes background concentration of PM2.5 = 3 µg/m<sup>3</sup>.

Recall that the mortality effects of an increase of short-term exposure by 10 µg/m<sup>3</sup> PM10 are assumed to have an increase in risk of about 0.6 to 1.0%, with a likely best estimate of 0.8% (example of calculation below by introducing  $\beta = 0.0008$  into Equation 1). Since the concentration–response function is practically linear, we can assume that the annual change in PM10 was derived from 365 daily changes of a similar magnitude. For this endpoint, the exposure population was assumed to be six million people, with a baseline mortality rate of 0.00558 deaths per person per year. We estimated the attributable deaths of outdoor air pollution by using the background concentration of 10 µg/m<sup>3</sup> PM10 as the baseline value, and the targeted avoidable deaths by using the proposed European Union Target Limit Value for 2010 of 20 µg/m<sup>3</sup> PM10 as the baseline. From Equation 1, the relative risk for estimating the attributable burden at the measured concentration of 70.28 µg/m<sup>3</sup> is:

$$RR = \exp[\beta(X - X_0)] = \exp[0.0008 \times (70.28 - 10)] = 1.0494$$

Using Equation 6 (equation for one city against the background value), the AF of deaths for a change of air quality to 10 µg/m<sup>3</sup>, or the attributable fraction of deaths to outdoor air pollution with the assumed background value is:

$$AF = (1.0494 - 1) / 1.0494 = 0.0471$$

The expected total number of cases of premature mortality from short-term exposure to PM10 was calculated using Equation 8. Therefore, the impact of the current ambient level of PM10 (70.28 µg/m<sup>3</sup>), relative to a background concentration of 10 µg/m<sup>3</sup>, is:

$E = 0.0471 \times 0.00558 \times 6\,000\,000) \approx 1580$  cases of premature deaths per year, with the lower and upper bound estimates of 1200 and 1960, respectively.

If we assume a “target” concentration of  $20\ \mu\text{g}/\text{m}^3$  PM10 as annual mean, the estimated effect is 1320 cases of premature deaths per year (lower and upper bounds of 1000 and 1640, respectively) that could be avoided if the air pollution level were reduced to  $20\ \mu\text{g}/\text{m}^3$ .

This same methodology can be used for respiratory mortality in children under 5 years of age associated with PM10, since a linear exposure model is used (Equation 1), and  $\beta = 0.0016$  (95% CI = 0.00034–0.0030; Table 1):

$$RR = \exp[0.0016 \times (70.28 - 10)] = 1.10.$$

From Equation 6:

$$AF = (1.10 - 1) / 1.10 = 0.091.$$

The total number of children under age five years amounts to 350 000, with a mortality rate of 0.00334 (both numbers based on data from the Thai Ministry of Health), which results in 1169 deaths per year. If we apply the regional ratio of acute respiratory infections (ARI) of 7.7%, the number of deaths due to ARI would amount to  $0.0077 \times 1169 = 90$  deaths from ARI per year. If we apply the calculated attributable fraction of 0.091 to ARI and to all-cause deaths we would obtain 8 and 106 deaths per year, respectively. This range is quite large, and the reality is likely to lie in between. As one of the original studies (Table 2), however, had been performed in Bangkok, it is likely that in this case the reality is closer to the upper boundary of 106 deaths rather than the lower boundary proposed.

To estimate the mortality effects of long-term exposure to PM2.5, we used concentration–response functions from Pope et al. (2002) (see Section 3.2), a log–linear function of exposure, all of the exposure data (i.e. for the years 1979–1983 and 1999–2000), and a background level of  $3\ \mu\text{g}/\text{m}^3$  of PM2.5 (likely background concentration). The ambient concentration of PM2.5 was estimated to be  $35.1\ \mu\text{g}/\text{m}^3$  ( $70.28\ \mu\text{g}/\text{m}^3 \times 0.5$ ). This was applied to the three million people in Bangkok older than age 30 years, with a cardiopulmonary mortality rate of 0.0023.

