

Environmental Burden of Disease Series, No. 1

Introduction and methods

Assessing the environmental burden of disease at national and local levels

Annette Prüss-Üstün
Colin Mathers
Carlos Corvalán
Alistair Woodward

Series Editors
Annette Prüss-Üstün, Diarmid Campbell-Lendrum, Carlos Corvalán, Alistair Woodward



World Health Organization
Protection of the Human Environment
Geneva 2003

WHO Library Cataloguing-in-Publication Data

Assessing the environmental burden of disease at national and local levels :
introduction and methods / edited by Annette Prüss-Üstün ... [et al.]

(Environmental burden of disease series ; no.1)

1.Environmental health 2.Environmental exposure 3.Risk factors 4.Cost of illness
5.Disability evaluation 6.Health status indicators 7.Risk assessment - methods
8.Manuals I.Prüss-Üstün, Annette. II.Series.

ISBN 92 4 154620 4
ISSN 1728-1652

(NLM Classification: WA 30)

Suggested citation

Prüss-Üstün A, et al. *Introduction and methods: assessing the environmental burden of disease at national and local levels*. Geneva, World Health Organization, 2003. (WHO Environmental Burden of Disease Series, No. 1).

© World Health Organization 2003

All rights reserved. Publications of the World Health Organization can be obtained from Marketing and Dissemination, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel: +41 22 791 2476; fax: +41 22 791 4857; email: bookorders@who.int). Requests for permission to reproduce or translate WHO publications – whether for sale or for noncommercial distribution – should be addressed to Publications, at the above address (fax: +41 22 791 4806; email: permissions@who.int).

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

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

The World Health Organization does not warrant that the information contained in this publication is complete and correct and shall not be liable for any damages incurred as a result of its use.

Printed by the WHO Document Production Services, Geneva, Switzerland.

Table of Contents

Preface	v
Affiliations and acknowledgements	vi
Summary.....	vii
1. The WHO guides on assessing the environmental burden of disease	1
1.1 Objective of the guides	1
1.2 Target readership	2
1.3 Content of this series	2
1.4 Adapting the guides to specific needs	2
1.5 Improving the evidence base	2
1.6 What evidence is missing?	3
1.7 Related activities.....	3
2. Background to assessing the environmental burden of disease.....	4
2.1 Why measure the EBD?	5
2.2 What are environmental risk factors and how are they categorized?	9
2.3 Attributable risk.....	12
2.4 Limitations of EBD studies	16
2.5 What are the links between EBD assessments and policy-making?	17
2.6 Data and indicators for EBD assessments	20
3. The Global Burden of Disease concept.....	27
3.1 Introduction	27
3.2 Summary measures of population health.....	27
3.3 Quantifying time lived with disability.....	28
3.4 Other social values.....	30
3.5 Calculation of DALYs with discounting and age weighting.....	32
3.6 Relating summary measures of health to the causes of loss of health.....	35
3.7 The GBD 2000 study – an analysis of global mortality patterns.....	36
3.8 The GBD 2000 study – epidemiological analyses for calculating YLD	36
3.9 Main findings from the GBD 2000 study.....	36
4. Methods for estimating the environmental burden of disease.....	41
4.1 General method.....	41
4.2 Alternative or counterfactual exposure.....	45
4.3 Choosing the study population for an EBD assessment	47

4.4	Estimating the EBD when NBD data are available	48
4.5	Estimating the EBD from disease-specific national health statistics when NBD data are not available	48
4.6	Estimating the EBD from limited national or local health statistics when NBD data are not available	49
4.7	Estimating the EBD using preliminary NBD estimates from WHO	50
4.8	Estimating the EBD for diseases that are caused by one risk factor	51
4.9	Estimating the EBD for diseases not assessed by national statistics or by WHO	52
4.10	Estimating uncertainty	53
	References	57
	Glossary of terms for the EBD series	60

Preface

To prevent disease and injury it is essential that their underlying causes (health risks) are quantitatively attributed. Together with information on the costs of interventions, their effectiveness and the socioeconomic context, such knowledge provides a rational basis for policy-setting. While quantitative studies have been performed for some health risks, few have assessed the disease burden from environmental risk factors and, traditionally, the studies have focused on a single risk factor. Recently, however, WHO analysed the global burden of disease from 26 risk factors, and the results were published in the World Health Report 2002 (WHO, 2002). The Environmental Burden of Disease (EBD) series of guides is based on the same methodological framework as used in the World Health report, and provides practical guidance on assessing the health impacts of environmental risk factors.

The guides, together with accompanying material, such as the spreadsheets available for certain risk factors at web site www.who.int/phe, should provide sufficient methodological information to perform the EBD assessments.

The EBD series of guides is composed of an introductory volume, and volumes that provide detailed guidance for assessing the health burden of specific environmental risk factors. Most of the guides focus on assessments of national and local populations, which are most relevant for policy-making. In some volumes, however, the global disease burden is assessed for certain health risks. 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. In this introductory volume, the methodological framework for quantitatively assessing health impacts at population level is described. It is recommended that the framework be adopted by other EBD studies, to ensure that estimates are both reliable and comparable.

Affiliations and acknowledgements

Annette Prüss-Üstün, Colin Mathers, Carlos Corvalán and Diarmid Campbell-Lendrum are from the World Health Organization, and Alistair Woodward is from the Wellington School of Medicine, New Zealand.

We would like to acknowledge the many experts around the world who, over several years, have contributed to the development of methods for estimating the disease burden of environmental risk factors. In particular, we would like to thank participants of the meeting, *Methodology for assessment of environmental burden of disease* (held in Buffalo, NY, USA in 2000), and participants of two regional meetings on the environmental burden of disease (Curitiba, Brazil in 2002; and Damascus, Syria in 2002). Peer reviewers of the sections have also provided invaluable comments.

The financial support of the US Environmental Protection Agency is also greatly appreciated. We gratefully acknowledge the editing by Kevin Farrell, and layout by Eileen Brown, who have put this document into its final form.

Abbreviations used

- BoD Burden of disease.
- EBD Environmental burden of disease.
- GBD Global burden of disease.
- NBD National burden of disease.
- YLL Years of life lost due to premature mortality.
- YLD Years lived with disability.

Summary

This introductory guide provides the background to, and a description of, the general method for assessing the disease burden caused by environmental risk factors. Subsequent guides address the disease burdens of specific environmental risk factors. To assess a disease burden, the health impact of disease and injury needs to be assessed quantitatively at population level. This may be measured in terms of the number of deaths, or as a summary measure of population health, such as the disability-adjusted life year (DALY).

Environmental burden of disease (EBD) studies assess the disease burden attributable to environmental risk factors, and are closely linked to assessments of the disease burden for individual diseases and injuries. Indeed, the burden of disease from disease and injury has been assessed at global level, and national level data are becoming available, which can be used in EBD studies. The results of disease burden studies are generally presented by gender and by age group, and are measured in terms of deaths and DALYs. The actual calculations for an EBD assessment are relatively simple once the input data (exposure and health outcomes) have been collected in a suitable format. The method can also be adapted to the health statistics that are available for the study population.

EBD assessments do not necessarily entail large costs. In many countries and regions, environmental health indicators are already routinely assessed, but are not yet processed into health information. Certain of these indicators can be used directly as input for EBD assessments, so additional assessments may not be necessary. The accuracy of EBD assessment will, however, depend on the quality of the data used as input.

Attributing the health impacts of environmental risk factors at population level can serve several public health activities. It can help to prioritize actions for preventing or reducing health impacts in the population, and by allowing the future health burden to be estimated, an EBD assessment can inform planning for preventive action. EBD assessments can also be used to estimate performance indicators for health-supporting environments, and identify high-risk groups in the population. Finally, EBD information can also be used to predict the health gains that interventions (including regulations) will bring to a population.

1. The WHO guides on assessing the environmental burden of disease

This guide is the first in a series about estimating the disease burden of environmental risk factors. It provides an introduction to the environmental factors that pose a risk to health, and outlines the general methods used to estimate the disease burden of these factors. It also introduces the Global Burden of Disease (GBD) concept (Murray & Lopez, 1996), describes National Burden of Disease (NBD) studies (Mathers et al., 2001) and provides a summary of environmental health indicators.

Other guides in the series focus on specific risk factors and on how to assess the associated disease burdens (Box 1). It is hoped that the guides will help to strengthen local capacity in the analysis and interpretation of environmental health data, and assist with decision-making at national level.

Box 1: Risk factors covered in the guides^a

- Ambient air
- Indoor air
- Lead
- Water, sanitation and hygiene
- Climate change
- Occupational factors:
 - injuries
 - noise
 - carcinogens
 - dusts
 - ergonomic stressors
 - sharps injuries in health-care workers
- Nutrition
- UV radiation
- Recreational water-quality
- Fluoride in drinking-water
- Arsenic in drinking-water
- Nitrates in drinking-water
- Community noise
- Poverty

^a The disease burden of the risk factors listed to the left in Box 1 has been assessed at global level (WHO, 2002), together with that of 16 other risk factors from areas such as lifestyle, diet-related risks, use of addictive substances, unsafe sex and unsafe health practices (Ezzati et al., 2003).

1.1 Objective of the guides

The objective of the guides is to provide practical information to countries on how to assess what fraction of a national or subnational disease burden is attributable to an environmental risk factor. To assess the disease burden of a risk factor, the harmful effects of the risk factor on human health must be estimated fully, as well as the distribution of the harmful effects in the population. Any estimates and assumptions used in the assessment should be stated explicitly. The outcome of the assessment is information that can be used: to guide policies and strategies both in the health sector and in the environmental sector; to monitor health risks; and to analyse the cost-effectiveness of interventions. For example, the information can highlight the contribution of major environmental risk factors to the total disease burden of a country or study population. Or, it can be used to estimate changes in the disease burden and avoidable disease burden, following interventions to reduce an environmental risk factor or to change behaviour.

More generally, an assessment of the environmental burden of disease (EBD) can be used to raise awareness and strengthen institutional capacity for reducing the impact of environmental health risks on the population. The EBD can be assessed for an entire country, or applied to the subnational level (e.g. a city or district), provided basic data are available for the chosen perimeter. EBD studies complement NBD studies, as well as studies of other behavioural or physical risk factors, such as alcohol intake, high levels of blood cholesterol, and unsafe sex.

1.2 Target readership

The target readership includes professionals, such as researchers in universities, government agencies or the private sector, and decision-makers at national or regional level, who are interested in quantitatively estimating the health impacts of environmental risk factors.

1.3 Content of this series

The Introduction (Sections 1–4) addresses the relevance of an EBD assessment to policy, as well as to the larger framework of environmental health assessment, management and evaluation. The general method for assessing the disease burden of a risk factor is also critically reviewed, as are alternative methods, and specific issues are evaluated, such as the units of measurement of disease burden, health valuation, and discounting of future outcomes. There is some guidance on adapting the methods to local needs and circumstances.

Further volumes in the series are composed of guides that help professionals quantify the EBD from some specific environmental risk factors. In these volumes, practical steps are described for performing the quantitative assessments of risk, and for processing the risk data into burden of disease (BoD) data. Data requirements are also given.

Of particular concern are the uncertainties around estimates and the interpretation of results. These issues are addressed in Section 4, and also in the volumes on specific risk factors. In the Annexes, global EBD estimates are given for 10 major environmental risk factors, including 5 occupational risk factors. The data are shown by region, by gender and for 8 age groups.

1.4 Adapting the guides to specific needs

The guidance provided in the series can be adapted to a country's specific needs, to available data sets, or to the desired degree of accuracy. If more locally-specific data, or new exposure-response relationships become available, these can also be used to complement or update the evidence given in the guides for the various risk factors.

1.5 Improving the evidence base

Over the past 10-20 years, significant progress has been made in the evidence base that links environmental risks to health. This was possible owing to new methods in data analysis, and to improvements in computer capacity and performance. Global databases on environmental conditions have also been developed recently. However, there has also been an increase in the level of awareness and concern about environmental degradation or change, and about the short-term and long-term impacts on health. A measure of the

progress that has been made is that, two decades ago, it probably would not have been possible to implement the EBD methods proposed in this series.

A developing evidence base also means that the methods proposed in the EBD guides should be updated as new links between health and the environment are uncovered. The new information could help to improve the accuracy of quantitative linkages between health and the environment, or improve the geographical applicability of data, or better describe the health impacts on poorly-assessed subgroups in a population (e.g. women, or people in a particular age range).

1.6 What evidence is missing?

Although current evidence on the relationship between exposure and disease is solid enough to develop quantitative estimates of the disease burden for a number of environmental risk factors, many other risk factors have not been well documented. In particular, it is easy to overlook risk factors with long latency periods or nonspecific outcomes; factors with exposures that are difficult to assess at population level; and factors that are distal to the outcome. And the absence of data does not necessarily mean that the BoD is negligible or absent. The results of risk factor assessments should therefore be interpreted with caution, and BoD assessments should be regarded as the best current estimates of the magnitude of health problems due to environmental factors.

1.7 Related activities

An assessment of the BoD *by risk factor* (which includes an EBD assessment) is closely linked to an assessment of the BoD *by disease*. Indeed, an EBD assessment is best performed after a NBD study has been developed (Mathers et al, 2001), but a prior NBD assessment is not essential for an EBD assessment.

2. Background to assessing the environmental burden of disease

An assessment of the BoD quantifies the amount of disease at population level. Ideally, the assessment should be carried out in an internally consistent way, and use common units of measurement, since this will allow data on the disease burden and risk factors to be compared between studies. Assessments carried out in this way would also allow the data to be compared for different population groups and across geographical regions. For this reason, a summary measure of population health is used in assessments, which serves as a common currency. Although various summary measures have been developed, the disability-adjusted life year (DALY) is most frequently used in this text. This measure combines the number of years of healthy life lost due to premature mortality and to disability. A detailed explanation of how the DALY is calculated is given in Section 3).

Traditional assessments of the number of healthy years lost have measured either the number of deaths due to disease, or the disease incidence, but not both, which makes it difficult to compare losses that occur at different ages, or from different causes of ill-health.

How does a death at age 20 years compare with a death at age 70 years? How do 200 acute respiratory infections compare to 400 cases of infectious diarrhoea? Summary measures of population health, such as the DALY, provide a framework for dealing with these difficult questions.

Assessments are internally consistent, provided that similar approaches are taken across diseases, risk factors and geographical regions. And the assessments should be coherent – for example, the total number of deaths estimated by a BoD study should not exceed the total number of deaths registered in a country. Death registers generally provide the most complete set of health statistics, but specific causes of death may be recorded in different ways. Many countries have carried out national BoD studies, which estimate the amount of ill-health (commonly measured in DALYs) that is attributable to different disease categories. These studies provide a basis for estimating the EBD, and provide an opportunity to compare health losses due to different risk factors or disease states. In such cases, the internal consistency is already ensured to a large extent. However, a national BoD study is not essential, and the fraction of deaths or morbidity caused by environmental factors can still be estimated without such data.

Main issues

- A BoD study quantifies the health gap at population level and can form the basis of an EBD assessment;
- Summary measures of population health make it possible to compare different estimates of the EBD by standardizing methodology;
- Estimates of the EBD should be internally consistent and use an explicit, commonly-applied methodology.

2.1 Why measure the EBD?

This section explores the rationale for assessing disease burden and outlines ways in which such information can contribute to policy formulation. An important reason for using the formal EBD approach is that it is open to scrutiny. The scientific input to policy decisions often includes important assumptions and judgments that are unspoken (or unwritten). This lack of clarity is why knowledgeable people, when faced with common problems, frequently come to different conclusions. The EBD approach helps to understand the reasons for divergent opinion, without which it will be difficult to develop effective policies.

For various reasons, the magnitude of the health problem from environmental factors needs to be estimated. Health ministries, researchers, bodies responsible for setting standards, scientific advisory groups, and international aid organizations all require such estimates. The questions posed in assessments of the health burden may seem relatively straightforward (e.g. “what is the impact of unvented house fires on the health of families?”), but the answers are often difficult to pin down. For example, it may not be immediately obvious how the exposures and outcomes should be defined (what is meant by the “health of families?”), or what time scale to use (are we dealing with current effects of past exposures, or future effects of current exposures?). The alternative scenario may also be unclear (what is the impact of unvented fires to be compared to? Open fires with chimneys? Wood-burning stoves? No fires at all?). Difficulties also arise in defining health outcomes (should asthma, pneumonia and ear infections be lumped together, or counted separately? Should deaths and nonfatal illnesses be combined to give a single measure of “impact”, and how?).

The EBD method provides a formalized, explicit approach, in which the choices of inputs are apparent. This allows the effects of different assumptions to be readily displayed (e.g. DALYs due to indoor air pollution from unvented fires can be calculated with and without age-weighting). In this way, the EBD approach is not just a tool for improving our understanding of environmental and health linkages – it also allows estimates from different sources to be compared and communicated in a standardized format. The steps for calculating the EBD are described in more detail in Section 4.

There are several other good reasons for performing EBD studies, including:

Prioritizing actions in health and the environment

EBD information supports decisions on priority actions in health and the environment. A common problem in both developed and developing countries is that resources are limited, and informed choices about health have to be made under circumstances where it may not be possible to achieve “safe environmental levels” of every known hazardous substance, given the available resources. For example, air pollution levels are regularly exceeded in most major urban centres, but available resources are not always adequate for modifying transport policies or technologies. Many beaches also do not meet the standards for bathing-water, but improved sewage treatment may not be affordable in the short term. EBD assessments do not replace decision-making in environmental health, but are designed to assist in the process of weighting the advantages and disadvantages of alternative interventions. The purpose is not to provide a fine-tuned calculus of priorities, but instead, an indication of the relative effects of environmental exposures.

Planning for preventive action

When planning to prevent or reduce problems associated with a high disease burden, the information provided by an EBD study is essential for prioritizing actions. EBD data about the effectiveness and cost-effectiveness of interventions (i.e. the dollar cost per unit reduction in environmental exposures or DALYs) will indicate the preventability and the relative costs of the disease burden, respectively. EBD data can also inform preventive actions by being used as input for infrastructure planning, for example, or for estimates of the future disease burden.

However, information other than EBD data also needs to be taken into account when planning preventive interventions. The social and ethical context, for example, should be considered systematically. Social considerations could include the ability of people to respond to their own needs, the social consequences of disease burden, and the priority placed on reducing health inequalities.

Assessing performance

Data from an EBD study can be used to calculate performance indicators for health-supporting systems and environments in a country or region. If a study has the necessary resolution, it can map out geographical or population-specific differences, and monitor trends. The performance indicators can be used to compare the developmental status of regions; they can be compared with other measures of the developmental status of a region; or can be used to compare regions of similar developmental status.

Comparing action and health gain

EBD information provides the opportunity to manage environmental risks from a new perspective. For example, since available resources do not always allow the risks from environmental exposure to be reduced to zero, government agencies might concentrate on risks that provide the best opportunities for gain, for a given investment of resources. EBD information is useful in such an evaluation, and allows the actual health gain of an action in environmental management (or related behaviour change) to be estimated. While most environmental health guidelines attempt to answer the question, “at which value can we reasonably expect that no observable health impacts will occur in an exposed population,” the EBD assessments in the guides answer the following:

- which of the environmental burdens generate the largest impacts on public health;
- by how much will the disease burden in a population be reduced if guidelines are implemented;
- which reductions in exposures would generate the greatest change in DALYs for a given cost;
- what is the cheapest way to achieve a given reduction in DALYs.

In this way, an EBD study provides the basis for estimating the benefits associated with an environmental health action, which is essential information when resources are limited.

