



PREVENTING DISEASE THROUGH HEALTHY ENVIRONMENTS

Towards an estimate of the environmental burden of disease



World Health
Organization

PREVENTING DISEASE THROUGH HEALTHY ENVIRONMENTS

Towards an estimate of the
environmental burden of disease

A. Prüss-Üstün and C. Corvalán

WHO Library Cataloguing-in-Publication Data

Prüss-Üstün, Annette.

Preventing disease through healthy environments. Towards an estimate of the environmental burden of disease. / Prüss-Üstün A, Corvalán C.

1. Environmental monitoring. 2. Cost of illness. 3. Risk factors. I. Corvalán, Carlos F. II. World Health Organization.

ISBN 92 4 159382 2

(NLM classification: WA 30.5)

© World Health Organization 2006

All rights reserved. Publications of the World Health Organization can be obtained from WHO Press, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel: +41 22 791 3264; 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 WHO Press, 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.

All reasonable precautions have been taken by the World Health Organization to verify the information contained in this publication. However, the published material is being distributed without warranty of any kind, either express or implied. The responsibility for the interpretation and use of the material lies with the reader. In no event shall the World Health Organization be liable for damages arising from its use.

The named authors alone are responsible for the views expressed in this publication.

Printed in France

TABLE OF CONTENTS

PREFACE	6
EXECUTIVE SUMMARY	8
1 INTRODUCTION	18
2 WHAT IS THE ENVIRONMENT IN THE CONTEXT OF HEALTH?	20
3 WHAT IS MEANT BY THE "ATTRIBUTABLE FRACTION" OF A RISK FACTOR?	24
4 METHODS	26
5 ANALYSIS OF ESTIMATES OF THE ENVIRONMENTAL ATTRIBUTABLE FRACTION, BY DISEASE	32
Respiratory infections	33
Diarrhoea	34
Malaria	34
Intestinal nematode infections	36
Trachoma	37
Schistosomiasis	37
Chagas disease	37
Lymphatic filariasis	38
Onchocerciasis	38
Leishmaniasis	39
Dengue	39
Japanese encephalitis	39
HIV/AIDS	40
Sexually transmitted diseases	41
Hepatitis B and hepatitis C	41
Tuberculosis	42
Perinatal conditions	43
Congenital anomalies	44
Malnutrition	44
Cancers	45
Neuropsychiatric disorders	46
Cataracts	47
Deafness	48
Cardiovascular diseases	48
Chronic obstructive pulmonary disease	49
Asthma	49
Musculoskeletal diseases	50
Road traffic injuries	50
Unintentional poisonings	51
Falls	52

Fires	52	
Drownings	52	
Other unintentional injuries	53	
Suicide	54	
Interpersonal violence	54	
Physical inactivity	55	
Other diseases	56	
6	GLOBAL RESULTS OF THE ANALYSIS	58
7	CONCLUSIONS	64
ANNEX 1	WHO Member States, by WHO subregion and mortality stratum	72
ANNEX 2	Global statistics produced by the analysis of the environmental disease burden	74
REFERENCES		90
ACKNOWLEDGEMENTS		102
CREDITS		104

LIST OF FIGURES

FIGURE 1	Definition of the environment	21
FIGURE 2	Probability distributions of five expert estimates for the attributable fraction of road traffic injuries	30
FIGURE 3	Overlay of individual expert estimates, CRA estimate, and pooled estimate for road traffic injuries in developing countries	31
FIGURE 4	Environmental disease burden, by WHO subregion	60
FIGURE 5	Diseases with the largest environmental contribution	60
FIGURE 6	Environmental disease burden in DALYs per 1000 people, by WHO subregion (2002)	61
FIGURE 7	Environmental disease burden in deaths per 100 000 people, by WHO subregion (2002)	61
FIGURE 8	Main diseases contributing to the environmental burden of disease, for the total population	62
FIGURE 9	Main diseases contributing to the environmental burden of disease among children 0-14 years	62

LIST OF TABLES

TABLE 1	Environmental risk factors and related diseases included in the CRA	27
TABLE A2.1	Attributable environmental fractions for each disease or disease group	75
TABLE A2.2	Indicative values for environmental attributable fractions, by specific environmental risk factor and disease or disease risk	80
TABLE A2.3	Deaths attributable to environmental factors, by disease and mortality stratum, for WHO regions in 2002	82
TABLE A2.4	Burden of disease (in DALYs) attributable to environmental factors, by disease and mortality stratum, for WHO regions in 2002	88

HOW MUCH DISEASE CAN BE PREVENTED THROUGH HEALTHIER ENVIRONMENTS?

This question lies at the heart of our global efforts to address the root causes of ill health through improved preventive health strategies - using the full range of policies, interventions and technologies in our arsenal of knowledge.

Previous World Health Organization studies have examined the aggregate disease burden attributed to key environmental risks globally and regionally, quantifying the amount of death and disease caused by factors such as unsafe drinking-water and sanitation, and indoor and outdoor air pollution.

Building from that experience, this present study examines how *specific diseases and injuries* are impacted by environmental risks, and which regions and populations are most vulnerable to environmentally-mediated diseases and injuries.

This report confirms that approximately one-quarter of the global disease burden, and more than one-third of the burden among children, is due to modifiable environmental factors. The analysis here also goes a step further, and systematically analyzes how different diseases are impacted by environmental risks... and by 'how much.' Heading that list are diarrhoea, lower respiratory infections, various forms of unintentional injuries, and malaria. This 'environmentally-mediated' disease burden is much higher in the developing world than in developed countries - although in the case of certain non-communicable diseases, such as cardiovascular diseases and cancers, the per capita disease burden is larger in developed countries. Children bear the highest death toll with more than 4 million environmentally-caused deaths yearly, mostly in developing countries. The infant death rate from environmental causes is 12 times higher in developing than in developed countries, reflecting the human health gain that could be achieved by supporting healthy environments.

This analysis details the health impacts of environmental risks across more than 80 diseases and injuries. Findings are particularly relevant to health care policymakers and practitioners. Our evolving knowledge about environment-health interactions can support the design of more effective preventive and public health strategies that reduce corresponding risks to health.

These estimates involved not only a systematic literature review in all of the disease categories addressed, but also a survey of more than 100 experts worldwide. As such, this analysis represents the result of a systematic process for estimating environmental burden of disease that is

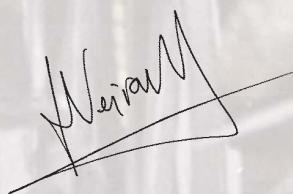
unprecedented in terms of rigor, transparency and comprehensiveness. It incorporates the best available scientific evidence on population risk from environmental hazards currently available. While not an official WHO estimate of environmental burden of disease, as such, it is an important input. More immediately, findings can be used to highlight the most promising areas for immediate intervention, and also gaps where further research is needed to establish the linkages and quantify population risk (burden of disease) for various environmental risk factors.

Many measures can indeed be taken almost immediately to reduce this environmental disease burden. Just a few examples include the promotion of safe household water storage and better hygiene measures, the use of cleaner fuels and safer, more judicious use and management of toxic substances in the home and workplace. At the same time, actions by sectors such as energy, transport, agriculture, and industry are urgently required, in cooperation with the health sector, to address the root environmental causes of ill health.

There is good news in this report, however. These findings underline the fact that environment is a platform for good health that we all share in common.

Acting together on the basis of coordinated health, environment and development policies, we can strengthen this platform, and make a real difference in human well-being and quality of life.

Coordinated investments can promote more cost-effective development strategies with multiple social and economic co-benefits, in addition to global health gains, both immediate and long term. Repositioning the health sector to act more effectively on preventive health policies, while enhancing intersectoral partnerships, is thus critical to addressing the environmental causes of disease and injury, meeting the Millennium Development Goals, and achieving better health for all.



Dr. Maria Neira
Director
Public Health and Environment
World Health Organization

PREVENTING DISEASE THROUGH HEALTHY ENVIRONMENTS

This global assessment provides quantitative estimates of 'burden of disease' from environmental factors across the major categories of reported diseases and injuries.

By focusing on the disease endpoint, and how various kinds of diseases are impacted by environmental influences, the analysis forges new ground in an understanding of interactions between environment and health. The estimates, in effect, reflect how much death, illness and disability could realistically be avoided every year as a result of reduced human exposures to environmental hazards.

Specifically considered here are "modifiable" environmental factors realistically amenable to change using available technologies, policies, and preventive and public health measures. These environmental factors include physical, chemical and biological hazards that directly affect health and also increase unhealthy behaviours (e.g. physical inactivity).

The analysis builds upon the Comparative Risk Assessment coordinated by WHO in 2002, which looked at the total burden of disease attributable to some of the most important environmental hazards, and upon other quantitative surveys of health impacts from the environment. When quantitative data were too scarce for meaningful statistical analysis, experts in environmental health and health care provided estimates. More than 100 experts from around the world contributed with reference to 85 categories of diseases and injuries. Estimates are quantified in terms of mortality from the attributable environmental fraction of each disease condition, and in terms of 'disability adjusted life years' (DALYs) – a weighted measure of death, illness and disability. While there are gaps in the reporting of many diseases at country level, this analysis makes use of the best available data on overall disease burden, globally and regionally, as reported by WHO (World Health Report, 2004).

The results and conclusions of this assessment are of particular relevance to the health-care sector, where policies and programmes generally address specific diseases or injuries. A better understanding of the disease impacts of various environmental factors can help guide policymakers in designing preventive health measures that not only reduce disease, but also reduce costs to the health-care system. The findings also are highly relevant to non-health sectors, whose activities influence many of the root environmental factors – such as air and water quality, patterns of energy use, and patterns of land use and urban design – which affect health and behaviour directly and indirectly.

Along with reducing disease burden, many of the same health sector and non-health sector measures that reduce environmental risks and exposures also can generate other co-benefits, e.g. improved quality of life and well-being, and even improved opportunities for education and employment. Overall, then, an improved environment also will contribute to achieving the Millennium Development Goals. A brief summary of specific findings is presented below, in terms of key questions that were explored.

1. HOW SIGNIFICANT IS THE IMPACT OF ENVIRONMENT ON HEALTH?

An estimated 24% of the global disease burden and 23% of all deaths can be attributed to environmental factors.

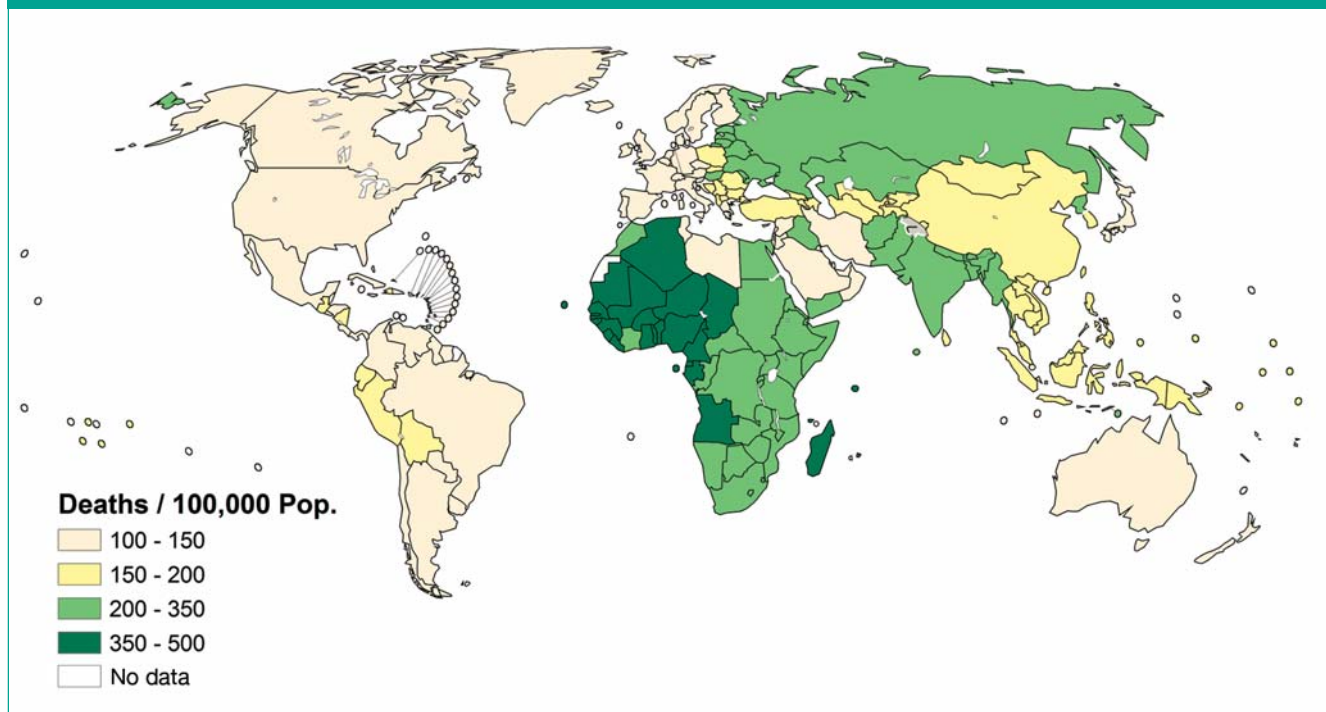
Of the 102 major diseases, disease groupings and injuries covered by the *World Health Report* in 2004, environmental risk factors contributed to disease burden in 85 categories. The specific fraction of disease attributable to the environment varied widely across different disease conditions.

Globally, an estimated 24% of the disease burden (healthy life years lost) and an estimated 23% of all deaths (premature mortality) was attributable to environmental factors. Among children 0–14 years of age, the proportion of deaths attributed to the environment was as high as 36%. There were large regional differences in the environmental contribution to various disease conditions – due to differences in environmental exposures and access to health care across the regions. For example, although 25% of all deaths in developing regions were attributable to environmental causes, only 17% of deaths were attributed to such causes in developed regions. Although this represents a significant contribution to the overall disease burden, it is a conservative estimate because there is as yet no evidence for many diseases. Also, in many cases, the causal pathway between environmental hazard and disease outcome is complex. Where possible, attempts were made to capture such indirect health effects. For instance, malnutrition associated with water-borne diseases was quantified, as was disease burden related to aspects of physical inactivity attributable to environmental factors (e.g. urban design). But in other cases, disease burden was not quantifiable even though the health impacts are readily apparent. For instance, the disease burden associated with changed, damaged or depleted ecosystems in general was not quantified.

Diseases with the largest absolute burden attributable to modifiable environmental factors included: diarrhoea; lower respiratory infections; 'other' unintentional injuries; and malaria.

- **Diarrhoea.** An estimated 94% of the diarrhoeal burden of disease is attributable to environment, and associated with risk factors such as unsafe drinking-water and poor sanitation and hygiene.
- **Lower respiratory infections.** These are associated with indoor air pollution related largely to household solid fuel use and possibly to second-hand tobacco smoke, as well as to outdoor air pollution. In developed countries, an estimated 20% of such infections are attributable to environmental causes, rising to 42% in developing countries.

ENVIRONMENTAL DISEASE BURDEN BY WHO SUBREGION (2002) ^a

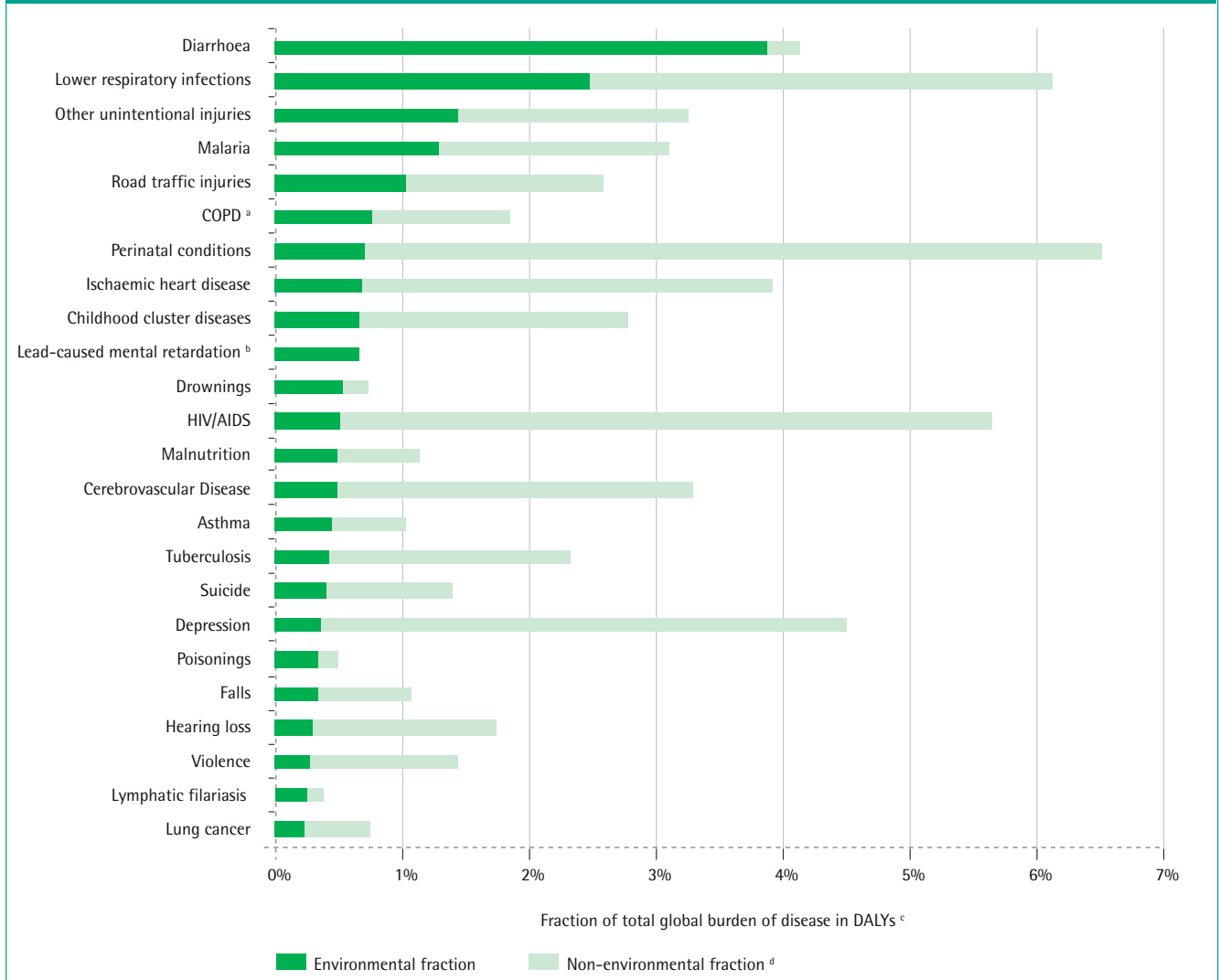


^a The disease burden is measured in deaths per 100 000 population for the year 2002. See Annex 1 for a list of the countries in each WHO subregion.

- **'Other' unintentional injuries.** These include injuries arising from workplace hazards, radiation and industrial accidents; 44% of such injuries are attributable to environmental factors.
- **Malaria.** The proportion of malaria attributable to modifiable environmental factors (42%) is associated with policies and practices regarding land use, deforestation, water resource management, settlement siting and modified house design, e.g. improved drainage. For the purposes of this study, the use of insecticide-treated nets was not considered an environmental management measure.

Environmental factors, such as inadequate pedestrian and cycling infrastructures, also make a significant contribution to injuries from road traffic accidents (40%). However, health impacts of certain longer term changes in urban geography and mobility patterns are yet to be measured. An estimated 42% of chronic obstructive pulmonary disease (COPD), a gradual loss of lung function, is attributable to environmental risk factors such as occupational exposures to dust and chemicals, as well as indoor air pollution from household solid fuel use. Other forms of indoor and outdoor air pollution – ranging from transport to second-hand tobacco smoke – also play a role. A list of the 24 diseases with the largest environmental contribution to overall burden is noted in the following figure. Detailed description of environmental factors and impacts on all diseases considered is provided in subsequent chapters, as are statistical tables and annexes covering global, and regional disease burden, as well as special sub-groups such as children.

DISEASES WITH THE LARGEST ENVIRONMENTAL CONTRIBUTION



^a Abbreviations: COPD = Chronic obstructive pulmonary disease.

^b Lead-caused mental retardation is defined in the WHO list of diseases for 2002, accessed at: www.who.int/evidence.

^c DALYs represents a weighted measure of death, illness and disability.

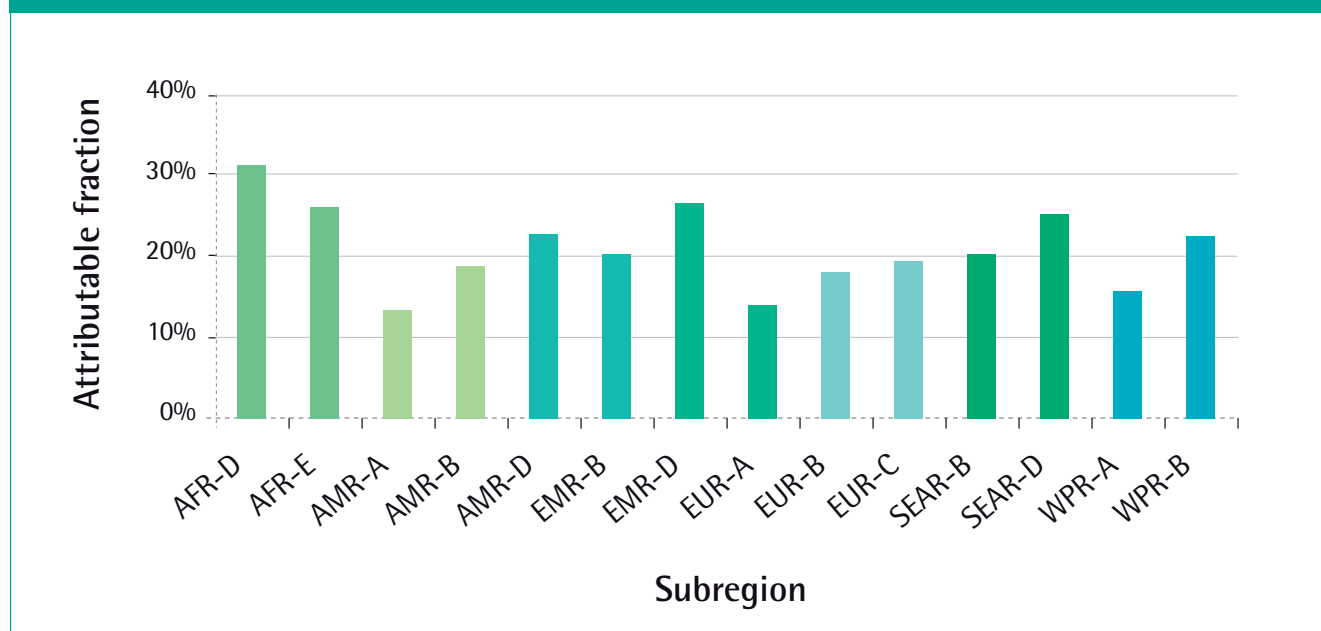
^d For each disease the fraction attributable to environmental risks is shown in dark green. Light green plus dark green represents the total burden of disease.

2. IN WHICH REGIONS OF THE WORLD IS HEALTH MOST AFFECTED BY ENVIRONMENTAL FACTORS, AND HOW?

Developing regions carry a disproportionately heavy burden for communicable diseases and injuries.

The largest overall difference between WHO regions was in infectious diseases. The total number of healthy life years lost per capita as a result of environmental burden per capita was 15-times higher in developing countries than in developed countries. The environmental burden per capita of diarrhoeal diseases and lower respiratory infections was 120- to 150-times greater in certain WHO developing country subregions as compared to developed country subregions. These differences arise from variations in exposure to environmental risks and in access to health care.

ENVIRONMENTAL DISEASE BURDEN, BY WHO SUBREGION ^a



^a The burden of disease is measured in DALYs. See Annex 1 for country groupings within WHO subregions.

No overall difference between developed and developing countries in the fraction of non-communicable disease attributable to the environment was observed.

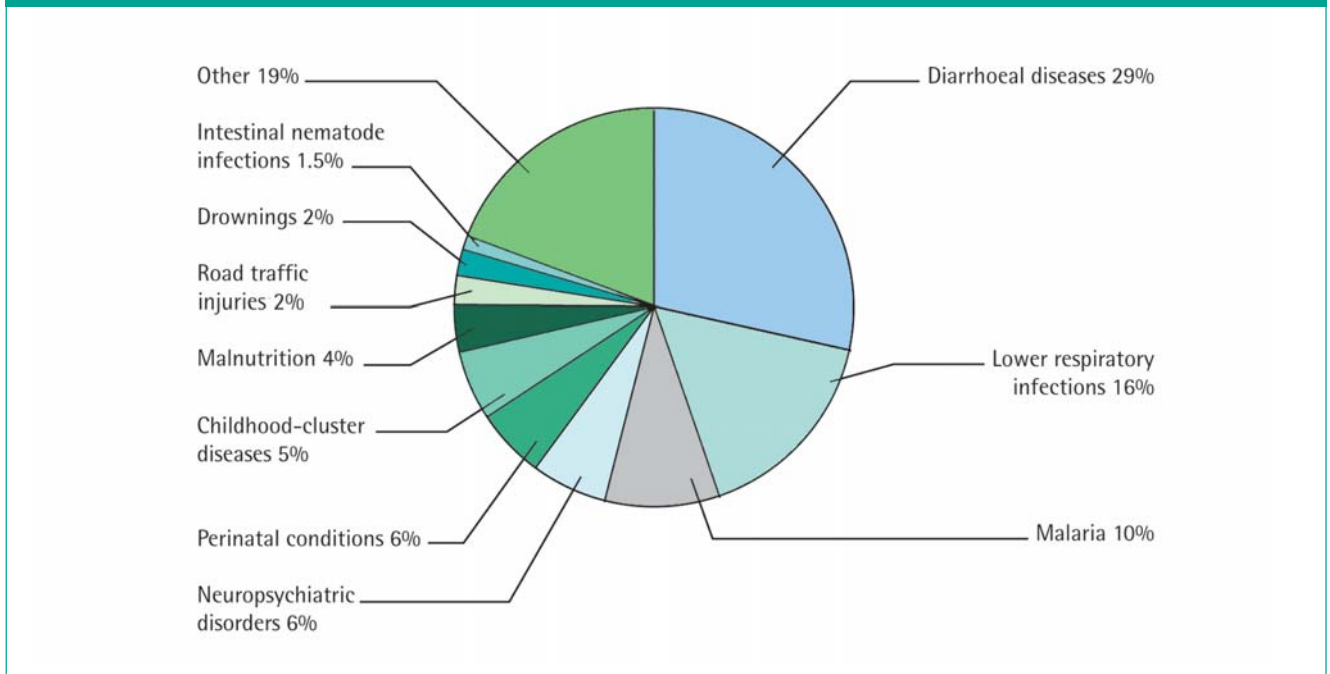
... However, in developed countries, the per capita impact of cardiovascular diseases and cancers is higher.

The number of healthy life years lost from cardiovascular disease, as a result of environmental factors, was 7-times higher, per capita, in certain developed regions than in developing regions, and cancer rates were 4-times higher. Physical inactivity is a risk factor for various non-communicable diseases including ischaemic heart disease, cancers of the breast, colon and rectum, and diabetes mellitus. It has been estimated that in certain developed regions such as North America, physical inactivity levels could be reduced by 31% through environmental interventions, including pedestrian- and bicycle-friendly urban land use and transport, and leisure and workplace facilities and policies that support more active lifestyles.

... Developing countries, meanwhile, carry a heavier burden of disease from unintentional injuries and road traffic injuries attributable to environmental factors.

In developing countries, the average number of healthy life years lost, per capita, as a result of injuries associated with environmental factors, was roughly double that of developed countries; the gap was even greater at the subregional level. For road traffic injuries, there was a 15-fold difference between the environmental burden of disease in the best performing and worst-performing subregions, and a 10-fold disparity for 'other' unintentional injuries.

