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During the twentieth century reliable cause-specific mortality statistics became available for many countries, culminating in the Global Burden of Disease project which, during the 1990s, provided estimates for different regions of the world (with, obviously, varying degrees of reliability) of the numbers of deaths due to major diseases, and of the amounts of “disability-adjusted” loss of healthy life from those diseases. The present study goes further, and seeks to estimate the amounts of death and disability due to the main avoidable causes of those diseases. Its preliminary conclusions underlay the 250-page World Health Organization report on “Reducing Risks” (2002), the aim of which was to summarize, for the first time, the amount of death and disability in each of 14 subregions of the world that is attributable not to particular diseases, but to particular avoidable risk factors.

Such attributions of causality throw up, of course, many more difficulties than were encountered in the previous studies of the Global Burden of Disease, which merely tried to classify deaths by the one main disease (or type of accident or violence) that underlay them. For, one death may have several avoidable causes. For example, if a poorly nourished child dies of measles, should “the cause” be thought of as exposure to the virus, or as the lack of measles vaccination (in that child or in the community), or as the poor diet (low in protein, energy and certain micronutrients) that prevented recovery from the illness? The most appropriate answer, if we want to prevent such deaths, is that each of these factors should be thought of as “a cause” of a certain proportion of the childhood deaths from measles. That is what the authors of the World health report 2002 tried to do, and in the present much more detailed series of monographs they explain to the interested (or disputative) reader much more about their main conclusions, and about how they reached those conclusions. This is important, because over the years some of the conclusions may need to be revised, as more detailed studies are undertaken or as exposure and disease patterns evolve.

For many decades it has been recognized increasingly clearly by those concerned with global health that much can affordably be achieved even in relatively poor countries if resources are directed to the major diseases of childhood and early adult life, and more recently the affordable avoidability of much other adult mortality and morbidity has been recognized.
(see *The Health of Adults in the Developing World*). Ten years ago, the 1993 World Bank report, *Investing in health* (together with its companion volume, *Disease Control Priorities in Developing Countries*) was extremely influential in consolidating these ideas and getting them accepted, and acted upon, by the major international economic institutions.

But, any such cost-effectiveness calculations require, among other things, reliable estimates of effectiveness, and the present report goes further than any other in providing estimates of just how much mortality and morbidity could be avoided by addressing particular causes of disease. In many parts of the world (the main exceptions being where political disruption or HIV predominate) the risk of premature death has been reduced by more than half over the past few decades, and premature death can be halved again over the next few decades if the major intervention options are pursued to control disease and injury, and their causes.

This book will greatly facilitate such progress. It is well organized, stimulating and is an important part of a political and scientific process that is already preventing many millions of deaths a year, and will prevent many more millions of deaths a year in the future.

Sir Richard Peto  
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A clear understanding of the role and relative magnitude of diseases, injuries and their underlying causes—and effective and affordable interventions to reduce them—should guide policies and programmes for health development. Over the centuries, the health of populations has improved because science has helped us understand the main causes of disease affecting large populations, and how technologies or programmes can be delivered to reduce hazards among those affected or at risk.

While the monitoring and analysis of diseases and mortality in populations has been largely undertaken by actuaries and demographers, much of the work on causes of disease has emanated from research in fields such as epidemiology, toxicology and physiology, which focus on micro-level analysis. By its very nature, this research has quantified hazards in the study population, with its specific characteristics. This body of knowledge has had tremendous application in reducing the established causes of disease, from smoking to iodine deficiency, in many populations. The broader, policy-relevant issue of population effects of exposure to risks, however, has remained under-explored relative to our documentation of established diseases. Thus, while there has been decades of epidemiological research into the leading causes of many major diseases, from childhood diarrhoea to ischaemic heart disease, there have been few attempts to estimate the population-level effects of various exposures, either for specific countries and groups of countries, or for the world and its major regions.

During the last quarter of the twentieth century, a number of works have addressed both the methodological and empirical aspects of population-wide effects of major causes of diseases. Examples include the development of cancer risk models and methods to forecast the health of ageing populations based on their causal determinants, and the estimates of mortality due to risk factors such as smoking, asbestos and childhood malnutrition. This gradual establishment of “risk assessment” or “risk quantification” has been driven partly by the academic curiosity of individual researchers and partly by the demands of regulatory agencies and public policy for better quantitative evidence on the health implications of certain risk exposures.