Identifying high-risk populations

An EBD study, or more generally, a BoD study, can identify the important contributions to health inequalities in high-risk populations. Although routine health statistics (e.g. mortality registrations) may point to population subgroups at high risk (e.g. women, the elderly, people low on the socioeconomic scale or ethnic groups), EBD estimates can help to understand the causes of inequalities in the subgroups by attributing health gaps to particular environmental exposures, and help to direct public health efforts accordingly (Box 2.1).

Box 2.1 Adjusting burden of disease estimates for equity^a

In 1999, a NBD^b study was carried out in New Zealand to identify the impact of over 100 major diseases and injuries, and 8 chronic disease risk factors. Two years later, a further analysis was conducted, to examine more closely the distribution of the disease burden between indigenous (Maori) and nonindigenous ethnic groups. In general, the health of non-Maoris is considerably better than that of Maoris. Age-specific mortality of non-Maoris, for instance, is approximately half that for Maoris.

The conventional DALY approach was modified using an “impact share” model. This takes into account the variation in the distribution of the BoD between Maori and non-Maori, and the extent to which that contributes to the total Maori/non-Maori gap in health status.

For each disease or risk factor, an equity adjustment factor was calculated as:

$$1 + [p(RR-1)/(p(RR-1)+1)]$$

where RR is the age-standardized DALY rate ratio (diseases) or prevalence rate ratio (risk factors), and p is the proportion of the total difference between Maori and non-Maori age-standardized DALY rates accounted for by the condition of interest. (See the Glossary for a definitions of these terms.)

The equity factor was multiplied by the number of DALYs lost for each disease or risk factor. The purpose of the adjustment was to assist policy-makers in setting priorities for health expenditure. The effect of including the adjustment for equity was to boost the relative importance of diseases and risk factors that weigh disproportionately on the health of Maori. This was apparent when the top 25 causes of DALYs lost in New Zealand were compared, with and without the adjustment for equity. Differences included:

- diabetes was ranked higher;
- a lack of physical activity was ranked lower as a risk factor;
- sudden infant death syndrome was included on the list;
- hearing disorders were no longer in the top 25 causes of disease.

^a Source: New Zealand Ministry of Health (2001)

Planning for future needs

It is possible to project exposures into the future and estimate trends in the EBD, even when there may be a long time-lag between exposure and the onset of disease. Such estimates of

the future burden give policy makers the option of shifting priorities proactively, if this is likely to be less costly than doing nothing until the disease burden is apparent. For example, on the basis of past exposures it is possible to project future trends in mesothelioma and other asbestos-related conditions. Moreover, the future EBD can be estimated for asbestos exposures, depending on different control strategies.

Assessing future scenarios

There have been several recent assessments of future global changes. For example, the Intergovernmental Panel of Climate Change produced a set of CO₂ emission scenarios that could lead to global temperature increases, and the United Nations Environment Programme produced a set of environmental scenarios in its Global Environmental Outlook. Such scenarios can be used to paint a picture of future population exposures, and to estimate health losses that may occur. Assessments of this kind carry a high level of uncertainty, but they do provide indications of what may happen if certain environmental conditions apply. Forward-looking assessments like these are important with problems such as climate change, where there are long time-lags between emission of greenhouse gases, changes in the climate, and subsequent health effects.

Setting priorities in health research

To some extent, an EBD assessment at national level can also foster priority-setting in health research. At present, resources spent on research and development do not always reflect the disease burden. This is apparent from the fact that only 10% of the total resources spent on health research is devoted to the health problems of 90% of the world's population (WHO, 1996). The following five criteria have been proposed for making rational decisions about allocating resources for research (WHO 1996):

1. what is the attributable BoD (this is addressed in this guide);
2. what are the main determinants of the BoD and its persistence;
3. what is the knowledge base of the disease (including the cost-effectiveness of interventions to reduce the disease burden);
4. what is the likelihood that cost-effective interventions can be developed;
5. what are the present resource flows for the risk factor or disease.

Relevancy to policy-making

EBD assessments may be more relevant than disease-based assessments for decision-making in environmental health policies, since the health gain from changes in environmental exposures can be directly estimated with these methods. The links between EBD information and policy are discussed in Section 2.5.

Main issues

The rational development of a health policy uses inputs from EBD studies in the following ways:

- EBD studies help to identify priority actions in health and the environment, as well as the effectiveness, cost-effectiveness, and social and ethical implications of an intervention;
- EBD studies allow policy actions or interventions to be based on estimated health gains, rather than on “safe environmental levels” of the risk factor alone;
- EBD studies help to assess the performance of a country;
- EBD studies can identify high-risk populations;
- EBD studies allow research to be prioritized.

2.2 What are environmental risk factors and how are they categorized?

Environmental causes of disease may be categorized in many ways, e.g. by referring to media which may carry hazards, as individual risk factors (agents), or according to the nature of the hazard.

Media that carry hazards include:

- water used for drinking, recreational activities or agricultural activities (such as irrigation);
- food;
- special environments that potentially carry hazards, such as agricultural environments, water resources, or wetlands;
- indoor and outdoor air.

Individual risk factors include:

- chemical substances;
- noise;
- radiation (ionizing, UV, electromagnetic).

These risk factors can be further divided into those in the occupational environment, or in the general environment (i.e. non-occupational environment). Many of the media risk factors and individual risk factors overlap. Risk factors also present different types of hazards, including:

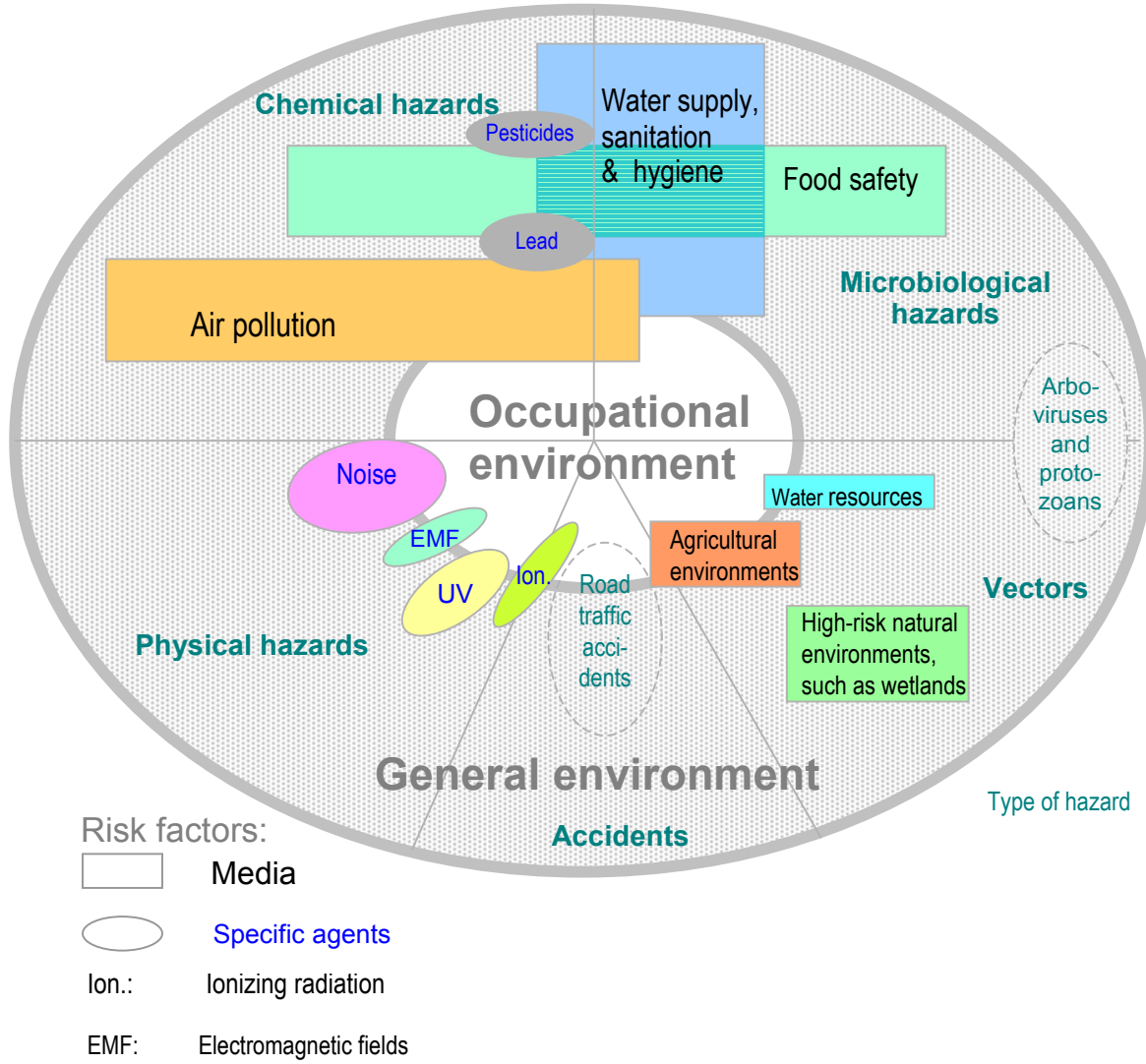
- chemical hazards;
- microbiological hazards;
- physical hazards;
- accidents;
- vectors.

The effects of environmental exposures on health depend on the social settings in which the exposures occur and on individual behaviours. Behavioural risk factors are sometimes

closely related to physical risk factors (e.g. hygiene is related to sanitation) and modify the health impacts of the physical risk factors. Indeed, the specific contributions of related behavioural and physical risk factors sometimes cannot easily be separated, as for example in the risk factor “water, sanitation and hygiene”.

As the primary aim of EBD estimates is to inform policy, the assessment of risk factors that are most directly relevant to policy would be the most useful. A representation of how the risk factors affect policy options, such as options for energy policy, transportation policy, or emission-reduction policy, would be ideal. Currently, evidence is generally compiled around media and agents (e.g. air quality, food), and EBD assessments focus on these categories because the data are accessible. Examples of major environmental risk factors, their categories, the types of hazards they carry and their overlaps are represented in Figure 2.1.

Figure 2.1 Environmental hazards and risk factors



Policy scenarios are linked to multiple distal causes and are therefore more complicated to assess (Murray & Lopez, 1999a). For example, transportation scenarios are linked to air pollution, accidents and noise, and the health effects of reduced physical activity. Similarly, energy policies may be linked to a variety of risk factors, including air pollution, accidents, radiation, water pollution etc., according to the selected technology.

Main issues

Environmental risk factors can be categorized according to:

- the media carrying the hazards;
- individual risk factors;
- the nature of the hazard;
- the general environmental or occupational environment.

EBD assessments that are related to policy scenarios would be most useful, but such assessments are complex.

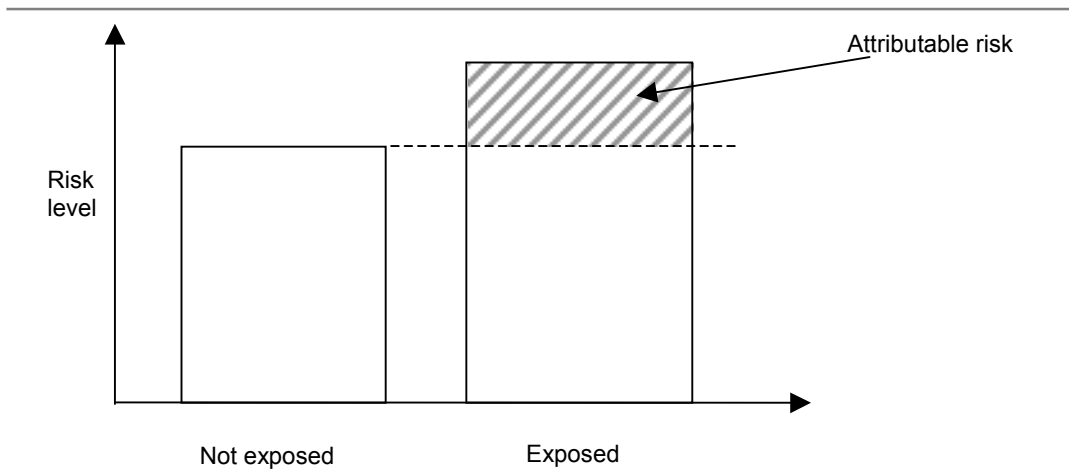
2.3 Attributable risk

BoD assessments strive to provide a quantitative answer to the question, “how big is this particular health problem?”. This requires two steps: establishing an appropriate measure of health status, and deciding how much ill-health can be attributed to a particular risk factor. Measures of health status are discussed in Section 4. In this section, we review attributable risk, and how it is applied to EBD calculations. More detailed descriptions of attributable risk can be found in the literature (WHO, 1993; Rothman & Greenland, 1998).

Attributable risk is one of the fundamental concepts underlying BoD assessments, and it involves the ideas of attribution and causal inference. Sometimes we can say, with confidence, that an occurrence of disease is due to a particular environmental exposure. Mesothelioma, for example, is a neoplasm of the lung that is seldom caused by anything other than asbestos. But this is an exception, and in most cases there are many possible causes of a disease. Gastroenteritis may result from drinking contaminated water, but it may also be caused by toxins in food, or pathogens spread from hand to mouth. Because most diseases have multiple origins, attributable risk cannot be applied at the level of an individual. If a child scores poorly on IQ tests this may be caused by exposure to lead, but in the case of an individual child it may be impossible to exclude other, plausible explanations (such as social disadvantage).

When individuals are grouped together, the task of attribution is more straightforward. If two groups are alike in all important respects, except that one group has been exposed to a factor of interest, then any difference in disease rates between the two groups is said to be attributable to (caused by) the exposure. The disease rates can be equated to risk levels for the populations, and the attributable risk for the factor calculated from the difference (Figure 2.2). Although such data do not distinguish between individuals who fell ill because they were exposed to the factor and those who fell ill from other causes, the data can be used to deduce what fraction of the total BoD would have been avoided if the exposure had not occurred.

Figure 2.2 In general, attributable risk is the difference in risk levels for “exposed” and “not exposed” populations

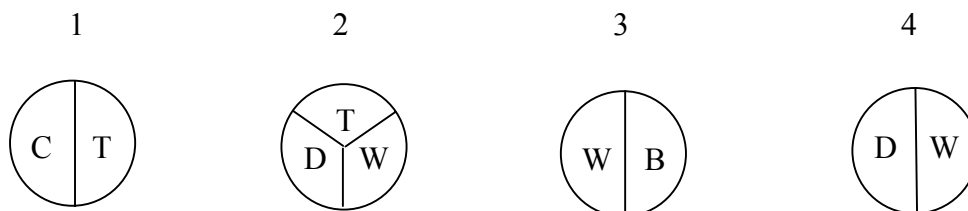


The proportion of the total burden of disease that is due to exposure to an environmental risk factor is called the attributable fraction for that risk factor. This gives an indication of how much ill-health might be avoided if exposure to the risk factor did not occur. What often causes confusion is that the attributable disease fractions for risk factors may not add up to unity. To understand why this might be so, consider an analogy in which the causes of disease are different routes that can be taken to reach a common destination, such as the different ways of travelling to work. An imaginary commuter has four ways of getting to work:

1. Cycle to the station, catch the train, and cycle to work.
2. Drive to the station, catch the train, and walk to work.
3. Walk to the bus stop, and catch the bus to work.
4. Drive to work and walk from the car-park.

It is assumed that the four routes are chosen with equal frequency (i.e. 25%), and that there is no replacement possible (if the trains are on strike one day, then the commuter simply misses a day of work). Also, no means of transportation by itself is sufficient to take the commuter all the way from home to work - they need to be used in combination - and none is necessary (i.e. without it, the commuter would never get to work).

If the methods of transportation are described as “component causes”, the four combinations of transportation that get the commuter to work can be diagrammed as follows:



where B = taking the bus, C= cycling, D = driving, T = taking the train, W = walking.

From the diagrams, it is simple to calculate the proportion of trips to work that use a particular transportation method. Walking, for example, is used in combinations 2, 3 & 4, or 75% (25%+25%+25%) of the trips. Driving is used in combinations 2 & 4, or 50% (25%+25%) of the trips. The corresponding values for taking the bus, cycling and taking the train are 25%, 25% and 50%, respectively. Each value represents the proportion of trips to work that would be prevented if that particular form of transportation were not available. The fact that these fractions add up to more than 200% does not mean that more than 200% of trips to work could be prevented by avoiding all exposures to buses, cycling, train travel etc. Rather, each fraction describes the change that would occur if that particular exposure were altered, while all others stayed the same.

Because these fractions are interdependent, they cannot simply be added up. Once one of the transportation methods has been removed, two things change. First, the frequency of the outcome (getting to work) is altered; and second, the proportions for the remaining transportation methods alter. For example, if bus travel were impossible, there would not only be 25% fewer trips to work, but the proportions for the remaining forms of transportation would also change. Since cycling would be used only in trip combination 1, it would be used in 33% of the trips (not 25%, as before), and trains, driving and walking would each be used in 67% of the trips. Another example of competing and interacting risks is given in Box 2.2.

Box 2.2 Competing risks^a

When analysing multiple risks it is natural to think that attributable disease fractions for individual risk factors should all total to 1.0 (or 100%). There are several reasons why this is not so in practice, and the disease fractions for individual risk factors can total to more than, or less than, 100%. Furthermore, the disease fractions may not be strictly additive. Examples of these situations are given below.

Perhaps, it is most easily seen that disease fractions for known risk factors might total less than 100% of the disease burden, given that our knowledge might be incomplete. In other words, if we have not identified all the risks associated with a disease, we may not be able to attribute 100% of the disease burden.

But surely, most people might say, disease percentages due to multiple attributable risks could never add to more than 100%? (After all, how can we prevent more disease than there actually is?). To illustrate how this can be so, consider a hypothetical situation of 1000 annual deaths from auto accidents along a dangerous stretch of highway. Studies show that the deaths would be reduced by 20% if headlight use was required during the day, 40% through stricter speed limits, 50% by installing more stop-lights, 90% by installing speed bumps, 98% by having a police officer accompany each car, and so on. Clearly, the total, 298%, is open-ended and reflects the detail with which we understand the problem, and our ingenuity in finding ways to deal with it. In this way, the diseases that could be prevented by removing various risk factors can add to more than 100%.

Another factor is that many important risk factors do not create disease cases by themselves, but act in conjunction with other hazards or conditions. Also, certain hazardous features of a risk factor can be compensated by other protecting circumstances. In other words, such risks factors are not usually completely independent, and changes in one will affect the others.

As a result, the attributable disease fractions for multiple risk factors may not be strictly additive. In the highway safety example, if we can save 100 lives by requiring daytime use of headlights, or 200 lives through stricter speed limits, could we save 300 lives by doing both? No, because once one intervention is implemented the overall situation changes and the remaining potential benefits of the other risk factors will be reduced. In this case, many of the 200 people whose lives might be saved in a speed-limit campaign might also have been saved by a campaign to use daytime headlights. Depending on the degree of non-independence, the remaining benefit of requiring daytime use of headlights might only be 50, for example. The total number of lives saved by instigating both campaigns would then be only 250, not 300.

^a Adapted from: Smith et al. (1999).

How could we apply this example to burden of disease assessments? The “destination” in this instance is generally a state of impaired health (measured in units such as DALYs), and the various forms of “transportation” are factors that combine in different ways to move an individual from good health to poor health. In other words, the fraction of a disease that is attributable to a risk factor tells us what health losses would be avoided if the risk factor were eliminated. (Commonly, the risk factor cannot be eliminated altogether and so the “avoidable” risk is calculated, in which case the counterfactual is not “no exposure”, but “minimum achievable exposure”).