MAIN DISEASES CONTRIBUTING TO THE ENVIRONMENTAL BURDEN OF DISEASE AMONG CHILDREN 0–14 YEARS ^a



^a The environmental disease burden is measured in disability-adjusted life years, a weighted measure of death, illness and disability (DALYs).

The results suggest that an important transition in environmental risk factors will occur as countries develop. For some diseases, such as malaria, the environmental disease burden is expected to decrease with development, but the burden will increase from other noncommunicable diseases, such as chronic obstructive pulmonary disease (COPD), to levels approximate with those seen in more developed regions of the world.

3. WHICH POPULATIONS SUFFER THE MOST FROM ENVIRONMENTAL HAZARDS TO HEALTH?

Children suffer a disproportionate share of the environmental health burden.

Globally, the per capita number of healthy life years lost to environmental risk factors was about 5-times greater in children under five years of age than in the total population. Diarrhoea, malaria and respiratory infections all have very large fractions of disease attributable to environment, and also are among the biggest killers of children under five years old. In developing countries, the environmental fraction of these three diseases accounted for an average of 26% of all deaths in children under five years old. Perinatal conditions (e.g. prematurity and low birth weight); protein-energy malnutrition and unintentional injuries – other major childhood killers – also have a significant environmental component, particularly in developing countries.

On average, children in developing countries lose 8-times more healthy life years, per capita, than their counterparts in developed countries from environmentally-caused diseases. In certain very poor regions of the world, however, the disparity is far greater; the number of healthy life years lost as a result of childhood lower respiratory infections is 800-times greater, per capita; 25-times greater for road traffic injuries; and 140-times greater for diarrhoeal diseases. Even these statistics fail to capture the longer term effects of exposures that occur at a young age, but do not manifest themselves as disease until years later.

4. WHAT CAN POLICYMAKERS AND THE PUBLIC DO ABOUT ENVIRONMENTAL RISKS TO HEALTH?

Public and preventive health strategies that consider environmental health interventions can be very important. Such interventions are cost-effective and yield benefits that also contribute to the overall well-being of communities.

Many environmental health interventions are economically competitive with more conventional curative health-sector interventions. Examples include phasing out leaded gasoline. Mental retardation due to lead exposures in general was estimated to be nearly 30 times higher in regions where leaded gasoline was still being used, as compared with regions where leaded gasoline had been completely phased out.

A key target of the Millennium Development Goals (MDG-7) is halving the proportion of people without sustainable access to safe drinking-water and sanitation by 2015. Globally, WHO has estimated that the economic benefits of investments in meeting this target would outweigh costs by a ratio of about 8:1. These benefits include gains in economic productivity as well as savings in health-care costs and healthy life years lost, particularly as a result of diarrhoeal diseases, intestinal nematode infections and related malnutrition.

Providing access to improved drinking-water sources in developing countries would reduce considerably the time spent by women and children in collecting water. Providing access to improved sanitation and good hygiene behaviours would help break the overall cycle of faecal-oral pathogen contamination of water bodies, yielding benefits to health, poverty reduction, well-being and economic development.

Reducing the disease burden of environmental risk factors will contribute significantly to the Millennium Development Goals.

Many Millennium Development Goals (MDGs) have an environmental health component; key elements are highlighted below.

GOAL 1 ERADICATE EXTREME POVERTY AND HUNGER

Minimizing exposures to environmental risk factors indirectly contributes to poverty reduction, because many environmentally mediated diseases result in lost earnings. Also, disability or death of one productive household member can affect an entire household. With respect to hunger, healthy life years lost to childhood malnutrition is 12-times higher per capita in developing regions, compared with developed regions. There was a 60-fold difference in WHO subregions with the highest and lowest malnutrition rates.

GOAL 2 ACHIEVE UNIVERSAL PRIMARY EDUCATION

Providing safe drinking-water and latrines at school (particularly latrines for girls) will encourage primary school attendance. Interventions that provide households with access to improved sources of drinking-water and cleaner household energy sources also improve student attendance, saving time that children would otherwise spend collecting water and/or fuel. The same interventions can save children from missing school as a result of illness or injury.

GOAL 3 PROMOTE GENDER EQUALITY AND EMPOWER WOMEN

Particularly in developing countries, access to improved drinking-water sources, cleaner household energy sources, and more generally, reduction of environmentally-attributable burden of childhood diseases, can save time women now spend in collection of fuel, water, and care for children who become sick. Time thus saved also can be invested by women in income-generating activities and education, thus contributing to the MDG goal of empowering women and promoting gender equality.

GOAL 4 REDUCE CHILD MORTALITY

The mortality rate in children under five years of age from environmentally-mediated disease conditions is 180 times higher in the poorest performing region, as compared with the rate in the best performing region. In terms of just diarrhoea and lower respiratory infections, two of the most significant childhood killers, environmental interventions could prevent the deaths of over 2 million children under the age of five every year, and thus help achieve a key target of this MDG – a two-thirds reduction in the rate of mortality among children in that age category.

GOAL 5 IMPROVE MATERNAL HEALTH

Environmental interventions can contribute to this MDG by providing a safe home environment, which is of great importance to the health of children and pregnant mothers. Conversely, a contaminated home environment is a threat to the mother and her unborn child. Childbirth, for example, requires safe water and sanitary conditions.

GOAL 6 COMBAT HIV/AIDS, MALARIA AND OTHER DISEASES

Results of this analysis indicate that over half a million people die every year from malaria, and over a quarter of a million people die from HIV/AIDS, as a result of environmental and occupational causes. A large proportion of malaria, in particular, may be attributable to readily modifiable environmental factors, such as land use, irrigation and agricultural practices.

GOAL 7 ENSURE ENVIRONMENTAL SUSTAINABILITY

Diarrhoeal diseases associated with a lack of access to safe drinking-water and inadequate sanitation result in nearly 1.7 million deaths annually. Household use of biomass fuels and coal by over one-half of the world's population, results in 1.5 million deaths a year from pollution-related respiratory diseases. Enhancing access to improved sources of drinking-water, sanitation, and clean energy are therefore key environmental interventions that can reduce pressures on ecosystems from water and air-borne contamination, and also improve health. Residents in fast-growing cities of the developing world may be exposed to the combined health hazards of unsafe drinking-water, inadequate sanitation, and indoor and outdoor air pollution. Reductions in such environmental exposures will both improve the health and the lives of urban slum dwellers – one of the key targets of MDG-7.

GOAL 8 DEVELOP A GLOBAL PARTNERSHIP FOR DEVELOPMENT

The underlying message of this study is that both the health sector and non-health sector actors can, and need, to take joint action to effectively address environmentally-mediated causes of disease. To do this global partnerships are essential. Many such alliances already exist in the field of children's environmental health; occupational health; in joint health sector and environment sector linkages; and in actions in the water, chemical and air pollution sectors. Such global partnerships need to be strengthened and reinforced, harnessing the full range of policy tools, strategies and technologies that are already available – to achieve the interrelated goals of health, environmental sustainability, and development.





INTRODUCTION

1

How much can the burden of disease be reduced by reducing environmental risks to health? If we can estimate the burden of disease from environmental risks, we also can evaluate the most important priorities for targeted environmental protection, while helping to promote the idea that sound environmental management plays a key role in protecting people's health.

Early estimates of the global disease burden attributable to the environment, derived partly on the basis of expert opinion, were in general agreement (WHO, 1997: 23%; Smith, Corvalán and Kjellström, 1999: 25–33%). A third major study of OECD countries, however, yielded significantly different results, concluding that only 2.1%–5.0% of the overall disease burden was attributable to the environment (Melse and de Hollander, 2001). This lower estimate can be explained both by the methodology used and research scope (e.g. occupational risk factors were not considered), and the different impact environmental risks have on health in developed countries – as compared to developing ones.

Even more recently, WHO developed a framework for a much more rigorous approach to burden of disease estimations. This project, known as the Comparative Risk Assessment (CRA), considered 6 environmental and occupational risk factors among a set of 26 environmental, occupational, social and behavioural risk factors having a major impact on population health (WHO, 2002). The total disease burden attributable to these risk factors was estimated across all 14 WHO subregions, 8 age groups, and by gender. The six environmental and occupational risk factors considered in the CRA were factors for which there was clear causal evidence that could be applied globally; for which global estimates of exposure could be obtained; and which had large impacts on people's health. However, this assessment remained limited in terms of the range of environmental risks assessed, and with respect to quantification of impacts in terms of specific health conditions.

The present analysis goes a step further, providing timely new estimates of burden of disease from a much broader range of environmental risk factors, and in terms of the categories of diseases and health conditions affected. The analysis makes use of the results from the CRA, complemented by extensive literature reviews and standardized surveys of expert opinions, in an approach that aims to improve scientific rigour and transparency. Focusing on modifiable environmental risks, the current assessment examines "how much" such factors affect various diseases and injuries – both in terms of premature mortality and in terms of overall disease burden as measured by DALY's (disability adjusted life years), a weighted measure of death and disability.

The definition of "modifiable" environmental risk factors include those reasonably amenable to management or change. Factors not readily modifiable were not considered here. The analysis considered most environmental risks and related diseases that could be quantified from available evidence. In some cases, however, disease burden from a known environmental risk was not quantifiable. This included certain diseases associated with changed, damaged or depleted ecosystems, and diseases associated with exposures to endocrine disrupting substances. The resulting analysis thus remains a conservative estimate of environmental disease burden.

This analysis provides timely new estimates of burden of disease from modifiable environmental risk factors.



Park in Shanghai.

Credit: Thomas Roetting/Still Pictures

WHAT IS THE ENVIRONMENT IN THE CONTEXT OF HEALTH?

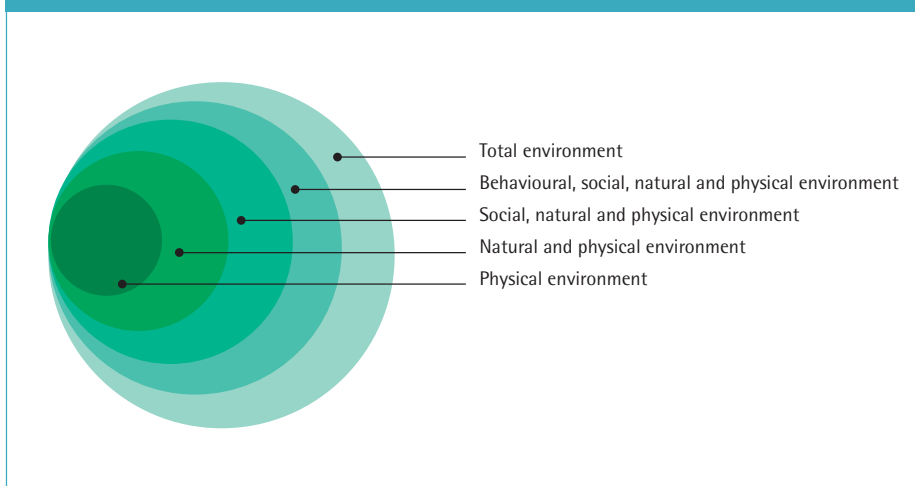
2



In the medical sense, the environment includes the surroundings, conditions or influences that affect an organism (Davis, 1989). Along these lines, Last (2001) defined the environment for the International Epidemiological Association as: "All that which is external to the human host. Can be divided into physical, biological, social, cultural, etc., any or all of which can influence health status of populations ...". According to this definition, the environment would include anything that is not genetic, although it could be argued that even genes are influenced by the environment in the short or long-term.

Figure 1 shows one way to represent the environment, from the most inclusive to the most restrictive definition (Smith, Corvalán and Kjellström, 1999).

FIGURE 1 DEFINITION OF THE ENVIRONMENT ^a



^a (Adapted from Smith, Corvalán and Kjellström, 1999)

For the purposes of environmental health, however, a more practical definition of the environment is needed, because environmental health action generally tries to change only the natural and physical environments and related behaviours (e.g. hand washing). Such interventions can rarely modify the social and cultural aspects of a community, which are usually independent of the environment (e.g. cultural pressures on lifestyle, unemployment). As a result, a more practical definition of the environment might be that given in Box 1.

A practical definition of the environment, targeted at what can be done through environmental health action, is needed.



Modern Tram line in France supports a healthier environment.

Credit: Martin Bond/Still Pictures

We can define 'environment' as "all the physical, chemical and biological factors external to the human host," as well as those factors impacting related behaviours.



Survivors of a flood in the Phillipines play in the debris of a polluted water site.

Credit: N. Dickinson/UNEP/Still Pictures

Box 1 A DEFINITION OF "ENVIRONMENT" FOR MEASURING THE ENVIRONMENTAL IMPACT ON HEALTH

The environment is all the physical, chemical and biological factors external to a person, and all the related behaviours.

This definition excludes behaviour not related to environment, as well as behaviour related to the social and cultural environment, and genetics.

For our analysis, we have limited the definition of environment further, to those parts of the environment that can be modified by short-term or longer-term interventions, so as to reduce the health impact of the environment (Box 2).

Box 2 THE DEFINITION OF "ENVIRONMENT" USED IN THIS STUDY

The environment is all the physical, chemical and biological factors external to the human host, and all related behaviours, but excluding those natural environments that cannot reasonably be modified.

This definition excludes behaviour not related to environment, as well as behaviour related to the social and cultural environment, genetics, and parts of the natural environment.

This definition thus aims to cover those parts of the environment that can be modified by environmental management. For onchocerciasis, for example, the definition of environment would include only that part of the environment that had been affected by man-made interventions (in this case, dams), and which could be modified by further intervention. Estimates of the environmental health impact would not include disease caused by vectors living in natural environments such as rivers, if those vectors could not be controlled by reasonable environmental interventions. Similarly, deaths and injuries of soldiers during war is not included here, even though they could be considered occupational, because no intervention could possibly provide a safe working environment.

Our definition of "environment" is thus not all-inclusive in terms of the natural environment, and includes only those aspects that are modifiable (not necessarily immediately, but with solutions that are already available). Factors that have been included in our definition of "environment", or excluded, are given in Box 3.

Box 3 EXAMPLES OF FACTORS INCLUDED IN, OR EXCLUDED FROM, OUR WORKING DEFINITION FOR “ENVIRONMENT”.

Included environmental factors are the modifiable parts (or impacts) of:

- pollution of air, water, or soil with chemical or biological agents;
- UV and ionizing radiation^a;
- noise, electromagnetic fields;
- occupational risks^b;
- built environments, including housing, land use patterns, roads;
- agricultural methods, irrigation schemes;
- man-made climate change, ecosystem change;
- behaviour related to the availability of safe water and sanitation facilities, such as washing hands, and contaminating food with unsafe water or unclean hands.

Excluded environmental factors are:

- alcohol and tobacco consumption, drug abuse;
- diet (although it could be argued that food availability influences diet);
- the natural environments of vectors that cannot reasonably be modified (e.g. in rivers, lakes, wetlands);
- impregnated bed nets (for this study they are considered to be non-environmental interventions);
- unemployment (provided that it is not related to environmental degradation, occupational disease, etc.);
- natural biological agents, such as pollen in the outdoor environment;
- person-to-person transmission that cannot reasonably be prevented through environmental interventions such as improving housing, introducing sanitary hygiene, or making improvements in the occupational environment.

^a Although natural UV radiation from space is not modifiable (or only in a limited way, such as by reducing substances that destroy the ozone layer), individual behaviour to protect oneself against UV radiation is modifiable. UV and other ionizing radiations are therefore included in our assessment of the environmental disease burden.

^b Occupational health risks also are directly related to physical, chemical and biological factors in the environment and related behaviours. This report focuses on such occupational risks as part of the general environment. For instance, in the context of the working definition for environmental factors used in this report, infections acquired by health care workers from needlestick injuries, as well sexually-transmitted diseases acquired in other occupational contexts, e.g. among commercial sex workers, are, for example, included in the analysis, as this refers to contact with infectious agents in the work environment, and related behaviour. Occupational health risks also may include the more distal economic and social determinants of occupational conditions, such as job security, which are however not fully addressed here.

Our definition of environment is further limited to include the consideration of only modifiable environmental factors, that is factors readily amenable to change.



Laying water and sewage lines in Bhutan, a measure that can facilitate access to safe drinking-water and improved sanitation.

Credit: Jorgen Schytte/Still Pictures

WHAT IS MEANT BY THE "ATTRIBUTABLE FRACTION" OF A RISK FACTOR?

3



If members of a community are exposed to a risk factor (e.g. agricultural pesticides) that causes health problems or deaths, and that risk factor is removed from the environment (e.g. by legislative action), we would expect that the overall number of health problems or deaths in the community would decline. The proportional reduction in the number of health problems or deaths as a result of reducing the risk factor is known as the "attributable fraction". In other words, it is the proportion of all health problems or deaths in the community that can be attributed to the risk factor (Miettinen, 1974; Greenland, 1984).

When calculating the disease burden attributable to an environmental risk factor (the attributable fraction), the simplest case is when exposure to the risk factor can be reduced to zero, but this is not always achievable in practice. For example, outdoor air pollution from particulate matter cannot be reduced to zero, because along with the particulates emitted by fossil fuel combustion, airborne particulate matter also occurs naturally (albeit at low levels). For this reason, this analysis considers how much disease burden would decrease if exposure to a risk factor were reduced, not to zero, but to some achievable level (the counterfactual or baseline level).

A second issue is the determination of what are "reasonably modifiable" environmental factors. Transport policy tradeoffs illustrate the difficulties implicit in such determinations. Banning cars entirely from cities as an air pollution reduction measure, for example, may not be practical or feasible, at least at present. However, the adoption of cleaner motor vehicle technologies and alternative modes of transport (e.g. rail, bus, cycling and walking) is very widely considered by policymakers. Such strategies would thus be considered as part of the modifiable environment, in the context of measures that could reduce urban air pollution and related diseases.

Often, disease burden is the result of diverse environmental, social and behavioural risk factors. The sum of these separate risk factors (attributable fractions) may add up to more than 100% – meaning that disease burden could be potentially reduced or eliminated by different forms of interventions. To decide on the best option, factors such as the cost-effectiveness of alternative interventions must be considered. However, environmental modification may offer several inherent advantages:

- preventing disease before it arises eliminates associated health-care treatment costs, and no burden is borne by the population;
- such interventions may be more generally sustainable (i.e. achieving a longer-term impact on health, as compared to medical treatment);
- environmental modification is often the most equitable option, generating benefits across broad groups or populations.

The 'attributable fraction' is the decline in disease or injury that could be achieved in a given population by reducing the risk.



Flooded neighborhood in the UK. Climate change can increase the risk of extreme weather events, leading to a range of health impacts, some of which are quantifiable, while others have not been measured.

Credit: Paul Glendell/Still Pictures



METHODS 4

The purpose of this analysis was to update and complete global estimates of the amount of disease that is attributable to the environment. We did this by combining existing evidence-based estimates of the disease burden with more approximate estimates for areas with limited evidence. In general, we gave priority to CRA results (WHO, 2002) and developed conservative estimates on the basis of additional approximate or qualitative estimates.

The CRA compares the global impact on health of 26 risk factors. The six environmental CRA risk factors included in our analysis are summarized in Table 1.

TABLE 1 ENVIRONMENTAL RISK FACTORS AND RELATED DISEASES INCLUDED IN THE CRA^a

Risk factors	Related diseases
Outdoor air pollution	Respiratory infections, selected cardiopulmonary diseases, lung cancer
Indoor air pollution from solid fuel use	COPD ^b , lower respiratory infections, lung cancer
Lead	Mild mental retardation, cardiovascular diseases
Water, sanitation and hygiene	Diarrhoeal diseases, trachoma, schistosomiasis, ascariasis, trichuriasis, hookworm disease
Climate change	Diarrhoeal diseases, malaria, selected unintentional injuries, protein-energy malnutrition
Selected occupational factors:	
injuries	Unintentional injuries
noise	Hearing loss
carcinogens	Cancers
airborne particulates	Asthma, COPD
ergonomic stressors	Low back pain

^a Source: Comparative Risk Assessment (WHO, 2002).

^b COPD: chronic obstructive pulmonary disease.

The risk factors in Table 1 are only some of the environmental risks that have health consequences, and not all the related diseases were addressed. Certain diseases or environmental risk factors were not included in our analysis, either because there was insufficient evidence at global level, or no global exposure estimates, or because the risk factor caused a relatively small disease burden. In the CRA, the global disease burden from all the environmental risk factors amounted to only 9.6% of the total disease burden.

The analysis uses results from the WHO Comparative Risk Assessment (2002), along with standardized surveys of expert opinion.



Farmworker in Asia exposed to pesticides while spraying crops without any protective gear.

Credit: Julio Etchart/Still Pictures

The attributable environmental fraction is a mean value, not necessarily applicable to any individual country.



Beaches are settings where a range of potential health risks, e.g. drownings and pollution of recreational waters, may be reduced by good environmental health policies and practices.

Credit: Philippe Hays/Still Pictures

To estimate the health impact of environmental risks worldwide, current CRA estimates for specific environmental factors needed to be completed and updated. We therefore conducted a survey of experts and asked them to provide estimates of the attributable fractions for specific diseases in their area of competence. More than 100 experts were selected on the basis of their international expertise in the area of each disease or risk factor of concern. The experts were identified either by the WHO unit responsible for the area, by other experts in the area, or as authors of key publications. We tried to balance the survey by including experts both from the disease and the risk factor perspectives, and who represented various regions, particularly when a risk factor showed significant geographical variation.

The experts were provided with summaries of information and references on each disease, as well as an initial estimate that was based on pooled estimates from the literature. CRA results also often provided partial results for one disease and a corresponding attributable risk. In total, 85 diseases and two risk factors were covered by the survey. The two risk factors were malnutrition and physical inactivity, and they were included because they are themselves influenced by environmental factors and have been linked quantitatively to various diseases (Bull et al., 2004; Fishman et al., 2004). Experts were asked to provide a point estimate and a 95% confidence interval for the attributable fraction. Experts were free to provide estimates by gender, age group or geographical region. A minimum of three independent expert opinions were obtained for each disease.

For each disease, it was assumed that the attributable fractions reported by the experts had a triangular probability distribution, defined by a maximum probability at the best estimate and the 95% confidence limits (Figure 2). These probability distributions were pooled, giving equal weighting to each distribution (i.e. to each expert reply), to obtain a combined probability distribution for the attributable fraction (Figure 3). The arithmetic mean of the combined probability distribution is the best estimate of the attributable fraction for the disease, with the new 95% confidence limits defined by the combined probability distribution (Figure 3). It is important to remember that the resulting attributable fraction is a mean value and is not applicable to any individual country, particularly if the associated risks vary significantly from country to country.

This method tends to overweight estimates at the extremes of a probability distribution, and confidence intervals are therefore generally large. For outliers, (i.e. estimates that do not overlap with any of the ranges provided by other experts), we used the outlier point estimate to define the upper or lower boundary (as relevant) of the pooled confidence interval, not the corresponding 95% confidence boundary for the outlier.

If CRA results were used, or other estimates with no specified uncertainty estimates, we used $\pm 30\%$ lower and upper boundaries around the best estimate to define the confidence intervals. No confidence intervals were used if the attributable fraction was 100%.

As an example, the method for analysing road traffic injuries in developing countries is outlined below. Five individual estimates, A-E, were obtained from expert replies (Figure 2), which were pooled to give a combined probability distribution for the attributable fraction for road traffic injuries (Figure 3). In this case, the CRA estimate was below the lower range of the combined probability distribution for expert replies. This was because the CRA estimate was obtained only for occupational causes of road injuries, which were a fraction of the many possible causes contributing to road traffic injuries (e.g. poor road design and maintenance, poor land use patterns).

To estimate the attributable fraction in terms of deaths and disability-adjusted life years (DALYs), the attributable fractions for each disease (obtained from the pooled expert estimates) was multiplied by the total number of deaths or DALYs for the disease in 2002. The global data were obtained from the WHO database¹, www.who.int/evidence, under "Burden of Disease Project", and "Global Burden of Disease Estimates", or from Annex Tables 2 and 3 of the World Health Report (WHO, 2004a). The global estimate of the attributable fraction for the environmental risk factors included in this study was then obtained by adding all disease-specific deaths and DALYs obtained in this way. To construct confidence intervals around the summary statistics, we used the software package @risk 4.5 for Excel (Palisade Europe UK Ltd., London) and simulation techniques (King, Tomz and Wittenberg, 2000), with the probability distributions for the individual attributable fractions as input.

STRENGTHS AND WEAKNESSES OF THE ANALYSIS

It is likely that our analysis underestimates the global burden of disease attributable to reasonably modifiable environmental causes, for several reasons. First, the experts generally derived their estimates on the basis of existing literature, yet only a fraction of environmental and occupational risks are adequately covered in the literature. There are many examples of risks that have not been adequately evaluated, including the effects of emerging risks (e.g. more intensive agricultural practices and zoonoses), the effects of many long-term chemical exposures on cancers or endocrine disorders, and the impact of electromagnetic and other exposures from new technologies. It was clear from the responses to our survey that the experts did not consider such poorly documented risk factors, as well as factors that are suspected to pose a risk, but for which there is no "hard" evidence.

¹ In the WHO database of disease statistics, diseases are grouped according to the International Classification of Diseases (WHO, 1992). Estimates are calculated for each gender, for eight age groups and 14 WHO subregions. The subregions are defined by geographical region and mortality stratum, and the country grouping is given in Annex 1.

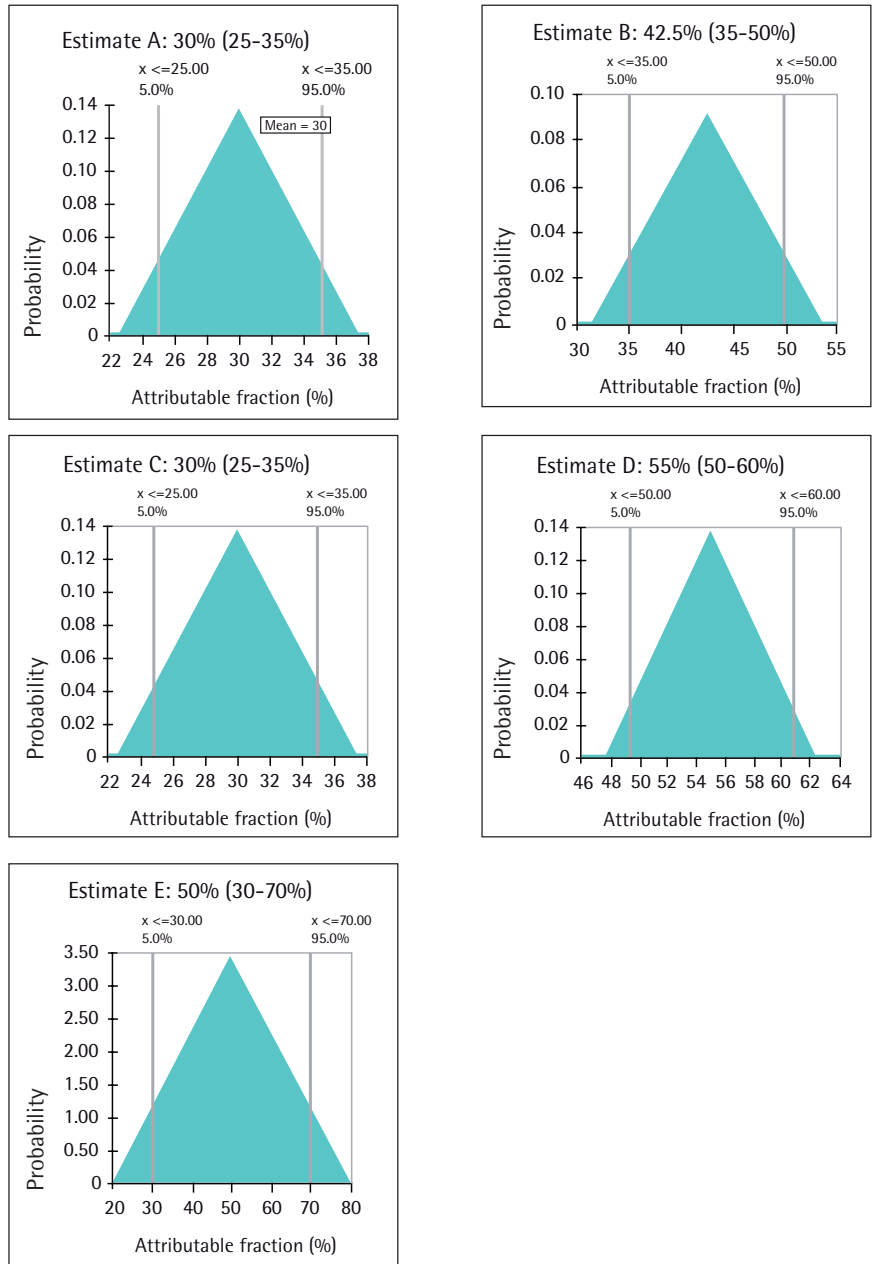
Our analysis underestimates the global burden of disease attributable to modifiable environmental factors, due to insufficient evidence regarding certain environmental risks.