From Equation 3 or Table 1:

$$RR = [(35.1 + 1) / (3 + 1)]^{0.1551} = 1.407.$$

And from Equation 6:

$$AF = 0.407 / 1.407 = 0.289.$$

Inserting these values into Equation 8:

$$E = 3\,000\,000 \times 0.0023 \times 0.289 = 1995.$$

We therefore estimate that there would be 1995 deaths from cardiopulmonary causes per year (95% CI = 800–3000) from long-term exposure to PM<sub>2.5</sub>. The long-term estimate should not be added to the estimate for short-term exposure, since this would double-count a portion, if not all, of the short-term cases. Rather, total cases of premature mortality are best approximated by applying the concentration–response function to long-term exposures only. For this example, estimated mortality due to short-term exposure is very close to mortality estimated from long-term exposure. Usually, effects of long-term exposure are several times higher. The roughly equivalent effect for Bangkok is due to the relatively small proportion of the population above age 30 years (50%) and the large difference in baseline mortality rates for all-cause versus cardiopulmonary mortality.

Since the effects of long-term PM exposure require several assumptions, we explored the sensitivity of the results for cardiovascular mortality to these assumptions. Table 6 provides a sensitivity analysis of the result for long-term PM exposure, using alternative estimates for: the shape of the concentration–response function (linear versus non-linear exposure function); the baseline concentration (3, 7.5 or 20 µg/m<sup>3</sup>); the ratio of PM<sub>2.5</sub> to PM<sub>10</sub> (0.50 or 0.65).

**Table 6** Sensitivity analysis of cardiopulmonary mortality related to long-term exposure, Bangkok, Thailand<sup>a</sup>

Case	Shape of exposure function	Background concentration [µg/m <sup>3</sup> PM <sub>2.5</sub> ]	PM <sub>2.5</sub> /PM <sub>10</sub> ratio	Relative risk	Attributable fraction	Annual number of deaths
1	Log-linear	7.5	0.5	1.25	0.201	1 388
2	Log-linear	3	0.5	1.41	0.289	1 996
3	Log-linear	7.5	0.65	1.30	0.232	1 602
4	Log-linear	3	0.65	1.46	0.317	2 187
5	Log-linear	20	0.65	1.13	0.117	804
6	Linear	7.5	0.5	1.28	0.219	1 509
7	Linear	7.5	0.65	1.41	0.289	1 994
8	Linear	3	0.65	1.46	0.317	2 187

<sup>a</sup> Data source: Thailand Ministry of Health and WHO mortality data for subregion SEAR B. For Bangkok, the subgroup of adults older than age 30 years is three million, with a baseline annual mortality rate of 0.0023. The concentration–response function is based on models using all available exposure data and the resultant relative risk is applied to the full range of concentrations, without an upper truncation.

The results of this sensitivity analysis show that by introducing different input parameters for the baseline concentration and for the PM<sub>2.5</sub>/PM<sub>10</sub> ratio, as well as a different concentration–response function, the number of expected cases of cardiopulmonary

mortality remained within the estimates calculated on the basis of the confidence intervals provided by the concentration–response function. For example, since much of the PM in Bangkok is likely to be fine particles, it made sense to assume the higher value of 0.65 for the PM<sub>2.5</sub>/PM<sub>10</sub> ratio, which generated a central estimate of 2187 deaths per year (95% CI = 890–3200). Using a similar methodology for lung cancer mortality (with, for example, 0.5 for the PM<sub>2.5</sub>/PM<sub>10</sub> ratio and a background concentration of PM<sub>2.5</sub> of 7.5 µg/m<sup>3</sup>), we obtained an estimate of 120 premature deaths per year (95% CI = 50–180).



## 8. Policy actions to reduce the burden

Quantifying the health risks associated with exposure to air pollution can be an important guide for policy-makers. The estimates will indicate the magnitude of the problem and provide the necessary perspective, so that air pollution control can be prioritised relative to other interventions that improve public health. Since many policy-makers are not aware of the array of health effects associated with exposure to outdoor air pollution, this quantification can be an effective educational tool. Creating an awareness of the health risks associated with air pollution is a crucial first step in developing successful control strategies. The quantification of risks will also provide an indication of the level of effort that is necessary in a given city, region, or control strategy.