For example, acute respiratory illness in childhood may result from one or more environmental factors, including second-hand smoke, indoor air pollution from other sources, crowding in the home, and attendance at group child-care. There are also factors that affect susceptibility to environmental threats, and some of these are known and can be measured, such as infant feeding (bottle or breast) and past history of chest illness. None of the environmental factors is a sufficient cause of acute respiratory illness (e.g. not all children whose parents smoke become ill), and none is a necessary cause (e.g. acute respiratory illness does not occur exclusively among children of smoking parents). Rather, it is a combination of environmental factors and causes of susceptibility that provides a sufficient cause of illness. Each individual case results from the combined effects of a number of environmental causes (not all identified). Removing one environmental factor (such as second-hand smoke) will reduce the frequency of acute respiratory illness, and may alter the fractions of the disease burden that may be attributed to the remaining risk factors.

2.4 Limitations of EBD studies

The limitations of EBD assessments should be considered in context, and include:

Important aspects of risk are not included

Priorities are not decided on the basis of numbers alone. Some risks pose specific environmental hazards that influence what individuals and populations regard as important. Factors such as equity, uncertainty, dread and degree of control can all play a role in shaping people’s attitudes. For example, the priority given to controlling spray drift (pesticides drifting into residential areas) is likely to be influenced more by the involuntary and inequitable features of exposure, rather than the resulting burden of disease. It is possible to weight EBD calculations in response to perceptions of different risks (e.g. Box 2.1), but this is a matter to be decided at local level.

EBD does not account for benefits other than health gain

This is important with environmental modifications that may have other kinds of social benefits. An intervention to protect water supplies, for example, can improve food production and lead to economic gains. Air pollution, too, can reduce particulates and other toxic emissions in periurban areas, but may also significantly boost agricultural production.

Sensitivity analyses can test the implications of different choices

BoD calculations involve judgements about standard life expectancy, severity (disability) weights, age weighting, and discounting over time. To see if these judgements make a difference to priorities for action, EBD data should be calculated using different assumptions.

The underlying causal processes may be, necessarily, simplified

Two aspects of complexity are relevant for EBD estimates. First, people are exposed to a complex mix of factors in the environment, and the interactions between these factors are often not well-understood and cannot be modelled satisfactorily in BoD calculations. For example, the effects on the lung of air pollutants such as ozone and NO₂ are greater when these factors are present together than when they are inhaled separately (in experimental chambers, for instance). Nevertheless, EBD estimates often treat pollutants individually, because little is known about joint effects. Second, BoD studies do not yet allow for comorbidity to be estimated, and outcomes tend to be dealt with singly, similar to the approach used with exposures. Yet when diseases occur together, the combined impact on the level of disability is likely to be different from the impact of a single disease.

Parameters that can be measured rapidly may be favoured over those that are difficult to measure

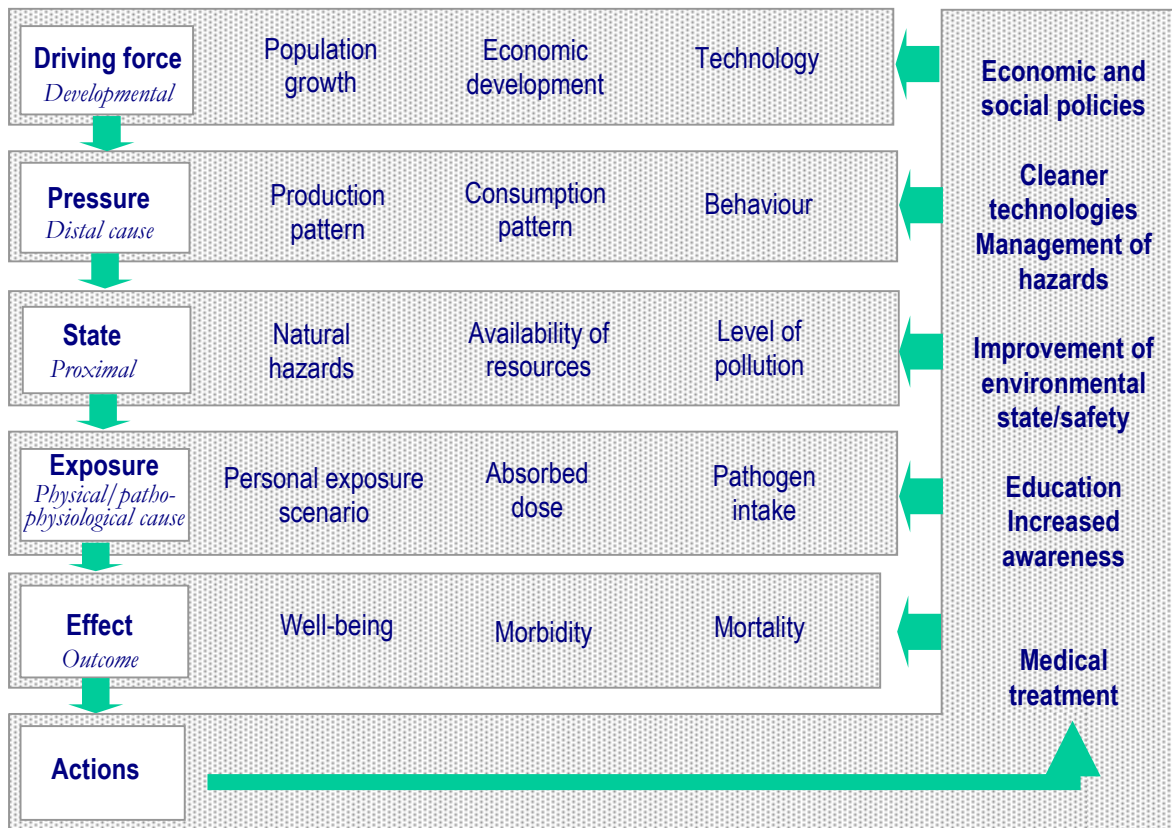
This caveat is relevant to all quantitative measures, but it is important that exposures and outcomes are not overlooked simply because the “right kind” of data are lacking. Examples range from simple health problems that are poorly documented (e.g. back pain), to the health effects of social and cultural dislocation.

2.5 What are the links between EBD assessments and policy-making?

In section 2.1, several links between EBD assessment and policy were highlighted. Basically, EBD assessments provide an important input to the development and evaluation of policies in the health sector and to activities of other sectors that directly manage or influence the determinants of health.

The DPSEEA (Driving-force – Pressure – State – Exposure – Effect – Action) framework (Figure 2.3; Kjellström & Corvalán, 1995; Corvalán, Briggs & Zielhuis, 2000) provides a hierarchical model that describes the actions of various causes that act, more or less directly, on health outcomes from environmental or related behavioural conditions. In addition, it displays the various levels of actions that can be taken to reduce health impacts.

Figure 2.3 Conceptual framework for environmental health assessment, management and evaluation

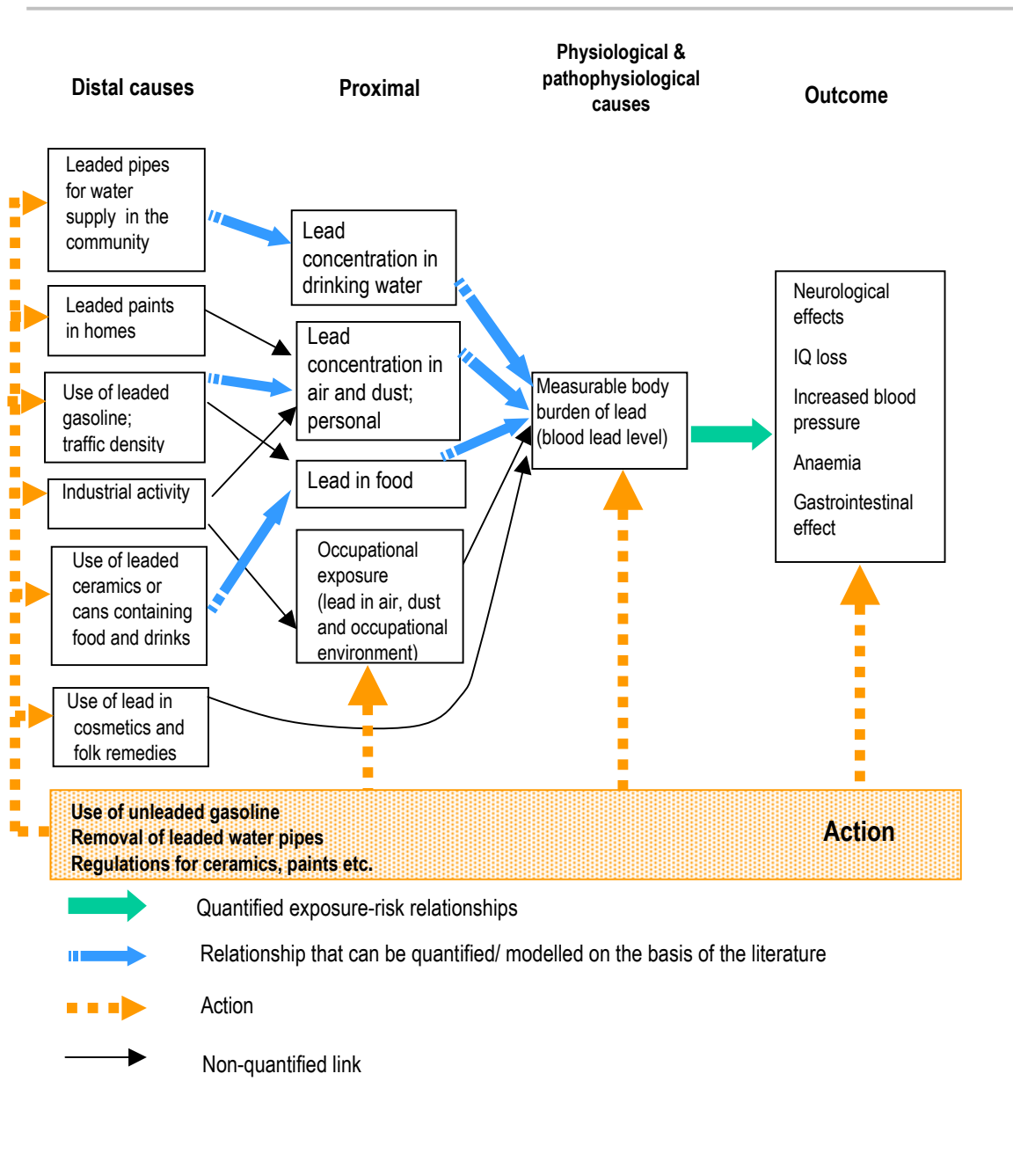


^a Adapted from WHO (1997).

The DPSEEA framework can be developed for most environmental risk factors and their associated health outcomes. This qualitative model can be developed further into a quantitative one if numerical functions are ascribed to the linkages between the various positions. It is then called a “causal web” and serves as a basis for EBD assessment (Murray & Lopez, 1999a). Ideally, policies act to reduce the causes of disease, and the subsequent impact of policies on the disease burden can be measured from the change in disease burden. In practice, however, it is often difficult to attribute changes in disease burden to particular policy interventions. Reasons include the lags that occur between policy implementation, exposure reduction and disease incidence, and the multicausal nature of disease. For example, lead in the environment is influenced by many factors, and the impact of regulations to remove lead from gasoline and paint on the rates of anaemia and high blood pressure may be difficult to discern (Figure 2.4).

Each of the volumes on risk factors start with the causal web, which constitutes a basis for EBD assessment. An example of a causal web for lead exposure is shown in Figure 2.4.

Figure 2.4 Causal web for lead exposure



This example of a causal web for lead highlights the relationships between causes that can be quantified and used to estimate the causal web, and how policy actions impact the framework. In this case, the available knowledge on quantitative information allows not only the disease burden caused by environmental exposure to lead to be estimated, but also a prediction of health gains according to policy actions. More detailed information on lead exposure is provided in the volume on lead.

Main issues

- An EBD assessment is an input to rational policy making in environmental health. Policies and interventions can modify health impacts from environmental risk factors at various levels.
- The DPSEEA (Driving-force – Pressure – State – Exposure – Effect – Action) framework is a hierarchical model linking measurable indicators to environmentally-caused diseases. Such a model can be developed quantitatively, and is then called a “causal web”.

2.6 Data and indicators for EBD assessments

Many countries collect a wealth of health and environmental data, but do not process them into quantitative information about the environmental health impacts on the population. The statistical data and trends may be used to formulate policies that aim at not exceeding “safe levels” of environmental pollutants, but this alone is not always directly relevant for policy-making or its evaluation. The key information that is needed to evaluate environmental health policies, or to assess the impact of an intervention, is an estimation of the health impacts.

With new methods for EBD assessment becoming available (such as those presented in this guide), the collection of environmental health data can be designed in such a way that the data can be directly processed into quantified health impacts. Selected indicators can thus be fitted into a framework for data assessment and management that includes collecting and processing the data, and using the information to inform policy and to provide feedback for evaluation. This would increase the value of the environmental data to policy-making in both the environmental and health sectors. A set of such indicators can be converted into disease burden estimates at global level (WHO, 2002), or can be used in guiding an assessment of the EBD at national or local level. These indicators are summarized in Table 2.1.

The selection and collection of data, and the processing of indicators into relevant public health information according to a comprehensive framework, will require close cooperation between the health and environmental sectors, since data on the quality of the environment are generally collected by the environmental sector, whereas environmental health activities are concentrated in the health sector. An EBD assessment does not necessarily require that additional data be collected, but rather a targeted collection of specific data that can be processed into information about the health impacts. Thus, although additional resources are not necessarily required, effective intersectoral collaborations are needed to translate information into health-promoting actions.

Table 2.1 Indicators that can be processed into disease burden estimates using existing methods^a

Area	Indicator ^b	Related diseases ^c or outcomes	Availability of indicator data
Water, sanitation and hygiene.	Water supply coverage. Sanitation coverage.	Diarrhoea; total fraction of schistosomiasis, hookworm disease, ascariasis, trichuriasis, trachoma.	Coverage at country level, in Water Supply and Sanitation Assessment 2000.
Ambient air pollution.	Annual mean concentrations of particulate matter (PM ₁₀ and PM _{2.5}).	Mortality from cardiopulmonary disease.	Measurements almost only available in developed countries; global exposure modelled by the World Bank; modelled and measured data cover more than 3000 cities.
Climate change: extreme temperatures.	Change in daily temperature distributions.	Cardiovascular diseases. Respiratory diseases (mortality only).	Based on current data and generated by climate models for missing data or projections.
Climate change: warming.	Change in monthly temperature.	Diarrhoea.	Based on current data and generated by climate models for missing data or projections.
Climate change: food production.	Change in temperature, rainfall and CO ₂ .	Malnutrition.	Generated on the basis of current data and climate models; food availability is modelled.
Climate change: coastal floods.	Sea level rise and frequency of coastal floods.	Deaths and injuries.	Generated on the basis of current data and climate models; model for frequency of coastal floods.
Climate change: inland floods, landslides.	Monthly rainfall exceeding the 1-in-10 year limit.	Deaths and injuries.	Generated on the basis of current data and climate model.
Climate change: vector-suitable environments.	Average monthly temperature, minimum annual temperature, average rainfall, resulting in area suitable for malaria transmission.	Malaria. Dengue.	Climate parameters based on observed and modelled data, and a vector model generates the transmission potential of malaria and dengue.
Indoor air pollution.	Household solid fuel use.	<i>Strong evidence:</i> – acute respiratory infections; – lung cancer; – chronic obstructive pulmonary disease. <i>Moderate evidence:</i> – asthma; – cataract; – tuberculosis.	Household solid fuels database and model for predicting fuel use.
Malnutrition ^d .	Stunting. Wasting. Underweight.	Child mortality. Contribution to acute respiratory diseases. Contribution to malaria.	WHO Global Database on Child Growth and Malnutrition, includes 400 000 measurements that are representative of 85% of the world's children.
Lead.	Blood lead.	Mild mental retardation. Loss of IQ points. Anaemia. Gastrointestinal symptoms.	Recent representative samples from 41 countries.

^a Source: WHO (2002a).

^b The disease burden estimated on the basis of the indicators generally does not capture all of the disease burden caused by the risk factor.

^c Expressed in mortality, attributable fraction of total disease group, disability-adjusted life years (DALYs).

^d Not directly measurable by environmental parameters, but could be related to them.

Main issues

- Environmental indicators that can be directly processed into health impact information may be of greater value for the environmental and health sectors than indicators that cannot be directly processed.
- Fitting indicators into a comprehensive framework of environmental assessment and management allows the state of the environment to be measured, as well as the health impacts and health gains of interventions.

What information is routinely collected?

To date, only a limited number of EBD methods have been developed for risk factors and the outcomes caused by those risk factors. However, information from currently assessed indicators could be converted into new EBD assessments. In this section we therefore provide an overview of the types of indicators that are currently being collected.

Countries, and national and international agencies, routinely collect information on indicators at different levels of aggregation, both on the environment and on health. The use and interpretation of indicators have been described as crucial links in the data-to-decision-making chain: measurements produce raw data; data are aggregated and summarized to provide statistics; statistics are analysed and re-expressed in the form of indicators; and indicators are then fed into the decision-making process (Wills & Briggs, 1995). An environmental health indicator can be understood as a measure which summarizes in easily understandable and relevant terms some aspect of the relationship between the environment and health that is amenable to action. It is a way of expressing the scientific knowledge about the link between environment and health in a form that can help decision-makers make more informed and more appropriate choices (Corvalán, Briggs & Zielhuis, 2000). The conversion of indicators into a summary measure of population health using a common metric, such as a DALY, provides additional input to decision-making. The following sections provide a summary of some types and sets of indicators.

Environmental indicators

An environmental indicator has been described as “a measurement, statistic or value that provides a proximate gauge or evidence of the effects of environmental management programs or the state or condition of the environment” (USEPA, 1994). As a result, the majority of environmental indicators developed so far describe the environment without any explicit or direct implications for health. Examples include indicators of atmospheric emissions, surface-water quality, designated areas, or threatened wildlife species. However, although the data are not collected specifically for health-related purposes, they can be converted into health measures in the framework of EBD assessments. In the context of human health, we are mostly concerned with indicators that measure human exposure to potential health risks, which allows the health impact of environmental risk factors to be evaluated.

Health indicators

The majority of health indicators describe the status of, or trends in, health, without any direct reference to the environment. Examples include simple measures of life expectancy, and cause-specific mortality rates where no attempt has been made to estimate health outcomes that are attributable to the environment. Health indicators have been used extensively to monitor the health of populations, both by international agencies at global, regional and country levels, and by countries at national and subnational levels. But many health outcomes can be related to environmental risk factors (e.g. disease related to the environment). Such a disease-oriented approach provides a means of monitoring and assessing the health outcomes of a wide range of environmental exposures. For example, the WHO Environmental Health Indicators Project has identified sets of indicators that focus on health outcomes and their associated risk factors (WHO, 1999). Summary measures of population health, e.g. expressed in DALYs, combine several health indicators into a single metric that characterizes population health, and describes the impacts of intervention.

Environmental health indicators

Environmental health is concerned with environmental factors that influence or directly affect human health (either positively or negatively). An environmental health indicator can thus be defined as “an expression of the link between environment and health, targeted at an issue of specific policy or management concern and presented in a form which facilitates interpretation for effective decision-making” (Corvalán, Briggs & Zielhuis, 2000). As such, it is more than either an environmental indicator or a health indicator: it is based upon a known or postulated relationship between environmental exposure and health. Examples of environmental health indicators used in water-quality studies are provided in Table 2.2.