Coastal slum in Asia prone to flooding and water pollution, occupied by families too poor to purchase houses further inland.

Credit: Mark Edwards/Still Pictures

FIGURE 2 PROBABILITY DISTRIBUTIONS OF FIVE EXPERT ESTIMATES FOR THE ATTRIBUTABLE FRACTION OF ROAD TRAFFIC INJURIES



Most experts considered the more immediate environmental risk to health, rather than more "distal" causes.

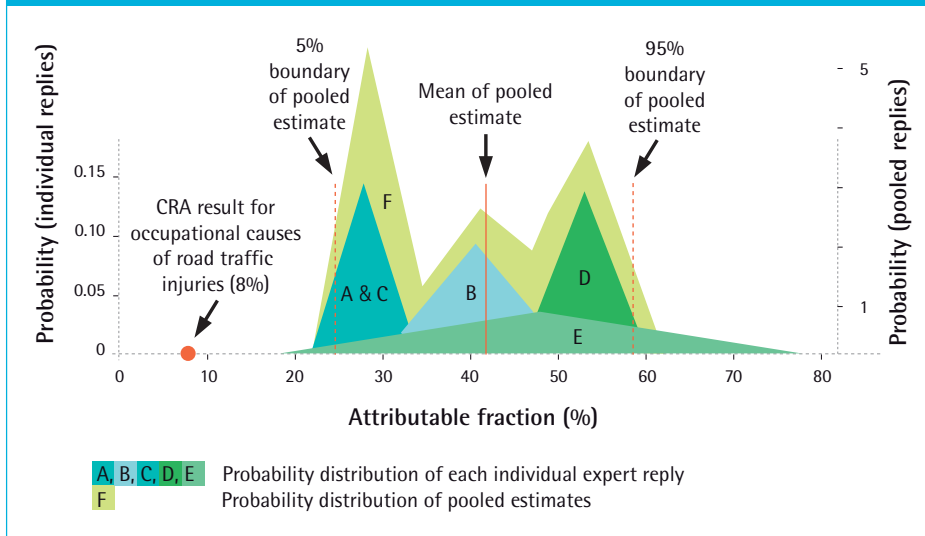


Urban environmental conditions and related behaviours interact, heightening exposure to traffic injury risk on a busy road in south-east Asia.

Credit: Jorgen Schytte/Still Pictures

Furthermore, most experts considered the more "proximal" or immediate environmental risk to health, rather than the more "distal" or "upstream" causes, which may again have some underlying environmental risks. For example, the incidence of tuberculosis in many countries is strongly driven by immigration from regions of the world with a high prevalence of tuberculosis. At the same time, environmental disruption associated with land degradation, water insecurity, and climate change-related events can have an important influence on population movement.

FIGURE 3 OVERLAY OF INDIVIDUAL EXPERT ESTIMATES, CRA ESTIMATE, AND POOLED ESTIMATE FOR ROAD TRAFFIC INJURIES IN DEVELOPING COUNTRIES



A composite graphic portraying (1) the CRA estimate for the fraction of road traffic injuries attributable to occupational factors; (2) individual expert estimates for attributable environmental fraction; (3) the resulting pooled estimate; (4) and the resulting mean estimate for road traffic injuries attributable to environmental factors in developing countries.

In this analysis, however, the effects of these more distal causes have not been taken into account. And this, too, may lead to an underestimate of the global health burden attributable to modifiable environmental factors.

Given the lack of information regarding many environmental risks and their impacts on health, we could have estimated the fraction of disease attributable to the environment by first estimating the causes of disease that are not environmental, and then attributing the remaining fraction to the environment. Such an approach, however, would have led to a much less conservative estimate.

UNCERTAINTIES

A large part of this analysis is based on surveys of expert opinion and, like many such analyses, the uncertainties of such estimates are relatively large. In part, this is because expert opinion generally reflects the evidence in the literature, which may not be homogeneous, can be region-specific, or incomplete. We have therefore provided not only point estimates, but also the likely ranges of the estimate. The uncertainties reflect the confidence intervals provided by the experts.

Uncertainties of estimates derived from surveys of experts are relatively large, so we have provided likely ranges as well as point estimates.



Separated bicycle lanes in Amsterdam; safer routes for cycling.

Credit: Argus/Still Pictures

ANALYSIS OF ESTIMATES OF THE ENVIRONMENTAL ATTRIBUTABLE FRACTION, BY DISEASE

U5



More than 100 experts participated in the survey and provided a total of about 200 quantitative replies (some experts provided environmental attributable fractions for several diseases or injuries). Other estimates of the environmental attributable fraction came from the CRA (WHO, 2002). We report the results for each disease or disease group in the following sections.

RESPIRATORY INFECTIONS

Indoor and outdoor air quality are two of the main environmental factors of concern for acute lower respiratory infections. Contributing risk factors include tobacco smoke, solid fuel use (Kirkwood et al., 1995; Smith et al., 2000), housing conditions and possibly hygiene. Previous estimates (WHO, 2002; Smith, Mehta and Maeusezahl-Feuz, 2004) showed that 36% of lower respiratory infections worldwide were attributable to solid fuel use alone, and 1% of all respiratory infections to outdoor air pollution (WHO, 2002; Cohen et al., 2004). In developed countries, solid fuel use was not significant, and environmental tobacco smoke may play a proportionally more important role in these countries. A study in Italy, for example, estimated that 21% of acute respiratory infections in the first two years of life were due to parental smoking (Forastiere et al., 2002).

A study in Europe determined that acute lower respiratory tract infections – attributable to indoor air pollution from solid fuel use alone – account for 4.6% of all deaths and 3.1% of all DALYs in children aged 0–4 (Valent et al., 2004).

Adding the effects of indoor and outdoor air pollution and other indoor conditions, at least 42% (95% Confidence Interval: 32–47%) of all lower respiratory infections were estimated to be attributable to the environment in developing countries. In developed countries, this rate was about halved to 20% (15–25%). It was more difficult to quantify the influence of other environmental factors (e.g. chilling, crowding), and the co-morbidities with other diseases that are partly attributable to the environment (e.g. malaria and diarrhoea), but they may add to the environmental health burden of lower respiratory infections.

The relationship of upper respiratory infections and otitis with environmental conditions was less well documented. In developing countries, about 24% (6–45%) of upper respiratory infections and otitis were attributable to environmental risk factors, such as outdoor and indoor air pollution, environmental tobacco smoke (Etzel et al., 1992; Stenstrom, Bernard and Ben-Simhon, 1993; California Environmental Protection Agency, 1997) and housing conditions. As with lower respiratory infections, the rate for upper respiratory infections and otitis was estimated to be lower in developed countries, at 12% (5–18%). Globally, more than 1.5 million deaths annually from respiratory infections are attributable to the environment (see Table A2.3).

Globally more than 1.5 million deaths annually from respiratory infections are attributable to the environment, including at least 42% of lower respiratory infections and 24% of upper respiratory infections in developing countries.



Cooking and heating with solid fuels over an open fire in Latin America. Many women and children in developing countries are thus exposed to very high concentrations of indoor air pollution, a major risk factor for respiratory infections.

Credit: Nigel Bruce/University of Liverpool

Globally, about 1.5 million deaths per year from diarrhoeal diseases are attributable to environmental factors, essentially water, sanitation and hygiene.



For a large part of the year, these settlements in Asia are surrounded by stagnant water. With no access to safe drinking water or basic sanitation, these children are constantly exposed to the risks of diarrhoea as well as other water-borne diseases.

Credit: Mark Edwards/Still Pictures

DIARRHOEA

A large proportion of diarrhoeal diseases is caused by faecal-oral pathogens. In the case of infectious diarrhoea, transmission routes are affected by interactions between physical infrastructure and human behaviours. If sanitation or related hygiene is poor, e.g. when hand washing facilities are inadequate, or when faeces are disposed of improperly, human excreta may contaminate hands, which can then contaminate food or other humans (person-to-person transmission). Faecal pathogens are frequently transferred to the waterborne sewage system through flush toilets or latrines, and these may subsequently contaminate surface waters and groundwater. Human excreta also can directly contaminate the soil and enter into contact with people; flies may carry pathogens from excreta to food, for example. Through these pathways, drinking-water, recreational water or food may be contaminated and cause diarrhoeal disease following ingestion. Animal excreta also transmit pathogens. The predominant route will depend upon the survival characteristics of the pathogen, as well as local infrastructure and human behaviour. Many interventions have proven efficient in interrupting the pathogen transmission cycle at various points.

WHO recently estimated that 88% of all cases of diarrhoea globally were attributable to water, sanitation and hygiene (WHO, 2002; Prüss-Üstün et al., 2004a). The risk factor was defined as "drinking-water, sanitation and hygiene behaviour", as well as aspects of food safety that are related to water, sanitation and hygiene (i.e. food contamination by unsafe water, or the lack of domestic hygiene). Very little disease was transmitted through pathways other than those associated with water, sanitation and hygiene, or food (e.g. airborne transmission), and about 94% (84–98%) of all cases of diarrhoea around the world were attributable to the environment, resulting in more than 1.5 million deaths annually, mainly in children. The estimate for developed countries (90%; 75–98%) was slightly smaller because there were fewer cases of infectious diarrhoea, although non-infectious diarrhoea formed a relatively higher proportion of all diarrhoea cases. Water, sanitation and hygiene also play an important role in malnutrition (covered in the subsection, Malnutrition). Diarrhoea, attributable to water and sanitation accounted for 5.3% of deaths and 3.5% of DALY's in European children aged 0-14 (Valent et al., 2004).

MALARIA

In humans, malaria is a disease caused by one of four parasite species belonging to the genus *Plasmodium*. The parasite is transmitted by the bite of an infected female mosquito of the genus *Anopheles*. The larval stages of *Anopheles* mosquitoes occur in a wide range of habitats, but most species share a preference for clean, unpolluted, stagnant or slowly moving fresh water (Muir, 1988).

There are three main approaches to the environmental management of malaria:

- *Modify the environment.* This approach aims to permanently change land, water or vegetation conditions, so as to reduce vector habitats.
- *Manipulate the environment.* This approach temporarily produces unfavourable conditions for vector propagation and therefore needs to be repeated.
- *Modify or manipulate human habitation or behaviour.* This approach aims to reduce contact between humans and vectors (WHO, 1982).

At the time these definitions were formulated, the third approach included the use of mosquito nets. The successful introduction of insecticide-treated mosquito nets has put them in a category of their own, and blurred the boundary between environmental management and chemical control. For the current survey, the use of mosquito netting was not considered to be environmental management.

An array of environmental modification and manipulation methods are available for vector control in general, and malaria control in particular (WHO, 1982). Important features of environmental management strategies are their non-toxicity, relative ease of application, cost-effectiveness and sustainability (Bos and Mills, 1987; Ault, 1994; Utzinger, Tozan and Singer, 2001). Strategies for malaria can be grouped into at least three distinct eco-epidemiological settings:

- malaria of deep forests and hills, including forest fringe malaria;
- rural malaria attributable to water resource development and management (e.g. irrigation and large dams), wetlands, rivers, streams and coasts;
- urban and periurban malaria.

The modification or manipulation of human habitation to reduce human contact with vectors can be used relatively easily in all eco-epidemiological settings except for forest areas, where such efforts are less feasible and therefore generally not recommended.

Environmental modification steps to control malaria include:

- drainage
- levelling land
- filling depressions, borrow pits, pools and ponds
- contouring reservoirs
- modifying river boundaries
- lining canals to prevent seepage
- constructing hydraulic structures, such as weirs, to avoid stagnant water.

Environmental management of malaria can involve modification or manipulation of the environment, as well as of human habitation and behaviour.



Anopheles stephensi, the urban vector of malaria in south Asia takes a blood meal. Different mosquito species transmitting a number of diseases breed in man-made environments. This makes environmental management an important component of vector control.

Credit: CDC/Jim Gathany

An estimated 42% of the global malaria burden, or half a million deaths annually, could be prevented by environmental management.



A health worker informs residents of an Ethiopian community about the value of environmental management in preventing malaria infection, with the aid of a health education poster.

Credit: WHO/TDR/Olivier Martel

In an urban environment, environmental modification options also include building drains and storm-drains, modifying house design (including gutters and roof drains), and installing wastewater management facilities. Other environmental tools for controlling malaria include water management (e.g. intermittent, or alternate wet and dry irrigation; sprinkler, drip or central pivot irrigation); vegetation management in rural settings; safe practices for storing domestic water; management of solid waste in and around urban environments; and the maintenance of water supply and sanitation in urban areas.

It was estimated that 42% (30–53%) of the global malaria burden, or half a million deaths annually, could be prevented by environmental management, although the fraction amenable to environmental management varied slightly, depending on the region: 36% (25–47%) in the Eastern Mediterranean Region; 40% (34–46%) in the Western Pacific Region; 42% (28–55%) in sub-Saharan Africa; 42% (30–54%) in the South-East Asia Region; 50% (38–63%) in the European Region; and 64% (51–77%) in the Region of the Americas. The potential of environmental management to reduce the disease burden of malaria differed according to the type of environment (i.e. deep forests and hills, rural settings, and urban and periurban settings). The differences can be explained by local differences in the behaviour of *Anopheles* species (e.g. biting and resting behaviour), and by the number and characteristics of their breeding sites (e.g. in urban areas there are generally fewer breeding sites and they are easier to get to for vector control).

INTESTINAL NEMATODE INFECTIONS

Ascariasis, trichuriasis and hookworm disease are all transmitted via soil and other media that are contaminated with excreta containing infective eggs or larvae. Transmission may take place near the home, or in a communal area with inadequate sanitation facilities and that is polluted with faeces. Transmission occurs when infective eggs are ingested, and in the case of hookworm disease, also when infective larvae penetrate the skin (Benenson, 1995). In addition, eggs may be found on uncooked food products contaminated with soil, faeces or wastewater. Transmission does not occur from person-to-person or from fresh faeces. Even if freshly excreted faeces are contaminated, it takes time for the parasite to develop and for the faeces to become infectious. These nematode infections can therefore be considered essentially 100% attributable to the environment, and they occur because of a lack of excreta management and inadequate hygiene practices (Prüss-Üstün et al., 2004a).

TRACHOMA

Trachoma is a chronic contagious eye disease that can result in blindness. It is caused by *Chlamydia trachomatis*, and all transmission routes are hygiene related (e.g. direct infection by flies, person-to-person contact from clothing used to wipe children's faces, etc.). Risk factors for the disease include lack of facial cleanliness, poor access to water supplies, lack of latrines, and a high number of flies (Benenson, 1995; Prüss-Üstün et al., 2004a). Trachoma-transmitting flies can be controlled by managing excreta and by making improvements to houses. Several environmental control measures are effective (Sutter and Ballard, 1983; Esrey et al., 1991; Emerson et al., 1999, 2000; Prüss and Mariotti, 2000), and trachoma can be considered to be almost 100% attributable to the environment.

SCHISTOSOMIASIS

Schistosomiasis is caused by infection with trematodes of the *Schistosoma* species. Most intermediate hosts of human *Schistosoma* parasites belong to three genera of snails; *Biomphalaria* and *Bulinus* are aquatic and *Oncomelania* is amphibious. Transmission occurs through human contact with water containing free-swimming larval forms, penetrating skin. Water is contaminated by infected humans excreting schistosome eggs in faeces or urine (Benenson, 1995). Current understanding of disease transmission indicates that disease burden is fully attributable to risk factors associated with water, sanitation and hygiene (Prüss-Üstün et al., 2004a).

CHAGAS DISEASE

Chagas disease (American trypanosomiasis) is caused by infection with the parasite *Trypanosoma cruzi*. The parasite is transmitted by various species of Mexican, and Central and South American triatomine bugs (Carcavallo et al., 1997; Coura et al., 2002), which have a range of resting and breeding places in and around houses. The disease can be controlled by interrupting transmission of the parasite. In the absence of effective drugs, an integrated vector management approach provides the best prevention and control option. Chagas disease burden can be reduced considerably by improving housing and by environmental management in peridomestic areas (Bos, 1990; Rozendaal, 1997; Rojas-De-Arias, 2001; Ramsey et al., 2003). Examples include structural improvements to houses (some triatomine bugs, e.g. *Triatoma infestans*, live in wall cracks), replacing palm-leaf roofs where *Rhodnius prolixus* is the vector, and cleaning or clearing wood stacks, goat corrals and chicken dens where *Triatoma dimidiata* tends to propagate. The global mean attributable fraction for Chagas disease was estimated to be 56% (31–80%) for environmental conditions that can be managed or manipulated.

Ascariasis, trichuriasis, hookworm disease, trachoma, schistosomiasis and Chagas disease could largely be prevented through improved hygiene, water and sanitation, and housing.



Rhodnius prolixus, here seen feeding on blood meals in a research setting, is among several species of triatomine bugs that transmit Chagas disease in central and south America. Housing improvement and peri-domestic environmental management are critical to sustained disease control.

Credit: Mark Edwards/Still Pictures

The disease burden of lymphatic filariasis, onchocerciasis and leishmaniasis could be reduced by improved water resource and waste management, and improved housing.



Children playing in a drain in an east African city are at an increased risk of water-associated diseases.

Credit: Ernst Tobisch/Still Pictures

LYMPHATIC FILARIASIS

This disease is caused by worms that live in the lymphatic system and whose larvae are transmitted by the bite of an infected mosquito. There are a number of distinct transmission pathways for this infection, which are linked to the ecological requirements of different vectors in different locations (Rozendaal, 1997; R. Bos, personal communication). In urban settings of south and south-east Asia and in the Americas, the predominant parasitic worm (*Wuchereria bancrofti*) is linked to organically polluted water (open sewage drains and waste-water treatment ponds) where *Culex quinquefasciatus* breeds (Meyrowitsch et al., 1998; Erlanger et al., 2005). In Africa, both *Culex* and *Anopheles gambiae* are key vectors in coastal areas, whereas inland *A. gambiae* complex and *A. funestus* are the main vectors. As a result, lymphatic filariasis is linked to fresh-water collections and irrigation schemes (Appawu et al., 2001; Erlanger et al., 2005). In parts of the Western Pacific region, filariasis is transmitted by *Aedes* species, including *A. polynesiensis* which breeds in crab holes. The less important *Brugia malayi* parasite, endemic mainly in India and Sri Lanka, is transmitted by mosquitoes belonging to the genus *Mansonia*, which propagate in the presence of aquatic weeds.

The variety of locations and vectors involved in this disease was reflected in the large differences in estimates for the environmental attributable fraction for the disease. In the South-East Asia Region and Western Pacific Region the attributable fraction was estimated to be 82% (50–98%), while in the Region of the Americas it was 70% (60–80%), derived mainly from considering urban environmental management. In the Africa Region, the attributable fraction was 40% (20–68%), and the resulting global average was 66% (35–86%).

ONCHOCERCIASIS

Onchocerciasis is caused by the pathogen, *Onchocerca volvulus*, which is transmitted by vectors (blackfly species belonging to the *Simulium damnosum* complex) that breed in rapidly flowing streams (Rozendaal, 1997; R. Bos, personal communication). In this analysis, only those breeding places in areas influenced by water resource projects were considered, particularly dams (e.g. building dams with a double-spillway design). Natural waters, which have limited opportunities for environmental management, were not considered. In this context, insecticide spraying of streams and rivers was not considered to be an environmental health action. Evidence suggests that disease transmission can be increased by forest degradation related to human activity, as deforested areas provide a favourable habitat for the vector of the more severe strain of the pathogen (Wilson et al., 2002; Adjami et al., 2004). The global environmental attributable fraction for this disease was estimated to be 10% (7–13%).

LEISHMANIASIS

Leishmaniasis is caused by parasitic protozoan species belonging to the genus *Leishmania*, which are transmitted by sandflies. Clinical manifestations are species-related, ranging from visceral to cutaneous to mucocutaneous. To some extent, leishmaniasis could be prevented in Africa and Asia by making improvements to housing. Houses with cracks in mud or masonry walls, as well as compounds where cattle are kept in close proximity to living quarters, provide breeding sites for the flies (Rozendaal, 1997; Desjeux, 2001; Bucheton et al., 2002; Moreira, 2003; R. Bos, personal communication). In these regions, the disease fraction attributable to the environment was estimated to be 27% (11–40%). In Central and South America, the vectors breed mainly in natural environments (e.g. forests), but increasingly transmission to humans occurs in and around houses (Campbell-Lendrum et al., 2001; Yadon et al., 2003). Interventions can be effective, such as those that improve housing. The global environmental attributable fraction for this disease was estimated to be 12% (1–30%).

DENGUE

Dengue and dengue haemorrhagic fever could be almost entirely prevented by good management of water bodies in and around houses, which are breeding sites for the main mosquito vector, *Aedes aegypti*. This species commonly breeds in temporary water-storage containers in the domestic (and sometimes the natural) environment, such as tanks and drums, plant pots, and also in standing water in solid waste, including tyres and discarded food containers. *Aedes albopictus* is an important secondary vector in some areas of the Western Pacific and South-East Asia Regions, while *Aedes polynesiensis*, which breeds in crab holes, transmits dengue on a number of Pacific islands. In such circumstances, the problem of dengue cannot be resolved simply by reducing or effectively managing *Aedes aegypti* breeding sites (Rozendaal, 1997; Heukelbach et al., 2001; R. Bos, personal communication). The global mean environmental attributable fraction for dengue was estimated to be 95% (90–99%).

JAPANESE ENCEPHALITIS

Vectors involved in the transmission of Japanese encephalitis include *Culex tritaeniorhynchus* and species belonging to the *C. gelindus* complex. This vector-borne disease could be efficiently prevented by environmental management, largely by managing irrigation areas (mainly rice fields) and their access to farm animals, pigs in particular (Rozendaal, 1997; Keiser et al., 2005; Bos, personal communication). The disease is therefore almost completely associated with the environment, with an estimated attributable fraction of 95% (90–99%).

Dengue and dengue haemorrhagic fevers could be almost entirely prevented by good management of water containers in and around houses.



Standing water in a Latin American town provides a potential breeding ground for the mosquito vectors of diseases associated with poor solid waste management.

Credit: WHO/TDR/Mark Edwards

Commercial sex workers and migrant workers are at increased risk of acquiring or transmitting the human immunodeficiency virus (HIV).



Prostitutes in Brazil have formed an association to educate themselves about AIDS.

Credit: Mark Edwards/Still Pictures

HIV/AIDS

Certain occupational groups are at increased risk of acquiring or transmitting the human immunodeficiency virus (HIV), which causes acquired immunodeficiency syndrome (AIDS). These include commercial sex workers, health-care workers who may be infected by sharps injuries or other exposures, and workers who spend part of the year away from their families (referred to as workers at "intermediate risk"). Workers at intermediate risk mainly include the uniformed workforce (e.g. policemen, the military), miners and truck drivers. Because they live away from their families part of the year, they are more likely to have sex with sex workers, and thus be at increased risk (Evian et al., 2004; UNAIDS/WHO, 2004; US Census Bureau and UNAIDS, 2004). Commercial sex workers may be at high risk, however they comprise a relatively small percentage of the general population. For example, commercial sex workers typically represent 0.4–1% of the general population in most regions, and 1–4% in sub-Saharan Africa and the industrializing part of the Western Pacific Region. The intermediate risk group is larger, generally 3% or more of the total population, but their risk is lower than that of commercial sex workers.

The fraction of HIV/AIDS attributable to occupation can be roughly estimated by comparing the adult prevalence rate in the general population with that in commercial sex workers (UNAIDS/WHO, 2004), or workers at intermediate risk (after accounting for competing risks, such as intravenous drug use). In adults, for example, the prevalence of HIV in commercial sex workers may be 2–20 times higher than in the general population, depending on the region. Only about 0.02% of the global HIV/AIDS burden is estimated to be caused by percutaneous injuries to health-care workers (Prüss-Üstün, Rapiti and Hutin, 2003).

The occupational-related attributable fraction for HIV/AIDS in adults was estimated to be 4–8% (2–13%) in most regions; it was lower in the developed part of the Western Pacific Region, at 2–3% (1–5%). In regions such as sub-Saharan Africa and the South-East Asia Region – which have higher rates of sex workers or very high rates of HIV in commercial sex workers as compared to the general population – the attributable fraction was estimated to be as high as 9–15% (4–20%). Globally, occupational causes accounted for about 9% (5–14%) of HIV transmissions, causing 250,000 deaths annually. This estimate only covers HIV transmission to workers, but infected workers may in turn infect members of the general population. In certain countries, the HIV epidemic may even be largely driven by commercial sex activities. The impact of prevention that is targeted to certain occupational groups may therefore be more far reaching than simply improving workers' health (the parameter used in this study to simplify quantification).

SEXUALLY TRANSMITTED DISEASES

The sexually transmitted diseases (STDs) considered in this section include only the main bacterial infections, syphilis, chlamydia and gonorrhoea. Viral infections are not covered, although human papilloma virus (HPV) infections are partly captured in the section on cancers, as they are linked to cervical and uterine cancer. Hepatitis B, hepatitis C and HIV/AIDS are covered in separate sections.

Workers in several occupations are at increased risk of infection. Sex workers, in particular, are at high risk for STDs compared with the general population (Riedner et al., 2003; Sugihantono et al., 2003; Xueref et al., 2003; Nessa et al., 2005), because disease can be transmitted during occupational activity. Another group of workers, including mainly the uniformed workforce and migrant workers (mainly truck drivers, policemen, military, sailors, miners, certain construction workers), are also at increased risk of infection because their work takes them away from home for extended periods and they are more likely to seek partners, particularly among commercial sex workers (Anonymous, 1994; Gawande et al., 2000). Although the transmission rates of STDs to sex workers and other workers at increased risk may be significant within the occupational group, such worker categories represent a relatively small fraction of the general population (typically 0.4–4% for sex workers, depending on the country), and only about 3% or more of all workers are at increased risk. The total attributable fraction for the occupational disease burden of the main bacterial STDs (syphilis, chlamydia and gonorrhoea) was estimated to be 7–9% (4–12%) for most regions, and about 20% (15–25%) for regions with high rates of sex workers, such as sub-Saharan Africa and the industrializing part of the Western Pacific Region. The global average for the attributable fraction of occupational STDs was about 17% (15–19%).

This estimate covers the transmission to workers, rather than infections to the general population by infected workers. Transmission to the general population from workers is potentially a major consequence of occupational transmission, and in some countries may even fuel the ongoing epidemic, but it is not considered here.

HEPATITIS B AND HEPATITIS C

Hepatitis B and hepatitis C have an occupational component, as certain occupational groups are at increased risk of infection with the hepatitis B virus (HBV) or hepatitis C virus (HCV) at work, or because of their working and living conditions. Many of the occupational groups at risk are the same as those at risk for occupational HIV infection and STDs. The groups include commercial sex workers, workers exposed to percutaneous injuries with contaminated sharp objects (e.g. nurses, doctors), and workers at

Each year, 16 400 hepatitis C and 65 600 hepatitis B infections occur in health workers as a result of injuries by contaminated sharp objects.



Intensive care nurse prepares for a procedure in a provincial hospital in Viet Nam.

Credit: Susan Wilburn

Crowding, and certain home or workplace exposures to air pollutants, are environmental factors that increase the burden of disease from tuberculosis.



A chest exam for tuberculosis.