Since PM comes from a variety of sources, the appropriate control strategies for PM will depend on the study area. Some particles are generated directly from sources such as cars, buses, trucks and smokestacks. In other cases, gases such as sulfur oxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>) and volatile organic compounds (VOC) interact with other compounds in the air to form particulate matter. “Coarse” particles are larger than 2.5 µm and generally come from sources such as vehicles travelling on unpaved roads, materials handling, and crushing and grinding operations such as cement manufacturing. “Fine” particles are less than 2.5 µm in diameter and result from fuel combustion in motor vehicles, power plants and industrial facilities, residential fireplaces, woodstoves, wildfires, burning of biomass and forest burning. Fine particles can also be formed in the atmosphere from gases such as SO<sub>2</sub>, NO<sub>2</sub>, and VOCs.

Control of PM can involve both institutional strategies mandated by local or federal governments, and individual changes in behaviour on the part of the impacted population. Examples of the former include putting control devices on motor vehicles, and upgrading combustion technology, especially for diesel engines and stationary sources such as power plants, industrial boilers and residential cooking and heating. Other activities may be regulated, such as the use of two-stroke engines, open burning of wastes and the uncontrolled burning of forests and agricultural fields.

Government can also promote the use of clean, renewable energy sources, such as solar and wind-powered energy, and can encourage movement away from the use of dirtier fuels such as coal. With regards to transportation, successful long-term air pollution control strategies increasingly focus on viable systems that provide an alternative to cars and diesel buses, including: rail, electric or alternative fuel-powered buses, and cycling/walking networks. Land use strategies that emphasize compact urban design around public transport and/or pedestrian and cycle networks can help to reduce travel distances and the need to travel by car. Along with reducing vehicle emissions, such strategies may yield many other important co-benefits for health in traffic injury prevention, noise reduction, creation of spaces for exercise and recreation, etc. Longer-term control strategies may be associated with transportation and land use planning. Finally, individuals may take the initiative in reducing pollution by opting to use mass transit or non-motorized transport, by conserving energy and by using appliances with cleaner technologies.

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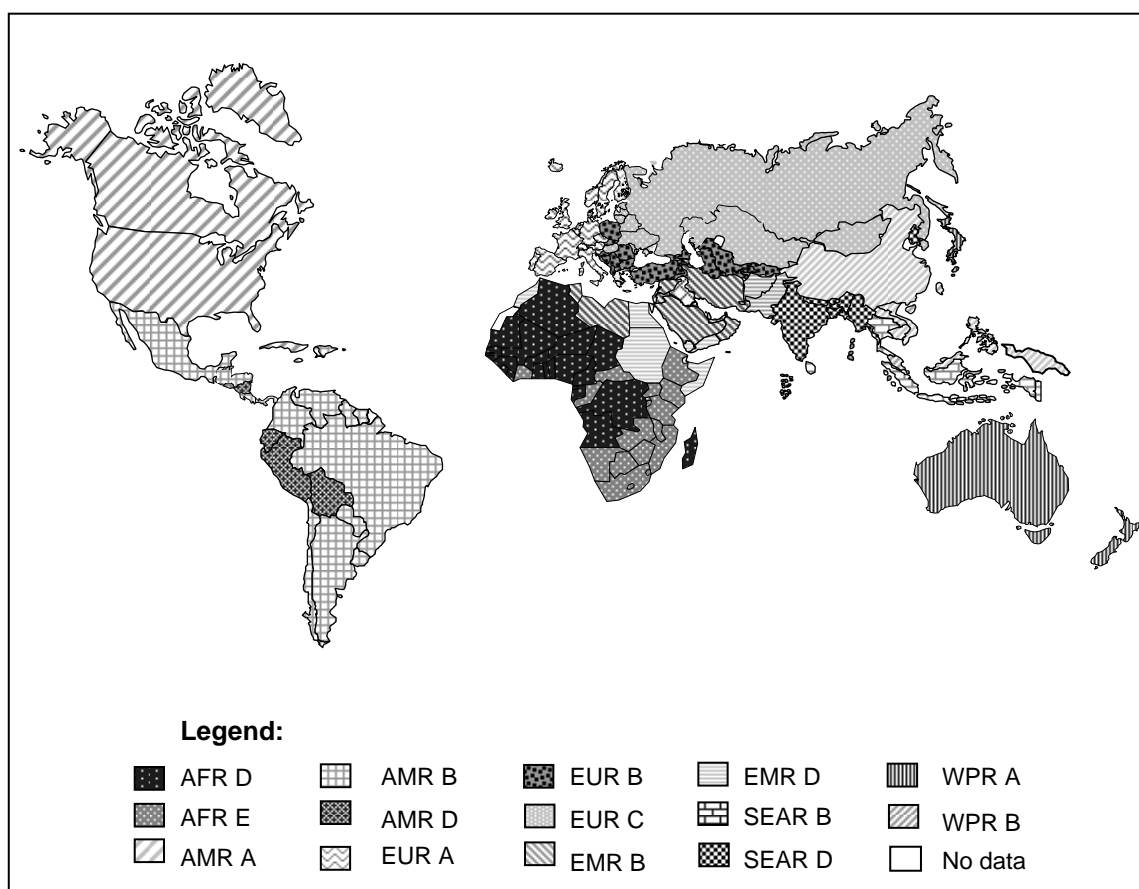