Table 2.2 Environmental health indicators within the DPSEEA framework: an example using microbiological contamination of water

	Descriptive indicator	Action indicator
Driving force	Level of poverty in the community.	Expenditure on water and sanitation improvements.
Pressure	Percent of households without a safe drinking-water supply.	Number of unserved households provided with clean water supply per year.
State	Coliforms in water.	Extent of water-quality surveillance and water treatment.
Exposure	Percentage of population exposed to hazardous water contaminants.	Extent of public education programmes on water hazards and treatment in the home.
Effect	Morbidity and mortality from diarrhoeal diseases.	Number of cases treated in hospitals and clinics.

Two general types of environmental health indicators can be distinguished: those based on exposure and those based on health effects. An exposure-based indicator (e.g. known levels of air pollution) can be used to estimate future risk, using current knowledge about an environmental hazard. Such indicators can be combined with a known environment-health relationship (e.g. the relationship between respiratory disease and levels of air pollution) to provide an estimate of the health impact. In contrast, an effect-based indicator (e.g. diarrhoea rates in the population) can be used to deduce the environmental cause of poor health, again based upon some epidemiological knowledge. For example, current diarrhoea death rates can be used to determine the number of cases that can be attributed to poor water quality.

Within the context of environmental health, “environmental” pertains to “all that which is external to the human host, including physical, biological, social, cultural, etc., aspects, any or all of which can influence the health status of populations” (Last, 2001). The environment therefore includes not only the general environment to which everyone is exposed, but also specific environments, such as the workplace and the domestic environment, where people spend a significant proportion of their time. Included among environmental health hazards are not only the immediate biological, chemical or physical factors that affect health, but also the underlying social, economic and technical conditions that give rise to (and modify) environmental health problems. An indicator that only describes the state of the environment, with no obvious link to health impacts of the environment, could not be considered an environmental health indicator. In the same vein, a pure health status indicator, with no obvious link to the environmental cause of health deterioration (or health improvement), also could not be considered an environmental health indicator.

To be useful, an environmental health indicator must relate to aspects of environmental health which are both relevant to the decision-maker, and directly or indirectly amenable to control. Given that the collection of information invokes costs, and that these costs will need to be justified, it will rarely make sense to collect information, or construct indicators, that will not be used to support policy. This means that most indicators are built around areas of existing policy: the policy imperative creates both the need for indicators and justifies the costs of constructing them. Some of the most valuable uses of indicators, however, are to help identify and assess new policy questions. This means that some indicators need to be developed in advance of a clear and definite policy need. For this reason, environmental health indicators need to be expressed in a way that is pertinent to, and understandable by, decision-makers. In many circumstances, this requires that the indicator be expressed in terms of the health risk associated with a specific environmental hazard, since this provides a universally recognizable “currency” that can be used to assess and compare different problems. The EBD approach is one means of introducing such a common currency.

WHO has compiled detailed lists of environmental health indicators, described in methodology sheets, that allow investigators to use a common approach and similar methods of data collection. Region-specific sets of indicators have also been developed (such as those prepared by the WHO Regional Office for Europe, available at http://www.who.dk/eprise/main/who/progs/ehi/indicators/20020319_1).

Main issues

- Useful indicators include environmental, health and environmental health indicators.
- Indicators may describe proximal or distal causes of disease.
- Various sets of indicators have been developed, not all of which can be converted into EBD information. However, the indicators may be useful for informing about other parameters that are important for decision-making
- Sets of currently compiled indicators provide potentially valuable data that can be converted into quantified and non-quantified information for policy-making.

3. The Global Burden of Disease concept

3.1 Introduction

The GBD concept, first published in 1996, constituted the most comprehensive and consistent set of estimates of mortality and morbidity yet produced (Murray & Lopez, 1996), and WHO now regularly develops GBD estimates at regional and global level for a set of more than 135 causes of disease and injury (Mathers et al., 2002; WHO, 2002a).

A GBD study aims to quantify the burden of premature mortality and disability for major diseases or disease groups, and uses a summary measure of population health, the DALY, to combine estimates of the years of life lost and years lived with disabilities. The data are also broken down by age, sex and region.

WHO also supports NBD studies to obtain country-specific estimates for input to national policy. The national studies are based on the GBD concept and the data can be used in EBD assessments to estimate the contributions that environmental risk factors make to the overall disease burden. Over 30 countries are now undertaking NBD studies. Guidelines, software tools, and data for NBD studies are available from WHO (Mathers et al., 2001).

3.2 Summary measures of population health

Summary measures of population health measure the health of a population by combining data on mortality and non-fatal health outcomes into a single number. Besides the DALY, several other such measures have been devised, including the Quality-Adjusted Life Year (QALY), the Disability-Adjusted Life Expectancy (DALE) and the Healthy Life Year (HeaLY) (Weinstein & Stason, 1977; Murray & Lopez, 1996; Hyder, Rotllant & Morrow, 1998; Murray, Salomon & Mathers, 2000). The benefits and challenges of these measures have been examined (Anand & Hanson, 1997; Williams, 1999; Murray & Lopez, 1999b, Murray, Salomon & Mathers, 2000; Murray et al., 2002). As the DALY has been the most widely-used measure, and can be applied across cultures, we will focus on it in this guide.

The DALY measures health gaps as opposed to health expectancies. It measures the difference between a current situation and an ideal situation where everyone lives up to the age of the standard life expectancy, and in perfect health. Based on life tables, the standard life expectancy at birth is set at 80 years for men and 82.5 for women.

The DALY combines in one measure the time lived with disability and the time lost due to premature mortality:

$$\text{DALY} = \text{YLL} + \text{YLD}$$

where:

YLL = years of life lost due to premature mortality.

YLD = years lived with disability.

The YLL metric essentially corresponds to the number of deaths multiplied by the standard life expectancy at the age at which death occurs, and it can be rated according to social preferences (see below). The basic formula for calculating the YLL for a given cause, age or sex, is:

$$YLL = N \times L$$

where:

N = number of deaths.

L = standard life expectancy at age of death (in years).

The DALY is based on the premise that the best approach for measuring the burden of disease is to use units of time. Having chosen units of time as the unit of measure, the burden of disease can still be calculated using incidence or prevalence measures. Time lost due to premature mortality is a function of the death rate and the duration of life lost due to a death at each age. Because death rates are incidence rates, there is no obvious alternative for mortality than to use an incidence perspective. By contrast, for non-fatal health outcomes, both incidence and prevalence measures have been routinely used. Thus, it is possible to calculate the number of healthy years of life lost because of people living in disease states, in terms of prevalent cases of disease in the population in the year of interest, or in terms of the incident stream of healthy years of life lost into the future for incident cases of the disease in the year of interest.

As noted above, the DALY measures the gap between the actual health status of a population and some “ideal” or reference status, using time as the measure. In developing the DALY indicator, Murray & Lopez (1996) identified two key value choices:

- how long “should” people in good health expect to live?
- how should we compare years of life lost through death, with years lived with poor health or disability of various levels of severity?

The first of these choices relates to the standard life expectancy used to calculate the YLL, and the second to the development of disability weights described in the following section.

3.3 Quantifying time lived with disability

There are at least two ways of measuring the aggregate time lived with a disability. One method is to take point prevalence measures of disability, adjusting for seasonal variation if present, and express them as an annual prevalence. The alternative is to measure the incidence of disabilities and the average duration of each disability. The product of the incidence and the duration will then provide an estimate of the total time lived with disability. This is the approach used for the DALY.

To estimate YLD on a population basis, the number of disability cases is multiplied by the average duration of the disease and a weight factor that reflects the severity of the disease on a scale from 0 (perfect health) to 1 (dead). The basic formula (without applying social preferences) for one disabling event is:

$$YLD = I \times DW \times L$$

where:

YLD = years lived with disability.

I = number of incident cases.

DW = disability weight.

L = average duration of disability (years)

To use time as a common currency for non-fatal health states and for YLL, time must be defined and measured for living in non-fatal health states. To place a value on the time lived in non-fatal health states, health state weights are used to formalize and quantify social preferences for different states of health. Depending on how these weights are derived, they are referred to as disability weights, QALY weights, health state valuations, health state preferences or health state utilities. Most such weights are measured as a number on a scale of 0-1, where 0 is assigned to a state comparable to death and 1 is assigned to a state of ideal health. This assignment for the QALY is inverted compared to that used for the DALY (where 0 = perfect health and 1 = death), because the QALY measures equivalent healthy years lived, whereas the DALY measures loss of health.

Although the disability weights used in DALY calculations quantify societal preferences for different health states, the weights do not represent the lived experience of any disability or health state, or imply any societal value for the person in a disability or health state. Rather, they quantify societal preferences for health states in relation to the societal ideal of good health. Thus, a weight for paraplegia of 0.57 does not mean that a person in this health state is “half dead”, that they experience their life as halfway between life and death, or that society values them less as a person compared to “healthy” people. It means that, on average, society judges a year with blindness (weight 0.43) to be preferable to a year with paraplegia (weight 0.57), and a year with paraplegia to be preferable to a year with unremitting unipolar major depression (weight 0.76). It also means that, on average, society would prefer a person to have a year in good health followed by death, than a year with paraplegia followed by death. Society would also prefer a person to live three years with paraplegia followed by death (3 years x 0.57 = 1.7 lost “healthy” years), than have one year of good health followed by death (2 lost years of good health).

Following the GBD terminology, and consistent with the WHO International Classification of Functioning, Disability and Health (ICF), the term “disability” is used broadly in BoD analyses to refer to departures from good or ideal health in any of the important domains of health. These include mobility, self-care, participation in usual activities, pain and discomfort, anxiety and depression, and cognitive impairment. In some contexts, “health” is understood to mean “absence of illness”, but in the context of summary measures of population health, health is given a broader meaning. As well as implying the absence of illness, it also means that there are no impairments or functional limitations due to previous illness or injury. Note that disability (i.e. a state other than ideal health) may be short-term or long-term. For example, a day with a common cold is a day with disability.

Ideally, any weighting to be used in BoD analyses or economic evaluations should measure preferences for clearly defined health states. The Global Burden of Disease Study 1990 asked small groups of participants (medical and public health experts) to make a judgement about the severity of the condition and the preference for time spent in each severity level. To a large extent, this was necessitated by the lack of population information on the severity distribution of most conditions at global and regional levels. Table 3.1 gives some examples of disability weights.

The Netherlands has carried out a project to measure weights for 53 diseases of public health importance, using a methodology consistent with the GBD study (Stouthard et al., 1997). This study used more-specific disease stages or severity levels, so that judgements about the distribution of disease stages or severity levels in the population were not required. In addition, the study defined each disease stage in terms of the associated

average levels of disability, handicap, mental well-being, pain and cognitive impairment, using a modified version of the EuroQol health status instrument.

The GBD 2000 project has adopted a similar approach to health state valuation, using a standard health state description based on eight core domains of health (mobility, self care, pain and discomfort, cognition, interpersonal activities, vision, sleep and energy, affect).

As part of the World Health Survey being conducted by WHO (WHO, 2003), revised disability weights will be developed during 2003 that are based on health state valuations from large representative population samples in over 70 countries.

Table 3.1 Examples of disability weights^a

Disease or sequelae	Mean disability weight (untreated form)	Mean disability weight (treated form)
AIDS	0.50	0.50
Infertility	0.18	0.18
Diarrhoea disease, episodes	0.11	0.11
Measles episode	0.15	0.15
Tuberculosis	0.27	0.27
Malaria, episodes	0.20	0.20
Trachoma, blindness	0.60	0.49
Trachoma, low vision	0.24	0.24
Lower respiratory tract infection, episodes	0.28	0.28
Lower respiratory tract infection, chronic sequelae	0.01	0.01
Cancers, terminal stage	0.81	0.81
Diabetes mellitus cases (uncomplicated)	0.01	0.03
Unipolar major depression, episodes	0.60	0.30
Alcohol dependence syndrome	0.18	0.18
Parkinson disease cases	0.39	0.32
Alzheimer disease cases	0.64	0.64
Post-traumatic stress disorder	0.11	0.11
Angina pectoris	0.23	0.10
Congestive heart failure	0.32	0.17
Chronic obstructive lung disease, symptomatic cases	0.43	0.39
Asthma, cases	0.10	0.06
Deafness	0.22	0.17
Benign prostatic hypertrophy	0.04	0.04
Osteoarthritis, symptomatic hip or knee	0.16	0.11
Brain injury, long-term sequelae	0.41	0.35
Spinal cord injury	0.73	0.73
Sprains	0.06	0.06
Burns (>60%) – long term	0.25	0.25

^a Adapted from Murray & Lopez (1996).

3.4 Other social values

All summary measures of population health involve explicit or implicit social value choices. In particular, the DALY measures the gap between the actual health status of a population and some “ideal” or reference status. In developing the DALY indicator, Murray & Lopez (1996) identified five value choices: in addition to the two discussed

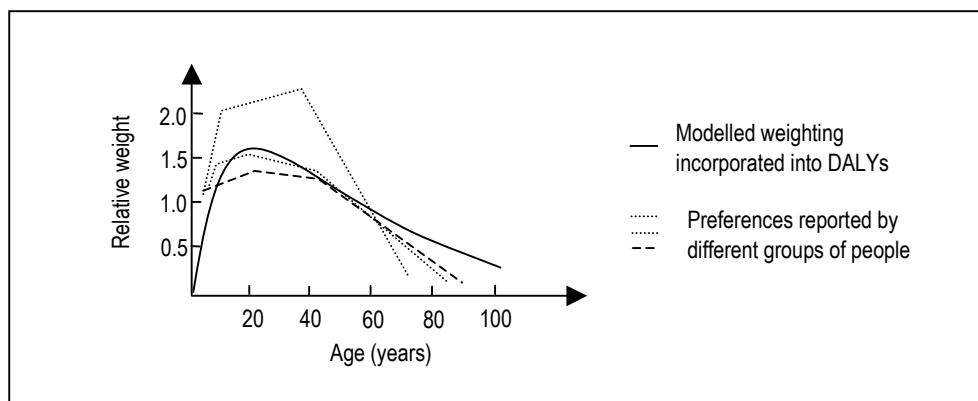
above (standard years of life lost for a death, and disability weights), they took into account three additional social choices:

- Is a year of healthy life gained now worth more to society than a year of healthy life gained sometime in the future, for instance in 20 years?
- Are lost years of healthy life valued more at some ages than others?
- Are all people equal? For a given age, do all people lose the same amount of health through death, even if current life expectancies vary between population groups?

In the GBD study, a year of healthy life lived at younger and older ages was weighted lower than for other ages. In other words, the GBD study chose to value a year of life in young adulthood more than a year in old age or infancy. This choice was based on a number of studies that have indicated there is a broad social preference to value a year lived by a young adult more highly than a year lived by a young child, or lived at older ages (Institute of Medicine, 1986; Murray, 1996). Not all such studies agree that younger and older ages should be given less weight, nor do they agree about the relative magnitude of the differences.

A general pattern of the valuation of health according to age is represented in Figure 3.1.

Figure 3.1 Relative value of a year of life lived, by age: reported preferences and modelling^a



^aAdapted from Murray & Lopez (1996).

The function used to model the relative age weights is:

$$X_w = Cx^{-\beta x}$$

where:
 X_w = weighted age (years)
 C = constant
 β = constant
 x = age (years)

Age weights are the most controversial value choice built into the DALY. Some find age weights unacceptable on equity grounds (every year of life is of equal value *a priori*), others on empirical grounds (the standard age weights do not reflect actual social values). Murray & Acharya (1997) have argued that age weights are not in themselves inequitable,

because everyone potentially lives through every age, and that they do reflect legitimate societal priorities.

Studies have shown that people have preferences regarding the moment at which death or disability occur (Murray, 1996; Murray & Acharya, 1997). People generally prefer a healthy year of life immediately, rather than in the future, if given the choice. The DALY measures the future stream of healthy years of life lost due to each incident case of disease or injury. It is thus an incidence-based measure, rather than a prevalence-based measure. To estimate the net present value of years of life lost, the GBD study applied a 3% time discount rate to years of life lost in the future. With this discount rate, a year of healthy life gained 10 years from now is worth 24% less than a year gained now.

For many years, a discount rate of 5% per annum has been standard in many economic analyses of health and in other social policy analyses, but recently environmentalists and renewable energy analysts have argued for lower discount rates for social decisions. The World Bank Disease Control Priorities study and the GBD project both used a 3% discount rate, and the US Panel on Cost-Effectiveness in Health and Medicine recently recommended that economic analyses of health also use a 3% real discount rate to adjust both costs and health outcomes (Gold et al., 1996). However, the panel also recommended that the sensitivity of the results to the discount rate should be examined.

Some recent NBD studies have chosen to include time discounting (see below), but not age weights, in the DALY calculations. Although WHO is continuing to report time-discounted and age-weighted DALYs as their standard, for the GBD 2000 study WHO is also making non-age-weighted DALYs available, with and without discounting. EBD studies may choose to weight or not to weight, depending on local preferences, but such studies should also compute standard age-weighted and discounted DALYs, so that the results can be compared with other, international studies.

3.5 Calculation of DALYs with discounting and age weighting

Discounting health with time reflects the social preference of a healthy year now, rather than in the future. To do this, the value of a year of life is generally decreased annually by a fixed percentage. For example, with a 3% discount rate, the YLL is:

$$YLL = \frac{N}{r} (1 - e^{-rL})$$

where:

N = number of deaths.

L = standard life expectancy at age of death (years).

r = discount rate (e.g. 3% corresponds to a discount rate of 0.03).

Similarly, the formula for YLD is:

$$YLD = \frac{I \times DW \times L (1 - e^{-rL})}{r}$$

where:

I = number of incident cases (-).
 DW = disability weight (-).
 L = duration of disability (years).
 r = discount rate.

If both age-weighting and discounting are applied, and the years between the event and the life expectancy are summed, the initially simple formulas for YLL and YLD become more complicated (formula for a single death). These formulas have also been programmed into calculation spreadsheet templates for DALYs that are available at the WHO website (see Annex 3.1).

$$YLL = \frac{KCe^{ra}}{(r + \beta)^2} [e^{-(r + \beta)(L + a)} [-(r + \beta)(L + a) - 1] - e^{-(r + \beta)a} [-(r + \beta)a - 1]] + \frac{1 - K}{r} (1 - e^{-rL})$$

where:

a = age of death (years).
 r = discount rate (usually 3%).
 β = age weighting constant (e.g. β=0.04).
 K = age-weighting modulation constant (e.g. K=1).
 C = adjustment constant for age-weights (e.g. C=0.1658).
 L = standard life expectancy at age of death (years).

Similarly, by replacing the standard life expectancy in the YLL formula by the duration of disease and by multiplying by the disability weight, the YLD formula becomes the following (for a single disabling event):

$$YLD = DW \left\{ \frac{KCe^{ra}}{(r + \beta)^2} [e^{-(r + \beta)(L + a)} [-(r + \beta)(L + a) - 1] - e^{-(r + \beta)a} [-(r + \beta)a - 1]] + \frac{1 - K}{r} (1 - e^{-rL}) \right\}$$

where:

a = age of death (years).
 r = discount rate (usually 3%).
 C, β, K = constants (see previous legend).
 L = duration of disability (years).
 DW = disability weight.

A calculation example for YLL is outlined in Box 3.1, using the spreadsheet template in Annex 3.1 to calculate DALYs. Similarly, an example calculation for YLD is provided in Box 3.2.

