

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](mailto: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

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