This book provides a comprehensive assessment of the health effects caused by a range of exposures that are known to be hazardous to human
health. Its origins lie in the expressed need by policy and advocacy groups for comparable data on risk factor exposure and effects in populations. The only previous attempt to quantify risk factor burden worldwide, the Global Burden of Disease (GBD) 1990 project, was affected by a lack of conceptual and methodological comparability across risk factors; the analysis of each risk was constrained by its own disciplinary tradition. It nonetheless stimulated debate about the crucial role of risk factor assessment as a cornerstone of the evidence base for public health action: for instance, the leading risk factor in 1990, malnutrition, accounted for substantially more disease burden worldwide than the leading cause of disease at that time, acute lower respiratory infections.

A key concern of the current work on risks to health is to provide a degree of conceptual and methodological consistency and comparability across risk factors. The results reported in this book, therefore, differ in a number of important ways from those of GBD 1990: a new analytical framework and consistent set of definitions on “risk factor exposure” have been used to enhance comparability; the number of exposures assessed has more than doubled; and the analyses have benefited from more recent and thorough research into causality and geographical variations in population exposures and health effects.

The scope of risks to health studied in this book covers many of the most important hazards to health addressed by various fields of scientific enquiry. Arguably, there are hundreds of risk exposures that are harmful to health; and there are important implications for better understanding the disease burden they cause across the world. We have selected only a relatively small number of exposures for quantification in this book, largely determined by the availability of scientific research about their prevalence and health effects in different parts of the world. It was also important to make choices about the definition of each risk factor. Given the close interrelationships among diet, exercise and physiological risks on the one hand, or among water, sanitation and personal hygiene on the other, the exact definition of what a “risk factor” is, itself requires careful attention. That a particular risk factor like dietary fat intake does not appear in this book does not, of course, imply that it is of limited relevance; or that exposure to lead has been assessed separately from urban air pollution does not override their close linkages. Rather, we have limited ourselves to risk factors for which there was good potential for satisfactory quantification of population exposure distributions and health effects using the existing scientific evidence and available data, and for which intervention strategies are available or might be envisioned to modify their impact on disease burden.

The chapters in Volumes 1 and 2 of this book fall into two broad categories: those that address specific risk factors, and those that provide conceptual, methodological or empirical links across risks. The book begins with a description of some of the important conceptual and methodological issues in quantifying risk factor burden in a consistent
and comparable framework. This is followed by twenty-two chapters, organized under six broad sections, each of which present the background and the scientific evidence and empirical findings for individual risks. These are followed by an attempt to quantify the distributions of some risks by poverty levels. While much is known about the relationship between poverty and health, it is undoubtedly too complex and population-specific to be adequately assessed in a single quantification effort. The research reported here is therefore limited to a simple mapping of risks by poverty, based on existing data. Following the risk factor chapters, the calculus of estimating the burden of disease attributable to each risk factor from exposure and hazard data is presented, followed by a chapter that summarizes the results for individual risk factors.

Many policies and programmes affect multiple risks simultaneously, motivating an assessment of the disease burden from multiple risk factors. The focus on joint exposures and hazards is particularly important because diseases and injuries are almost always caused by multiple risk factors, which may act together on disease processes, or have effects mediated through each other. We have therefore included two chapters on the joint effects of multiple risk factor exposures. The final chapter of the book provides conclusions and recommendations for future research, based on the analytical findings presented in the book, as well as the gaps in data and scientific knowledge that increased uncertainty in quantifying risk factor burden reported here.

The specific risk factor chapters have been grouped according to clusters of exposures likely to be of similar scientific or policy interest. Volume 1 begins with four chapters on childhood and maternal undernutrition, which collectively cause a significant proportion of the childhood infectious disease burden worldwide. With substantial reductions in child mortality over the past few decades in many countries, the focus of scientific enquiry has progressively moved to improving our understanding of the causes of disease and injury among adults. The next five chapters address the various distal (e.g. exercise), more proximal (e.g. overweight and obesity), and physiological (e.g. suboptimal cholesterol levels) risks that are clustered together under the label of nutrition and physical activity. The last section in Volume 1 and the first section in Volume 2, addictive substances and sexual and reproductive health, include the major lifestyle and behavioural risks that are widespread in many societies and, despite being the subject of scientific enquiry and public health intervention for decades, present a range of complexities in risk quantification.