Box 3.1 Example of a Microsoft Excel worksheet for calculating YLL

DALY Parameters

0.03 Discount rate (r)	Standard discount rate is 0.03
0.04 Beta (b)	Standard age weights use beta=0.04
0.1658 Constant (C)	Standard age weights use C=0.1658
-0.07 -(b+r)	
0 K	K=0 (no age weights) to 1 (full age weights)

YLL for diarrhoea in Afro E^a

	Population	Deaths	Deaths per 1000	Av. Age at death	Standard LE		YLLs	YLL per 1000
Males								
0-4	28 798 446	185 041	6.43	1.0	79.1	1.000	5 592 777	194.2
5-14	46 759 585	4 781	0.10	9.6	70.8	1.000	140 302	3.0
15-29	46 561 308	4 159	0.09	22.6	57.9	1.000	114 211	2.5
30-44	25 954 027	6 633	0.26	37.6	43.0	1.000	160 316	6.2
45-59	12 912 750	16 114	1.25	52.6	28.7	1.000	310 027	24.0
60-69	4 393 171	12 381	2.82	65.6	17.2	1.000	166 352	37.9
70-79	1 936 466	8 118	4.19	75.6	10.1	1.000	71 002	36.7
80+	417 445	1 518	3.64	85.6	5.3	1.000	7 389	17.7
Total	167 733 198	238 745	1.42	12.5	68.1		6 562 376	39.1
Females								
0-4	28 397 245	141 678	4.99	1.0	81.6	1.000	4 314 133	151.9
5-14	46 568 440	8 555	0.18	9.6	73.4	1.000	253 613	5.4
15-29	46 558 897	7 066	0.15	22.6	60.5	1.000	197 223	4.2
30-44	26 115 846	8 207	0.31	37.6	45.9	1.000	204 520	7.8
45-59	13 765 772	13 773	1.00	52.6	31.7	1.000	281 639	20.5
60-69	5 173 647	6 818	1.32	65.6	20.0	1.000	102 528	19.8
70-79	2 533 372	6 053	2.39	75.6	12.1	1.000	61 468	24.3
80+	700 406	2 116	3.02	86.6	5.8	1.000	11 321	16.2
Total	169 813 625	194 265	0	12.9	70.3		5,426 445	32.0

^aAfro E includes 21 countries of sub-Saharan Africa that have the highest mortality pattern

Box 3.2 Example of a Microsoft Excell Worksheet for calculating YLD (single sequela)

DALY Parameters								
	0.03	Discount rate (r)						
	0.04	Beta (b)			Standard discount rate is 0.03			
	0.1658	Constant (C)			Standard age weights use beta = 0.04			
	-0.07	-(b+r)			Standard age weights use C = 0.1658			
	1	K			K=0 (no age weights) to 1 (full age weights)			
YLD for Alzheimer and other dementias								
Australia	Population (x100 000)	Incidence	Incidence per 100 000	Age at onset	Duration	Disability weight	YLDs	YLD per 100 000
Males								
0-4	6.66	0	0	2.5	0.0	0.512	0.0	0.0
5-14	13.39	0	0	10	0.0	0.512	0.0	0.0
15-24	13.64	0	0	20	0.0	0.512	0.0	0.0
25-34	14.31	0	0	30	0.0	0.512	0.0	0.0
35-44	14.03	0	0	40	0.0	0.512	0.0	0.0
45-54	11.72	117	10	50	23.7	0.512	913.5	78.0
55-64	7.74	665	86	59.9	14.5	0.512	3 087.7	399.1
65-74	6.14	1 828	298	69.8	9.2	0.512	4 766.0	776.8
75+	3.46	6 918	2 001	80.7	3.8	0.512	6 404.1	1 852.2
	91.08	9 529	105	76.8	5.8	0.51	15 171	166.6
Females								
0-4	6.31	0	0	2.5	0.0	0.512	0.0	0.0
5-14	12.75	0	0	10	0.0	0.512	0.0	0.0
15-24	13.12	0	0	20	0.0	0.512	0.0	0.0
25-34	14.31	0	0	30	0.0	0.512	0.0	0.0
35-44	14.08	0	0	40	0.0	0.512	0.0	0.0
45-54	11.37	114	10	50	28.3	0.512	960.7	84.5
55-64	7.64	657	86	60	18.4	0.512	3 519.9	460.6
65-74	6.82	2 052	301	69.9	11.9	0.512	6 439.1	944.5
75+	5.62	11 482	2 043	81.3	4.3	0.512	11 557.7	2 056.5
	92.03	14 305	155	78.4	6.2	0.51	22 477.3	244.2

3.6 Relating summary measures of health to the causes of loss of health

BoD analysis quantifies loss of health in any of the important domains of health, including mobility, self-care, participation in usual activities, pain and discomfort, anxiety and depression, and cognitive impairment. Diseases and injuries are understood as proximal causes of loss of health, and risk factors and environmental determinants as distal causes of loss of health.

One fundamental goal in constructing summary measures is to identify the relative magnitude of different health problems according to causes (including diseases, injuries and risk factors). There are two widely used ways to attribute cause: categorical attribution and counterfactual analysis.

In categorical attribution, an event such as death is attributed to a single cause according to a defined set of rules. Thus, a death resulting from a combination of malnutrition and measles is categorically attributed either to malnutrition or to measles, according to the

rules of the International Classification of Diseases. Such rules inevitably involve grey areas and degrees of arbitrariness in dealing with multicausality and comorbidity.

In counterfactual analysis, the contribution of a disease, injury or risk factor is estimated by comparing the current and future levels of a summary measure with the levels that would be expected under some alternative hypothetical scenario. For example, we could ask what the BoD would be if no one in the population had ever smoked. By comparing this estimate with the actual current burden, we can estimate the attributable burden of tobacco smoking.

Health gap measures use categorical attribution to attribute the fatal and non-fatal burden of diseases and injuries to an exhaustive and mutually exclusive set of disease and injury categories. In contrast, counterfactual analysis is generally used in health gap measures to attribute the BoD to health determinants and risk factors. For EBD studies, the use of counterfactual analysis to estimate the disease burden associated with risk factors is described in Section 4 and in the guides for specific risk factors.

3.7 The GBD 2000 study – an analysis of global mortality patterns

New life tables and detailed distributions of the causes of death have been developed for all 191 WHO Member States for the years 2000. The data are based on a systematic review of all available evidence from surveys, censuses, sample registration systems, population laboratories, and national vital registration systems on the levels and trends of child and adult mortality (Mathers et al., 2002). Complete or incomplete vital registration data, together with sample registration systems, cover 72% of global mortality. Survey data and indirect demographic techniques provide information on the levels of child and adult mortality for the remaining 28% of estimated global mortality. Separate estimates have been made of the numbers and distributions of deaths due to HIV/AIDS in countries with a substantial HIV epidemic.

3.8 The GBD 2000 study – epidemiological analyses for calculating YLD

The key to estimating YLD is to develop comprehensive and consistent estimates for the incidences and point prevalences of diseases. WHO programme participation in the development and finalization of the estimates ensures that final estimates reflect all information and knowledge available to WHO. A wide range of data sources are used for the analysis of incidence, prevalence and YLD, including disease registers, population surveys, epidemiological studies and health facility data (Mathers et al., 2002).

Work is underway to document and publish the epidemiological reviews underlying the GBD 2000 estimates. Draft documentation is available on the WHO website at www.who.int/evidence/nbd together with regional summary tables of the GBD 2000.

3.9 Main findings from the GBD 2000 study

Version 2 estimates of the GBD 2000 study are listed in the World Health Report 2002. This report also presents an analysis of the attributable burden for 20 major risk factors. Methods and results for the GBD 2000 study are described in more detail in Mathers et al. (2002). The main findings of the GBD studies can be summarized as a ranking of diseases

(Table 3.2) and risk factors (Table 3.3), according to their global importance in deaths and DALYs.

Table 3.2 Disease rankings according to the GBD 2000 study (version 2)^a

Disease	Deaths (thousands)	As % of total deaths	DALYs (thousands)	As % of total DALYs
Ischaemic heart disease	7 033	12.6%	57 626	4.0%
Lower respiratory infections	6 164	11.1%	91 160	6.3%
Cerebrovascular disease	5 344	9.6%	45 088	3.1%
Chronic obstructive pulmonary disease	2 621	4.7%	29 371	2.0%
HIV/AIDS	2 570	4.6%	79 992	5.5%
Perinatal conditions	2 505	4.5%	98 424	6.8%
Diarrhoeal diseases	2 020	3.6%	63 346	4.4%
Tuberculosis	1 569	2.9%	35 302	2.4%
Road traffic accidents	1 203	2.2%	38 061	2.6%
Trachea, bronchus, lung cancers	1 201	2.1%	11 195	0.8%

^a Sources: Mathers et al. (2002); WHO (2002b).

Table 3.3 Selected risk factor rankings ^a

Risk factor	DALYs (thousands)	As % total DALYs	Deaths (thousands)	As % total deaths
Underweight	137 801	9.4%	3 748	6.6%
Unsafe sex	91 869	6.3%	2 886	5.1%
Blood pressure	64 270	4.4%	7 141	12.6%
Tobacco	59 081	4.0%	4 907	8.7%
Alcohol	58 323	4.0%	1 804	3.2%
Unsafe water, sanitation and hygiene	54 158	3.7%	1 730	3.1%
Cholesterol	40 437	2.8%	4 415	7.8%
Indoor smoke from solid fuels	38 539	2.6%	1 619	2.9%
Iron deficiency	35 057	2.4%	841	1.5%
Overweight	33 415	2.3%	2 591	4.6%
Zinc deficiency	28 034	1.9%	789	1.4%
Low fruit and vegetable intake	26 662	1.8%	2 726	4.8%
Vitamin A deficiency	26 638	1.8%	778	1.4%
Physical inactivity	19 092	1.3%	1 922	3.4%
Occupational risk factors for injury	13 125	0.9%	310	0.5%
Lead exposure	12 926	0.9%	234	0.4%
Illicit drugs	11 218	0.8%	204	0.4%
Unsafe health care injections	10 461	0.7%	501	0.9%
Lack of contraception	8 814	0.6%	149	0.3%
Childhood sexual abuse	8 235	0.6%	79	0.1%
Urban air pollution	7 865	0.5%	799	1.4%
Climate change	5 517	0.4%	154	0.3%
Occupational noise	4 151	0.3%	0	0.0%
Occupational airborne particulates	3 038	0.2%	243	0.4%
Occupational carcinogens	1 421	0.1%	146	0.3%
Occupational ergonomic stressors	818	0.1%	0	0.0%

^a Source: WHO (2002a)

Main Issues

- The GBD study is the most comprehensive and consistent set of estimates of morbidity and mortality by age, sex and region.
- The DALY is a summary measure of population health, combining mortality and disability.
- The DALY measures a health gap, relative to an “ideal” life expectancy of 80 years for men and 82.5 years for women.
- The DALY is the sum of years of life lost and years of life lived with disability.
- A disability weight is used to characterize each disease or sequelae.
- Social preferences for the point in time or age at which a death or disability occurs are incorporated into DALY calculations.

Annex 3.1: An example of a DALY calculation template

(available at www.who.int/evidence/nbd, under “other files”)

1	A	B	C	D	E	F	G	H	I	J	K	
2	DISEASE:	[Enter Disease/Sequela name]						Updated: [Date]				
3	REGION:	[Enter Country or Region name]						By: [Name]				
4	PERIOD:	[Enter reference year]						Status: [Preliminary, draft, final]				
5												
6												
7	THIS TEMPLATE ENABLES CALCULATION OF YLL (See Part A below in rows 26 to 104)											
8	YLD (See Part B below in rows 107 to 135)											
9	DALYs (See Part C below in rows 138 to 152)											
10												
11	IF YOU HAVE MORE THAN ONE SEQUELA FOR A DISEASE, CREATE A COPY OF THIS TEMPLATE											
12	FOR EACH SEQUELA AND ADD THE DALYs FOR ALL SEQUELAE.											
13												
14	1. Enter disease name, region and period in the yellow cells above.											
15	2. Enter update information in the purple cells above right.											
16	3. If required, change discount and age weight parameters for DALY calculation in the grey box below.											
17	4. If required, change age groups (insert additional rows if needed, and adjust lookup formulae for standard LE)											
18												
19	DALY Parameters											
20	0.03 Discount rate (r)						Standard discount rate is 0.03					
21	0.04 Beta (b)						Standard age weights use beta=0.04					
22	0.1658 Constant (C)						Standard age weights use C=0.1658					
23	-0.07 -(b+)											
24	0 K						K=0 (no age weights) to 1 (full age weights)					
25												
26	A. YLL template											
27	A1. Enter population data in yellow cells (Column B) below.											
28	A2. Enter numbers of deaths for 5 year age groups in green cells (Column C) below. (or death rates in next column and calculate numbers of deaths)											
29	A3. If necessary, modify average ages at death (blue column (E)). This may be important for lowest and highest age groups.											
30		Population	Deaths	Deaths per 1,000	Av. Age at death	Standard LE		YLLs	YLL per 1,000			
31												
32												
33												
34	Males											
35	0	1,500,000	2,000	1.33	0.1	79.9	1.000	60,607	40.4			
36	1-4	6,000,000	0	0.00	2.6	77.8	1.000	-	0.0			
37	5-9	7,250,000	0	0.00	7.3	73.1	1.000	-	0.0			
38	10-14	7,500,000	0	0.00	12.9	67.5	1.000	-	0.0			
39	15-19	7,500,000	1,000	0.13	18.1	62.4	1.000	28,203	3.8			
40	20-24	7,500,000	1,000	0.13	22.5	57.9	1.000	27,468	3.7			
41	25-29	7,500,000	1,000	0.13	27.5	53.0	1.000	26,536	3.5			
42	30-34	7,500,000	1,000	0.13	32.6	48.0	1.000	25,428	3.4			
43	35-39	7,250,000	1,000	0.14	37.5	43.1	1.000	24,181	3.3			
44	40-44	7,000,000	2,000	0.29	42.6	38.1	1.000	45,407	6.5			
45	45-49	6,500,000	3,000	0.46	47.7	33.2	1.000	63,071	9.7			
46	50-54	6,000,000	4,000	0.67	52.6	28.5	1.000	76,672	12.8			
47	55-59	5,500,000	5,000	0.91	57.6	23.9	1.000	85,394	15.5			
48	60-64	5,000,000	6,000	1.20	62.7	19.5	1.000	88,605	17.7			
49	65-69	4,000,000	7,000	1.75	67.7	15.4	1.000	86,314	21.6			
50	70-74	3,000,000	8,000	2.67	72.6	11.8	1.000	79,672	26.6			
51	75-79	2,000,000	8,000	4.00	77.5	8.8	1.000	62,081	31.0			
52	80-84	1,000,000	7,000	7.00	82.4	6.4	1.000	40,608	40.6			
53	85+	500,000	6,000	12.00	89.0	3.9	1.000	21,991	44.0			
54	Total	100,000,000	63,000	0.63	63.9	20.5		842,238	8.4			
55	Females											
56	0	1,500,000	2,000	1.33	0.1	82.4	1.000	61,045	40.7			
57	1-4	6,000,000	0	0.00	2.6	80.3	1.000	-	0.0			
58	5-9	7,250,000	0	0.00	7.4	75.6	1.000	-	0.0			
59	10-14	7,500,000	0	0.00	12.6	70.4	1.000	-	0.0			
60	15-19	7,500,000	1,000	0.13	17.9	65.2	1.000	28,613	3.8			
61	20-24	7,500,000	1,000	0.13	22.6	60.5	1.000	27,910	3.7			
62	25-29	7,500,000	1,000	0.13	27.5	55.7	1.000	27,072	3.6			
63	30-34	7,500,000	1,000	0.13	32.6	50.7	1.000	26,048	3.5			
64	35-39	7,250,000	1,000	0.14	37.5	45.9	1.000	24,926	3.4			
65	40-44	7,000,000	2,000	0.29	42.7	41.0	1.000	47,169	6.7			
66	45-49	6,500,000	3,000	0.46	47.7	36.2	1.000	66,219	10.2			
67	50-54	6,000,000	4,000	0.67	52.6	31.6	1.000	81,688	13.6			
68	55-59	5,500,000	5,000	0.91	57.7	26.9	1.000	92,301	16.8			
69	60-64	5,000,000	6,000	1.20	62.6	22.5	1.000	98,259	19.7			
70	65-69	4,000,000	7,000	1.75	67.6	18.2	1.000	98,177	24.5			
71	70-74	3,000,000	8,000	2.67	72.6	14.1	1.000	92,178	30.7			
72	75-79	2,000,000	8,000	4.00	77.6	10.5	1.000	72,305	36.2			
73	80-84	1,000,000	7,000	7.00	82.6	7.5	1.000	47,198	47.2			
74	85+	500,000	6,000	12.00	90.0	4.3	1.000	23,941	47.9			
75	Total	100,000,000	63,000	0	64.0	22.7		915,051	9.2			
76												

Annex 3.1: An example of a DALY calculation template (cont'd)

	A	B	C	D	E	F	G	H	I	J	K
78											
79	A1. YLL in study age groups										
80											
81		Population	Deaths	Deaths per 1,000	Av. Age at death			YLLs	YLL per 1,000		
82											
83											
84	Males										
85	0-4	7,500,000	2,000	0.3	0.1			60,607	8.1		
86	5-14	14,750,000	-	0.0				-	0.0		
87	15-29	22,500,000	3,000	0.1	22.7			82,207	3.7		
88	30-44	21,750,000	4,000	0.2	38.8			95,016	4.4		
89	45-59	18,000,000	12,000	0.7	53.5			225,138	12.5		
90	60-69	9,000,000	13,000	1.4	65.4			174,919	19.4		
91	70-79	5,000,000	16,000	3.2	75.0			141,752	28.4		
92	80+	1,500,000	13,000	8.7	85.5			62,599	41.7		
93	Total	100,000,000	63,000	0.6	63.9			842,238	8.4		
94											
95	Females										
96	0-4	7,500,000	2,000	0.3	0.1			61,045	8.1		
97	5-14	14,750,000	-	0.0				-	0.0		
98	15-29	22,500,000	3,000	0.1	22.7			83,595	3.7		
99	30-44	21,750,000	4,000	0.2	38.9			98,143	4.5		
100	45-59	18,000,000	12,000	0.7	53.5			240,208	13.3		
101	60-69	9,000,000	13,000	1.4	65.3			196,437	21.8		
102	70-79	5,000,000	16,000	3.2	75.1			164,484	32.9		
103	80+	1,500,000	13,000	8.7	86.0			71,140	47.4		
104	Total	100,000,000	63,000	0.6	64.0			915,051	9.2		
105											
106											
107	B. YLD template										
108	B1. Enter population data in yellow cells (Column B) below (if have not entered them above for YLL).										
109	B2. Enter incidence rates, age at onset and duration in green cells (Columns D, E and F)										
110	B3. Enter disability weights in blue cells (Column G) below.										
111											
112		Population	Incidence	Incidence per 1,000	Age at onset (years)	Duration (years)	Disability Weight	YLDs	YLD per 1,000		
113											
114											
115	Males										
116	0-4	7,500,000	0	0	2.5	0.0	0.500	-	0.0		
117	5-14	14,750,000	0	0	10.0	0.0	0.500	-	0.0		
118	15-29	22,500,000	0	0	22.5	0.0	0.500	-	0.0		
119	30-44	21,750,000	0	0	37.5	0.0	0.500	-	0.0		
120	45-59	18,000,000	0	0	52.5	0.0	0.500	-	0.0		
121	60-69	9,000,000	18,000	2	65.0	10.0	0.500	77,755	8.6		
122	70-79	5,000,000	50,000	10	75.0	5.0	0.500	116,077	23.2		
123	80+	1,500,000	45,000	30	85.0	3.0	0.500	64,552	43.0		
124	Total	100,000,000	113,000	1.1	77.4	5.0	0.50	258,383	2.6		
125											
126	Females										
127	0-4	7,500,000	0	0	2.5	0.0	0.500	-	0.0		
128	5-14	14,750,000	0	0	10.0	0.0	0.500	-	0.0		
129	15-29	22,500,000	0	0	22.5	0.0	0.500	-	0.0		
130	30-44	21,750,000	0	0	37.5	0.0	0.500	-	0.0		
131	45-59	18,000,000	0	0	52.5	0.0	0.500	-	0.0		
132	60-69	9,000,000	27,000	3	65.0	10.0	0.500	116,632	13.0		
133	70-79	5,000,000	75,000	15	75.0	5.0	0.500	174,115	34.8		
134	80+	1,500,000	60,000	40	85.0	3.0	0.500	86,069	57.4		
135	Total	100,000,000	162,000	1.6	77.0	5.1	0.50	376,816	3.8		
136											
137											
138	C. Total DALYS = YLL+YLD										
139											
140		Population	Males		Population	Females		Persons			
141			DALYs	DALYs per 1,000		DALYs	DALYs per 1,000	Population	DALYs	DALYs per 1,000	
142											
143	Age										
144	0-4	7,500,000	60,607	8.1	7,500,000	61,045	8.1	15,000,000	121,652	8.1	
145	5-14	14,750,000	-	-	14,750,000	-	-	29,500,000	-	-	
146	15-29	22,500,000	82,207	3.7	22,500,000	83,595	3.7	45,000,000	165,801	3.7	
147	30-44	21,750,000	95,016	4.4	21,750,000	98,143	4.5	43,500,000	193,159	4.4	
148	45-59	18,000,000	225,138	12.5	18,000,000	240,208	13.3	36,000,000	465,346	12.9	
149	60-69	9,000,000	252,674	28.1	9,000,000	313,068	34.8	18,000,000	565,742	31.4	
150	70-79	5,000,000	257,829	51.6	5,000,000	338,599	67.7	10,000,000	596,428	59.6	
151	80+	1,500,000	127,151	84.8	1,500,000	157,208	104.8	3,000,000	284,359	94.8	
152	Total	100,000,000	1,100,621	11.0	100,000,000	1,291,867	12.9	200,000,000	2,392,487	12.0	

4. Methods for estimating the environmental burden of disease

4.1 General method

Although the basic approach for estimating the EBD is common to every environmental risk factor, the calculations will vary according to the information available in the country, and the form in which this information is held. Specific information needed for the EBD calculations is laid out in each section, but information on the local epidemiology of disease will also be needed.