Credit: WHO/TBP/Davenport

intermediate risk (e.g. migrant workers, members of the uniformed workforce, miners, truck drivers and sailors) who spend time away from home and are more likely to seek out the services of sex workers.

Although HBV is highly sexually transmissible, it is not always possible to dissociate this route of transmission from other means, as studies of commercial sex workers have shown. Nevertheless, hepatitis B is generally more prevalent in commercial sex workers than in the general population, for countries in which HBV prevalence is low or intermediate (Ishi et al., 2001; Camejo, Mata and Diaz, 2003; Mak et al., 2003; P. Van Damme, personal communication). Sexual transmission of HCV between monogamous partners is likely to be low compared with other causes (Neumayr et al., 1999; Vandelli et al., 2004), but may account for as much as 10–20% of all HCV infections (Alter, 1997). Occupational transmission of HCV to sex workers has a low public-health priority and was not considered here.

It was estimated that the attributable fractions for occupational HBV and HCV infections in health-care workers are 0.3% of the global hepatitis C burden, corresponding to 16 400 HCV infections per year, and 0.3% of the global hepatitis B burden, corresponding to 65 600 HBV infections per year (Prüss-Üstün, Rapiti and Hutin, 2003). The total attributable fraction for occupational HBV infections in adults was estimated to be 3% (1–4%) in regions with low or intermediate hepatitis B prevalence (i.e. most regions, besides sub-Saharan Africa, China, parts of south-east Asia and selected countries). In high-prevalence regions, only infections of health-care workers from sharps injuries were considered.

TUBERCULOSIS

The risk of infection by *Mycobacterium tuberculosis* and progression to disease depends not only on the human host, but on a range of environmental factors. For instance, crowding in households or other settings, may favour casual transmission of the causal pathogen, increasing the likelihood of prolonged close contact between susceptible people and infectious tuberculosis cases (Antunes and Waldman, 2001; Lienhardt, 2001; Clark, Riben and Nowgesic, 2002). Malnutrition increases the risk of progression to tuberculosis, and worsens the prognosis, because it compromises the immune system (Byrd, Mehta and Roy, 2002; Zachariah et al., 2002). Exposure to indoor smoke from solid fuels (Mishra, Retherford and Smith, 1999; Desai, Mehta and Smith, 2004) and environmental tobacco smoke both have been associated with increased tuberculosis rates, but how they are associated is not well understood.

Certain occupational groups are at increased risk of tuberculosis, particularly miners exposed to airborne particles such as silica or coal dust (Trapido et al., 1998; Williams et al., 1998; Corbett et al., 1999, 2000; Davies et al., 2001; Rom and Garay, 2003), and workers handling asbestos (Segarra-Obiol, Lopez-Ibanez and Perez Nicolas, 1983). Health-care workers who come into contact with tuberculosis patients are at increased risk of infection (Takeda, Robazzi and Lavrador, 2001; Anonymous, 2004; Jelip et al., 2004). In some settings, such as hospitals and prisons, tuberculosis rates are particularly high (Braun et al., 1987; Eyob et al., 2002).

For most of the world, it was estimated that about 19% (6–41%) of the total tuberculosis burden was attributable to the environment, although in areas where the HIV epidemic had a large impact on tuberculosis incidence it was likely that environmental factors had a smaller effect. In parts of Africa that are strongly affected by HIV/AIDS, for example, the attributable fraction for tuberculosis associated with the environment was estimated to be only 14% (6–24%). Although tuberculosis may have a strong environmental component, this does not mean that the best way to control the epidemic is through environmental management. It is clear, however, that managing environmental risk factors could significantly reduce the disease burden of tuberculosis.

PERINATAL CONDITIONS

For the purpose of this study, perinatal conditions principally include low birth weight, prematurity, birth asphyxia and birth trauma. This definition is relatively narrow as it excludes stillbirths, malformations and other conditions affecting liveborn infants that may be affected by environmental factors.

Higher rates of low-birth-weight infants were observed for mothers exposed to the environmental risks of air pollution, tobacco smoke or various chemicals (Ritz and Yu, 1999; Seidler et al., 1999; Chen and Omaye, 2001; Boy, Bruce and Delgado, 2002; Desai, Mehta and Smith, 2004; Maisonet et al., 2004). In Italy, for example, it was estimated that environmental tobacco smoke alone accounted for 7.9% of all low birth weights (Forastiere et al., 2002). In developing countries, exposures to environmental hazards such as: unsafe water and inadequate sanitation; unsafe nutrition (itself related to poor water and sanitation); or maternal exposure to pesticides or other chemicals, constitute important risks to infant health, increasing the mortality rate for low-birth-weight and preterm infants (Zhang, Cai and Lee, 1992; Taha and Gray, 1993; Longnecker et al., 2001). Birth asphyxia and trauma could be caused by a low maternal Body Mass Index, however the contribution of these risk factors to the overall infant mortality rate is probably low.

Infants of mothers exposed to air pollution, environmental tobacco smoke and other chemicals had higher rates of low birth weight.



Active and passive smoking during coffee break in an office in Europe.

Credit: Mike Schroeder/Still Pictures

Malnutrition probably plays a role in more than half of all child deaths; 50% of malnutrition's disease burden is attributable to the environment.



Measuring head circumference as part of a survey into children's health and nutrition in India.

Credit: Mark Edwards/Still Pictures

It was estimated that environmental causes accounted for 6% (2–10%) of all adverse perinatal conditions in developed countries, and for 11% (3–25%) in developing countries (where exposures to environmental risks were estimated to be higher). It should be noted that the relationship between environmental exposures and perinatal conditions is relatively poorly documented, particularly in developing countries.

CONGENITAL ANOMALIES

Congenital anomalies include conditions such as abdominal wall effects, anencephalies, anorectal atresia, cleft lip or palate, oesophageal atresia, heart anomalies, spina bifida or Down's syndrome. Some have been linked to environmental or workplace exposures of pregnant women to chemicals or radioactivity, and to ambient air pollution (Reznik et al., 1992; Czeizel et al., 1993; Nurminen, 1995; Ritz et al., 2002). It was estimated that 5% (2–10%) of all congenital anomalies were attributable to environmental causes.

MALNUTRITION

Malnutrition has been used to refer both to overnutrition and undernutrition, but in this analysis we use the term exclusively to refer to undernutrition, measured as poor anthropometric status. Individual nutritional status depends on the food that an individual eats, his or her general health, and the physical environment. In all three aspects, poor water and sanitation play an important role in malnutrition (WHO, 2005a), and several infectious diseases associated with malnutrition, including diarrhoea and other diseases caused by intestinal parasites, are related to poor water, sanitation, hygiene and food safety (Martorell, Mendoza and Castillo, 1988; WHO, 1995; Prüss-Üstün et al., 2004a). It has also been shown that the levels of water and sanitation services significantly affect Z-scores and weight gain in infants (Esrey, Habicht and Casella, 1992; Esrey, 1996; Checkley et al., 2004).

Malnutrition is also related to feeding habits (Motarjemi et al., 1993), while the influence of a genetic component on nutritional status may only be small (Habicht et al., 1974; WHO, 2000; Bhandari et al., 2004). Land degradation and soil pollution, as well as climate change, can also contribute to malnutrition to a certain extent. It was estimated that climate change accounted for 2% of the health burden of malnutrition (WHO, 2002). Overall, 50% (39–61%) of the health burden of malnutrition was estimated to be attributable to the environment, and in particular to poor water, sanitation and hygiene. Malnutrition causes vulnerability and increases the risk of adverse health outcomes, particularly in children.

Malnourished children tend to have more frequent episodes of severe diarrhoea and are more susceptible to infectious diseases, such as respiratory infections and meningitis. Malnourished children have a poorer prognosis for almost all infectious diseases (except HIV), and malnutrition is thought to play a role in more than half of all child deaths worldwide (Pelletier, 1994; Schroeder and Brown, 1994), which makes malnutrition one of the most important risk factors for children globally (Fishman et al., 2004). In this analysis, we included the infectious disease burden of malnutrition in children that is associated with the environment.

CANCERS

Malignant neoplasms at several sites of the body have been associated with exposures to occupational and environmental risk factors. Although cancers from environmental causes cannot be distinguished from cancers from other causes, as for many other diseases, the contributions of environmental causes have been highlighted by analysing differences in cancer incidences by geography and over time, and by studying cancer rates in migrant populations (IARC, 1990). The effects of occupational carcinogens have been particularly well documented, with 28 agents considered to be definite, 27 agents probable, and 113 agents possible occupational carcinogens (Siemiatycki et al., 2004).

Lung cancer causes the largest disease burden of all cancers globally, or about 15% of the burden of all cancers. By far the largest risk factor for lung cancer is smoking, at 66% (WHO, 2002). About 9% of the disease burden of lung cancer has been attributed to occupation (WHO, 2002; Concha-Barrientos et al., 2004), about 5% to outdoor air pollution (WHO, 2002; Cohen et al., 2004) and 1% to exposure to indoor smoke from solid fuels (Smith, Corvalán and Maeusezahl-Feuz, 2004). Other exposures are also likely to pose a risk. These include exposure to environmental tobacco smoke (Taylor et al., 2001; IARC, 2004); radon (Lubin and Boice, 1997; Committee on Health Risks of Exposure to Radon, 1999; Pavia et al., 2003); and occupational exposure to ionizing radiation (IARC, 1992), asbestos, and other chemicals (e.g. chromium, nickel, cadmium).

Smoking may have an additive or multiplicative effect with some environmental exposures (Williams and Sandler, 2001). In Finland, for example, work-related factors accounted for 24% of lung cancer mortality in adults, but for only 8% of all cancer fatalities in adults (Nurminen and Karjalainen, 2001). It was estimated that environmental factors account for 31% of the global disease burden of lung cancer and 30% (6–55%) of the disease burden in developed countries, for both men and women. In developing countries, the attributable environmental fractions were 33% (6–65%) for men, and 25% (6–37%) for women.

Smoking is the largest risk factor for lung cancer, but environmental causes also account for an estimated 31% of global lung cancer burden.



A schoolgirl with a face mask for protection from smokestack pollution emissions of factories in her neighbourhood in the eastern Mediterranean region.

Credit: Munir NASA/UNEP/Still Pictures

Melanomas are linked to excessive UV exposure, yet we continue to deplete the ozone layer and fail to use personal protection.



Encouraging children in Australia to wear hats on the beach as part of the national SLIP SLAP SLOP campaign to increase awareness about health risks arising from excessive exposure to the sun's rays.

Credit: Mark Edwards/Still Pictures

The second most important neoplasm in terms of disease burden is stomach cancer, particularly in developing countries. Stomach cancer is associated with *Helicobacter pylori* infection, which is relatively common in developing countries, and transmission may be facilitated by poor sanitation and crowding. Other neoplasms, such as leukaemia, have been associated with chemical agents. For example, 2% of the leukaemia disease burden was attributed to occupational exposures to chemicals whose carcinogenic properties have been clearly established, such as benzene and ethylene oxide (WHO, 2002; Concha-Barrientos et al., 2004). There is also good evidence linking melanomas to excessive UV exposure – yet we continue to deplete the ozone layer, fail to use personal protection, and indulge in other risky behaviour leading to excessive UV exposure.

Other risk factors for cancer include aflatoxins in food (liver cancer), asbestos in drinking water (several cancers, including skin cancer), and human papilloma viruses (cervical cancer, can be occupationally transmitted to sex workers). Also, HIV-related Kaposi's sarcoma can be occupationally transmitted and is associated with lymphoma. Other environmental or occupational exposures have been associated with various neoplasms, but the quantitative evidence could not be established. In developed countries, it was estimated that 16% (10–34%) of cancers in men (other than lung cancers), and 13% (10–23%) in women, were attributable to the environment. In developing countries, the corresponding attributable fractions were 18% (10–45%) in men and 16% (10–35%) in women. The uncertainty surrounding these estimates is due to the fact that evidence linking specific environmental and occupational exposures to various cancers was incomplete. Globally, about 19% (12–29%) of all cancers were estimated to be attributable to the environment, resulting in 1.3 million deaths each year.

NEUROPSYCHIATRIC DISORDERS

This large group of diseases includes disorders such as Alzheimer and other dementias, bipolar affective disorders, Parkinson disease, schizophrenia, epilepsy, alcohol use and drug use disorder, multiple sclerosis, insomnia, migraine, panic disorder, post-traumatic stress disorder, and lead-induced mild mental retardation. Of all the neuropsychiatric disorders, unipolar depressive disorder causes the largest disease burden. Many of these conditions have a small-to-moderate link to the environment or occupation. Depression has been linked to occupational stress (Tennant, 2001), insomnia to noise exposure (Passchier-Vermeer and Passchier, 2000; Franssen and Kwekkeboom, 2003) and, more recently, conditions such as Parkinson disease have been linked to exposure to chemicals (Huang, de la Fuente-Fernandez and Stroessl, 2003; Tan et al., 2004).

Parkinson disease also could be linked to occupational head trauma, as could epilepsy. Drug use and alcohol disorder have been linked to the occupational environment, such as coca growing, or working in the entertainment or alcohol industry (Wilhelm et al., 2004). Post-traumatic stress disorders have been linked to disasters such as floods, earthquakes and fires, which could be partly prevented by environmental measures. Dams and land-use patterns could be used to control flooding, for example, and materials could be used to build sturdier houses that could better weather the effects of fire or earthquake. Insomnia has an environmental and occupational component, mainly through exposure to noise, or occupational stress.

The loss of IQ points caused by exposure to lead in early childhood can lead to mild mental retardation, and this environmental contribution to the disease burden is captured in the neuropsychiatric group of disorders. It was estimated that about 800 000 children were affected by exposure to lead each year (WHO, 2002; Prüss-Ustün et al., 2004). Other disorders may be associated with population density in urban settings and poor quality of the local environment. In Finland, for example, it was estimated that 4% of mental disorders and 3% of nervous system diseases were linked to occupation (Nurminen and Karjalainen, 2001). Overall, the environmental contribution to the disease burden of neuropsychiatric disorders was relatively modest, and the attributable fraction was estimated to be only 13% (10–16%). The neuropsychiatric diseases with the largest environmental components included insomnia, migraine, post-traumatic stress disorder, epilepsy (in developing countries), and alcohol use disorder, with attributable fractions ranging between 10% and 20%. Those for depression, epilepsy (in developed countries) and Parkinson disease ranged from 5% to 10%, while other neuropsychiatric diseases contributed less than 5% of the environmental burden.

CATARACTS

Cataracts have been associated with exposure to sunlight and environmental tobacco smoke (Hollows and Moran, 1981; Collman et al., 1988; Taylor et al., 1988; West, 1992; West et al., 1998; McCarthy, Nanjan and Taylor, 2000), as well as to smoke from solid household fuels (Mohan et al., 1989; Zodpey and Ughade, 1999; Desai, Mehta and Smith, 2004), and with dehydration from diarrhoea that is largely attributable to environmental causes (Minissian, Mehra and Jones, 1984; Minissian, Mehra and Verrey, 1989; Bhatnagar et al., 1991). However, more work is needed in these areas. Globally, about 5% of cortical cataracts have been associated with exposure to UV radiation (Lucas, 2004). In total, it was estimated that 7% (5–10%) of all cataracts are attributable to environmental risks.

In the year 2000, about 800 000 children were affected by lead exposure, leading to lower IQ and potential mild mental retardation.



A child directly exposed to tailpipe emissions from an automobile, which may heighten environmental exposure to lead in countries where leaded gasoline has not yet been phased out.

Credit: Harmut Schwarzbach/Still Pictures

Some 2.5 million people die every year from cardiovascular disease attributable to environmental factors, including work-related stress as well as chemical, air pollution, and environmental tobacco smoke exposures.



Processing fish in the USA. Workplace conditions, including the demands of assembly line work, can be a factor in stress.

Credit: Steven Kazlowski/Still Pictures

DEAFNESS

Hearing loss leading to deafness can be caused by occupational exposure to high levels of noise. The attributable fraction for the disease burden of occupational deafness was estimated to be 16% of the global average disease burden for all causes of deafness (WHO, 2002; Concha-Barrientos et al., 2004).

CARDIOVASCULAR DISEASES

Cardiovascular diseases have been associated with environmental risks such as air pollution, (Pope et al., 2002), risks in the workplace, exposure to chemicals such as lead (Schwartz, 1995) and exposure to environmental tobacco smoke (Kaur et al., 2004). Lead exposure, for example, can increase blood pressure, which in turn increases the risk of cardiovascular disease. Lead exposure was estimated to account for 2% of the ischaemic heart disease burden and 3% of the cerebrovascular disease burden (WHO, 2002; Prüss-Üstün et al., 2004).

Exposures to outdoor air pollution accounted for approximately 2% of the global cardiopulmonary disease burden (WHO, 2002; Cohen et al., 2004). Several other risk factors, such as low mineral content in drinking-water, are suspected of being associated with cardiovascular diseases, but evidence is still being developed and debated (WHO, 2006a).

Other environmental risks can be generated by stressful conditions in the workplace and ischaemic heart disease has been linked to stress at work (Bosma et al., 1988; WHO, 2002). Stressful workplace conditions include an imbalance in the effort-reward mix, long work hours, shiftwork, psychosocial stressors and physical exertion (Karasek et al., 1988; Johnson, Hall and Theorell, 1989; Belkic et al., 2004; Rosengren et al., 2004).

In Finland, it was estimated that occupational risks accounted for 17% of the deaths from ischaemic heart disease, and 11% of those from stroke (Nurminen and Karjalainen, 2001). In the USA, about 12% of the ischaemic heart disease burden was related to occupation, for the age group 20–69 years. This estimate was based on the specific risks of job control, noise, shift work and environmental tobacco smoke at work (Steenland et al., 2003). In Denmark, it has been estimated that about 16% of the cardiovascular disease burden could be prevented in men with non-sedentary occupations, and 22% in women with non-sedentary occupations. These figures increased to 51% and 55%, respectively, if men and women with sedentary work were included in the analysis (Olsen and Kristensen, 1991). In total, 16% (7–23%) of the total burden of cardiovascular disease was attributed to the environment, corresponding to 2.5 million deaths per year.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a slowly progressing disease characterized by a gradual loss of lung function. In terms of total disease burden, the most important risk factor is active smoking, estimated to contribute to 36% of the global disease burden of COPD (WHO, 2002). Most other risk factors are occupational or environmental, including dusts and chemicals in the workplace, air pollution, and environmental (second hand) tobacco smoke (National Heart, Lung and Blood Institute, 2005). Occupational exposures to airborne particulates, for example, were responsible for 12% of the global COPD disease burden (WHO, 2002; Concha-Barrientos et al., 2004), and exposures to indoor smoke from solid fuels accounted for a further 22% (WHO, 2002; Smith, Corvalán and Maeusezahl-Feuz, 2004). Outdoor air pollution accounted for 3% of cardiopulmonary mortality (Cohen et al., 2004).

Globally, it was estimated that 42% (37–47%) of the COPD disease burden could be attributed to the environment. This estimate was derived by combining CRA estimates (WHO, 2002), which did not include environmental risks such as environmental tobacco smoke, with those of the experts, which did account for the additional environmental risks (Baldacci and Viegi, 2002; Gauderman et al., 2004; Vineis et al., 2005). The CRA estimates (which covered 90% of the risks included in the expert survey results), were adjusted to cover additional environmental risks such as environmental tobacco smoke (second-hand smoke), using the expert opinion survey results, to give a final distribution for the estimated total disease burden from COPD.

The attributable fractions for COPD risk factors vary significantly between countries and by gender, a result of differences in the main risk factors to which people are exposed. In countries where solid fuel is widely used in homes for cooking or heating, indoor smoke levels can be high, and mean attributable fractions often exceeded 40%, with higher values for women than for men. In more developed regions, with little reliance on burning solid fuel in the home, mean attributable fractions were between 10% and 30%, with higher values for men because of occupational exposures to smoke.

ASTHMA

Asthma development and exacerbation can be triggered by a variety of indoor and outdoor environmental exposures. Indoor exposures to dampness, dust mites and fungal allergens may account for 20% of asthma prevalence (Melse and de Hollander, 2001). Indoor smoke from solid fuels (Mohamed et al., 1995; Xu, Niu and Christian, 1996; Desai et al., 2004) and environmental tobacco smoke (Etzel, 2003; Tatum and Shapiro, 2005) are also significant triggers for asthma symptoms and attacks.

An estimated 42% of COPD is attributable to environmental risks; occupational exposures to airborne particulates were responsible for 12% of this global COPD disease burden.



Protecting worker health from environmental dust at a cement plant in Africa.

Credit: Jorden Schytte/Still Pictures

Asthma can be triggered or exacerbated by indoor or outdoor exposures to dampness, dust mites, fungal allergens, environmental tobacco smoke and air pollution.



Child using inhaler during an asthma attack (UK).

Credit: Mike Jackson/Still Pictures

Outdoor environmental exposures, such as to poor air quality (e.g. smog), are also known to exacerbate asthma (Koenig, 1999; Etzel, 2003). Occupational exposures alone accounted for about 11% of the total disease burden from asthma (WHO, 2002; Concha-Barrientos et al., 2004). Total environmental exposures were estimated to account for 44% (26–53%). The estimate for environment exposures did not include outdoor exposure to pollen, as this is not realistically modifiable.

MUSCULOSKELETAL DISEASES

Musculoskeletal diseases included in this study were the main categories of rheumatoid arthritis, osteoarthritis, low back pain, gout, and a group of "other musculoskeletal diseases". Low back pain is associated with exposure to ergonomic stressors at work, and it has been estimated that occupational exposures accounted for 37% of the global burden of disease from low back pain (WHO, 2002; Concha-Barrientos, et al., 2004). The mean attributable fraction was generally higher for men than for women (41% versus 32%), because men were more frequently engaged in occupations that exposed them to risk.

Rheumatoid arthritis and osteoarthritis have both been linked to occupational risks, such as exposure to vibrations, repetitive trauma, knee bending or lifting heavy weights. The incidences of these diseases are higher in occupational groups such as farmers, truck drivers and unskilled workers (Maetzel et al., 1997; Lievense et al., 2001; Khuder, Peshimam and Agraharam, 2002; Kirkhorn, Greenlee and Reeser, 2003; Rossignol et al., 2003; Olsson et al., 2004; Yoshimura et al., 2004). It was estimated that environmental factors account for 17% (7–29%) of the disease burden from rheumatoid arthritis and 20% (13–26%) of that from osteoarthritis. The group of "other musculoskeletal diseases" includes other forms of arthritis, arthropaties, joint disorders, systemic connective tissue disorders, muscle and soft tissue disorders. Evidence indicates that these diseases are also linked to occupational conditions, and it was estimated that 15% (7–23%) of the disease burden for this group of musculoskeletal diseases was attributable to occupational risk factors.

ROAD TRAFFIC INJURIES

The frequency of road traffic injuries can be influenced by environmental conditions related to land use policies and practices; inappropriate road design (road environment); urban structure and density (layout and hierarchy of road systems and residential areas); and poor matches of road design and vehicles. Other environmental issues include poor street lighting and signs, poor road maintenance and narrow roads (Qin et al., 2004). Traffic calming measures, such as one-way streets, road narrowing, speed

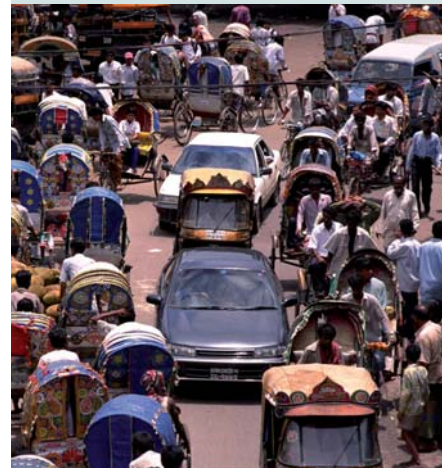
limits, street closures and speed bumps, may be effective in reducing injury (Forjuoh and Li, 1996; Elvik, 2001; Bunn et al., 2003; Mohan, 2004). Other successful measures include designating segregated bicycle lanes on urban roads, introducing barriers along the roadside, and pedestrian crossings (Forjuoh and Li, 1996; Peden et al., 2004; Racioppi et al., 2004).

The CRA study estimated that occupational factors contributed 6% of the global disease burden of road traffic injuries (WHO, 2002; Concha-Barrientos et al., 2004). It is relatively difficult to assess the contribution of environmental factors using intervention studies, because most such studies must be implemented within an existing built environment, with only minor constructional modifications possible. Also, longer-term environmental changes, such as modifications to the urban geography, density or road layout, or changes in the use of motor vehicles, could not be measured. Despite these limitations, it was estimated that 25% (12–59%) of road traffic injuries in Western Europe were attributable to the environment, 17% (5–50%) in Australia, North America and Japan, and 42% (26–60%) in developing countries. The global average for road accidents attributable to environmental factors was 40% (25–57%).

UNINTENTIONAL POISONINGS

Unintentional poisonings analysed in this study were poisonings by chemicals or other noxious substances, including drugs, and toxic vapours or gases. Suicides and homicides, attempted or actual, as well as drug abuse, and other intentional poisonings were not included in this category. Food poisonings, or contact with venomous animals or plants, were analysed, but under a separate category ("other unintentional injuries"). Many unintentional poisonings could be avoided if toxic chemicals were handled and stored safely, and if users were educated about the dangers of products and medications (e.g. by providing them with clear information about using medications) (McGuigan, 1999). Nevertheless, some poisonings from accidental drug overuse or negligence still occur, even when chemical safety measures are implemented and adequate information/education provided. These poisonings were not considered to be related to occupation or environment. It was estimated that 68% (46–84%) of poisonings in adults were attributable to occupation or the environment, and 85% (60–98%) in children. The figure is greater for children because certain behavioural and developmental factors specific to this group also make them more vulnerable to environmental risks associated with poisonings. For adults and children combined, environmental risk factors accounted for an average of 71% (52–85%) of all unintentional poisonings.

Traffic calming measures and improved design for cyclists and pedestrians can help reduce road traffic injuries.



Motorized traffic weaves its way through crowds of pedestrians and three-wheelers in a busy Asian city.

Credit: WHO

Falls, risk of fire and other forms of unintentional injury could be reduced through improvements to housing, recreational and built environments, as well as workplace settings.



Construction worker in Argentina. Building site safety is an important factor in worker health in developed and developing countries.

Credit: Mark Edwards/Still Pictures

FALLS

The number of falls could be reduced by improving the housing environment (e.g. by installing window guards or grab rails, removing slippery surfaces, and replacing lighting), by limiting access to building sites, and by improving the safety of recreational environments (Forjuoh and Li, 1996; Cryer, 2001; Gillespie et al., 2003; Millward, Morgan and Kelly, 2003; WHO Regional Office for Europe, 2004). In developed countries, about 26% (16–47%) of all falls were attributed to the environment. Although there were few data for developing countries, the corresponding figure was estimated to be 31% (17–60%). Approximately 12% of all falls worldwide occurred at work (WHO, 2002; Concha-Barrientos, 2004).

FIRES

Risks for domestic fires include the types of materials used to build the house, and the types of home furnishings (e.g. tapestry, upholstery, furniture). In developing countries, building materials are often poorly regulated, and the use of unsafe stoves, open fires or kerosene candles in the house is not uncommon. Some interventions within the built environment, such as installing smoke alarms, were successful in reducing fire-related injuries (Cryer, 2001; Millward, Morgan and Kelly, 2003).

Globally, an estimated 7% (5–9%) of fire-related injuries had environmental causes, with workplace factors accounting for about 2% (WHO, 2002; Concha-Barrientos, 2004).

DROWNINGS

Drownings can be caused by environmental factors, such as risks in the recreational environment and in the built environment (e.g. unprotected wells or house cisterns) (Celis, 1991), by floods, or by non-environmental factors such as alcohol consumption (Giustini et al., 2003; WHO, 2003; Centers for Disease Control and Prevention, 2004). In low-income countries, transportation on waterways is also a hazardous undertaking, owing to a lack of safety measures and regulations, and may play a major role in drownings.