The risk factors that are a part of the physical environment of households (e.g. indoor air pollution from household solid fuel use), communities (e.g. urban air pollution), or specific subgroups (e.g. occupational risk factors) are the next group of risks assessed in Volume 2. The next two chapters, childhood sexual abuse and contaminated medical injection...
tions, do not fall into any of the above broad categories and are presented independently. These two chapters, each representing a risk factor that affects multiple important diseases, illustrate the potential for risk assessment as an analytical tool for improving the public health evidence base across a wide spectrum of health concerns.

In each of the specific risk factor chapters, the authors have provided a definition of the risk factor and introduced an “exposure variable” that best reflects the distribution of hazards in the population. The complexity of disease causation mechanisms (e.g. sexual behaviour and sexually transmitted infections), and the limitations posed by available data and epidemiological studies (e.g. physical inactivity or indoor smoke from solid fuels) have been important factors in the choice of exposure variable. Coupled with this is the choice of a “theoretical-minimum-risk population exposure distribution”, which can serve as a consistent baseline for assessing attributable disease burden across difference risks. For some risks such as smoking or childhood abuse, the theoretical-minimum-risk population exposure distribution is obviously zero exposure for the whole population; for others the choice of baseline exposure distribution is less obvious, either because zero exposure is not definable (e.g. blood pressure) or because it may not lead to the lowest risk level in some populations (e.g. alcohol). Each chapter includes current estimates of exposure distributions by age and sex for 14 epidemiological subregions. The chapters also examine in detail the evidence for health outcomes, including the evidence for causality and the estimates of hazard (disease-specific) associated with each level of exposure. Each chapter then concludes with summary results of the burden of disease and injury in 2000 attributable to the risk factor, and when possible using existing evidence and knowledge, estimates of projected future exposure to the risk.

The CD-ROM attached contains detailed tables on the various components of disease burden (i.e. deaths, years of life lost [YLL] due to premature mortality, and disability-adjusted life years [DALYs]) attributable to each risk factor by age, sex and the 14 epidemiological subregions of the world used by the World Health Organization (WHO) in the World health report 2002. The 191 Member States of WHO were divided into five mortality strata on the basis of their levels of child mortality (under five years of age) and 15–59-year-old male mortality. When these mortality strata are applied to the six WHO regions, they produce 14 epidemiological subregions, which are used throughout this book (Table 1).

This book is the culmination of over four years of scientific enquiry and data collection, collectively known as the comparative risk assessment (CRA) project, coordinated by WHO and involving over 100 scientists worldwide. The book is also one of the several planned outputs of the GBD 2000 project which includes multiple analytical and empirical perspectives on global population health. The importance of the collaborative effort in the CRA project goes beyond having leading
### Table 1: The 14 GBD epidemiological subregions

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

A: very low child mortality and very low adult mortality; B: low child mortality and low adult mortality; C: low child mortality and high adult mortality; D: high child mortality and high adult mortality; E: high child mortality and very high adult mortality. High-mortality developing subregions: AFR-D, AFR-E, AMR-D, EMR-D and SEAR-D. Low-mortality developing subregions: AMR-B, EMR-B, SEAR-B, WPR-B. Developed subregions: AMR-A, EUR-A, EUR-B, EUR-C and WPR-A. This classification has no official status and is for analytical purposes only.
researchers for multiple risk factors working simultaneously on the same project. Rather, the interactions of these researchers, with a core network of scientists applying a common analytical framework and methods, has ensured greater consistency and comparability in using and evaluating scientific evidence across risks. As a result, our understanding of the comparative extent of disease burden caused by various exposures worldwide has advanced, and key areas of scientific enquiry necessary to better inform policies to reduce risks have been elucidated. Health advocates and those entrusted with policy and programme development to promote better health now have a more comparable empirical assessment of the hazards to health worldwide, and thus a firmer basis for public health action. We hope that the methodological and empirical findings reported in these volumes will indeed serve as the stimulus for global, regional and national policy action to reduce key hazards to health for decades to come.

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Christopher J.L. Murray