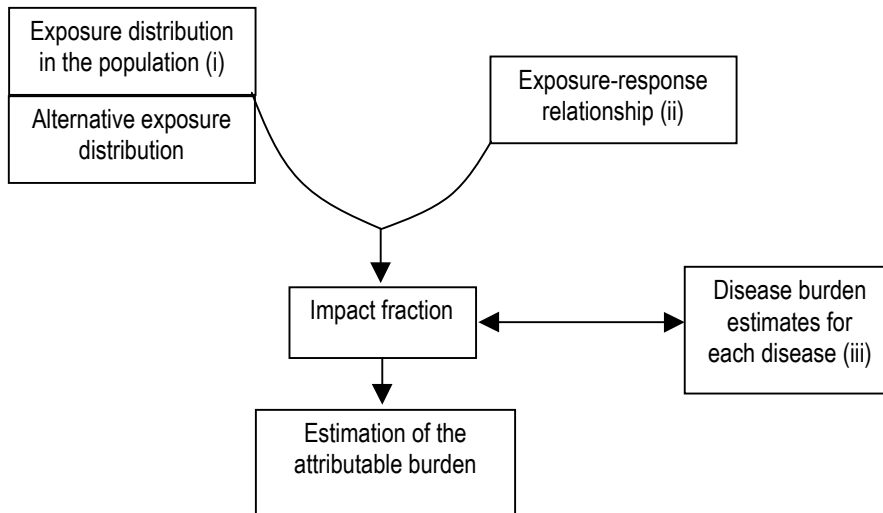
An assessment of the EBD requires the following data for each risk factor:

- i. the distribution of risk factor exposure within the study population;
- ii. the exposure-response relationship for the risk factor;
- iii. the DALYs lost to disease for the risk factor of interest (or other epidemiological information, such as mortality rates or disease incidence, if DALYs are not available).

The distribution of the risk factor exposure in the population (i), and the exposure-response information (ii), are combined into an impact fraction, which is applied to the disease estimates (iii). The impact fraction is the percentage of the population risk that can be attributed to hazardous exposures or risky behaviours, multiple levels of exposure, or to incomplete elimination of exposure. When exposure is measured in terms of increasing levels of pollutants, the approach is called an exposure-based approach (Figure 4.1). For example, exposure to outdoor air pollution is commonly reported as continuous or categorical increases in ambient air pollution.

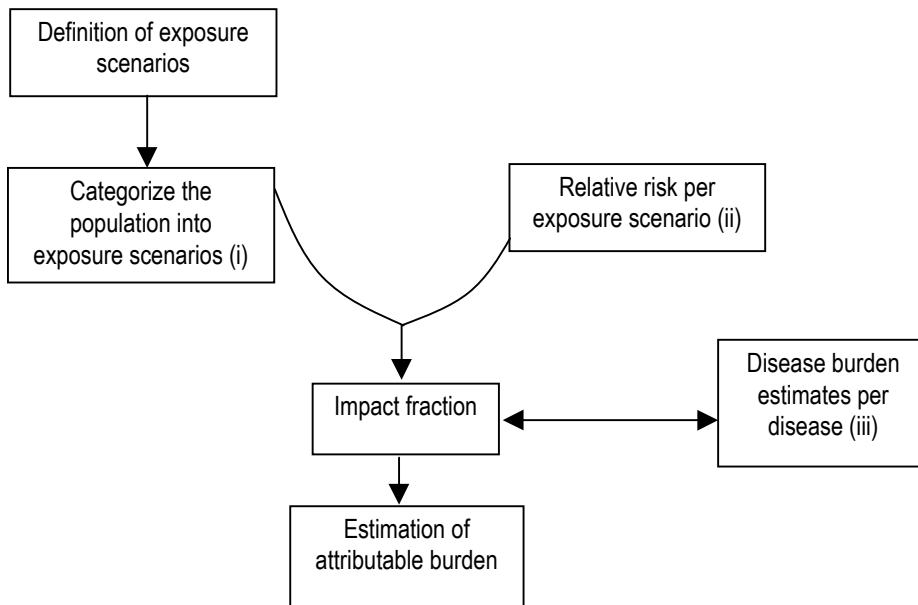
If it is not possible to specify a continuous numerical relationship between the proximal cause of disease and the disease outcomes, for example because of competing relationships between exposures, an alternative approach is to select characteristic exposure scenarios (Figure 4.2). In this way, the study population can be divided into defined exposure scenarios, each of which has a corresponding specific health risk. For example, in the area of water, sanitation and hygiene there are often no direct measurements of drinking-water quality for the entire population of a country. Therefore, exposure scenarios are defined on the basis of more distal causes, such as access to improved water supply and improved sanitation (Prüss-Üstün et al., 2003).

Figure 4.1 Calculating the EBD using an exposure-based approach^a



^a The Roman numerals in parentheses refer to the EBD requirements listed above.

Figure 4.2 Calculating the EBD using a scenario-based approach^a



^a The Roman numerals in parentheses refer to the EBD requirements listed above.

For the exposure assessment (i, above), the following steps are generally followed for each risk factor:

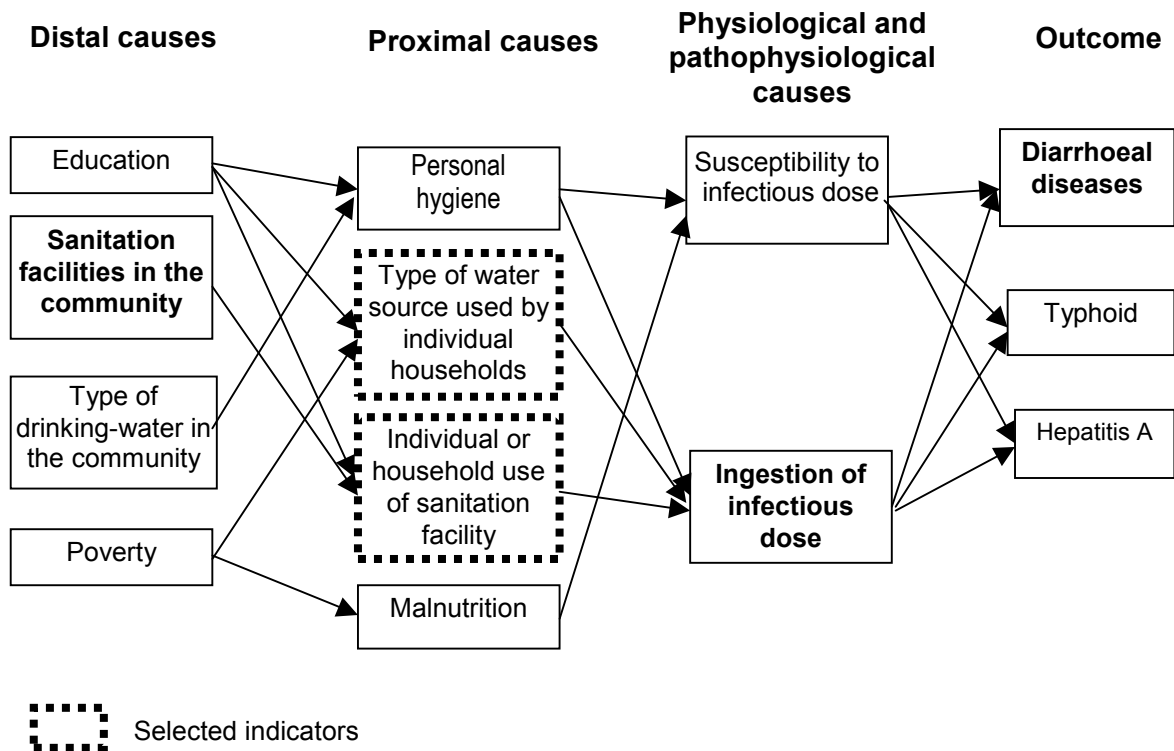
- Step 1* Outline the distal and proximal causes of the disease and their interactions (the causal web) as a framework for assessment.
- Step 2* Choose an indicator to assess exposure. The choice of exposure indicator will be determined by the metric used to assess the exposure-risk relationship.
- Step 3* Define the levels of exposure, and the distribution of the population among the exposure levels, including the unexposed level. Exposure can also be assessed as a continuous variable.

A graphical outline of distal and proximal causes (a causal web) provides a framework for selecting exposure indicators and for displaying the corresponding exposure-risk relationships. An example of the interrelated causes for the transmission of faecal-oral diseases is outlined in Figure 4.3. In this example, the risk of transmission of faecal-oral pathogens in the study population could be assessed by using an indicator that measured access to a water source or sanitation facility. The access, or lack of access, to improved water or sanitation facilities, then define scenarios and the population is distributed into the scenarios. In this way, the exposure distribution is rather simple, and can be summarized as percentages of the population in each scenario. A quantitative example of this approach for calculating the attributable BoD for diarrhoeal disease is outlined in Box 4.1.

The exposure-response relationship (step 2 above) is usually derived from a comprehensive review of the epidemiological literature. If there are good reasons to think that generalized results from other populations cannot be applied directly to the local setting, then local information may be used instead (e.g. if some feature of the local population, such as level of malnutrition, means that the people suffer unusually severe effects from any given level of exposure to the risk factor). Alternatively, schemes exist for combining local studies and global estimates. The type of epidemiological studies that can be used will depend on the parameters needed to assess the EBD of specific risk factors (see the volume in this series on the specific risk factor of interest), and on the quality and size of the study. In any case, an epidemiological study should use an exposure indicator that matches the measure of exposure available for the local population.

Population exposure data (i, above) and exposure-response results (ii, above) are then combined to estimate the population disease burden (iii, above). The calculations can be performed according to one of the methods explained in the following sections. Impact fractions can be applied to NBD estimates to derive the DALYs, or the deaths attributable to the risk factor.

Figure 4.3 Example of a causal web for the faecal-oral transmission of pathogens



The BoD attributable to a specified change in level of a risk factor can be estimated using the formula for the impact fraction (Last, 2001):

$$IF = \frac{\sum P_i RR_i - \sum P'_i RR_i}{\sum P_i RR_i}$$

(Equation 1)

where:

- IF = Impact fraction.
- P_i = Proportion of the population in exposure category i .
- P'_i = Proportion of the population in exposure category i after an intervention or other change.
- RR_i = Relative risk at exposure category i compared to the reference level.

This formula applies to any situation in which a population is distributed into graded levels of exposure. It can be used to estimate the impact of changing the exposure from one distribution to another, for example through a public health intervention. The same formula can also be used to estimate the fraction of the disease burden that is attributable to the risk factor, relative to some alternative or counterfactual level (see below). The counterfactual level could be the minimum disease burden achievable in a given time frame. If the risk factor were to be completely removed, or if exposed populations were to be compared with

unexposed populations, the BoD reduction can be calculated from a simplified form of the above formula:

$$IF = \frac{\sum P_i RR_i - 1}{\sum P_i RR_i} \quad (\text{Equation 2})$$

A generalized potential impact fraction can be calculated for an exposure distributed continuously across the study population (Mathers et al., 2001). However, this approach has not been used to assess environmental risk factors, but rather risks from other areas:

$$IF = \frac{\int_{x=0}^m RR(x)P(x)dx - \int_{x=0}^m RR(x)P'(x)dx}{\int_{x=0}^m RR(x)P(x)dx}$$

(Equation 3)

where:	
x	= Exposure level.
P(x)	= Population distribution of exposure.
P'(x)	= Alternative ("counterfactual") population distribution of exposure.
RR(x)	= Relative risk at exposure level x compared to the reference level.

To calculate the fraction of disease attributable to a risk factor for any defined population, the total disease burden for the population (in deaths and DALYs) is multiplied by the impact fraction. If there is evidence that the exposure distribution or the relative risks differ between subpopulations (such as by age or gender), then the impact fraction should be calculated separately for each subpopulation:

$$\text{Attributable burden}_{(\text{age, sex})} = IF \times \text{total burden}_{(\text{age, sex})} \quad (\text{Equation 4})$$

If the exposure-response relationships are different for mortality and morbidity, the impact fractions should be calculated separately for YLL and YLD. For example, if the case-fatality rate for diarrhoeal disease caused by exposure to unsafe water, sanitation or hygiene is different from that for the average diarrhoeal disease, then the impact fraction should be different for mortality than for DALYs and incidence. This is due to the fact that the relative risks for diarrhoea incidence and diarrhoea mortality would be different.

An example of how to estimate the disease burden attributable to water, sanitation and hygiene for an entire region (AmrD) is outlined in Box 4.1, and a comparison of this disease burden with other disease burdens is given in Box 4.2.

4.2 Alternative or counterfactual exposure

A counterfactual analysis compares current levels of exposure to the exposure that would occur under an alternative, hypothetical scenario. It may be helpful to specify more than one reference scenario. For example, policy-makers may be interested in the health gains that would accrue both from a readily achievable alternative exposure distribution in the population, and from an exposure distribution in the population that would result in the lowest possible disease burden attributable to the risk factor (also called the theoretical minimum exposure).

Box 4.1 Example calculation of the burden of diarrhoeal disease (YLL) attributable to water, sanitation and hygiene^a

Determine the population distribution for the exposure scenarios:

Improved water supply and improved sanitation^b (Scenario IV^c): 68%

Improved water supply, no improved sanitation (Scenario Vb): 7%

No improved water supply and no basic sanitation (Scenario VI): 25%

In this region, no significant part of the population is in any other scenario.

Calculate the relative risks associated with the scenarios:

The reference group for the risk scenarios is defined as: no transmission of diarrhoeal disease through unsafe water, sanitation and hygiene, but transmission only through other exposures. Also, the relative risks used in this example are global averages (Prüss-Üstün et al., 2003), but they could be made more specific by using region-specific data.

Scenario IV: RR=6.9

Scenario Vb: RR=8.7

Scenario VI: RR=11

Process the data

Using the formula for the impact fraction, $IF = \frac{\sum P_i RR_i - 1}{\sum P_i RR_i}$ gives:

$$IF = \frac{(68\%*6.9+7\%*8.7+25\%*11)-1}{68\%*6.9+7\%*8.7+25\%*11} = 87.6\%$$

Calculate the disease burden for diarrhoea

For 2000, the total disease burden of diarrhoea for AmrD was 863 000 DALYs (Mathers et al., 2002). The diarrhoeal disease burden attributable to water, sanitation and hygiene is then 87.6% of this figure, or 756 000 DALYs.

In this particular example, no proportion of the population was unexposed. Had there been an unexposed fraction (P_i), this fraction would have been taken into account in the impact fraction formula (with $RR_i = 1$).

^a The example is given for the AmrD region which is comprised of Bolivia, Ecuador, Guatemala, Haiti, Nicaragua and Peru.

^b Improved water supply and sanitation were defined according to the type of technology (WHO/UNICEF/WSSCC, 2000). Improved water supplies included household connections, public standpipes, boreholes, protected dug wells, and spring or rainwater collection. Improved sanitation included connections to a public sewer or septic system, pour-flush latrines or pit latrines.

^c For a definition of the scenarios see Prüss-Üstün et al. (2003).

Box 4.2 The burden of diarrhoeal disease from poor-quality water, sanitation and hygiene, compared to the burden of other diseases^a

In 2000, the disease burden in AmrD for diarrhoeal disease associated with poor-quality water, sanitation and hygiene was 756 000 DALYs. For comparison, the burden of several other diseases or risk factors in the region are listed below:

Diseases:

HIV:	720 000 DALYs
All cancers:	883 000 DALYs
Nutritional deficiencies;	503 000 DALYs

Risk factors:

Tobacco:	65 000 DALYs
Alcohol:	959 000 DALYs

^a Sources for regional disease burdens: Mathers et al. (2002); WHO (2002a).

The counterfactual exposure level may also be an exposure that cannot be avoided, and thus a disease burden that cannot be avoided. For example, although in theory man-made pollution could be reduced, it may never be possible to reduce the natural exposure to pollutants. For exposure to lead, for example, a counterfactual exposure from natural sources could equal the blood lead levels in pre-industrial humans (0.016 µg/dl). In this particular case, such a low exposure probably does not induce a disease burden, but if it did, then the avoidable burden would amount to the total burden caused by lead minus the burden that is unavoidable.

Another way to define the counterfactual exposure is to define it as the level at which no health effects are observed in the population. For example, at blood lead levels as low as 0-1 µg/dl no health effects have yet been shown. Using this definition, the counterfactual exposure is the theoretical minimum exposure.