Many drownings could be prevented by known interventions. These include public education and awareness programmes, improving recreational environments in the vicinity of water bodies, enforcing regulations related to water bodies (e.g. to install physical barriers, or to maintain prevention and rescue services), and enforcing regulations for occupational safety (Norris and Wilson, 2003; WHO, 2003).

Although floods are natural events that are exacerbated by climate change (McMichael et al., 2004), many drownings related to flooding could be prevented by dams, appropriate land use patterns, or even longer-term actions to limit climate change. Some alcohol-related drownings could be avoided by implementing safety measures in recreational environments, and by targeted education. Drowning rates have decreased significantly in developed countries over the past decade, coinciding with a period in which interventions related to recreational environments and to education were emphasized. In Italy, for example, drownings were reduced by 75% (Giustini et al., 2003).

For developed countries, it was estimated that 54% (30–76%) of drownings were attributable to the environment or to occupation. In developing countries, where recreational safety, water transportation safety, and flood control were less developed, the corresponding figure was higher (74%; 48–92%). About 1% of all drownings occurred at work (WHO, 2002; Concha-Barrientos, 2004).

OTHER UNINTENTIONAL INJURIES

This category includes a range of injuries that occur in a variety of circumstances and settings, many of which relate to the environment. The injuries are mainly sustained from:

- contact with mechanical forces (including sports equipment and agricultural machinery);
- off-road transportation accidents;
- animal bites and contact with venomous plants;
- exposure to ionizing radiation or electric currents;
- suffocation;
- natural forces (e.g. floods, storms, periods of excessively hot or cold weather, earthquakes);
- contact with hot substances;
- complications from medical and surgical care.

About 18% of all injuries in this category were attributable to occupation (WHO, 2002; Concha-Barrientos et al., 2004). Another 0.4% were attributable to floods caused by climate change (WHO, 2002; McMichael et al., 2004).

Although floods and earthquakes are natural events, both were included in this analysis because their consequences could in part be ameliorated by environmental measures (e.g. dams, land use patterns, action to limit climate change, use of adequate building materials). In developed countries, it was estimated that 30% (20–40%) of all injuries in this category were attributable to the environment, and 45% (22–76%) in developing countries.

More than 50% of all drownings can be prevented by improving environmental safety and management.



Crowds at a beach in the United Kingdom.

Credit: David Woodfall/Still Pictures

SUICIDE

Environmental factors affecting access to pesticides or guns may help facilitate suicide. Suicides also may be associated with work-related stress and with stress related to the built or degraded environment (Boxer, Burnett and Swanson, 1995).

Methods commonly used in suicide include: ingesting pesticides or other chemicals, drowning, hanging, shooting (Lester and Murrell, 1980; Kellermann et al., 1992), and gassing with car exhaust, domestic gas (Kreitman, 1976), or charcoal fumes (Chung and Leung, 2001). Modifying the environment, by improving chemical safety, detoxifying domestic gas or limiting access to guns, may therefore significantly reduce suicide incidence (Farmer and Rohde, 1980; Brent et al., 1991; Bertolote, 1993; Bowles, 1995; Leenaars et al., 2000).

The methods of self-harm differed significantly between regions (Farmer and Rohde, 1980; Clarke and Lester, 1987; Gunnell and Eddleston, 2003). As a result, estimates of the fraction of the suicide burden that could be affected by modifying the environment also vary. For example, in rural China, Malaysia, Sri Lanka and Trinidad, the primary method of suicide was ingestion of pesticides (Gunnell and Eddleston, 2003). In certain parts of the USA, gunshot was the main method (Lester and Murrell, 1980; Kellermann et al., 1992), while in England and Wales hanging was a common method (Wilkinson and Gunnell, 2000).

Estimates of the fraction of suicide injuries that could be attributed to the environment were: 22% (7–43%) in the Europe and the Eastern Mediterranean regions, 24% (20–30%) in North America, 18% (15–20%) in Latin America, 36% (20–50%) in developing regions of Asia, 16% (10–30%) in developed areas of the Western Pacific Region, and 10% (5–15%) in the Africa Region (although this estimate was based on few data). Globally, an average of 30% (22–37%) of all suicides were attributable to the environment.

INTERPERSONAL VIOLENCE

Various environmental factors influence interpersonal violence; interventions in the physical environment could thus reduce levels of interpersonal violence. Examples include reducing access to dangerous items (e.g. type of bar glassware), or reducing access to firearms through safe storage (Hemenway and Miller, 2000; Slovak, 2002). Street lighting may reduce violence by increasing visibility and raising the perceived risk of performing a violent act (Welsh and Farrington, 2004). Certain urban design and land use patterns also may reduce tensions and crime (André and Platteau, 1998).

Environmental interventions such as improved street lighting can help reduce the level of interpersonal violence.



Young women walk along a well-lit street in Moscow.

Credit: Transit/Still Pictures

Exposure to certain substances such as lead, can affect neuropsychological development and cognitive functioning, which could also increase delinquent behaviour (Needleman et al., 1996; Nevin, 2000; Dietrich et al., 2001; Stretesky and Lynch, 2001, 2004; Needleman et al., 2002).

Certain dietary choices, including fish consumption, balanced intake of micronutrients, and a good nutritional status overall also have been associated with reduced rates of violent behaviour (Schoenthaler et al., 1997; Hibbeln, 2001; Gesch et al., 2002; Schrauzer, 2002; Liu et al., 2004; Gesch, 2005). Although the consumption of seafood and other nutrients may have environmental components (e.g. related to the availability of fish, or the lithium content of drinking-water), such consumption was not considered to be "environmental" in this study, as the links between nutritional choices, micronutrient intake and the environment are very difficult to assess.

In developed countries, environmental factors accounted for 16% (3–28%) of the injuries from interpersonal violence, and for about 19% (7–31%) in developing countries. The global average was also about 19% (7–31%), as the majority of injuries from violence occurred in developing countries.

PHYSICAL INACTIVITY

Physical inactivity is a risk factor for noncommunicable diseases including: ischaemic heart disease and stroke; cancers of the breast, colon and rectum; and diabetes mellitus. For these diseases, the attributable fraction for physical inactivity varied between 10% and 22% globally, depending on the disease (WHO, 2002; Bull et al., 2004). The prevalence of physical inactivity can be modulated by the environment, via factors that encourage physical activity (Brownson et al., 2001; Craig et al., 2002; De Bourdeaudhuij, Sallis and Saelens, 2003; Eyster et al., 2003; Huston et al., 2003; Ewing, 2005).

It was estimated that 17% (11–23%, depending on the region) of the global population was inactive, defined as "doing no or very little physical activity at work, home, for transport or in discretionary time". In addition, 41% (32–52%, depending on the region) of the global population had insufficient activity levels (Bull et al., 2004). Insufficient activity has been defined as: "doing some physical activity, but less than 150 minutes of moderate-intensity physical activity, or 60 minutes of vigorous-intensity physical activity, a week, accumulated across work, home, transport or discretionary domains." This means that more than half of the global population gets insufficient physical activity to protect them from related risks causing death, chronic morbidity and disability from a range of noncommunicable diseases. Inactivity levels could be reduced by designing environments that are more conducive to physical activity in the workplace, at home, in transport and in leisure time.

More than half of the global population gets insufficient physical activity to protect them from a range of non-communicable diseases, including heart disease; cancers of the breast, colon and rectum; and diabetes mellitus.



Cyclists at rush hour in Shanghai. Cycling, a very beneficial form of physical exercise, is still a popular means of daily travel in Asia, but there is increasing competition for road space from motor scooters and motor vehicles.

Credit: Julio Etchart/Still Pictures

Transport and urban design measures can reduce the level of physical inactivity, which is a risk factor for several diseases, including heart disease, stroke, cancer and diabetes.



Good public transport systems also can stimulate “active travel” by pedestrians and cyclists going to and from transit stations.

Credit: Topfoto/Image Works

Certain built environments (Transport Research Board, 2005), or related policy measures, may facilitate more active lifestyles, particularly by encouraging walking and cycling. Modifiable factors in the built environment include land use mix and densities, access to key destinations and facilities, transport infrastructure, and building design. Pedestrian-friendly and bicycle-friendly environments include side-walks, ample building setbacks, walking and cycling paths, parks, bus shelters, and streets that are easy to cross. The level of car use in a population is related to the built environment and also has been linked to physical inactivity and obesity (Frank, Andresen and Schmid, 2004). Therefore, measures that discourage reliance on a car also may encourage physical activity, and reduce physical inactivity.

Higher car-related taxes are one such measure that can discourage car use and promote physical activity. In Denmark and the UK, for example, traffic modification measures increased the number of cyclists by about 20% in urban areas (Mayor of London - Transport for London, 2004; Odense Municipality, 2004). In certain developing regions, the potential impact of environmental interventions on physical inactivity levels is likely to be low because rates of motor vehicle ownership are low and much routine travel is by foot or bicycle. Nevertheless, many developing regions are undergoing rapid urbanization and motorization. Thus, the impact of environmental factors on physical activity levels may be very dynamic.

It has been estimated that inactivity levels could be reduced by 31% (12–59%) for North America and developed areas of the Western Pacific region, 27% (12–58%) for the European region, 20% (8–38%) for China, 18% (11–34%) for the Latin America and the Caribbean region, and 13% (3–35%) in other developing regions. Globally, 19% (13–27%) of current inactivity levels could be prevented by environmental interventions.

OTHER DISEASES

Not all environmental risk factors and related diseases have been included in this analysis. Some diseases and disease groups were not significantly linked to the modifiable environment under the definition used. In other cases, the evidence was too incomplete to make a reasonably sound estimate of quantifiable health impacts. For example, population health impacts associated with environmental exposures to endocrine-disrupting substances were not deemed to be quantifiable at present. Injuries related to wartime conflicts also were not considered. Examples of other diseases or health conditions associated with environmental risks, but where quantification of those health impacts was not deemed to be feasible, include obesity, anaemia, and iodine deficiency.



The global estimate of the environmental disease burden is conservative, because the health impacts of many risk factors and related diseases still cannot be quantified.

GLOBAL RESULTS OF THE ANALYSIS

6



The global results of our analysis are given in detail in Annex 2, Tables A2.1–A2.4. The data are broken down by WHO sub-region, by disease or injury category, and by gender and age group (in some cases). The estimated attributable fractions for diseases and injuries considered are provided in Table A2.1, and indicative values for the attributable fractions are outlined by disease and environmental risk factor in Table A2.2. Tables A2.3 and A2.4 detail the environmental disease burden in deaths and DALYs, respectively. The data are broken down by disease or injury category, and by WHO subregion.

From an analysis of the results in Section 5, it was estimated that environmental risk factors contribute to 24% (21–27%) of the global burden of disease from all causes (in DALYs), and to 23% (21–25%) of all deaths. The environmental attributable fraction is slightly higher for men than for women (25%; 22–28%, versus 22%; 20–25%), partly because occupational risks were higher in men. It was greatest for WHO subregion AFR D³(31%; 28–36%), but was in the 25–30% range for subregions EMR D, SEAR D and AFR E (Figure 4). It was lowest in the industrialized subregions, at only 16% (15–18%). If the global mean value for the environmental disease burden (24%) appears relatively large compared with some subregional values, it is because the global mean value was strongly weighted by the high environmental attributable fractions for disease in the developing subregions, where most of the disease burden is found.

In children 0–4 years old, 36% (31–40%) of the overall disease burden is attributable to modifiable environmental risk factors, while that fraction is 34% among children 0–14 years of age. In terms of mortality, the environmental attributable fraction is 37% for children 0–4 years of age, and 36% for children 0–14 years. The big killers are diarrhoea, malaria and respiratory infections, which together contributed to 24% of all deaths in children under 15 years of age. Other important environmental risks to children include perinatal conditions, protein-energy malnutrition and unintentional injuries. The attributable fractions generated by this analysis are shown in Table A2.1 (Annex 2).

Indicative values for the corresponding environmental disease burdens are shown by risk factor in Annex 2 (Table A2.2). The corresponding environmental disease burdens, in deaths and DALYs, are shown by disease for each of the WHO subregions (Tables A2.3, A2.4 in Annex 2). The main diseases, and their contributions to the total environmental disease burden, are represented in Figures 8 and 9 for the global population and for children, respectively.

Environmental factors contribute to 23% of all deaths worldwide and 36% of all deaths among children 0–14 years old.

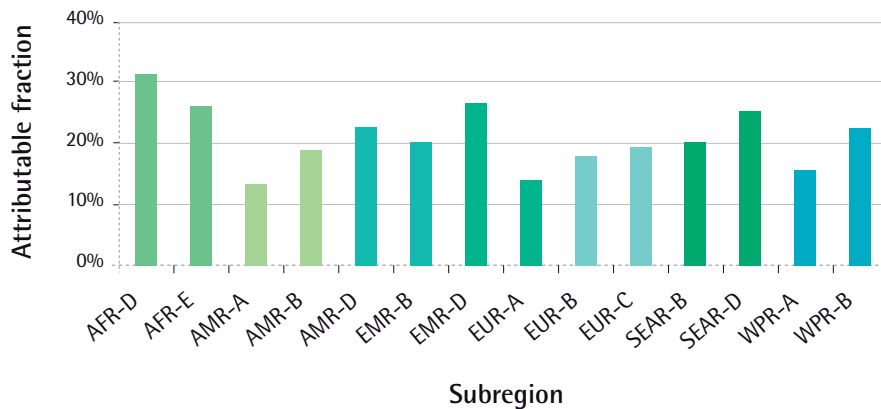


Taking a blood sample from an infant at a local health clinic (Brazil).

Credit: Mark Edwards/Still Pictures

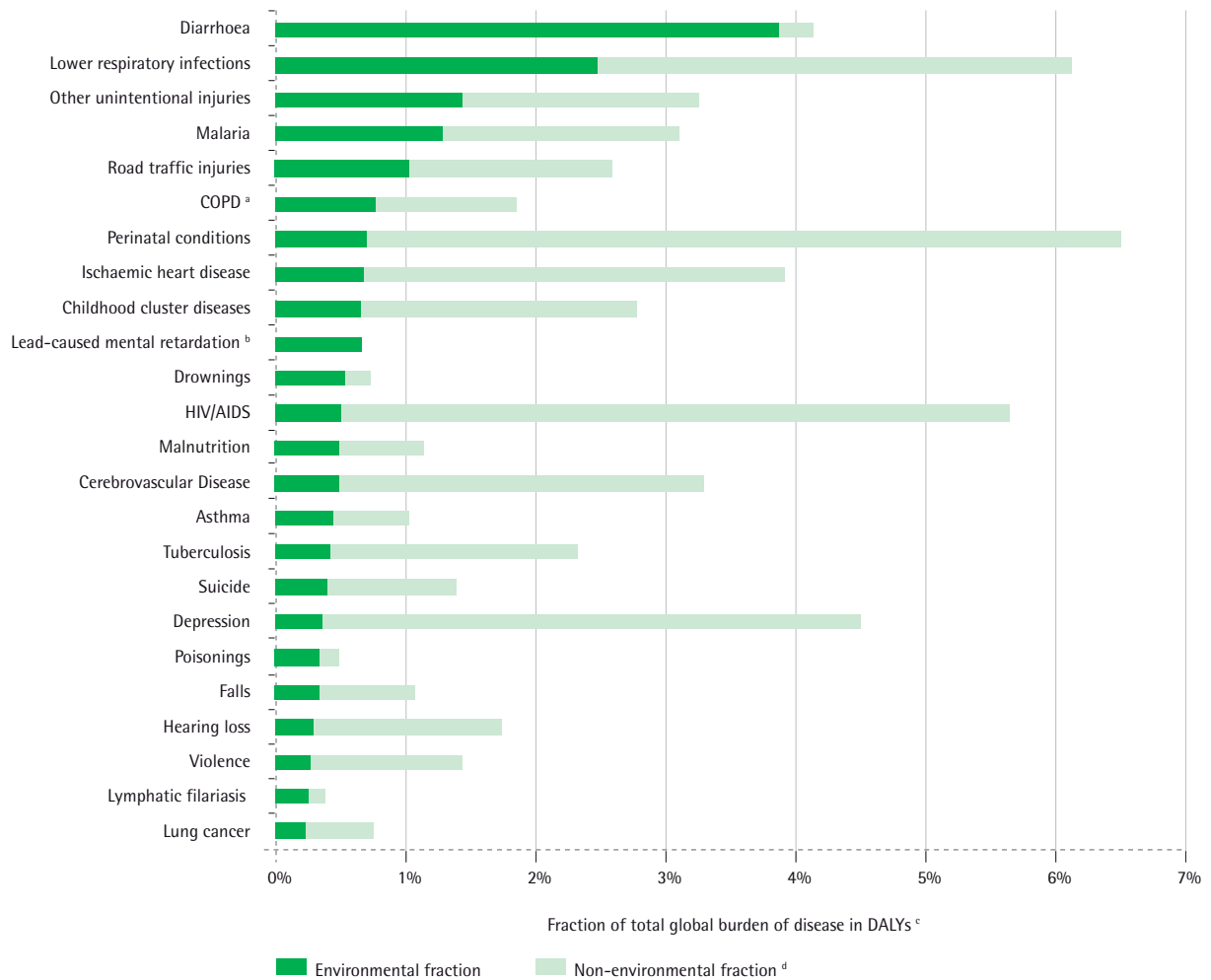
³ See Annex 1 for a list of the countries in each WHO subregion.

FIGURE 4 ENVIRONMENTAL DISEASE BURDEN, BY WHO SUBREGION ^a



^a The burden of disease is measured in DALYs. See Annex 1 for country groupings within WHO subregions.

FIGURE 5 DISEASES WITH THE LARGEST ENVIRONMENTAL CONTRIBUTION



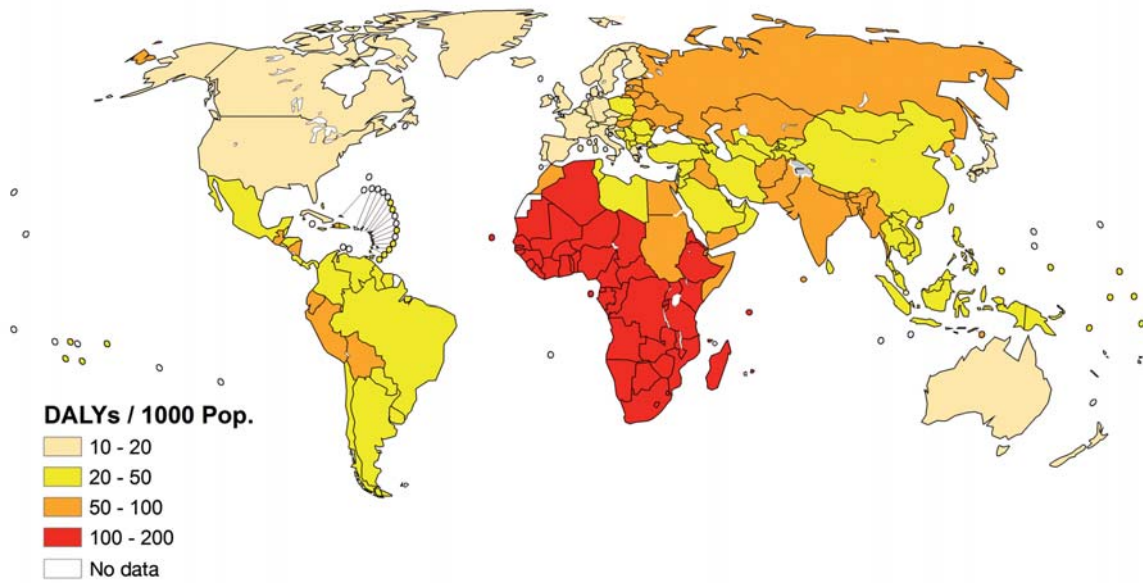
^a Abbreviations: COPD = Chronic obstructive pulmonary disease.

^b Lead-caused mental retardation is defined in the WHO list of diseases for 2002, accessed at: www.who.int/evidence.

^c DALYs represent a weighted measure of death, illness and disability.

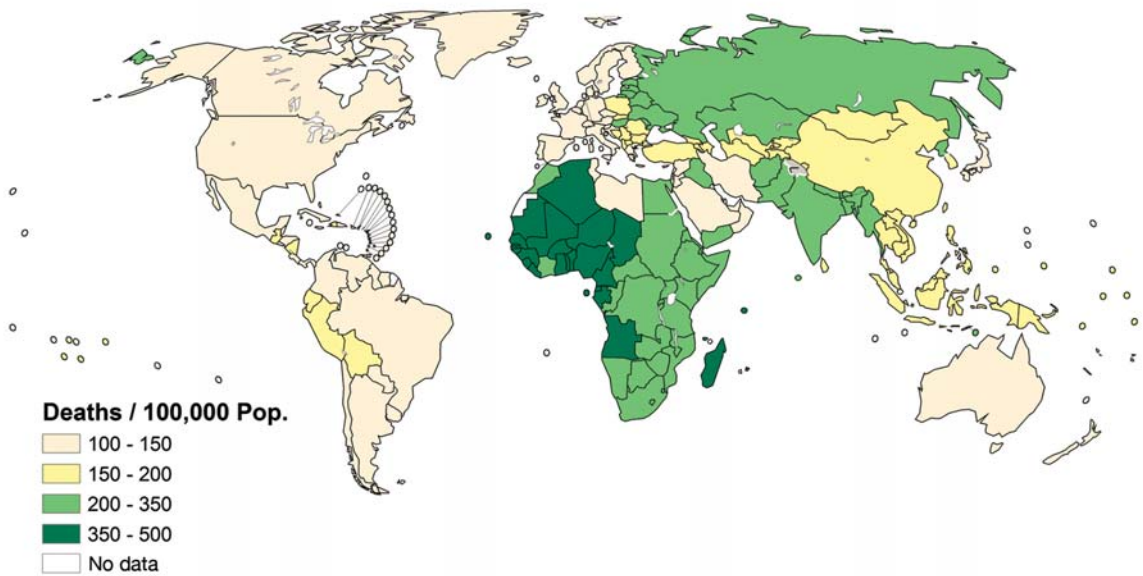
^d For each disease the fraction attributable to environmental risks is shown in dark green. Light green + dark green represents the total burden of disease.

FIGURE 6 ENVIRONMENTAL DISEASE BURDEN IN DALYS PER 1000 PEOPLE, BY WHO SUBREGION (2002)^a



^a See Annex 1 for a list of the countries in each WHO subregion.

FIGURE 7 ENVIRONMENTAL DISEASE BURDEN IN DEATHS PER 100 000 PEOPLE, BY WHO SUBREGION (2002)^a



^a See Annex 1 for a list of the countries in each WHO subregion.

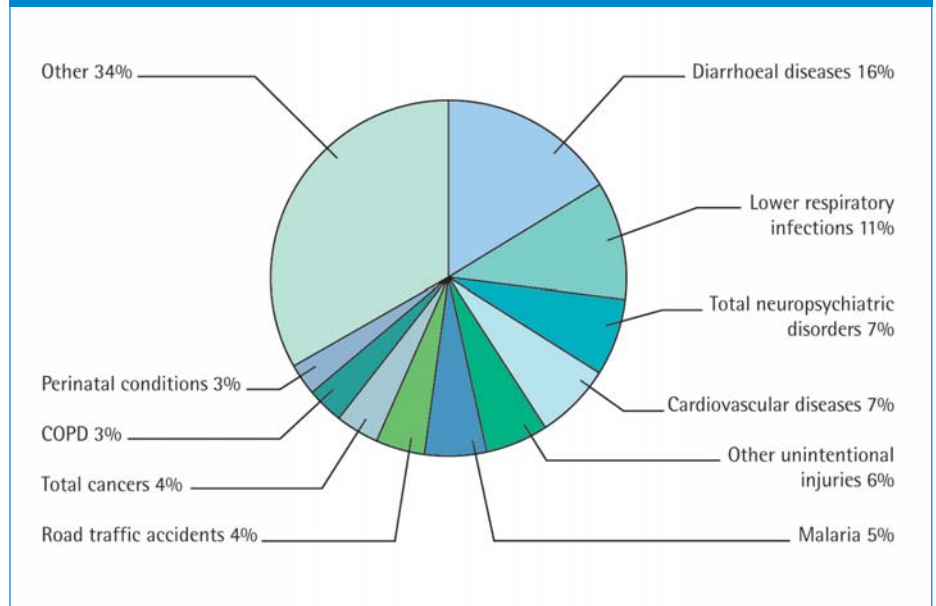
An estimated 24% of all deaths in children under 15 are due to environmentally-related diarrhoea, malaria and respiratory infections; these same three killers also represent the largest share of the childhood environmental disease burden.



Weighing a baby in Ghana as part of a project monitoring nutritional status.

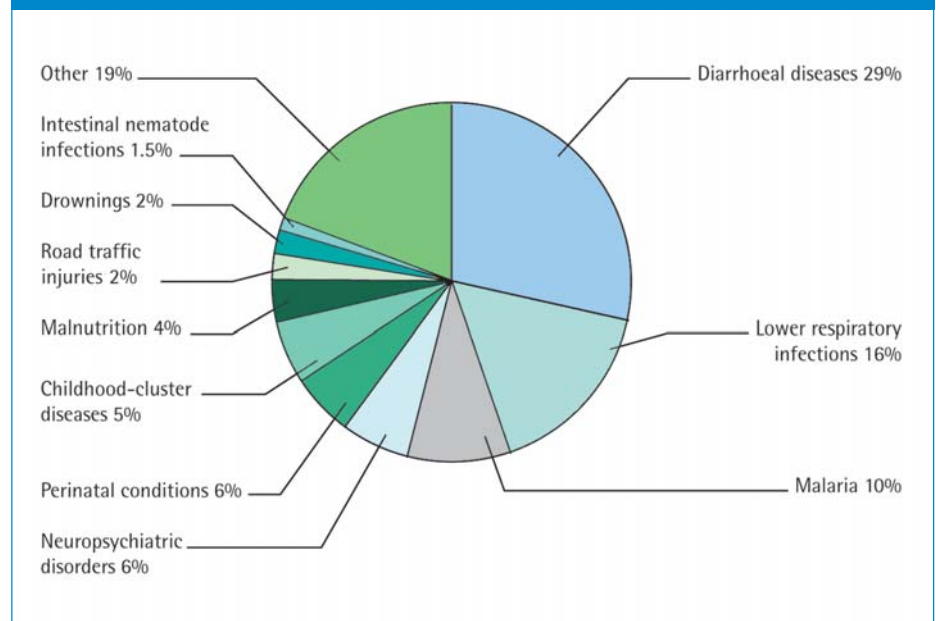
Credit: Martha Cooper/Peter Arnold, Inc.

FIGURE 8 MAIN DISEASES CONTRIBUTING TO THE ENVIRONMENTAL BURDEN OF DISEASE, FOR THE TOTAL POPULATION ^a



^a COPD = chronic obstructive pulmonary disease.

FIGURE 9 MAIN DISEASES CONTRIBUTING TO THE ENVIRONMENTAL BURDEN OF DISEASE, AMONG CHILDREN 0-14 YEARS ^a



^a The environmental disease burden is measured in disability-adjusted life years, a weighted measure of death, illness and disability (DALYs).





CONCLUSIONS 7

Health and environmental issues have been included in several high-level initiatives, including the United Nations Millennium Declaration, and regional interministerial conferences on health and the environment. Despite the visibility of these issues, the importance of environmental health interventions in preventing disease is not always fully appreciated. As the present analysis shows, modifying environmental risk factors for disease and injury could significantly reduce the disease burden of a country. Several conclusions emerge from this study, which are listed below under four main areas.

Nearly one quarter of the global disease burden is attributable to the modifiable environment

- Of the 102 major diseases reported in the World Health Report 2004 (WHO 2004a), 85 are partly caused by exposures to environmental risk factors. The environmental attributable fractions for the diseases varied widely, but, in total, environmental causes contributed to 24% of the number of years of healthy life lost to disease, and to 23% of the mortality associated with the diseases. Although this is a significant contribution to the overall disease burden, it is a conservative estimate because there is as yet no evidence for many diseases.
- For some diseases, the complexity of the causal pathways was sufficient reason to be excluded from this analysis, or for the attributable fractions not to reflect the true impacts. Although we made an attempt to capture many of these complex links, such as those diseases mediated by the environmental components of malnutrition and physical inactivity, some could not be included. Among them was the disease burden associated with changed, damaged or depleted ecosystems, which was not quantifiable, even though the associated health effects are readily apparent (WHO 2005b).