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## Annex 1 Summary results of the global assessment of disease burden from outdoor air pollution

A global analysis of the disease burden caused by exposure to outdoor air pollution was performed on the basis of the same approach as described in this guide (WHO, 2002; Cohen et al., 2004). The analysis was performed for the year 2000, and for 14 WHO subregions of the world, grouped as shown in Figure A1 and Table A1, and by age and sex groups.

**Figure A1** Subregional country groupings for the global disease burden



This is only a schematic representation. The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries.

**Table A1** Country groupings for global assessment according to WHO subregions<sup>a</sup>

Subregion <sup>b</sup>		WHO Member States
AFR	D	Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Niger, Nigeria, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo.
AFR	E	Botswana, Burundi, Central African Republic, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Eritrea, Ethiopia, Kenya, Lesotho, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United Republic of Tanzania, Zambia, Zimbabwe.
AMR	A	Canada, Cuba, United States of America.
AMR	B	Antigua and Barbuda, Argentina, Bahamas, Barbados, Belize, Brazil, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Grenada, Guyana, Honduras, Jamaica, Mexico, Panama, Paraguay, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Uruguay, Venezuela.
AMR	D	Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru.
EMR	B	Bahrain, Cyprus, Iran (Islamic Republic of), Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates.
EMR	D	Afghanistan, Djibouti, Egypt, Iraq, Morocco, Pakistan, Somalia, Sudan, Yemen.
EUR	A	Andorra, Austria, Belgium, Croatia, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom.
EUR	B	Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgyzstan, Poland, Romania, Slovakia, Tajikistan, The Former Yugoslav Republic of Macedonia, Turkey, Turkmenistan, Uzbekistan, Yugoslavia.
EUR	C	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation, Ukraine.
SEAR	B	Indonesia, Sri Lanka, Thailand.
SEAR	D	Bangladesh, Bhutan, Democratic People's Republic of Korea, India, Maldives, Myanmar, Nepal, Timor Leste.
WPR	A	Australia, Brunei Darussalam, Japan, New Zealand, Singapore.
WPR	B	Cambodia, China, Cook Islands, Fiji, Kiribati, Lao People's Democratic Republic, Malaysia, Marshall Islands, Micronesia (Federated States of), Mongolia, Nauru, Niue, Palau, Papua New Guinea, Philippines, Republic of Korea, Samoa, Solomon Islands, Tonga, Tuvalu, Vanuatu, Viet Nam

<sup>a</sup> Source: WHO (2002).

<sup>b</sup> Subregions: AFR = Africa; AMR = Americas; EMR = Eastern Mediterranean; EUR = Europe; SEAR = South-East Asia; WPR = Western Pacific; A: Very low child, very low adult mortality; B: Low child, low adult mortality; C: Low child, high adult mortality; D: High child, high adult mortality; E: High child, very high adult mortality.

Exposure was based on a empirical model using data on urban concentrations of PM10 and PM2.5. The model was developed by the World Bank and is summarized in section 4.2. Using this model, PM10 was estimated for every city in the world with a population above 100 000. Smaller cities and rural areas were not included in the estimates. Exposure was then estimated for each of the WHO subregions using a population-weighted average of the city estimates. A summary of exposure is provided in Table A2.