4.3 Choosing the study population for an EBD assessment

The study population is the population for which the disease burden is calculated. This guide mainly addresses the population of a nation or region of a country. The study population needs to be selected according to the following criteria:

- *Policy relevance.* The study population should be relevant to the policy needs of the country or region. For example, to address the impact of outdoor air pollution on health, the relevant population might be that of a city, or the urban population of a country. For national policy-making, however, the most common choice of population will be that of the entire country.
- *Data availability.* Information about exposures and outcomes may be available only for a subgroup. Outdoor air pollution, for example, may be monitored in some cities, but not in others, and not at all in rural areas. If information is missing, assumptions will need to be made about the exposures if all cities, or the entire national population, are to be included in the assessment. On the other hand, health data may exist for an entire country, but the data may not be subdivided by demographical or geographical

variables. In such cases, it may be best to perform an assessment for the whole country only. EBD assessments below national level necessarily assume that a particular disease burden is distributed equally throughout the population. However, this is unlikely to be the case, because exposures are likely to be different throughout the country, for example.

- *Population subgroups.* The impact of a risk factor may be limited to a single population subgroup (e.g. the elderly, children, asthmatics, and those immunosuppressed). Also, data may be available for only one of these groups.

4.4 Estimating the EBD when NBD data are available

If a NBD study has already been carried out, the attributable BoD for an environmental risk factor can be obtained simply by multiplying the attributable disease fraction for the risk factor by the total disease burden for the corresponding disease assessed in the NBD study (see Equation 4). NBD studies can be a useful sources of data for EBD assessments, since the former attempt to estimate the disease burden of the major diseases or disease groups at country level (generally, in terms of deaths and DALYs), and the data are sorted by age and gender. However, NBD studies can take months to carry out and involve teams of people with diverse skills. For detailed guidance on how to carry out a NBD study see Mathers et al. (2001). The main result of an NBD consists of a list of the burdens of more than 100 diseases or disease groups, by age group and gender, expressed in deaths and DALYs.

4.5 Estimating the EBD from disease-specific national health statistics when NBD data are not available

If a NBD study has not been carried out, it is still possible to estimate the impact fraction for an environmental risk factor. In such cases, national or subnational mortality and morbidity statistics can be used with the method described in Section 4.4. An example of how to calculate the attributable mortality from diarrhoea that is associated with poor water, sanitation and hygiene is outlined in Box 4.3.

Box 4.3 Estimating the mortality in AmrD from diarrhoea attributable to poor water, sanitation and hygiene^a

The fraction of diarrhoeal disease attributed to poor water, sanitation and hygiene was estimated to be 87.6% for AmrD (Box 4.1). This fraction can be applied to national or subnational mortality and morbidity statistics, provided that the relative risks are the same for mortality and for morbidity. In 2000, for example, the total mortality from diarrhoea in AmrD was 26 000 deaths. The number of these deaths that can be attributed to poor water, sanitation and hygiene can be estimated by multiplying the total number of deaths from diarrhoea by the attributable fraction for the risk factor (87.6%), which amounts to 22 800 deaths.

^a Derived from Mathers et al. (2002). See also Box 4.1.

Such results may certainly be useful for policy-making, as mortality from one risk factor can be directly compared to the number of deaths caused by other risk factors or diseases. However, it is difficult to compare risk factors that induce diseases that are not fatal, but nevertheless cause a high degree of disability. Examples include hearing impairment from exposure to occupational noise, and occupational back pain. In such cases, the risk factors should be expressed in a common unit (such as DALYs lost, or health service dollars spent).

In the absence of a NBD study, an additional problem of internal inconsistency between various figures and health statistics may arise. This could happen for a number of reasons, including the coherence between cases and deaths from the various studies; registers that were not checked; and the total deaths summed for different diseases may not have been checked against the total number of deaths in a country (death certificates, hospital registers etc.). Also, the attributable burden of a specific disease caused by a risk factor should not be unrealistically large, or larger than, the entire “disease envelope” (i.e. the total disease burden of the disease in the study population).

4.6 *Estimating the EBD from limited national or local health statistics when NBD data are not available*

It is more difficult to carry out accurate EBD assessments if little is known about the health problems related to environmental risk factors. However, it may be possible to develop a preliminary estimate of the proportion of the disease burden attributable to the environment. Surveys could be used to estimate the incidence or prevalence of the disease(s) of interest, or it may be possible to use pre-existing regional estimates of disease burden. Such results may be less accurate than national estimates, but if the country has similar epidemiological, socioeconomic and cultural characteristics to the rest of the region, the results are likely to provide good preliminary estimates of the BoD attributable to environmental factors (Box 4.4).

Box 4.4 Acute lower respiratory infections (ALRI) in children under five years old in India^a

In India, 81% of children younger than five years old are exposed to indoor smoke from solid fuels. The relative risk for ALRI in exposed children of this age group is 2.3. According to Equation 2, the resulting attributable fraction is 51% (i.e. 51% of ALRI in children younger than five years old in India is attributable to indoor smoke from solid fuels).

If the total ALRI in India in this age group were not known, a preliminary estimate could be obtained from the per capita rate of ALRI for SearD (which includes India). For example, the total disease burden for ALRI in SearD for children 0-4 years old is 24 007 000 DALYs and 682 000 deaths (Mathers et al., 2002). As 81.6% of the population of SearD is in India, it can be assumed that 81.6% of the disease burden will also occur in India:

$81.6\% \times 24\,007\,000 \text{ DALYs} = 19\,590\,000 \text{ DALYs}$ of ALRI in children 0-4 years old in India.

$81.6\% \times 682\,000 \text{ deaths} = 557\,000 \text{ deaths}$ of ALRI in children 0-4 years old in India.

To calculate the fraction of this ALRI burden that is attributable to indoor smoke from solid fuels, the above figures are multiplied by the attributable fraction for ALRI from indoor smoke, which is 51% in this case. The disease burden from ALRI in children 0-4 years old in India caused by exposure to indoor smoke from solid fuels is thus estimated to be:

$51\% \times 19\,590\,000 \text{ DALYs} = 9\,991\,000 \text{ DALYs}$.

$51\% \times 557\,000 \text{ deaths} = 284\,000 \text{ deaths}$.

^a Source: Desai, Smith & Mehta (2003).

A similar technique can be applied if the NBD is known, but the burden for the city or district is not known. Provided that the country has a relatively homogeneous geographical disease distribution, the subregional disease burden could be estimated.

4.7 Estimating the EBD using preliminary NBD estimates from WHO

To develop regional estimates of burden of disease *per disease*, each year WHO compiles country health statistics and the results of epidemiological studies, and publishes the regional estimates in the statistical annexes of the World Health Report. Mortality estimates are based on analysis of latest available national information on levels of mortality and cause distributions. YLD estimates are based on the GBD 2000 analyses of incidence, prevalence, duration and severity of conditions for the relevant epidemiological subregion, together with national and subnational level information available to WHO (Mathers et al., 2002). The GBD 2000 uses the latest population estimates for WHO Member States prepared by the UN Population Division (UN, 2001).

For each Member State, the data compiled by WHO have been used to develop internally consistent estimates of mortality, incidence, prevalence, duration and DALYs for over 130 major disease causes. The WHO summary tables represent *prior* estimates of the NBD and are intended to provide a starting point for more in-depth analyses by NBD teams. It is hoped that such national studies will lead, in turn, to improvements in the GBD 2000 estimates at national, regional and global levels. Prior estimates for WHO Member States are now available upon official request to WHO (contact Colin Mathers at

matheresc@who.int, Epidemiology and Burden of Disease unit, Evidence for Health Policy). An example of the type of information available from the WHO prior estimates is provided in Annex 4.1 for the region AmrD (which includes Bolivia, Ecuador, Guatemala, Haiti, Nicaragua and Peru). However, WHO prior estimates cover only one country.

Current WHO prior estimates of the NBD are based on Version 2 results for the GBD 2000 study (Mathers et al., 2002), as published in the World Health Report 2002 (WHO, 2002). Revised prior estimates based on Version 3 results of the GBD 2000 will be available in late 2003. These prior estimates should be interpreted as best estimates, based on the available evidence in mid-2002, rather than as official estimates of Member States. Prior estimates are always open to revision on the basis of additional data.

Documentation and GBD 2000 summary tables are available on the WHO website (www.who.int/evidence/bod), together with software tools and a NBD manual that provides guidelines for conducting an NBD study (Mathers et al., 2002). For further information, contact Colin Mathers at matheresc@who.int, Epidemiology and Burden of Disease unit, Evidence for Health Policy.

4.8 Estimating the EBD for diseases that are caused by one risk factor

Some diseases are caused by one risk factor (although the risk factors themselves may cause more than one disease). Examples include schistosomiasis, hookworm disease and ascariasis, which are defined by the presence of particular microorganisms. Owing to the characteristics of these pathogens, the diseases can be said to be caused entirely by the risk factor “water, sanitation and hygiene”. The health burden of such diseases can then be attributed entirely to the corresponding risk factor, and the attributable fraction need not be determined, since it is defined as 100%. The entire health burden associated with these pathogens can thus be added to other burdens that are attributable to the same risk factor. For example, the schistosomiasis burden can easily be estimated from the disease statistics, and added to the attributable burden of diarrhoeal diseases (see Box 4.5). In other words, the EBD associated with a risk factor is the sum of all attributable disease burdens caused by that risk factor.

Box 4.5 Diseases caused entirely by one risk factor

Assuming that schistosomiasis, ascariasis, trachoma, trichuriasis and hookworm disease are entirely attributable to the risk factor “water, sanitation and hygiene” (Prüss-Üstün et al., 2003), the DALYs associated with each of these diseases can be added directly to the attributable burden of diarrhoeal disease, another outcome of the risk factor. For AmrD in 2000, the attributable burden of diarrhoeal disease was 756 000 DALYs (Box 4.1), and the DALYs associated with the pathogen-based diseases were (Mathers et al., 2002):

Schistosomiasis:	9 000 DALYs
Trachoma:	0 DALYs
Ascariasis:	26 000 DALYs
Trichuriasis:	46 000 DALYs
Hookworm disease:	20 000 DALYs
<hr/>	
Subtotal for above diseases:	101 000 DALYs
Subtotal for diarrhoeal disease:	756 000 DALYs
<hr/>	
Total, including diarrhoeal disease:	857 000 DALYs
<hr/>	

If only the incidence and the mortality are known, the DALYs could be estimated if additional information were available, including the age of onset of disease, its duration and the disability weight of the disease. A DALY calculation template, and list of disability weights used for the yearly estimates of the GBD (WHO, 2002), are available at the WHO web site:

(http://www3.who.int/whosis/menu.cfm?path=whosis,burden,burden_manual,burden_manual_other&language=english).

This template should be used in connection with the *National Burden of Disease Manual* at:

(<http://www3.who.int/whosis/menu.cfm?path=whosis,burden&language=english>).

4.9 Estimating the EBD for diseases not assessed by national statistics or by WHO

In certain cases, an environmental risk factor may cause a disease that has not been assessed in the country or region of concern, nor has it been compiled as a WHO prior estimate (see Section 4.7). In certain countries, for example, statistics on the occurrence of silicosis, skin lesions from exposure to arsenic, and certain neoplasms associated with environmental exposures may not be available. Neither has WHO assessed these diseases as a specific category. However, for each of these diseases an incidence rate is known for specific exposure levels, and thus the attributable incidence for each disease can be estimated directly:

Attributable incidence = Number of people exposed x Incidence rate *(Equation 5)*

For certain risk factors and associated diseases, the number of attributable cases, and thus the attributable incidence, is better assessed than the relative risks between exposed and non-exposed populations. This is relatively rare, as the literature often reports relative risks, and there is generally no assessment of the occurrence of disease by cause. There are, however, a few exceptions, one being occupational injuries. A number of countries have a register or reporting system for occupational injuries or occupational fatalities. The data are often listed by type of injury, occupation, or industrial activity sector. By knowing the number of workers, the occupational injury rate can be assessed. In this way, the number of injuries and their fatalities can be directly estimated, and there is no need to estimate an attributable fraction. Countries having no such register, or an incomplete register, could apply injury rates from neighbouring countries, which are likely to have similar working conditions. More information on this approach can be found in the EBD series volume on occupational health.

Once the incidence rate or fatality rate has been estimated by this “direct” method, the number of DALYs could be estimated using the DALY calculation template, provided that the age-specific and gender-specific incidences are known, as well as the duration of disease or injury and its severity weight (see Section 4.8 and Section 3 for further information).

4.10 Estimating uncertainty

The uncertainty around BoD estimates can be influenced by many parameters, including:

- the exposure assessment and associated data sources (e.g. the number of people exposed at each level);
- the exposure-response relationship (i.e. the shape of the function or values associated with each scenario, and whether there are any threshold effects);
- the model combining exposure with exposure-risk information (e.g. the EBD method itself may introduce uncertainty, depending upon the specific approach used to assess the risk factor);
- the total disease burden *per disease* for the study population;
- the method(s) used to extrapolate data to subpopulations.

For each of these parameters there are a variety of sources that could potentially contribute to uncertainty. For example, uncertainty linked to exposure may arise because the exposure assessment did not use a sample that was representative of the study population as a whole. The sample size, the choice of exposure indicator, or the measurement accuracy can also introduce uncertainty or errors. Similarly, the uncertainty linked to the model can come at numerous steps, such as applying the exposure-risk relationship to the study population, or matching exposure measurements with the measure used in the exposure-risk relationship. While the impact of some sources of uncertainty can be estimated (e.g. those due to small sample size), for others it is very difficult to estimate their effects on the accuracy of the results (e.g. the uncertainty associated with extrapolating the exposure-risk relationship to data-poor population groups).

It can therefore be difficult to provide a reliable interval of uncertainty for EBD estimates. If uncertainties for the main parameters in national or local assessments have been quantified, a Monte Carlo analysis can be used to provide uncertainty estimates for an EBD, but this is rarely the case. As there is no other straightforward mechanism to capture uncertainty around the best estimate, a more approximate approach may be chosen. For example, “low” and “high” estimates can be obtained from the lower and upper confidence intervals for the relative risks used in the method, and from the confidence intervals of exposure estimates obtained through surveys or other empirical techniques. The effects of different combinations of low and high estimates on the final results can then be examined. Such evaluations of uncertainties have no statistical meaning, but they do illustrate the range of health impacts that could be caused by an environmental risk factor. Practical approaches for estimating the low and high values of a disease burden are provided in the series volumes on individual risk factors.

E. Nutritional deficiencies	503	156	20	31	10	5	3	3	2	229	129	29	55	38	9	5	6	2
1. Protein-energy malnutrition	277	122	8	9	3	2	1	2	1	149	106	10	2	1	1	2	3	2
2. Iodine deficiency	22	12	4	0	0	0	0	0	0	16	5	1	0	0	0	0	0	0
3. Vitamin A deficiency	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4. Iron-deficiency anaemia	201	21	7	22	7	3	2	1	0	63	17	18	52	37	7	3	3	1
Other nutritional disorders	4	1	0	0	0	0	0	0	0	2	1	0	0	0	1	0	0	0
Noncommunicable diseases	8383	731	378	1031	685	676	367	229	74	4171	749	416	1000	676	649	380	251	92
A. Malignant neoplasms	883	15	29	48	55	83	68	48	13	359	20	24	53	115	159	85	53	14
1. Mouth and oropharynx cancers	14	1	0	1	1	2	2	1	0	8	1	0	1	1	1	1	1	0
2. Oesophagus cancer	8	0	0	0	0	1	2	1	0	5	0	0	0	0	1	1	1	0
3. Stomach cancer	140	0	0	4	12	20	18	12	3	69	1	0	5	13	19	16	12	3
4. Colon and rectum cancers	37	0	0	2	3	5	3	2	1	16	0	0	1	4	6	4	3	1
5. Liver cancer	66	0	1	5	7	9	6	4	1	32	1	1	3	5	10	7	5	1
6. Pancreas cancer	21	0	0	0	2	4	3	2	0	10	0	0	0	1	3	3	2	1
7. Trachea, bronchus, lung cancers	26	0	0	0	2	6	5	2	0	15	0	0	1	2	4	3	1	0
8. Melanoma and other skin cancers	12	0	1	1	1	2	1	1	0	6	0	0	0	1	1	1	1	0
9. Breast cancer	58	0	0	0	0	0	0	0	0	0	0	0	2	18	26	9	3	1
10. Cervix uteri cancer	74	0	0	0	0	0	0	0	0	0	0	0	8	25	27	9	5	1
11. Corpus uteri cancer	53	0	0	0	0	0	0	0	0	0	0	0	5	17	19	8	4	1
12. Ovary cancer	16	0	0	0	0	0	0	0	0	0	0	0	2	3	7	2	1	0
13. Prostate cancer	32	0	0	1	1	4	10	11	4	32	0	0	0	0	0	0	0	0
14. Bladder cancer	6	0	0	0	0	1	1	1	0	4	0	0	0	0	1	1	0	0
15. Lymphomas, multiple myeloma	51	0	3	5	6	8	4	3	0	30	0	2	5	4	5	3	2	1
16. Leukaemia	90	7	14	14	6	4	2	1	0	48	8	11	10	6	5	2	1	0
Other malignant neoplasms	177	6	9	15	15	17	13	7	2	83	8	9	12	15	24	15	9	2
B. Other neoplasms	22	2	2	2	2	2	1	1	0	12	2	1	2	1	2	1	1	0
C. Diabetes mellitus	223	0	1	9	25	34	19	10	2	102	1	2	9	24	40	26	16	4
D. Endocrine disorders	254	60	14	19	12	9	4	2	1	120	61	14	23	15	13	5	3	1
E. Neuropsychiatric conditions	2876	152	163	691	291	100	28	12	5	1443	149	191	634	293	112	34	15	7
1. Unipolar depressive disorders	866	0	49	131	94	37	9	1	0	321	0	47	233	175	70	16	3	0
2. Bipolar disorder	172	0	5	73	6	0	0	0	0	84	0	5	76	7	0	0	0	0
3. Schizophrenia	204	0	17	77	6	1	0	0	0	101	0	2	84	15	1	0	0	0
4. Epilepsy	148	5	20	31	14	5	1	1	0	77	6	19	28	12	5	1	0	0
5. Alcohol use disorders	338	0	15	150	83	23	3	1	0	275	0	3	35	18	4	1	0	0
6. Alzheimer and other dementias*	57	0	0	0	6	8	6	4	0	25	0	0	0	0	7	10	9	5
7. Parkinson disease	7	0	0	0	1	1	1	0	0	4	0	0	0	1	1	1	0	0
8. Multiple sclerosis	15	0	1	3	2	0	0	0	0	6	0	1	5	2	0	0	0	0
9. Drug use disorders	228	0	2	122	40	7	0	0	0	171	0	1	39	14	3	0	0	0
10. Post-traumatic stress disorder	31	0	0	6	3	1	0	0	0	10	0	0	11	7	2	0	0	0
11. Obsessive-compulsive disorder	84	0	1	26	9	3	1	0	0	40	0	0	30	8	5	1	0	0
12. Panic disorder	83	0	1	26	0	1	0	0	0	27	0	2	52	0	1	0	0	0
13. Insomnia (primary)	47	0	1	8	7	3	1	0	0	21	0	1	11	8	4	2	0	0
14. Migraine	146	2	19	20	5	0	0	0	0	45	7	78	5	10	0	0	0	0
15. Mental retardation, lead-caused	225	114	0	0	0	0	0	0	0	114	111	0	0	0	0	0	0	0
Other neuropsychiatric disorders	225	31	32	20	19	12	4	2	0	119	24	30	24	15	8	2	2	1
F. Sense organ diseases	298	0	0	5	32	52	41	20	3	153	0	0	8	21	45	44	23	4
1. Glaucoma	6	0	0	0	0	1	1	0	0	2	0	0	0	0	2	1	0	0
2. Cataracts	114	0	0	0	5	20	20	10	2	57	0	0	0	3	16	21	14	3
3. Vision disorders, age-related	32	0	0	2	4	3	1	0	11	0	0	0	3	6	6	3	1	0
4. Hearing loss, adult onset	145	0	0	2	23	29	18	8	1	81	0	0	5	12	21	19	7	1
Other sense organ disorders	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
G. Cardiovascular diseases	1006	39	15	58	84	129	95	73	30	523	48	16	53	61	106	83	76	39
1. Rheumatic heart disease	11	0	0	1	1	1	0	0	0	4	0	1	2	2	2	0	0	0
2. Hypertensive heart disease	118	1	0	3	8	17	14	11	5	58	1	1	3	7	15	14	13	7
3. Ischaemic heart disease	294	1	1	10	25	54	42	31	9	174	2	1	7	11	33	30	24	11
4. Cerebrovascular disease	277	4	3	16	24	39	27	19	7	139	5	4	15	21	37	26	23	9
5. Inflammatory heart diseases	24	1	1	4	2	3	1	1	0	12	2	1	4	2	2	1	1	0
Other cardiovascular diseases	282	33	9	24	25	15	10	11	9	137	38	9	22	19	17	12	16	13
H. Respiratory diseases	761	128	60	86	43	35	20	16	6	393	132	71	71	29	28	17	13	6
1. Chronic obstructive pulmonary disease	86	11	1	3	12	5	5	5	2	44	20	1	4	5	4	3	4	2