The environmental disease burden is not distributed evenly across the world, and some regions carry a disproportionately heavy burden for specific diseases.

- The differences arise from variations in exposures to environmental risks, and in access to health care.
- The largest differences were observed for infectious diseases, where total number of healthy life years lost per capita was 15-times higher in developing countries than in developed countries. The environmental burden of lower respiratory infections and diarrhoeal diseases was 120 and 150 times higher, respectively, in the most-impacted WHO subregions, as compared to those least impacted.

Developing regions carry a disproportionately heavy share of the environmental disease burden.



Public square in southeast Asia; inadequate access to, and management of, water and sanitation, impacts environmental health risks.

Credit: UNEP/Tarlok Chawala

On average, children in developing countries lost 8-times more healthy life years than their counterparts in developed countries from environmentally-related diseases. For some key diseases, the gap is far greater.



Children in Latin America stand by a dug out canoe full of freshly-caught fish; the poor in developing countries often are directly dependent on locally-available foods, and environmental degradation can threaten vital nutritional sources.

Credit: Sean Sprague/Still Pictures

- For noncommunicable diseases, there was no overall difference between developed and developing countries, but in countries of developed subregions per capita numbers of healthy life years lost from cardiovascular disease are up to 7-times higher than in countries of less developed subregions, and cancer rates are 4-times higher, when comparing best and least-performing regions.
- In developing countries, the per capita average number of healthy life years lost to injuries was roughly double that in developed countries, but differences were even larger at the subregional level. For road traffic accidents, there was a 15-fold difference between the best-performing and worst-performing subregions, and a 10-fold disparity for other unintentional injuries.
- The above results indicate that an important transition in environmental risk factors will occur as countries develop. For some diseases, such as malaria, the environmental disease burden is expected to decrease with development, but the burden will increase from other noncommunicable diseases, such as COPD, and approximate levels seen in the more developed regions of the world.

Children suffer a disproportionate share of the environmental health burden

- Globally, the per capita number of healthy life years lost to environmental risk factors was about 5-fold greater in children under five years of age than in the total population. The difference was even greater (7–10 -fold greater) for major diseases, such as upper and lower respiratory infections, diarrhoea, malaria and malnutrition. The differences would have been larger if the noncommunicable diseases rates for children had not been very low.
- On average, children in developing countries lost 8-times more healthy life years than their counterparts in developed countries, but for key diseases the differences were astounding. For childhood cluster diseases, the per capita rates in developing countries were over 70-times higher than in developed countries. At subregional level, the differential was greater than 25 for road traffic injuries, 140 for diarrhoeal diseases, and 800 for lower respiratory infections.
- Although these statistics are alarming, they do not capture the longer-term effects of exposures that occur at a young age, but do not manifest themselves as disease until years after the exposure.

Interventions can be cost-effective and have benefits that go well beyond health, and contribute to the overall well-being of communities.

- Many environmental health interventions are economically competitive with other kinds of health-sector interventions. Examples include phasing out leaded gasoline, which produces adverse effects on cognitive function and on the productivity of a country. Mental retardation caused by exposure to lead from all sources (not just from leaded gasoline) was estimated to be nearly 30-times higher in regions where leaded gasoline was still being used, compared with regions where leaded gasoline had been completely phased out.
- Environmental health interventions often yield benefits that go beyond the immediate health improvements. A key target of the Millennium Development Goals is halving the proportion of people without sustainable access to safe drinking water and sanitation by 2015. Globally, WHO has estimated that the overall economic benefits of meeting this target, essential to reducing rates for diarrhoea, intestinal nematode infections and malnutrition, would outweigh the investment cost by a ratio of 8:1 (WHO and UNICEF, 2005). In addition, it has been estimated that providing safe drinking-water and improved sanitation to a developing country household would result in an average gain of 60 minutes per household per day, e.g. in terms of time spent collecting water (WHO, 2004b). Safe sanitation also helps break the cycle whereby faecal-oral pathogens often infiltrate drinking-water. Consequent reductions in environmental pollution can not only benefit households, but also fisheries, the food industry, those engaged in water-based recreational activities, as well as the health sector (from avoided health-care costs), and the labour sector (from fewer work days lost to illness).
- Many of the actions affecting determinants of health come from outside the health sector, which highlights the importance of cooperation between sectors when undertaking activities to reduce the environmental health burden. Also, health-sector costs are increasing, and often demands cannot be met, so without cross-sector cooperation it is unlikely that progress will be sustainable in many health areas.

Many environmental health interventions are economically competitive with other kinds of health-sector interventions.



Street Scene in Curitiba, Brazil, where intensive investment in sustainable transport, urban green spaces and urban waste and water management, has reduced environmental risks to health and improved quality of life.

Credit: GTZ

Reducing environmental risks to health also contributes to poverty reduction, while supporting other MDG goals such as access to education and gender equality.



Muslim girls in a classroom.

Credit: M. Nimsiri/UNEP/Still Pictures

Reducing the disease burden of environmental risk factors will contribute significantly to the Millennium Development Goals.

Many Millennium Development Goals (MDGs) have an environmental health component, some of which are highlighted below.

Goal 1 Eradicate extreme poverty and hunger

- Minimizing exposures to environmental risk factors indirectly contributes to reducing poverty, because many environmentally mediated diseases cause lost earnings. If occupational disease, injury or death eliminates the only source of income for a family, this leads to increased poverty and disease for the entire family.
- For developing regions, the mean per capita rate of healthy life years lost to childhood malnutrition is 12-times higher than for developed regions, and there is a 60-fold difference between the per capita rates for the WHO subregions with the highest and lowest malnutrition rates.

Goal 2 Achieve universal primary education

- Environmental health intervention helps to achieve this goal in several ways. Providing clean water and latrines at school (particularly latrines for girls) will encourage primary school students to come to school. Interventions that provide water and fuel for houses will also improve student attendance, because children (often girls) will not need to spend time collecting water and/or fuel for the home. Also, children often look after younger siblings who fall sick from polluted water, or from respiratory disease caused by burning solid fuels in poorly ventilated houses, causing them to miss school. Interventions to improve household ventilation, for example, or to provide clean water, will alleviate the health burden from these risk factors and free children from having to act as caregiver to younger siblings. This should help to improve school attendance and contribute to the MDG.

Goal 3 Promote gender equality and empower women

- Although there were no great differences between the overall rates of environmentally mediated diseases for men and for women, women are disadvantaged in many aspects. In developing countries, women are more likely to be involved in collecting safe water for the family, and in looking after children who may be sick from environmental risk factors such as polluted water or polluted indoor air (from using biomass fuels to cook and heat).

Time invested in these chores lost from activities that could improve the nutritional standard and health of the entire family. Interventions that alleviate such environmental risk factors would therefore free up some of the time women spend in roles such as caretaking and water collection, for income generation or educational activities. This, in turn, contributes to the MDG of empowering women and promoting gender equality.

Goal 4 Reduce child mortality

- The environmentally-linked mortality rate in children under five years of age was 180-times higher in the poorest performing region compared with that in the best performing region. Improving the environment could thus help to reach the MDG, to reduce by two thirds the mortality rate among children under five years old.

Goal 5 Improve maternal health

- Environmental interventions can contribute to this MDG by providing a safe home environment, which is of great importance to the health of children and pregnant mothers. An unprotected or contaminated home environment is a threat to the mother and her unborn child. Childbirth, for example, requires safe water and sanitary conditions.

Goal 6 Combat HIV/AIDS, malaria and other diseases

- Every year, there are over half a million deaths from malaria worldwide and over a quarter of a million deaths from HIV/AIDS that are related to environmental and occupational causes. Targeted environmental interventions could reduce the impact of major diseases such as these and help to achieve the MDG. Environmental interventions could also reduce the number of deaths from diarrhoea and lower respiratory infections by over 3 million each year. With the exception of HIV/AIDS, all of these diseases affect children in large number, and even HIV/AIDS can have a major indirect impact on the health of children.

Goal 7 Ensure environmental sustainability

- Providing sustainable sources of safe water and clean energy are key environmental interventions that contribute to this MDG. The potential health gains from these interventions can be appreciated from the global statistics for 2002: 1.1 billion people, mostly in developing countries, were still using potentially harmful sources of water, and 2.6 billion people lacked even a simple improved latrine (WHO and UNICEF, 2004). Diarrhoeal diseases, caused mainly by a lack of clean water and inadequate sanitation, contribute to nearly 1.7 million deaths a year.

Improving the environment can help reduce the child mortality rate, improve maternal health, and combat HIV/AIDS, malaria and other diseases.



Family life, The Phillipines.

Credit: Mark Edwards/Still Pictures

Both the health sector and non-health sector actors can, and should, take joint action to address environmentally-mediated causes of disease.

Over half of the world's population still relies on biomass fuels and coal to meet their energy needs, resulting in 1.5 million deaths a year from respiratory diseases (a combination of lower respiratory infections, chronic obstructive pulmonary disease and lung cancer) (WHO 2006b).

- Environmental interventions will likely have a great impact on improving the health of slum dwellers, who are among those most affected by the combined health hazards associated with polluted water, inadequate sanitation, urban ambient air pollution, and indoor air pollution from solid fuel use.

Goal 8 Develop a global partnership for development

In summary, environmental health interventions can make a valuable and sustainable contribution towards reducing the global disease burden and improving the well-being of people everywhere. Many interventions can be cost-effective and have benefits beyond improving people's health, benefits such as helping to alleviate poverty and reducing gender inequalities.

Both the health sector and non-health sector actors can, and need, to take joint action to effectively address environmentally-mediated causes of disease. To do this global partnerships are essential. Many such alliances already exist in the field of children's environmental health; occupational health; in joint health sector and environment sector linkages; and in actions in the water, chemical and air pollution sectors. Such global partnerships need to be strengthened and reinforced, harnessing the full range of policy tools, strategies and technologies that are already available – to achieve the interrelated goals of health, environmental sustainability, and development.



Slum housing poses various risks to health that need to be tackled together by the health and non-health sectors (Latin America).

Credit: Mark Edwards/Still Pictures



ANNEX 1

A group of people, including children and adults, are standing on a metal bridge over a river. One person is using a long pole to collect a water sample from the river. The scene is outdoors under a blue sky with clouds. The water in the river is dark and reflects the bridge and the people on it. The bridge has a metal railing and is supported by wooden posts. The people are dressed in casual summer clothing. The overall atmosphere is bright and sunny.

WHO Member States, by WHO subregion and mortality stratum

ANNEX 1 WHO MEMBER STATES, BY WHO SUBREGION AND MORTALITY STRATUM ^a

Subregion and mortality stratum	Description	Broad grouping	Member States
Africa			
AFR-D	Africa with high child and high adult	High-mortality developing	Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Niger, Nigeria, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo.
AFR-E	Africa with high child and very high adult mortality	High-mortality developing	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.
Americas			
AMR-A	Americas with very low child and very low adult mortality	Developed	Canada, Cuba, United States of America.
AMR-B	Americas with low child and low adult mortality	Low-mortality developing	Antigua and Barbuda, Argentina, Bahamas, Barbados, Belize, Brazil, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Grenada, Guyana, Honduras, Jamaica, Mexico, Panama, Paraguay, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Uruguay, Venezuela.
AMR-D	Americas with high child and high adult mortality	High-mortality developing	Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru.
South-East Asia			
SEAR-B	South-East Asia with low child and low adult mortality	Low-mortality developing	Indonesia, Sri Lanka, Thailand.
SEAR-D	South-East Asia with high child and high adult mortality	High-mortality developing	Bangladesh, Bhutan, Democratic People's Republic of Korea, India, Maldives, Myanmar, Nepal, Timor-Leste.
Europe			
EUR-A	Europe with very low child and very low adult mortality	Developed	Andorra, Austria, Belgium, Croatia, Cyprus, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom.
EUR-B	Europe with low child and low adult mortality	Developed	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.
EUR-C	Europe with low child and high adult mortality	Developed	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation, Ukraine.
Eastern Mediterranean			
EMR-B	Eastern Mediterranean with low child and low adult mortality	Low-mortality developing	Bahrain, Iran (Islamic Republic of), Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates.
EMR-D	Eastern Mediterranean with high child and high adult mortality	High-mortality developing	Afghanistan, Djibouti, Egypt, Iraq, Morocco, Pakistan, Somalia, Sudan, Yemen.
Western Pacific			
WPR-A	Western Pacific with very low child and very low adult mortality	Developed	Australia, Brunei Darussalam, Japan, New Zealand, Singapore.
WPR-B	Western Pacific with low child and low adult mortality	Low-mortality developing	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

^a Source: WHO (2004a).

ANNEX 2

Global statistics
produced by the analysis
of the environmental
disease burden

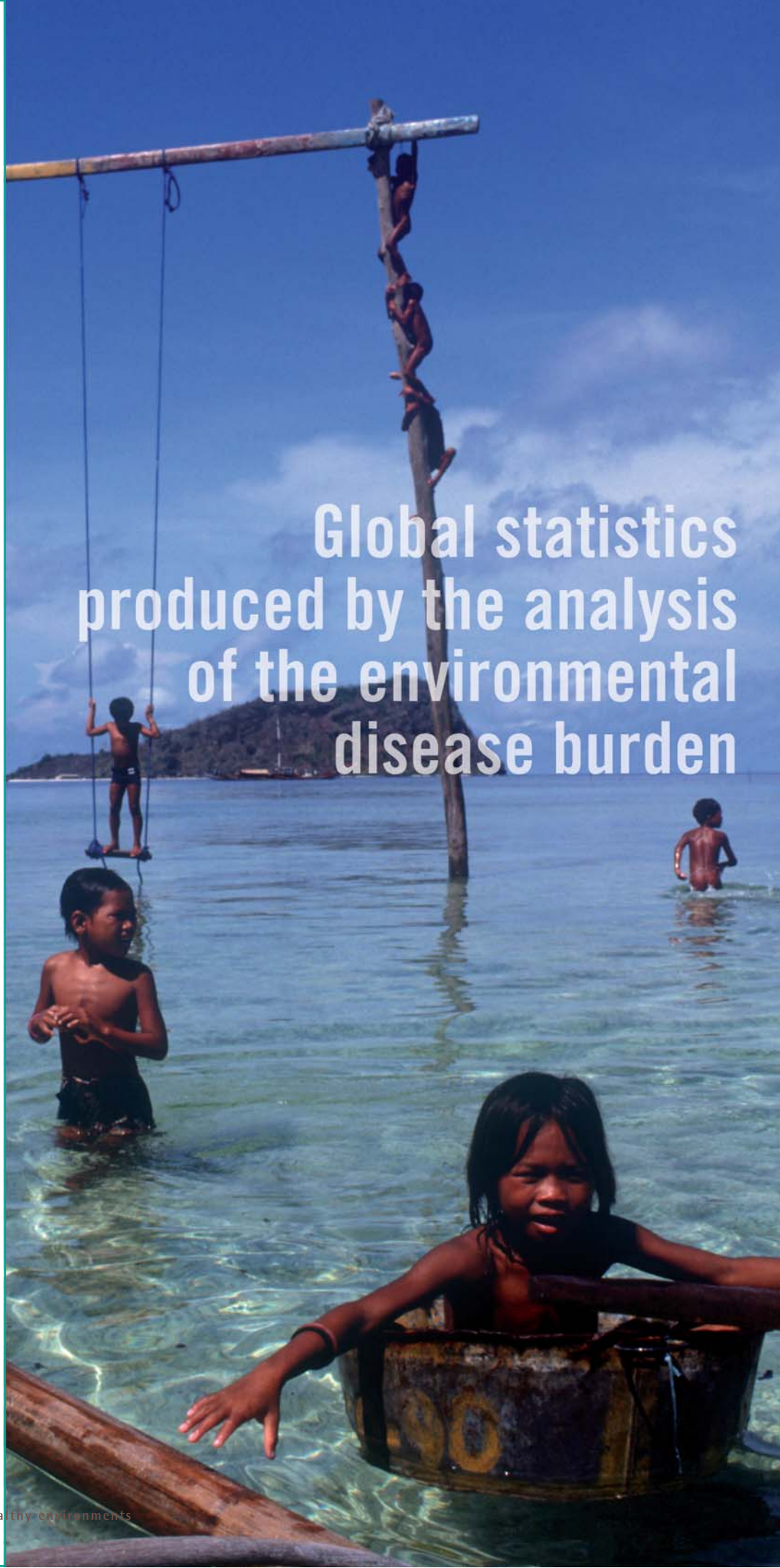


TABLE A2.1 ATTRIBUTABLE ENVIRONMENTAL FRACTIONS FOR EACH DISEASE GROUP >>>

Disease or disease group	Attributable fraction (%)		
	Mean	95% Confidence interval	
		5%	95%
<i>Lower respiratory infections, global</i>	41	32	47
developing countries	42	32	47
developed countries	20	15	25
<i>Upper respiratory infections and otitis, global</i>	25	14	38
developing countries	24	6	45
developed countries	12	5	18
<i>Diarrhoea, global</i>	94	84	98
developing countries	94	84	98
developed countries	90	75	98
<i>Malaria, global</i>	42	30	53
Americas	64	51	77
Eastern Mediterranean	36	25	47
Europe	50	38	63
South-East Asia	42	30	54
Sub-Saharan Africa	42	28	55
Western Pacific	40	34	46
<i>Intestinal nematode infections</i>	100	-	-
<i>Trachoma</i>	100	-	-
<i>Schistosomiasis</i>	100	-	-
<i>Chagas disease</i>	56	31	80
<i>Lymphatic filariasis, global</i>	66	35	86
Africa	40	20	68
Americas	70	60	80
Asia and Western Pacific	82	50	98
<i>Onchocerciasis</i>	10	7	13
<i>Leishmaniasis</i>	26	12	40
Africa and Asia	27	11	40
Central and Latin America	12	1	30
<i>Dengue</i>	95	90	99
<i>Japanese encephalitis</i>	95	90	99
<i>HIV/AIDS^a, global</i>	9	5	14
Industrialized regions of Europe, North America ^b	8	2	13
Developed regions of Western Pacific ^b	2–3	1	5
Eastern Mediterranean ^b	5	2	13
Industrializing regions of Europe ^b	7	2	13
Industrializing regions of Western Pacific ^b	5	2	13
Latin America and the Caribbean ^b	5	2	13
South-East Asia ^b	15	4	20
Sub-Saharan Africa ^b	9	4	20

>>> TABLE A2.1 ATTRIBUTABLE ENVIRONMENTAL FRACTIONS FOR EACH DISEASE GROUP

Disease or disease group	Attributable fraction (%)		
	Mean	95% Confidence interval	
		5%	95%
<i>Sexually transmitted diseases, global</i> (main bacterial infections only)	17	15	19
Eastern Mediterranean, industrializing regions of Europe, Western Pacific, Latin America and Caribbean ^b	8	4	12
Industrialized regions of Europe, South-East Asia ^b	9	4	12
Developing regions of Western Pacific ^b	23	15	25
Sub-Saharan Africa ^b	19	15	25
<i>Hepatitis B, global</i> ^b	1	1	3
Low and intermediate HBV prevalence regions ^b	2	1	4
<i>Tuberculosis, global</i>	18	9	35
Parts of Africa strongly affected by HIV/AIDS	14	6	24
Other regions	19	6	41
<i>Perinatal conditions, global</i>	11	3	24
Developing countries	11	3	25
Developed countries	6	2	10
<i>Congenital anomalies</i>	5	2	10
<i>Malnutrition</i>	50	39	61
<i>Childhood-cluster diseases</i> ^c	24	17	31
<i>Meningitis</i> ^c	11	8	14
<i>Cancers, global</i>	19	12	29
Lung cancer, men, developing countries	33	6	65
Lung cancer, women, developing countries	25	6	37
Other cancer, men, developing countries	18	10	45
Other cancer, women, developing countries	16	10	35
Lung cancer, men, developed countries	30	6	55
Lung cancer, women, developed countries	30	6	55
Other cancer, men, developed countries	16	10	34
Other cancer, women, developed countries	13	10	23
<i>Neuropsychiatric disorders, global</i>	13	10	16
Depression	8	3	17
Bipolar affective disorder	4	0	8
Schizophrenia	4	1	10
Epilepsy, developed countries	8	2	14
Epilepsy, developing countries	23	2	55
Alcohol use disorder	10	2	20
Alzheimer and other dementias	4	1	9
Parkinson's disease	5	1	9
Multiple sclerosis	3	0	9
Drug-use disorders	3	0	9
Post-traumatic stress disorder	19	4	40
Obsessive-compulsive disorder	3	0	8
Panic disorder	6	0	17
Insomnia	20	6	40
Migraine	10	1	27
Other	9	3	17

Disease or disease group	Attributable fraction (%)		
	Mean	95% Confidence interval	
		5%	95%
<i>Cataracts</i>	7	5	10
<i>Deafness</i> ^{d, e}	16	11	21
Africa, males	23	16	30
Africa, females	12	8	16
North America, males	12	8	16
North America, females	5	4	7
Latin America and the Caribbean, males	19	13	25
Latin America and the Caribbean, females	9	6	12
Eastern Mediterranean, males	20	14	26
Eastern Mediterranean, females	11	8	14
Europe subregion A, males	13	9	17
Europe subregion A, females	5	4	7
Europe subregions B,C, males	24	17	31
Europe subregions B,C, females	13	9	17
South-East Asia subregion B, males	23	16	30
South-East Asia subregion B, females	16	11	21
South-East Asia subregion D, females	24	17	31
South-East Asia subregion D, females	9	6	12
Industrialized regions, Western Pacific, males	9	6	12
Industrialized regions, Western Pacific, females	6	4	8
Industrializing regions, Western Pacific, males	26	18	34
Industrializing regions, Western Pacific, females	15	11	20
<i>Cardiovascular diseases</i>	14	7	23
<i>Chronic obstructive pulmonary disease, mean</i> ^e	42	37	47
Africa subregion D, males	45	32	59
Africa subregion D, females	68	48	88
Africa subregion E, males	42	29	55
Africa subregion E, females	60	42	78
North America, males	22	15	29
North America, females	6	4	8
Latin America and Caribbean subregion B, males	29	20	38
Latin America and Caribbean subregion B, females	27	19	35
Latin America and Caribbean subregion D, males	38	27	49
Latin America and Caribbean subregion D, females	47	33	61
Eastern Mediterranean subregion B, males	24	17	31
Eastern Mediterranean subregion B, males	14	10	18
Eastern Mediterranean subregion B, males	38	27	49
Eastern Mediterranean subregion B, males	60	42	78
Europe subregion A, males	22	15	29
Europe subregion A, females	5	4	7
Europe subregion B, males	29	20	38
Europe subregion B, females	32	22	42
Europe subregion C, males	26	18	34
Europe subregion C, females	19	13	25
South-East Asia subregion D, males	36	25	47
South-East Asia subregion D, females	66	46	86
Industrialized regions, Western Pacific, males	27	19	35
Industrialized regions, Western Pacific, females	9	6	12
Industrializing regions, Western Pacific, males	47	33	61
Industrializing regions, Western Pacific, females	64	45	83

>>> TABLE A2.1 ATTRIBUTABLE ENVIRONMENTAL FRACTIONS FOR EACH DISEASE GROUP

Disease or disease group	Attributable fraction (%)		
	Mean	95% Confidence interval	
		5%	95%
<i>Asthma</i>	44	26	53
<i>Musculoskeletal diseases, global^f</i>	17	13	22
Low back pain ^{d, e}	37	26	48
Africa, males	36	25	47
Africa, females	30	21	39
North America, males	35	25	46
North America, females	25	18	33
Latin America and Caribbean subregion B, males	41	29	53
Latin America and Caribbean subregion B, females	23	16	30
Latin America and Caribbean subregion D, males	34	24	44
Latin America and Caribbean subregion D, females	18	13	23
Eastern Mediterranean subregion B, males	31	22	40
Eastern Mediterranean subregion B, males	12	8	16
Eastern Mediterranean subregion B, males	36	25	47
Eastern Mediterranean subregion B, males	25	18	33
Europe subregion A, males	34	24	44
Europe subregion A, females	22	15	29
Europe subregions B,C, males	44	31	57
Europe subregions B,C, females	36	25	47
South-East Asia, males	43	30	56
South-East Asia, females	34	24	44
Industrialized regions of the Western Pacific, males	38	27	49
Industrialized regions of the Western Pacific, females	27	19	35
Industrializing regions of the Western Pacific, males	44	31	57
Industrializing regions of the Western Pacific, females	38	27	49
Osteoarthritis	20	13	26
Rheumatoid arthritis	17	7	29
Other musculoskeletal diseases	15	7	23
<i>Road traffic accidents, global</i>	40	25	57
Developing countries	42	26	60
Europe	25	12	59
North America, industrialized regions of Western Pacific	17	5	50
<i>Unintentional Poisonings, global</i>	71	52	85
Adults	68	46	84
Children	85	60	98
<i>Falls, global</i>	31	16	60
Developing countries	31	17	60
Developed countries	26	16	47
<i>Fires</i>	7	3	11
<i>Drownings, global</i>	72	46	91
Developing countries	74	48	92
Developed countries	54	30	76
<i>Other unintentional injuries, global</i>	44	22	74
Developing countries	45	22	76
Developed countries	30	20	40
<i>Suicide, global</i>	30	22	37

Disease or disease group	Attributable fraction (%)		
	Mean	95% Confidence interval	
		5%	95%
Africa	10	5	15
Developing regions of Asia	36	20	50
Developed regions of Western Pacific	16	10	30
Eastern Mediterranean and Europe	22	7	43
Latin America and the Caribbean	18	15	20
North America	24	20	30
<i>Interpersonal violence, global</i>	19	7	31
Developing countries	19	7	31
Developed countries	16	3	28
<i>Physical inactivity, global</i>	19	13	27
Europe	27	12	58
Industrializing regions of Western Pacific	20	8	38
Latin America and the Caribbean	18	11	34
North America and developed regions of Western Pacific	31	12	59
Other developing regions	13	3	35

^a Abbreviations: HBV = hepatitis B virus; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome.

^b Data were taken from adult populations only.

^c Fraction of diseases induced by malnutrition related to the environment.

^d From the Comparative Risk Assessment (Concha-Barrientos et al., 2004).

^e See Annex 1 for a list of countries in each WHO subregion.

^f Gout was included in musculoskeletal diseases, but its environmental attributable fraction was zero.