**Table A2** Population-weighted predicted PM<sub>10</sub> and percentiles of the distribution of estimated PM<sub>10</sub> µg/m<sup>3</sup>)

Subregion	Mean	Percentiles of PM <sub>10</sub> distribution				
		5%	25%	50%	75%	95%
AFR-D	68	32	43	61	72	84
AFR-E	39	30	35	39	44	58
AMR-A	25	24	25	25	25	25
AMR-B	37	35	36	38	39	42
AMR-D	51	37	43	48	53	58
EMR-B	40	23	30	34	39	48
EMR-D	110	62	78	99	110	127
EUR-A	26	25	26	26	27	28
EUR-B	48	41	44	46	48	50
EUR-C	31	21	25	29	33	38
SEAR-B	108	39	86	105	129	151
SEAR-D	84	73	80	84	88	96
WPR-A	32	27	30	32	34	37
WPR-B	89	73	83	89	96	104
World	60	51	56	58	62	65

Adapted from Cohen et al., 2004.

The exposure data were combined with relative risks similar to those summarized in this document to determine AFs and burdens. As in this report, the health outcomes examined included adult cardiovascular mortality and lung cancer associated with long-term exposure to PM<sub>2.5</sub>, all-cause mortality for all ages associated with short-term exposure to PM<sub>10</sub>, and infant and childhood mortality associated with PM<sub>10</sub> exposure. The resulting disease burdens from outdoor air pollution for the 14 WHO subregions is summarized in Table A3. A breakdown by disease, age group and sex is further detailed in Tables A4 and A5.

The results indicated that outdoor air quality is an issue in many urbanized areas of the world, with particularly high values in the big cities of developing regions (Cohen et al., 2004). The analysis suggested that outdoor air pollution accounted for 1.4% of the total mortality, amounting to 800 000 deaths, and 0.5% of all DALYs. The burden predominantly occurs in developing countries with 49% of the attributable burden occurring in WPR B and 19% in SEAR D. Furthermore, 81% of the mortality and 49% of DALYs are estimated to occur in people 60 years and older. These estimates consider only mortality and not morbidity, and therefore underestimate the real burden.

**Table A3** Mortality and DALYs<sup>a</sup> attributable to outdoor air pollution for 14 WHO subregions<sup>b</sup>

Subregion	Attributable mortality (thousands)	Percentage of total mortality in the subregion	Attributable DALYs (thousands)	Percentage of total DALYs in the subregion
AFR D	22	0.5%	319	0.2%
AFR E	10	0.2%	166	0.1%
AMR A	28	1.0%	200	0.4%
AMR B	30	1.2%	307	0.4%
AMR D	5	1.0%	53	0.3%
EMR B	8	1.2%	91	0.4%
EMR D	51	1.5%	636	0.6%
EUR A	23	0.6%	151	0.3%
EUR B	38	1.9%	338	0.8%
EUR C	46	1.3%	370	0.6%
SEAR B	32	1.5%	339	0.6%
SEAR D	132	1.1%	1 513	0.4%
WPR A	18	1.6%	114	0.7%
WPR B	355	3.5%	3 272	1.3%
World	799	1.4%	7 865	0.5%

<sup>a</sup> Abbreviations: DALYs = disability-adjusted life years.<sup>b</sup> Source: WHO (2002).**Table A4** Selected population attributable fractions from outdoor air pollution<sup>a</sup>

Disease	Male (%)	Female (%)	Both sexes (%)
Cardiopulmonary diseases <sup>b</sup>	2	2	2
Respiratory infections	1	1	1
Trachea/bronchus/lung cancers	5	6	5

<sup>a</sup> Source: WHO (2002).<sup>b</sup> Selected cardiovascular and pulmonary diseases.

**Table A5** Attributable mortality and DALYs<sup>a</sup> from outdoor air pollution, by age group and sex<sup>b</sup>

	Age group (years)				Sex	
	0-4	5-14	15-59	60+	Male	Female
Distribution of attributable deaths (% of attributable events)	3	0	16	81	51	49
Distribution of attributable DALYs (% of attributable events)	12	0	40	49	56	44

<sup>a</sup> Abbreviations: DALYs = disability-adjusted life years.

<sup>b</sup> Source: WHO (2002).