I. Digestive diseases	781	57	24	60	83	152	58	27	7	468	49	18	53	46	76	41	24	7
1. Peptic ulcer disease	39	0	1	5	5	6	4	2	1	24	0	1	3	3	4	3	2	1
2. Cirrhosis of the liver	272	2	2	14	41	90	32	13	2	197	3	0	5	10	28	18	9	2
3. Appendicitis	15	2	1	2	2	1	0	0	0	8	2	1	1	1	1	0	0	0
Other digestive diseases	454	52	20	39	35	55	21	12	4	238	43	16	44	32	43	20	13	4
J. Genitourinary diseases	267	21	9	15	17	45	15	11	5	137	23	13	24	18	22	14	11	5
1. Nephritis and nephrosis	170	13	7	12	15	16	11	8	3	85	16	10	13	9	15	11	8	3
2. Benign prostatic hypertrophy	27	0	0	0	0	24	1	1	1	27	0	0	0	0	0	0	0	0
Other genitourinary system disease	70	8	2	3	2	5	3	2	1	25	8	3	12	9	7	3	2	1
K. Skin diseases	42	2	1	4	3	2	2	2	1	16	3	1	5	6	4	3	2	1
L. Musculoskeletal diseases	296	3	9	15	33	29	13	7	1	111	2	18	45	43	39	23	12	2
1. Rheumatoid arthritis	81	0	3	5	4	3	1	0	0	17	1	10	25	17	8	2	1	0
2. Osteoarthritis	113	0	0	3	14	17	10	6	1	50	0	0	1	12	22	17	9	1
3. Gout	19	0	0	1	10	5	1	0	0	17	0	0	0	1	1	0	0	0
4. Low back pain*	23	1	3	4	3	1	0	0	0	13	1	3	2	3	1	0	0	0
Other musculoskeletal disorders	59	2	4	2	2	2	1	1	0	14	1	5	17	11	7	2	2	0
M. Congenital anomalies	529	245	8	7	1	0	0	0	0	262	252	7	6	1	1	0	0	0
1. Abdominal wall defect	4	2	0	0	0	0	0	0	0	2	2	0	0	0	0	0	0	0
2. Anencephaly	18	6	0	0	0	0	0	0	0	6	12	0	0	0	0	0	0	0
3. Anorectal atresia	4	2	0	0	0	0	0	0	0	2	2	0	0	0	0	0	0	0
4. Cleft lip	5	3	0	0	0	0	0	0	0	3	2	0	0	0	0	0	0	0
5. Cleft palate	3	2	0	0	0	0	0	0	0	2	2	0	0	0	0	0	0	0
6. Oesophageal atresia	3	1	0	0	0	0	0	0	0	1	2	0	0	0	0	0	0	0
7. Renal agenesis	1	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
8. Down syndrome	58	30	1	0	0	0	0	0	0	31	27	0	0	0	0	0	0	0
9. Congenital heart anomalies	245	114	5	5	1	0	0	0	0	126	109	5	4	1	0	0	0	0
10. Spina bifida	55	23	0	0	0	0	0	0	0	23	31	0	0	0	0	0	0	0
Other Congenital anomalies	132	61	2	1	0	0	0	0	0	66	63	2	2	0	0	0	0	0
N. Oral conditions	145	7	41	13	4	4	4	1	0	73	6	40	13	4	4	5	1	0
1. Dental caries	128	6	41	12	3	1	0	0	0	65	6	40	12	3	1	0	0	0
2. Periodontal disease	3	0	0	0	0	1	0	0	0	1	0	0	0	0	1	0	0	0
3. Edentulism	12	0	0	0	0	2	4	0	0	6	0	0	0	0	2	4	1	0
Other oral diseases	2	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
I Injuries	2239	192	237	707	356	138	32	12	3	1676	130	125	191	67	32	10	6	2
A. Unintentional injuries	1688	188	215	438	235	103	27	11	2	1218	129	111	133	53	28	10	6	2
1. Road traffic accidents	458	18	46	141	88	34	7	3	1	339	12	26	47	21	9	3	2	0
2. Poisonings	20	2	1	5	4	2	1	0	0	14	2	1	2	1	0	0	0	0
3. Falls	128	10	20	34	12	5	2	1	0	84	6	17	13	3	2	1	1	0
4. Fires	37	8	4	5	2	1	0	0	0	22	7	3	3	1	1	0	0	0
5. Drownings	112	18	13	32	14	5	1	1	0	84	12	7	5	2	1	0	0	0
6. Other unintentional injuries	932	131	131	221	115	55	16	6	1	675	90	56	62	25	14	5	3	1
B. Intentional injuries	551	4	22	269	121	35	5	1	0	458	1	14	58	14	4	1	0	0
1. Self-inflicted injuries	98	0	3	32	14	5	1	0	0	55	0	5	32	4	1	0	0	0
2. Violence	448	4	18	236	106	29	4	1	0	400	1	7	26	9	3	1	0	0

References

- Anand S, Hanson K (1997) Disability-adjusted life years: a critical review. *Journal of Health Economics*, 16:695-702.
- Corvalán C, Briggs D, Zielhuis G (2000) *Decision-making in environmental health - from evidence to action*. London, E & FN Spon for the World Health Organization.
- Desai M, Smith K, Mehta S (2003) Guide for assessment of environmental burden of disease at national and local level: indoor smoke from solid fuels. In: Prüss-Üstün A, Woodward A, Corvalán C, eds. *The environmental burden of disease series*, Geneva, World Health Organization.
- Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. (2003) *Comparative quantification of health risks: global and regional burden of disease due to selected major risk factors*. Geneva, World Health Organization.
- Gold MR, Siegel JE, Weinstein MC, Russell LB (1996) *Cost-effectiveness in health and medicine*. New York, Oxford University Press.
- Hyder AA, Rotllant G, Morrow RH (1998) Measuring the burden of disease: healthy life years. *American Journal of Public Health*, 88:196-202.
- Institute of Medicine (1986) *New vaccine development. Establishing priorities. Volume II. Disease importance in developing countries*. Washington DC, National Academy Press.
- Kjellström T, Corvalán C (1995) Framework for the development of environmental health indicators. *World Health Statistics Quarterly*, 48:144-154.
- Last JM (2001) *A dictionary of epidemiology*. New York, Oxford University Press/International Epidemiological Association.
- Mathers CD, Stein C, Ma Fat D, Rao C, Inoue M, Tomijima N, Berbard C, Lopez AD, Murray CJL (2002) *Global Burden of Disease 2000: version 2 methods and results*. Geneva, World Health Organization (Global Programme on Evidence for Health Policy Discussion Paper No. 50). (Internet communication at web site <http://www.who.int/evidence/bod>).
- Mathers CD, Vos T, Lopez AD, Ezzati M (2001) *National burden of disease studies: a practical guide. Edition 2.0*. Geneva, World Health Organization, Global Programme on Evidence for Health Policy.
- Murray CJ (1996) Rethinking DALYs. In: Murray CJ, Lopez AD, eds. *The global burden of disease*. Geneva, World Health Organization, Harvard School of Public Health, World Bank.
- Murray CJ, Acharya AK (1997) Understanding DALYs. *Journal of Health Economics*, 16:703-730.
- Murray CJL, Lopez AD (1996) *The Global Burden of Disease*. Geneva, World Health Organization, Harvard School of Public Health, World Bank.
- Murray CJL, Lopez AD (1999a) On the comparable quantification of health risks: lessons from the Global Burden of Disease study. *Epidemiology*, 10(5):594-605.

- Murray CJL, Lopez AD (1999b) *Progress and directions in refining the global burden of disease approach*. Geneva, World Health Organization (GPE Discussion Paper No 1).
- Murray CJL, Salomon JA, Mathers CD (2000) A critical examination of summary measures of population health. *Bulletin of the World Health Organization*, 78(8):981-994.
- Murray CJL, Salomon JA, Mathers CD, Lopez AD (2002) *Summary measures of population health: concepts, ethics, measurement and applications*. Geneva, World Health Organization.
- New Zealand Ministry of Health (2001) *Evidence-based health objectives for the New Zealand Health Strategy*. Wellington, Ministry of Health (Internet communication of 2001 at web site <http://www.moh.govt.nz>).
- Prüss-Üstün A, Kay D, Fewtrell L, Bartram J (2003) Water, sanitation and hygiene. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks: global and regional burden of disease due to selected major risk factors*. Geneva, World Health Organization (in press).
- Rothman KJ, Greenland S (1998) *Modern epidemiology*, 2nd ed. Philadelphia, Lippincott, Williams & Wilkins.
- Smith KR, Corvalán CF, Kjellström T (1999) How much global ill-health is attributable to environmental factors? *Epidemiology*, 10(5):573-584.
- Stouthard M, Essink-Bot M, Bonsel G, Barendregt J, Kramers P (1997) *Disability weights for diseases in the Netherlands*. Rotterdam, Department of Public Health, Erasmus University.
- UN (2001) *World population prospects: the 2000 revision*. New York, United Nations.
- USEPA (1994) *A conceptual framework to support the development and use of environmental information*. Washington, US Environmental Protection Agency.
- Weinstein MC, Stason WB (1977) Foundations of cost effective analysis for health and medical practices. *New England Journal of Medicine*, 296:716-721.
- WHO (1993) *Basic epidemiology*. Geneva, World Health Organization.
- WHO (1996) *Investing in health research and development. Report of the ad hoc committee on health research relating to future intervention options*. Geneva, World Health Organization.
- WHO (1997) *Health and environment in sustainable environment. Five years after the Earth Summit*. Geneva, World Health Organization.
- WHO (1999) *Environmental health indicators: framework and methodologies*. Geneva, World Health Organization (WHO/SDE/OEH/99.10).
- WHO (2002a) *World Health Report 2002 - reducing risks, promoting healthy life*. Geneva, World Health Organization.
- WHO (2002b) *Burden of disease project: mortality and DALYs*. Geneva, World Health Organization (Internet communication of 2002 at web site www.who.int/whosis).
- WHO (2003) *WHO World Health Survey*. Geneva, World Health Organization (Internet communication of May 2003 at web site <http://www3.who.int/whs/>).

WHO, UNICEF, WSSCC (2000) *Global water supply and sanitation assessment 2000 report*. Geneva, WHO/UNICEF/Water Supply and Sanitation Collaborative Council.

Williams A (1999) Calculating the global burden of disease: time for a strategic reappraisal? *Health Economics*, 8:1-8.

Wills J, Briggs D (1995) Developing indicators for environment and health. *World Health Statistics Quarterly*, 48(2):155-163.

Glossary of terms for the EBD series

Attributable fraction^a The portion of the incidence rate of a given outcome in a given population that is identified as due to a given exposure.

Consequently, that portion of the incidence rate could be reduced if causative exposure were eliminated.

Attributable burden Burden of a given disease in a given population that is identified as due to a specific exposure.

Consequently, that portion of disease burden in the population that could be reduced if causative exposure were eliminated.

Avoidable burden^b The portion of burden of a given disease that could be reduced if levels of exposure to a given risk factor were reduced to an alternative, achievable exposure distribution.

Confidence interval (and 95% CI)^a The computed interval with a given probability, e.g. the 95%, that the true value of a variable such as a mean, proportion, or rate is contained within the interval.

Confounding^{c,d} Concomitant exposure with the exposure being studied in the population, associated both with the disease and the exposure being studied. *Confounding can lead to either the observation of apparent differences between study groups when they do not truly exist or, conversely, the observation of no differences when they do exist.*

Counterfactual exposure An alternative exposure distribution used as baseline for estimating the burden of disease caused by the exposure distribution of interest. The disease burden caused by a risk factor is estimated by comparing the burden caused by current and future levels of exposure to the exposure levels that would be expected under some alternative hypothetical scenario.

Covariate^a A variable that is possibly predictive of the outcome under study. A covariate may be of direct interest to the study or may be a confounding variable of effect modifier.

Comparative Risk Assessment (CRA) A systematic counterfactual approach to estimating health gaps (q.v.) (or changes in health expectancy) causally attributable to a risk factor or a group of risk factors. The underlying approach is the same as the one used in environmental burden of disease assessment.

Disability adjusted Life Year (DALY) An indicator of life expectancy combining mortality and morbidity into one summary measure of population health to account for the number of years lived in less than optimum health. It is a *health gap* (q.v.) measure developed for calculating the *global burden of disease* (q.v.).

Epidemiology^a The study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control of health problems.

Environmental tobacco smoke (ETS)^a A type of air pollution due to burning of tobacco, especially sidestream smoke. ETS is a confirmed carcinogen^e.

Geometric mean^a A measure of central tendency. This is calculated by adding the logarithms of the individual values, calculating their arithmetic mean, and converting back by taking the antilogarithm.

Global burden of disease (GBD) An estimate of health gaps (q.v.) for a comprehensive set of disease and injury causes, and for major risk factors, in the world populations using all available mortality and health data and methods to ensure internal consistency and comparability of estimates. The WHO Global Burden of Disease 2000 project estimates health gaps using DALYs (q.v.) for 14 subregions of the world for the year 2000 and subsequent years.

Gross national product (GNP) The total income produced in a country's economy, including goods and services produced abroad.

Health-adjusted life expectancy (HALE) Any of a number of summary measures which use explicit weights to combine health expectancies for a set of discrete health states into a single indicator estimating the expectation of equivalent years of good health. Also referred to as 'Healthy life expectancy'.

Healthy life years (HeaLYs) A health gap (q.v.) measure calculated on the basis of the incidence of pathological processes and the future non-fatal health outcomes and mortality from those processes.

Health gap An estimate of the difference between the current population health and a normative goal for population health. It is a summary measure of population health

Impact fraction^a A generalisation of the attributable fraction that accommodates both hazardous and protective exposures, multiple levels of exposure or incomplete elimination of exposure.

Life expectancy Any summary measure of population mortality that estimates the expectation of years of life.

Meta-analysis^a A statistical synthesis of the data from separate but similar, i.e. comparable, studies, leading to a quantitative summary of the pooled results. A frequent application has been the pooling of results from a set of randomized controlled trials, none in itself necessarily powerful enough to demonstrate statistically significant differences, but in aggregate capable of doing so. Meta-analysis has a qualitative component, i.e. application of a predetermined criteria of quality (e.g. completeness of data, absence of biases), and a quantitative component, i.e. integration of the numerical information. The aim is to integrate the findings, pool the data, and identify the overall findings of results. An essential prerequisite is that the studies must stand up to critical appraisal, and various biases.

Monte Carlo simulation^f An analysis of a sequence of events using random numbers to generate possible outcomes in an iterative process. This technique can be used to simulate uncertainty and variability. Each probability distribution is sampled in a manner that

reproduces the distribution's shape. The distribution of the values calculated for the model outcome therefore reflects the probability of the values that could occur.

Odds ratio^a The ratio of two odds, an odds being a ratio of probabilities (in this instance, the ratio of the probability of occurrence of an event to the probability of non-occurrence). When considering the following:

	Exposed	Unexposed
Disease	<i>a</i>	<i>b</i>
No disease	<i>c</i>	<i>d</i>

The odds ratio is ad/bc .

Perinatal^a Pertaining to a period commencing at 22 completed weeks (154 days) of gestation (the time when the birth weight is normally 500g) and ending seven completed days after birth. (ICD-10)

Person-time^a A measurement combining persons and time as the denominator in incidence and mortality rates when, for varying periods, individual subjects are at risk of developing disease or dying. It is the sum of periods of time at risk for each of the subjects.

The most widely used measure is person-years. With this approach, each subject contributes only as many years of observation to the population at risk as the period over which that subject has been observed; a subject observed over one year contributes 1 person-year, a subject observed over a 10-year period contributes 10 person-years. This method can be used to measure incidence rate over extended and variable time periods.

Probability (random) sample^a A representative subset of a population made of randomly selected individuals.

All individuals have a known chance of selection. They may all have an equal chance of being selected. If a stratified sampling method is used, the rate at which individuals from several subsets are sampled can be varied so as to produce greater representation of some classes than of others.

Quality-adjusted life expectancy (QALE) A form of HALE (q.v.) based on a question on activity restriction in the Canada Health Survey.

Relative risk (RR)^a The ratio of risk of disease or death among the exposed to the risk among the unexposed; this usage is synonymous with risk ratio.

Alternatively, the ratio of the cumulative incidence rate in the exposed to the cumulative incidence rate in the unexposed, i.e. the rate ratio.

Standard deviation (SD)^a A statistical measure of dispersion, or variation, in a frequency distribution.

It is the most widely used measure of dispersion. It is equal to the positive square root of the variance. The mean tells where the values for a group are centred. The standard deviation is a summary of how widely dispersed the values are around this centre.

Socioeconomic status (SES)^a A description of a person's position in society, which may be expressed on an ordinal scale (i.e. classification into qualitative categories) using such criteria as income, educational level attained, occupation, value of dwelling place, etc.

Summary measures of population health Indicators that summarize the health of a population into a single number. They combine information about mortality and population health states. They may summarize either average health level or inequality for a population.

Years lived with disability (YLD) The component of the DALY (q.v.) that measures the lost years of healthy life through living in states of less than full health.

Years of Life Lost (YLL) The component of the DALY (q.v.) that measures the years lost through premature mortality.

^a Adapted from: Last (2001) *A dictionary of epidemiology*. New York, Oxford University Press/International Epidemiological Association.

^b Adapted from: *Comparative risk assessment: interim guidelines*. Geneva, World Health Organization, Comparative Risk Assessment Working Group, 2000 (draft available at website <http://www.ctr.u.auckland-ac-nz/CRA/>).

^c Adapted from: *Basic Epidemiology*. (1993) Geneva, World Health Organization.

^d Hennekens CH, Buring JE (1987) *Epidemiology in medicine*. Boston, MA, Little, Brown and Company.

^e Environmental Protection Agency (1992) *Respiratory health effects of passive smoking: lung cancer and other disorders*. Washington, DC, Office of Health and Environmental Assessment.

^f Vose d (2000) *Risk analysis*, 2nd ed. Chichester, John Wiley & Sons, Ltd.