TABLE A2.2 INDICATIVE VALUES FOR ENVIRONMENTAL ATTRIBUTABLE FRACTIONS, BY SPECIFIC ENVIRONMENTAL RISK FACTOR AND DISEASE RISK ^a

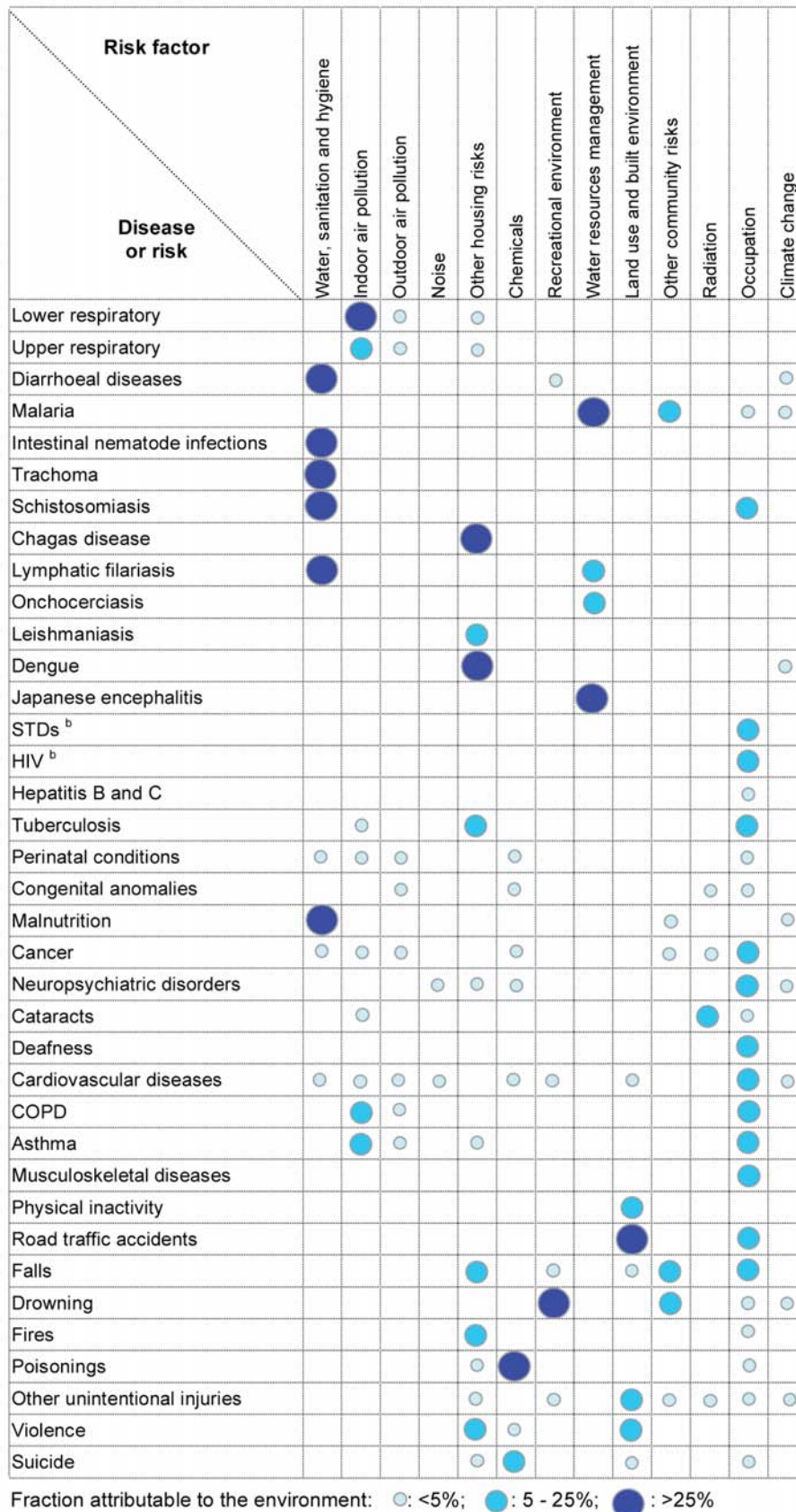




TABLE A2.3 DEATHS ATTRIBUTABLE TO ENVIRONMENTAL FACTORS, BY DISEASE AND MORTALITY STRATUM,

Disease/disease group	AFRICA						THE AMERICAS				
	Total		Children 0–14 years		Developed Countries	Developing Countries	Mortality stratum		Mortality stratum		
	(000)	% ^c	(000)	% ^c	(000)	(000)	High child, high adult (000)	High child, very high adult (000)	Very low child, very low adult (000)	Low child, low adult (000)	High child, high adult (000)
Population (000)	6 224 985		1 830 140		1 366 867	4 858 118	311 273	360 965	333 580	445 161	73 810
Total deaths	57 029		11 945		13 430	43 599	4 657	6 007	2 720	2 701	541
Total environmental deaths ^b	13 295		4 259		2 302	10 994	1 415	992	420	485	121
% of total deaths	23		36		17	25	30	17	15	18	22
Lower respiratory infections	1 516	11.5	823	19.3	113	1 403	234	217	14	43	19
Upper respiratory infections and otitis	20	0.1	10	0.2	2	18	2	2	0	0	0
Diarrhoeal diseases	1 682	12.8	1 512	35.5	18	1 664	329	334	2	31	20
Malaria	526	4.0	482	11.3	0	526	232	241	0	1	0
Intestinal nematode infections ^d	12	0.1	8	0.2	0	12	1	2	0	0	1
Trachoma	0	0.0	0	0.0	0	0	0	0	0	0	0
Schistosomiasis	15	0.1	0	0.0	0	15	1	1	0	1	0
Chagas disease	8	0.1	0	0.0	0	8	0	0	0	6	2
Lymphatic filariasis	0	0.0	0	0.0	0	0	0	0	0	0	0
Onchocerciasis	0	0.0	0	0.0	0	0	0	0	0	0	0
Leishmaniasis	14	0.1	6	0.1	0	14	1	1	0	0	0
Dengue	18	0.1	14	0.3	0	18	0	0	0	1	1
Japanese encephalitis	13	0.1	7	0.2	0	13	0	0	0	0	0
HIV/AIDS (>15 years)	259	2.0	NA	NA	4	256	43	149	1	3	2
STDs (>15 years) ^c	35	0.3	NA	NA	0	35	7	6	0	0	0
Hepatitis B (>15 years)	3	0.0	NA	NA	0	3	0	0	0	0	0
Tuberculosis	285	2.2	17	0.4	14	271	27	31	0	5	4
Perinatal conditions	270	2.1	270	6.3	8	262	31	30	1	15	3
Congenital anomalies	27	0.2	1	0.0	3	24	1	2	1	2	0
Malnutrition ^f	74	0.6	74	1.7	0	74	17	22	0	4	2
Childhood-cluster diseases ^{g h}	276	2.1	276	6.5	0	276	76	54	0	0	1
Meningitis ^h	13	0.1	13	0.3	1	12	1	1	0	0	1
Cancer, total	1 385	10.6	15	0.3	550	836	35	39	125	78	12
Lung cancer	380	2.9	0	0.0	182	198	2	3	51	15	1
Other cancers ⁱ	1 006	7.7	15	0.3	368	637	33	36	74	63	11
Neuropsychiatric disorders, total	91	0.7	11	0.3	28	62	6	7	10	5	1
Depression	1	0.0	0	0.0	0	1	0	0	0	0	0
Bipolar affective disorder	0	0.0	0	0.0	0	0	0	0	0	0	0
Schizophrenia	1	0.0	0	0.0	0	1	0	0	0	0	0
Epilepsy	28	0.2	8	0.2	3	25	4	5	0	1	0
Alcohol use disorder	9	0.1	0	0.0	4	5	0	0	1	1	0
Alzheimer & other dementias	16	0.1	0	0.0	9	7	0	0	4	0	0
Parkinson's disease	5	0.0	0	0.0	2	2	0	0	1	0	0
Multiple sclerosis	1	0.0	0	0.0	0	0	0	0	0	0	0
Drug use disorders	2	0.0	0	0.0	1	2	0	0	0	0	0
Post-traumatic stress disorder	0	0.0	0	0.0	0	0	0	0	0	0	0
Obsessive-compulsive disorder	0	0.0	0	0.0	0	0	0	0	0	0	0
Panic disorder	0	0.0	0	0.0	0	0	0	0	0	0	0
Insomnia	0	0.0	0	0.0	0	0	0	0	0	0	0
Migraine	0	0.0	0	0.0	0	0	0	0	0	0	0
Mental retardation ^j	5	0.0	2	0.0	2	4	0	0	1	0	0
Other	23	0.2	3	0.1	8	15	1	2	3	2	0

Disease/disease group	EASTERN MEDITERRANEAN		EUROPE		SOUTH-EAST ASIA		WESTERN PACIFIC		
	Mortality stratum		Mortality stratum		Mortality stratum		Mortality stratum		
	Low child, Low adult	High child, High adult	Very low child, Very low adult	Low child, low adult	Low child, High adult	Low child, Low adult	High child, High adult	Very low child, very low adult	Low child, low adult
Population (000)	142 528 (000)	360 296 (000)	415 323 (000)	222 846 (000)	239 717 (000)	298 234 (000)	1 292 598 (000)	155 400 (000)	1 562 136 (000)
Total deaths	706	3 446	3 920	1 865	3 779	2 191	12 466	1 146	10 794
Total environmental deaths ^h	145	915	602	348	743	466	3 198	189	2 713
% of total deaths	20	27	15	19	20	21	26	17	25
Lower respiratory infections	9	134	34	25	20	50	544	19	153
Upper respiratory infections and otitis	0	2	0	0	1	1	5	0	7
Diarrhoeal diseases	14	229	2	12	1	38	527	1	143
Malaria	1	20	0	0	0	5	22	0	5
Intestinal nematode infections ^d	0	1	0	0	0	1	4	0	1
Trachoma	0	0	0	0	0	0	0	0	0
Schistosomiasis	0	9	0	0	0	0	0	0	4
Chagas disease	0	0	0	0	0	0	0	0	0
Lymphatic filariasis	0	0	0	0	0	0	0	0	0
Onchocerciasis	0	0	0	0	0	0	0	0	0
Leishmaniasis	0	1	0	0	0	0	10	0	0
Dengue	0	1	0	0	0	3	8	0	4
Japanese encephalitis	0	2	0	0	0	0	8	0	3
HIV/AIDS (>15 years)	0	2	0	0	2	7	44	0	4
STDs (>15 years) ^c	0	0	0	0	0	0	3	0	0
Hepatitis B (>15 years)	0	0	0	0	0	0	1	0	0
Tuberculosis	1	25	1	4	8	26	86	1	67
Perinatal conditions	3	30	1	4	2	9	103	0	38
Congenital anomalies	1	3	1	1	1	1	7	0	6
Malnutrition ^f	1	10	0	0	0	2	12	0	3
Childhood-cluster diseases ^{g h}	0	34	0	0	0	5	99	0	8
Meningitis ^h	0	5	0	1	0	0	3	0	2
Cancer, total	15	37	194	61	105	52	173	66	395
Lung cancer	3	6	60	20	32	13	42	19	112
Other cancers ⁱ	12	31	134	41	73	39	131	47	283
Neuropsychiatric disorders, total	3	7	11	3	4	5	17	1	11
Depression	0	0	0	0	0	0	1	0	0
Bipolar affective disorder	0	0	0	0	0	0	0	0	0
Schizophrenia	0	0	0	0	0	0	0	0	0
Epilepsy	0	2	0	1	1	1	7	0	4
Alcohol use disorder	0	0	1	0	1	0	1	0	1
Alzheimer and other dementias	0	0	4	0	0	0	3	0	2
Parkinson's disease	0	0	1	0	0	0	0	0	1
Multiple sclerosis	0	0	0	0	0	0	0	0	0
Drug use disorders	0	1	0	0	0	0	1	0	0
Post-traumatic stress disorder	0	0	0	0	0	0	0	0	0
Obsessive-compulsive disorder	0	0	0	0	0	0	0	0	0
Panic disorder	0	0	0	0	0	0	0	0	0
Insomnia	0	0	0	0	0	0	0	0	0
Migraine	0	0	0	0	0	0	0	0	0
Mental retardation ^j	2	2	0	0	0	0	0	0	0
Other	0	2	3	1	1	3	4	1	2

>>> TABLE A2.3 DEATHS ATTRIBUTABLE TO ENVIRONMENTAL FACTORS, BY DISEASE AND MORTALITY

Disease/disease group	AFRICA						THE AMERICAS				
	Mortality stratum						Mortality stratum				
	Total	Children 0–14 years		Developed Countries	Developing Countries	High child, high adult	High child, very high adult	Very low child, very low adult	Low child, low adult	High child, high adult	
<i>Population (000)</i>	6 224 985	1 830 140	1 366 867	4 858 118	311 273	360 965	333 580	445 161	73 810		
	(000)	% ^c	(000)	% ^c	(000)	(000)	(000)	(000)	(000)	(000)	
Cataracts	0	0.0	0	0.0	0	0	0	0	0	0	
Deafness	0	0.0	0	0.0	0	0	0	0	0	0	
Cardiovascular diseases ^k	2 571	19.6	18	0.4	1 006	1 566	75	82	166	123	14
COPD	1 312	9.9	0	0.0	78	1 234	29	32	20	26	5
Asthma	106	0.8	5	0.1	23	82	5	6	2	4	1
Musculoskeletal diseases, total	12	0.1	0	0.0	5	7	1	0	2	1	0
Low back pain	1	0.0	0	0.0	0	0	0	0	0	0	0
Osteoarthritis	1	0.0	0	0.0	0	0	0	0	0	0	0
Rheumatoid arthritis	3	0.0	0	0.0	1	2	0	0	0	0	0
Other musculoskeletal diseases	8	0.1	0	0.0	3	5	0	0	1	1	0
Road traffic accidents	467	3.6	74	1.7	56	412	40	41	9	31	4
Poisonings	243	1.9	30	0.7	86	158	15	13	10	1	0
Falls	123	0.9	12	0.3	32	91	3	3	5	4	0
Fires	22	0.2	5	0.1	2	20	2	1	0	0	0
Drownings	277	2.1	106	2.5	33	244	27	21	2	11	2
Other unintentional injuries	402	3.1	83	1.9	80	323	32	25	9	26	8
Suicide	258	2.0	4	0.1	51	207	2	2	9	5	0
Violence	105	0.8	6	0.1	16	88	11	15	3	22	2

Disease/disease group	EASTERN MEDITERRANEAN		EUROPE			SOUTH-EAST ASIA		WESTERN PACIFIC	
	Mortality stratum		Mortality stratum			Mortality stratum		Mortality stratum	
	Low child, Low adult	High child, High adult	Very low child, Very low adult	Low child, low adult	Low child, High adult	Low child, Low adult	High child, High adult	Very low child, very low adult	Low child, low adult
<i>Population (000)</i>	142 528	360 296	415 323	222 846	239 717	298 234	1 292 598	155 400	1 562 136
	(000)	(000)	(000)	(000)	(000)	(000)	(000)	(000)	(000)
Cataracts	0	0	0	0	0	0	0	0	0
Deafness	0	0	0	0	0	0	0	0	0
Cardiovascular diseases ^k	43	120	249	167	364	90	504	59	515
COPD	3	40	22	14	18	49	280	5	772
Asthma	1	6	5	4	10	9	34	2	16
Musculoskeletal diseases, total	0	0	2	0	1	1	1	1	2
Low back pain	0	0	0	0	0	0	0	0	0
Osteoarthritis	0	0	0	0	0	0	0	0	0
Rheumatoid arthritis	0	0	0	0	0	0	0	0	1
Other musculoskeletal diseases	0	0	1	0	0	1	1	0	1
Road traffic accidents	24	31	11	9	24	30	92	2	120
Poisonings	2	8	4	4	67	5	60	1	52
Falls	2	6	15	3	8	5	34	2	34
Fires	1	2	0	0	1	1	12	0	1
Drownings	4	15	2	5	20	11	61	3	92
Other unintentional injuries	8	21	10	14	42	12	118	5	73
Suicide	2	5	11	5	20	13	74	7	104
Violence	1	4	1	1	12	5	16	0	12

Note: Other diseases considered in the analysis but not listed in Table A2.3 include hepatitis C, leprosy and diabetes mellitus, as their environmental contributions to the disease burden are negligible and/or they are secondary to another risk factor.

^a Abbreviations: COPD = chronic obstructive pulmonary disease; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome; STDs = sexually transmitted diseases.

^b Total of all deaths attributable to environmental risk factors. Subtotals for the diseases/disease groups do not add up to 100% because minor diseases are not listed.

^c Percentage of all deaths attributable to environmental risk factors.

^d Intestinal nematode infections include ascariasis, trichuriasis and hookworm disease in this analysis.

^e STDs include syphilis, chlamydia and gonorrhoea in this analysis.

^f Totals include only deaths attributable to protein-energy malnutrition, and not those caused by the consequences of malnutrition.

^g Childhood cluster diseases include pertussis, poliomyelitis, diphtheria, measles and tetanus in this analysis.

^h Induced by malnutrition related to the environment.

ⁱ Other cancers include the 16 other cancer categories as listed in the World Health Report 2004.

^j Only deaths from the consequences of lead-caused mental retardation.

^k Cardiovascular diseases include rheumatic heart disease, hypertensive heart disease, ischaemic heart disease, cerebrovascular disease and inflammatory heart disease in this analysis.

TABLE A2.4 BURDEN OF DISEASE (IN DALYS) ATTRIBUTABLE TO ENVIRONMENTAL FACTORS, BY DISEASE

Disease/disease group	AFRICA						THE AMERICAS				
	Mortality stratum										
	Total	Children 0–14 years		Developed Countries	Developing Countries	High child, high adult	High child, very high adult	Very low child, very low adult	Low child, low adult	High child, high adult	
Population (000)	6 224 985	1 830 140	1 366 867	4 858 118	311 273	360 965	333 580	445 161	73 810		
	(000)	% ^c	(000)	% ^c	(000)	(000)	(000)	(000)	(000)	(000)	
Total DALYs	1490 126		544 534		213 574	1 276 552	160 415	200 961	46 868	81 589	17 130
Total environmental DALYs ^b	353 572		187 839		34 911	318 660	50 121	52 488	6 370	15 394	3 882
% of total DALYs	24		34		16	25	31	26	14	19	23
Lower respiratory infections	37 084	10.5	29 878	15.9	1 173	35 912	7 620	6 663	67	704	403
Upper respiratory infections and otitis	806	0.2	669	0.4	64	742	90	85	6	38	16
Diarrhoeal diseases	57 966	16.4	53 925	28.7	776	57 190	10 812	10 945	93	1 399	702
Malaria	19 241	5.4	17 984	9.6	11	19 230	8 350	8 647	0	55	16
Intestinal nematode infections ^d	2 948	0.8	2 884	1.5	3	2 945	809	329	1	66	101
Trachoma	2 320	0.7	13	0.0	0	2 319	486	726	0	162	2
Schistosomiasis	1 698	0.5	560	0.3	1	1 697	621	713	0	74	0
Chagas disease	370	0.1	0	0.0	5	366	0	0	5	270	96
Lymphatic filariasis	3 791	1.1	1 213	0.6	1	3 790	391	415	0	6	1
Onchocerciasis	56	0.0	15	0.0	0	56	44	11	0	0	0
Leishmaniasis	553	0.2	280	0.1	2	551	56	47	0	5	1
Dengue	586	0.2	512	0.3	0	586	1	4	0	30	36
Japanese encephalitis	671	0.2	459	0.2	0	671	0	0	0	0	0
HIV/AIDS (>15 years)	7 594	2.2	NA	NA	135	7 459	1 274	4 390	34	94	65
STDs (>15 years) ^e	1 950	0.6	NA	NA	35	1 915	298	297	6	35	4
Hepatitis B (>15 years)	94	0.0	NA	NA	2	92	0	0	0	1	1
Tuberculosis	6 341	1.8	691	0.4	290	6 050	737	848	2	94	76
Perinatal conditions	10 666	3.0	10 666	5.7	330	10 336	1 200	1 158	48	611	121
Congenital anomalies	1 473	0.4	42	0.0	152	1 321	75	112	37	123	20
Malnutrition ^f	7 446	2.1	7 446	4.0	94	7 352	1 263	1 439	9	248	106
Childhood-cluster diseases ^{g h}	10 064	2.8	10 064	5.4	21	10 043	2 717	1 945	0	11	25
Meningitis ^h	675	0.2	675	0.4	31	643	49	64	0	14	30
Cancer, total	14 504	4.1	549	0.3	4 987	9 517	399	450	1 112	855	133
Lung cancer	3 439	1.0	11	0.0	1 519	1 919	23	35	401	140	8
Other cancers ⁱ	11 065	3.1	538	0.3	3 468	7 597	376	415	711	714	124
Neuropsychiatric disorders, total	24 448	6.9	11 327	6.0	4 051	20 397	1 515	1 542	1 135	2 883	449
Depression	5 334	1.5	480	0.3	1 238	4 097	160	184	416	466	71
Bipolar affective disorder	504	0.1	26	0.0	84	420	29	34	19	38	6
Schizophrenia	626	0.2	64	0.0	92	534	30	35	21	49	8
Epilepsy	1 625	0.5	608	0.3	127	1 498	163	204	14	173	33
Alcohol use disorder	1 988	0.6	25	0.0	750	1 238	24	66	245	344	33
Alzheimer and other dementias	417	0.1	28	0.0	201	216	7	7	52	26	3
Parkinson's disease	77	0.0	1	0.0	40	37	2	2	12	3	0
Multiple sclerosis	48	0.0	5	0.0	15	33	2	1	4	3	1
Drug use disorders	214	0.1	5	0.0	67	147	19	19	23	24	7
Post-traumatic stress disorder	641	0.2	11	0.0	141	500	29	33	35	40	6
Obsessive-compulsive disorder	159	0.0	20	0.0	36	123	13	15	7	18	3
Panic disorder	378	0.1	13	0.0	68	310	20	24	16	29	5
Insomnia	705	0.2	18	0.0	208	497	29	33	55	66	10
Migraine	786	0.2	341	0.2	195	591	20	26	52	77	15
Mental retardation ^j	9 925	2.8	9 843	5.2	499	9 426	912	788	82	1 427	229
Other	1 021	0.3	365	0.2	292	729	59	70	82	97	19

Disease/disease group	EASTERN MEDITERRANEAN		EUROPE			SOUTH-EAST ASIA		WESTERN PACIFIC	
	Mortality stratum		Mortality stratum			Mortality stratum		Mortality stratum	
	Low child, Low adult	High child, High adult	Very low child, Very low adult	Low child, low adult	Low child, High adult	Low child, Low adult	High child, High adult	Very low child, very low adult	Low child, low adult
	Population (000)	Population (000)	Population (000)	Population (000)	Population (000)	Population (000)	Population (000)	Population (000)	Population (000)
Total DALYs	24 074	115 005	51 725	37 697	60 900	62 463	364 110	16 384	248 495
Total environmental DALYs ^b	4 876	30 635	7 255	6 864	11 870	12 638	92 372	2 552	56 255
% of total DALYs	20	27	14	18	19	20	25	16	23
Lower respiratory infections	206	4 070	125	584	332	698	12 499	64	3 047
Upper respiratory infections and otitis	12	79	7	25	23	29	174	3	219
Diarrhoeal diseases	531	7 578	97	454	91	1 387	17 618	40	6 218
Malaria	33	777	1	10	0	211	955	0	185
Intestinal nematode infections ^d	1	225	0	1	0	135	669	2	611
Trachoma	91	283	0	0	0	0	168	0	400
Schistosomiasis	29	197	0	0	0	3	4	0	55
Chagas disease	0	0	0	0	0	0	0	0	0
Lymphatic filariasis	0	49	0	1	0	195	2 401	0	332
Onchocerciasis	0	1	0	0	0	0	0	0	0
Leishmaniasis	4	62	0	1	0	2	363	0	13
Dengue	9	19	0	0	0	85	278	0	125
Japanese encephalitis	0	78	0	0	0	27	262	0	303
HIV/AIDS (>15 years)	3	68	15	2	83	184	1 231	0	151
STDs (>15 years) ^c	11	43	6	11	9	34	197	3	91
Hepatitis B (>15 years)	0	3	0	1	0	0	13	0	0
Tuberculosis	21	544	9	81	191	591	2 027	7	1 113
Perinatal conditions	146	1 195	30	176	72	377	3 946	4	1 582
Congenital anomalies	50	184	30	37	37	63	408	11	286
Malnutrition ^f	83	869	8	49	22	322	2 153	5	870
Childhood-cluster diseases ^{g h}	5	1 237	0	19	0	200	3 577	1	327
Meningitis ^h	6	242	0	31	0	1	129	0	109
Cancer, total	184	519	1 600	676	1 093	617	1 999	507	4 361
Lung cancer	31	59	485	200	310	129	422	124	1 071
Other cancers ⁱ	153	461	1 115	476	783	487	1 577	383	3 290
Neuropsychiatric disorders, total	605	1 670	1 074	733	814	1 183	5 087	294	5 463
Depression	100	298	327	209	206	230	1 403	80	1 185
Bipolar affective disorder	14	31	22	17	16	25	108	9	134
Schizophrenia	18	40	23	22	17	41	143	9	169
Epilepsy	28	117	20	44	44	85	438	5	257
Alcohol use disorder	5	4	218	62	176	32	159	48	572
Alzheimer and other dementias	4	10	80	16	22	15	49	31	95
Parkinson's disease	2	3	14	3	5	2	10	6	13
Multiple sclerosis	1	2	5	2	3	2	9	1	11
Drug use disorders	16	24	22	6	14	5	24	2	9
Post-traumatic stress disorder	16	37	40	24	25	35	139	16	166
Obsessive-compulsive disorder	6	11	8	9	9	6	27	2	25
Panic disorder	10	24	18	14	13	20	84	7	94
Insomnia	7	33	71	24	32	24	175	26	121
Migraine	14	43	76	27	24	35	177	15	183
Mental retardation ^j	330	918	57	199	145	570	1 962	15	2 290
Other	32	74	71	54	62	57	181	23	139

>>> TABLE A2.4 BURDEN OF DISEASE (IN DALYS) ATTRIBUTABLE TO ENVIRONMENTAL FACTORS, BY

Disease/disease group	AFRICA						THE AMERICAS				
	Total		Children 0-14 years		Developed countries	Developing countries	Mortality stratum		Mortality stratum		
							High child, very high adult	Very low child, very low adult	Low child, low adult	High child, high adult	
	(000)	% ²	(000)	% ²	(000)	(000)	(000)	(000)	(000)	(000)	(000)
Population (000)	6 224 985		1 830 140		1 366 867	4 858 118	311 273	360 965	333 580	445 161	73 810
Cataracts	1 806	0.5	19	0.0	42	1 765	178	191	9	83	18
Deafness	4 284	1.2	0	0.0	696	3 589	155	186	120	157	21
Cardiovascular diseases ^k	23 238	6.6	714	0.4	7 332	15 906	787	867	1 177	1 165	137
COPD	11 654	3.3	0	0.0	1 124	10 530	264	297	251	384	71
Asthma	6 745	1.9	2 785	1.5	1 107	5 638	420	555	349	694	123
Musculoskeletal diseases, total	5 161	1.5	0	0.0	1 433	3 729	201	220	285	368	49
Low back pain	855	0.2	0	0.0	139	716	42	48	27	50	7
Osteoarthritis	2 941	0.8	0	0.0	875	2 066	119	128	166	169	20
Rheumatoid arthritis	740	0.2	0	0.0	236	504	20	21	50	81	12
Other musculoskeletal diseases	626	0.2	0	0.0	183	443	21	22	42	68	10
Road traffic accidents	15 295	4.3	4 036	2.1	1 574	13 720	1 505	1 457	238	1 062	143
Poisonings	5 235	1.5	1 117	0.6	1 714	3 521	435	368	227	46	12
Falls	5 102	1.4	1 965	1.0	789	4 313	194	145	97	205	42
Fires	800	0.2	330	0.2	53	746	76	63	7	9	3
Drownings	7 871	2.2	3 845	2.0	736	7 135	779	624	63	333	59
Other unintentional injuries	21 465	6.1	7 724	4.1	2 550	18 915	2 080	1 659	253	1 322	377
Suicide	6 189	1.8	301	0.2	1 053	5 135	39	50	191	115	14
Violence	4 015	1.1	381	0.2	537	3 478	443	553	100	1 070	68

DISEASE AND MORTALITY STRATUM, FOR WHO REGIONS IN 2002 ^a

Disease/disease group	EASTERN MEDITERRANEAN		EUROPE			SOUTH-EAST ASIA		WESTERN PACIFIC	
	Mortality stratum		Mortality stratum			Mortality stratum		Mortality stratum	
	Low child, Low adult	High child, High adult	Very low child, Very low adult	Low child, low adult	Low child, High adult	Low child, Low adult	High child, High adult	Very low child, very low adult	Low child, low adult
<i>Population (000)</i>	142 528	360 296	415 323	222 846	239 717	298 234	1 292 598	155 400	1 562 136
	(000)	(000)	(000)	(000)	(000)	(000)	(000)	(000)	(000)
Cataracts	41	134	8	11	12	249	542	2	328
Deafness	83	244	164	141	226	332	1 151	44	1 261
Cardiovascular diseases ^k	449	1 382	1 447	1 346	2 939	996	5 584	424	4 538
COPD	70	450	242	184	225	691	3 300	221	5 003
Asthma	123	443	308	158	132	271	1 684	159	1 325
Musculoskeletal diseases, total	80	209	370	273	352	275	887	154	1 440
Low back pain	13	44	32	31	34	47	198	14	267
Osteoarthritis	43	109	229	165	214	178	460	101	840
Rheumatoid arthritis	14	32	69	41	53	19	130	23	175
Other musculoskeletal diseases	10	23	39	35	51	30	100	15	158
Road traffic accidents	764	1 125	305	264	713	994	3 130	54	3 539
Poisonings	53	193	86	88	1 287	110	1 180	25	1 125
Falls	161	385	195	153	298	212	1 599	46	1 370
Fires	20	76	4	11	30	32	425	2	42
Drownings	113	477	40	122	475	291	1 714	35	2 745
Other unintentional injuries	603	1 717	281	700	1 209	796	6 808	107	3 553
Suicide	56	164	195	117	432	338	2 193	118	2 166
Violence	45	195	21	48	362	177	500	6	427

- ^a Abbreviations: COPD = chronic obstructive pulmonary disease; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome; STDs = sexually transmitted diseases.
- ^b Total of all DALYs attributable to environmental risk factors. Subtotals for the diseases/disease groups do not add up to 100% because minor diseases are not listed.
- ^c Percentage of all DALYs attributable to environmental risk factors.
- ^d Intestinal nematode infections include ascariasis, trichuriasis and hookworm disease in this analysis.
- ^e STDs include syphilis, chlamydia and gonorrhoea in this analysis.
- ^f Totals include only DALYs attributable to protein-energy malnutrition, and not those caused by the consequences of malnutrition.
- ^g Childhood cluster diseases include pertussis, poliomyelitis, diphtheria, measles and tetanus in this analysis.
- ^h Induced by malnutrition related to the environment.
- ⁱ Other cancers include the 16 other cancer categories as listed in the World Health Report 2004.
- ^j Only DALYs from the consequences of lead-caused mental retardation.
- ^k Cardiovascular diseases include rheumatic heart disease, hypertensive heart disease, ischaemic heart disease, cerebrovascular disease and inflammatory heart disease in this analysis.

REFERENCES

- Adjami AG, Toe L, Bissan Y, Bugri S, Yameogo L, Kone M, Katholi CR, Unnasch TR (2004). The current status of onchocerciasis in the forest/savannah transition zone of Cote d'Ivoire. *Parasitology*, 128(Pt 4):407–414.
- Alter MJ (1997). The epidemiology of acute and chronic hepatitis C. *Clinics in Liver Disease*, 1(3):559–568, vi–vii.
- André C, Platteau JP (1998). Land relations under unbearable stress: Rwanda caught in the Malthusian trap. *Journal of Economic Behavior and Organization*, 34(1):1–47.
- Anonymous (1994). HIV and STD prevalence among bus and truck drivers in Cameroon. *AIDS Analysis Africa*, 4(5):2.
- Anonymous (2004). Tuberculosis – the 3rd most frequent infectious disease for health care personnel. *Krankenflege Journal*, 42(1–2):15.
- Antunes JL, Waldman EA (2001). The impact of AIDS, immigration and housing overcrowding on tuberculosis deaths in Sao Paulo, Brazil, 1994–1998. *Social Science and Medicine*, 52(7):1071–1080.
- Appawu MA, Dadzie SK, Baffoe-Wilmot A, Wilson MD (2001). Lymphatic filariasis in Ghana: entomological investigation of transmission dynamics and intensity in communities served by irrigation systems in the upper east region of Ghana. *Tropical Medicine and International Health*, 6(7):511–516.
- Ault SK (1994). Environmental management: a re-emerging vector control strategy. *American Journal of Tropical Medicine and Hygiene*, 50: 35–49.
- Baldacci S, Viegi G (2002). Respiratory effects of environmental pollution: epidemiological data. *Monaldi Archives for Chest Disease*, 57(3–4):156–160.
- Belkic KL, Landsbergis PA, Schnall PL, Baker D (2004). Is job strain a major source of cardiovascular disease risk? *Scandinavian Journal of Work, Environment and Health*, 30(2):85–128.
- Benenson AS (1995). Control of communicable disease manual, 16th ed. Washington, DC, American Public Health Association.
- Bertolote J (1993). *Guidelines for the primary prevention of mental, neurological and psychosocial disorders: suicide*. Geneva, World Health Organization.
- Bhandari N, Bahl R, Taneja S, de Onis M, Bhan MK (2004). Growth performance of affluent Indian children is similar to that in developed countries. *Bulletin of the World Health Organization*, 80:189–195.
- Bhatnagar R, West KP Jr, Viatile S, Sommer A, Joshi S, Venkataswamy G (1991). Risk of cataract and history of severe diarrheal disease in southern India. *Archives of Ophthalmology*, 109(5):696–699.
- Bos R (1990). The importance of peridomestic environmental management for the control of vectors of Chagas disease. *Revista Argentina de Microbiología*, [Argentine Journal of Microbiology], 20(1):58–62.
- Bos R, Mills A (1987). Financial and economic aspects of environmental management for vector control. *Parasitology Today*, 3:160–163.
- Bosma H, Peter R, Siegrist J, Marmot M (1998). Two alternative job stress models and the risk of coronary heart disease. *American Journal of Public Health*, 88(1):68–74.
- Bowles J (1995). Suicide in Western Samoa: an example of a suicide prevention program in a developing country. In: Diekstra R, Gulbinat RW, De Leo D, Kienhorst I, eds. *Preventive strategies on suicide*. Leiden, Netherlands, Brill.

- Boxer PA, Burnett C, Swanson N (1995). Suicide and occupation: a review of the literature. *Journal of Occupational and Environmental Medicine*, 37(4):442–452.
- Boy E, Bruce N, Delgado H (2002). Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. *Environmental Health Perspective*, 110(1):109–114.
- Brent DA, Perper JA, Allman CJ, Moritz GM, Wartella ME, Zelenak JP (1991). The presence and accessibility of firearms in the homes of adolescent suicides. A case-control study. *Journal of the American Medical Association*, 266(21):2989–2995.
- Brownson RC, Baker EA, Housemann RA, Brennan LK, Bacak SJ (2001). Environmental and policy determinants of physical activity in the United States. *American Journal of Public Health*, 91(12):1995–2003.
- Bucheton B, Kheir MM, El-Safi SH, Hammad A, Mergani A, Mary C, Abel L, Dessein A (2002). The interplay between environmental and host factors during an outbreak of visceral leishmaniasis in eastern Sudan. *Microbes and Infection*, 4(14):1449–1457.
- Bull FC, Armstrong TP, Dixon T, Ham S, Neiman A, Pratt M (2004). Physical inactivity. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization.
- Bunn F, Collier T, Frost C, Roberts I, Wentz R (2003). Traffic Calming for the prevention of road traffic injuries: systematic review and meta analysis. *Injury Prevention*, 9: 200–204.
- Braun MM, Morse DL, Maguire B, Broaddus R (1987). Tuberculosis and the acquired immunodeficiency syndrome in prisoners. *Journal of the American Medical Association*, 257:1471–1472.
- Byrd Jr RP, Mehta JB, Roy TM (2002). Malnutrition and pulmonary tuberculosis. *Clinical Infectious Diseases*, 35:634–635.
- California Environmental Protection Agency (1997). *Health effects of exposure to environmental tobacco smoke*. Sacramento, CA, Office of Environmental Health Hazard Assessment.
- Camejo MI, Mata G, Diaz M (2003). Prevalencia de hepatitis B, hepatitis C y sífilis en trabajadoras sexuales de Venezuela. [Prevalence of hepatitis B, hepatitis C and syphilis in female sex workers in Venezuela]. *Revista de Salud Publica [Journal of Public Health]*, 37(3):339–344.
- Campbell-Lendrum D, Dujardin JP, Martinez E, Feliciangeli MD, Perez JE, Passerat de Silans LNM, Desjeux P (2001). Domestic and peridomestic transmission of American cutaneous leishmaniasis: changing epidemiological patterns present new control opportunities. *Memórias do Instituto Oswaldo Cruz, [Archives of the Oswaldo Cruz Institute]* 96(2):159–162.
- Carcavallo R, Galindez I, Jurberg J, Lent H (1997). *Atlas of Chagas disease vectors in the Americas*. Rio de Janeiro, Brasil, Editora Fiorescruz.
- Celis A (1991). Drowning in Jalisco: 1983–1989. Asfixia por inmersión en Jalisco: 1983–1989 *Salud Publica de Mexico, [Public Health of Mexico]*, 33(6):585–589.
- Centers for Disease Control and Prevention (2004). Nonfatal and fatal drownings in recreational water settings – United States, 2001–2002. *Morbidity and Mortality Weekly Report*, 53(21):447–452.
- Checkley W, Gilman RH, Black RE, Epstein LD, Cabrera L, Sterling CR, Moulton LH (2004). Effect of water and sanitation on childhood health in a poor Peruvian periurban community. *Lancet*, 363(9403):112–118.
- Chen L, Omaye ST (2001). Air pollution and health effects in northern Nevada. *Reviews on Environmental Health*, 16(2):133–149.
- Chung WSD, Leung CM (2001). Carbon monoxide poisoning as a new method of suicide in Hong Kong. *Psychiatric Services*, 52(6):836–838.
- Clark M, Riben P, Nowgesic E (2002). The association of housing density, isolation and tuberculosis in Canadian First Nations Communities. *International Journal of Epidemiology*, 31(5):940–945.
- Clarke R, Lester D (1987). Toxicity of car exhausts and opportunity for suicide: comparison between Britain and the United States. *Journal of Epidemiology and Community Health*, 41:114–120.

Cohen AJ, Anderson HR, Ostro B, Pandey KD, Krzyzanowski M, Künzli N, Gutschmidt K, Pope CA III, Romieu I, Samet JM, Smith KR (2004). Urban air pollution. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization.

Collman GW, Shore DL, Shy CM, Checkoway H, Luria AS (1988). Sunlight and other risk factors for cataracts: an epidemiologic study. *American Journal of Public Health*, 78(11): p. 1459–62.

Committee on Health Risks of Exposure to Radon (Beir VI) (1999). *Health effects of exposure to radon*. National Research Institute, National Academic Press, Washington DC.

Concha-Barrientos M, Imel Nelson D, Driscoll T, Steenland NK, Punnett L, Fingerhut MA, Prüss-Üstün A, Leigh J, Tak SW, Corvalán C (2004). Selected occupational risk factors. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization.

Corbett EL, Churchyard GJ, Clayton T, Herselman P, Williams B, Hayes R, Mulder D, De Cock KM (1999). Risk factors for pulmonary mycobacterial disease in South African gold miners. A case-control study. *American Journal of Respiratory and Critical Care Medicine*, 159:94–99.

Corbett EL, Churchyard GJ, Clayton TC, Williams BG, Mulder D, Hayes RJ, De Cock KM (2000). HIV infection and silicosis: the impact of two potent risk factors on the incidence of mycobacterial disease in South African miners. *AIDS*, 14:2759–2768.

Coura JR, Junqueira AC, Fernandes O, Valente SA, Miles MA (2002). Emerging Chagas disease in Amazonian Brazil. *Trends in Parasitology*, 18(4):171–176.

Craig CL, Brownson RC, Cragg SE, Dunn AL (2002). Exploring the effect of the environment on physical activity: a study examining walking to work. *American Journal of Preventive Medicine*, 23(2 Suppl.):36–43.

Cryer C (2001). What works to prevent accidental injury amongst older people. *Report to the Health Development Agency (HAD)*, London. Canterbury, UK, University of Kent, Centre for Health Services.

Czeizel AE, Elek C, Gundy S, Metneki J, Nemes E, Reis A, Sperling K, Timar L, Tusnady G, Viragh Z (1993). Environmental trichlorfon and cluster of congenital abnormalities. *Lancet*, 341(8844):539–542.

Davies JCA, Williams BG, Debeila MA, Davies DA (2001). Asbestos-related lung disease among women in the Northern Province of South Africa. *South African Journal of Science*, 97:87–92.

Davis FA (1989). *Tabler's cyclopedic medical dictionary*. Philadelphia, PA, FA Davis Company.

De Bourdeaudhuij I, Sallis JF, Saelens BE (2003). Environmental correlates of physical activity in a sample of Belgian adults. *American Journal of Health Promotion*, 18(1):83–92.

Desai MA, Mehta S, Smith KR (2004). *Indoor smoke from solid fuels: Assessing the environmental burden of disease at national and local levels*. Geneva, World Health Organization (WHO Environmental Burden of Disease Series, No. 4).

Desjeux P (2001). The increase in risk factors for leishmaniasis worldwide. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 95(3):239–243.

Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL (2001). Early exposure to lead and juvenile delinquency. *Neurotoxicology and Teratology*, 23(6):511–518.

Elvik R (2001). Area-wide urban traffic calming schemes: a meta-analysis of safety effects. *Accident, Analysis and Prevention*, 33:327–336.

Emerson PM, Lindsay SW, Walraven GE, Faal H, Bogh C, Lowe K, Bailey RL (1999). Effect of fly control on trachoma and diarrhoea. *Lancet*, 353: 1401–1403.

Emerson PM, Cairncross S, Bailey RL, Mabey DC (2000). Review of the evidence base for the 'F' and 'E' components of the SAFE strategy for trachoma control. *Tropical Medicine and International Health*, 5(8):515–527.

Erlanger TE, Keiser J, Caldas de Castro M, Bos R, Singer BH, Tanner M, Utzinger J (2005). Effect of water resource development and management on lymphatic filariasis, and estimates of populations at risk. *American Journal of Tropical Medicine and Hygiene* (in press).

- Esrey SA (1996). Water, waste, and well-being: a multicountry study. *American Journal of Epidemiology*, 143(6):608–623.
- Esrey SA, Habicht JP, Casella G (1992). The complementary effect of latrines and increased water usage on the growth of infants in rural Lesotho. *American Journal of Epidemiology*, 135(6):659–666.
- Esrey SA, Potash JB, Roberts L, Shiff C (1991). Effects of improved water supply and sanitation on ascariasis, diarrhoea, dracunculiasis, hookworm infection, schistosomiasis, and trachoma. *Bulletin of the World Health Organization*, 69(5):609–621.
- Etzel RA (2003). How environmental exposures influence the development and exacerbation of asthma. *Pediatrics*, 112(1 Pt. 2):233–239.
- Etzel RA, Pattishall EN, Haley NJ, Fletcher RH, Henderson FW (1992). Passive smoking and middle ear effusion among children in day care. *Pediatrics*, 90(2 Pt. 1):228–232.
- Evian C, Fox M, MacLeod W, Slotow SJ, Rosen S (2004). Prevalence of HIV in workforces in southern Africa, 2000–2001. *South African Medical Journal*, 94(2):125–130.
- Ewing R (2005). Can the physical environment determine physical activity levels? *Exercise and Sport Sciences Reviews*, 33(2):69–75.
- Eyler AA, Brownson RC, Bacak SJ, Housemann RA (2003). The epidemiology of walking for physical activity in the United States. *Medicine and Science in Sports and Exercise*, 35(9):1529–1536.
- Eyob G, Gebeyhu M, Goshu S, Girma M, Lemma E, Fontanett A (2002). Increase in tuberculosis incidence among the staff working at the Tuberculosis Demonstration and Training Centre in Addis Ababa, Ethiopia: a retrospective cohort study (1989–1998). *International Journal of Tuberculosis and Lung Disease*, 6:85–88.
- Farmer R, Rohde J (1980). Effects of availability and acceptability of lethal instruments on suicide mortality. *ACTA Psychiatrica Scandinavica (Copenhagen)*, 62:436–446.
- Fishman SM, Caulfield LE, de Onis M, Blössner M, Hydner AA, Mullany L, Black RE (2004). Childhood and maternal underweight. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL eds. *Comparative quantification of health risks*, Volume 1. Geneva, World Health Organization.
- Forastiere F, Lo Presti E, Agabiti N, Rapiti E, Perucci CA (2002). Impatto sanitario dell'esposizione al fumo di sigaretta in Italia. [Health impact of exposure to environmental tobacco smoke in Italy]. *Epidemiologia e Prevenzione [Epidemiology and Prevention]*, 26(1):18–29.
- Forjuoh SN, Li G (1996). A review of successful transport and home injury interventions to guide developing countries. *Social Science and Medicine*, 43(11):1551–1560.
- Frank L, Andresen, M, Schmid T (2004). Obesity relationships with community design, physical activity, and time spent in cars. *American Journal of Preventive Medicine*, 27: 87–96.
- Franssen AEM, Kwekkeboom JMI (2003). Effecten van geluid door wegverkeer op de slaap. Een systematische review van studies in de woonomgeving. [Effects from road traffic noise on sleep. A systematic review of field studies]. Bilthoven: Report 715120010.
- Gauderman WJ, Avol E, Gililand F, Vora DH, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurman F, Rappaport E, Margolis H, Bates D, Peters J (2004). The effect of air pollution on lung development from 10 to 18 years of age. *The New England Journal of Medicine*, 351(11):1057–1067.
- Gawande AV, Vasudeo ND, Zodpey SP, Khandait DW (2000). Sexually transmitted infections in long distance truck drivers. *Journal of Communicable Diseases*, 32(3):212–215.
- Gesch B (2005). The potential of nutrition to promote physical and behavioural well-being. In: Huppert FA, Baylis N, Keverne B, eds. *The science of well-being*. Oxford, Oxford University Press.
- Gesch CB, Hammond SM, Hampson SE, Eves A, Crowder MJ (2002). Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners. *British Journal of Psychiatry*, 181:22–28.
- Gillespie LD, Gillespie WJ, Robertson MC, Lamb SE, Cumming RG, Rowe BH (2003). Interventions for preventing falls in elderly people. *Cochrane Database of Systematic Reviews*, 4:CD000340.

- Giustini M, Ade P, Taggi F, Funari E (2003). Incidenti nelle aree di balneazione. [Accidents in recreational waters]. *Annali dell'Istituto Superiore di Sanita, [Annals of the Superior Sanitary Institute]*, 39(1):69–76.
- Greenland S (1984). Bias in methods for deriving standardized morbidity ratio in and attributable fraction estimates. *Statistics in Medicine*, 3:131–141.
- Grosse SD, Matte TD, Schwartz J, Jackson RJ (2002). Economic gains resulting from the reduction in children's exposure to lead in the United States. *Environmental Health Perspectives*, 110(6):563–569.
- Gunnell D, Eddleston M (2003). Suicide by ingestion of pesticide: a continuing tragedy in developing countries. *International Journal of Epidemiology*, 32(6):902–909.
- Habicht JP, Martorell R, Yarbrough C, Malina RM, Klein RE (1974). Height and weight standards for preschool children: How relevant are ethnic differences in growth potential? *Lancet*, 1(7858):611–614.
- Hemenway D, Miller M (2000). Firearm availability and homicide rates across 26 high-income countries. *Journal of Trauma*, 49(6):985–988.
- Heukelbach J, de Oliveira FA, Kerr-Pontes LR, Feldmeier H (2001). Risk factors associated with an outbreak of dengue fever in a favela in Fortaleza, north-east Brazil. *Tropical Medicine and International Health*, 6(8):635–642.
- Hibbeln JR (2001). Seafood consumption and homicide mortality. A cross-national ecological analysis. *World Review of Nutrition and Dietetics*, 88:41–46.
- Hollows F, Moran D (1981). Cataract – the ultraviolet risk factor. *Lancet*, 2(8258):1249–1250.
- Huang Z, de la Fuente-Fernandez R, Stroessl AJ (2003). Etiology of Parkinson's disease. *Canadian Journal of Neurological Sciences*, 30(Suppl. 1):S10–18.
- Huston SL, Evenson KR, Bors P, Gizlice Z (2003). Neighborhood environment, access to places for activity, and leisure-time physical activity in a diverse North Carolina population. *American Journal of Health Promotion*, 18(1):58–69.
- IARC (1990). *Cancer: Causes, occurrence and control*. Lyon, France WHO International Agency for Research on Cancer (IARC Scientific Publication No. 100).
- IARC (2004). *Tobacco smoke and involuntary smoking*. Lyon, France WHO International Agency for Research on Cancer (IARC Monographs on the evaluation of carcinogenic risks to humans, Volume 83).
- Ishi K, Suzuku F, Saito A, Yoshimoto S, Kubota T (2001). Prevalence of human immunodeficiency virus, hepatitis B and hepatitis C virus antibodies and hepatitis B antigen among commercial sex workers in Japan. *Infectious Diseases in Obstetrics and Gynecology*, 9(4):215–219.
- Jelip J, Mathew GG, Yusin T, Dony JF, Singh N, Ashaari M, Lajanin N, Shanmuga Ratnam C, Yusof Ibrahim M, Gopinath D (2004). Risk factors of tuberculosis among health-care workers in Sabah, Malaysia. *Tuberculosis*, 84(1–2):19–23.
- Johnson JV, Hall EM, Theorell T (1989). Combined effects of job strain and social isolation on cardiovascular disease morbidity and mortality in a random sample of the Swedish male working population. *Scandinavian Journal of Work, Environment and Health*, 15(4):271–279.
- Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL (1988). Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *American Journal of Public Health*, 78(8):910–918.
- Kaur S, Cohen A, Dolor R, Coffman CJ, Bastian LA (2004). The impact of environmental tobacco smoke on women's risk of dying from heart disease: a meta-analysis. *Journal of Women's Health (Larchmont)*, 13(8):888–897.
- Keiser J, Maltese MF, Erlanger TE, Bos R, Tanner M, Singer BH, Utzinger J (2005). Effect of irrigated rice agriculture on Japanese encephalitis, including challenges and opportunities for integrated vector management. *Acta Tropica*, (in press).
- Kellermann AL, Rivara FP, Somes G, Reay DT, Francisco J, Gillentine Banton J, Prodzinski J, Fligner C, Hackman BB (1992). Suicide in the home in relationship to gun ownership. *The New England Journal of Medicine*, 327(7):467–472.

- Khuder SA, Peshimam AZ, Agraharam S (2002). Environmental risk factors for rheumatoid arthritis. *Reviews on Environmental Health*, 17(4):307–315.
- King G, Tomz M, Wittenberg J (2000). Making the most of statistical analyses: Improving interpretation and presentation. *American Journal of Political Science*, 44(2):341–355.
- Kirkhorn S, Greenlee RT, Reeser JC (2003). The epidemiology of agriculture-related osteoarthritis and its impact on occupational disability. *WMJ:State Medical Society of Wisconsin*, 102(7):38–44.
- Kirkwood BR, Gove S, Rogers S, Lob-Levyt J, Arthur P, Campbell H (1995). Potential interventions for the prevention of childhood pneumonia in developing countries: a systematic review. *Bulletin of the World Health Organization*, 73(6):793–798.
- Koenig JQ (1999). Air pollution and asthma. *Journal of Allergy and Clinical Immunology*, 104(4 Pt. 1):717–722.
- Kreitman N (1976). The coal gas story. *British Journal of Preventive and Social Medicine*, 30:86–93.
- Last JM (2001). *A dictionary of epidemiology*, 4th ed. New York, Oxford University Press, International Epidemiological Association.
- Leenaars A, Cantor C, Connolly J, EchoHawk M, Gailiene D, He ZX, Kokorina N, Lester D, Lopatin AA, Rodriguez M, Schlebusch L, Takahashi Y, Vijayakumar L, Wenckstern S (2000). Controlling the environment to prevent suicide: international perspectives. *Canadian Journal of Psychiatry - Revue Canadienne de Psychiatrie*, 45(7):639–644.
- Lester D, Murrell ME (1980). The influence of gun control laws on suicidal behaviour. *American Journal of Psychiatry*, 137(1):121–122.
- Lienhardt C (2001). From exposure to disease: the role of environmental factors in susceptibility to and development of tuberculosis. *Epidemiological Reviews*, 23(2):288–300.
- Lieverse A, Bierma-Zeinstra S, Verhagen A, Verhaar J, Koes B (2001). Influence of work on the development of osteoarthritis of the hip: a systematic review. *Journal of Rheumatology*, 28(11):2520–2528.
- Liu J, Raine A, Venables PH, Mednick SA (2004). Malnutrition at age 3 years and externalizing behavior problems at ages 8, 11, and 17 years. *American Journal of Psychiatry*, 161(11):2005–2013.
- Longnecker MP, Klebanoff MA, Zhou H, Brock JW (2001). Association between maternal serum concentration of the DDT metabolite DDE and preterm and small-for-gestational-age babies at birth. *Lancet*, 358(9276):110–114.
- Lubin JH, Boice JD Jr (1997). Lung cancer risk from residential radon: meta-analysis of eight epidemiologic studies. *Journal of the National Cancer Institute*, 89(1):49–57.
- Lucas R (2006). *The global burden of disease from exposure to UV radiation*. Geneva, World Health Organization, (in press).
- Maetzel A, Makela M, Hawker G, Bombardier C (1997). Osteoarthritis of the hip and knee and mechanical occupational exposure – a systematic overview of the evidence. *Journal of Rheumatology*, 24(8):1599–1607.
- Maisonet M, Correa A, Misra D, Jaakkola JJ (2004). A review of the literature on the effects of ambient air pollution on fetal growth. *Environmental Research*, 95(1):106–115.
- Mak R, Traen A, Claeysens M, Van Renterghem L, Leroux-Roels G, Van Damme P (2003). Hepatitis B vaccination for sex workers: do outreach programmes perform better? *Sexually Transmitted Infections*, 79(2):157–159.
- Martorell R, Mendoza F, Castillo R (1988). Poverty and stature in children. In: Waterlow JC, ed. *Linear growth retardation in less developed countries*. New York, Raven Press (Nestle Nutrition Workshop Series volume 14, pp. 57–73.)
- Mayor of London - Transport for London (2004). *Congestion charging: Impacts monitoring. Second annual report April 2004*. London, Transport for London (http://www.tfl.gov.uk/tfl/cc_london/cc_monitoring-2nd-report.shtml, accessed October 2004).
- McCarty CA, Nanjan MB, Taylor HR (2000). Attributable risk estimates for cataract to prioritize medical and public health action. *Investigative Ophthalmology and Visual Science*, 41(12):3720–3725.
- McGuigan MA (1999). Common culprits in childhood poisoning: epidemiology, treatment and parental advice for prevention. *Paediatric Drugs*, 1(4):313–324.

McMichael AJ, Campbell-Lendrum D, Wilkinson P, Wilson T, Nicholls R, Hales S, Tanser F, Le Sueur D, Schlesinger M, Andronova N (2004). Global climate change. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization.

Melse JM, de Hollander AEM (2001). *Environment and health within the OECD region: lost health, lost money*. Background document to the OECD Environmental Outlook. Bilthoven, RIVM (National Institute of Public Health and the Environment), (<http://www.rivm.nl/bibliotheek/rapporten/402101001.pdf>, accessed August 2005).

Meyrowitsch DW, Nguyen DT, Hoang TH, Nguyen TD, Michael E (1998). A review of the present status of lymphatic filariasis in Vietnam. *Acta Tropica*, 70(3):335–347.

Michigan State University (2005). *The dictionary of epidemiology*. East Lansing, Michigan (<http://www.msu.edu/~borsenag/dictionary.htm>, accessed May 2005).

Miettinen OS (1974). Proportion of disease caused or prevented by a given exposure, trait or intervention. *American Journal of Epidemiology*, 99:325–332.

Millward L, Morgan A, Kelly M (2003). *Prevention and reduction of accidental injury in children and older people*. UK, Health Development Agency.

Minassian DC, Mehra V, Jones BR (1984). Dehydrational crises from severe diarrhoea or heatstroke and risk of cataract. *Lancet*, 1(8380):751–753.

Minassian DC, Mehra V, Verrey JD (1989). Dehydrational crises: a major risk factor in blinding cataract. *British Journal of Ophthalmology*, 73(2):100–105.

Mishra VK, Retherford RD, Smith KR (1999). Biomass cooking fuels and prevalence of tuberculosis in India. *International Journal of Infectious Diseases*, 3(3):119–129.

Mohamed N, Ng'ang'a L, Odhiambo J, Nyamwaya J, Menzies R (1995). Home environment and asthma in Kenyan schoolchildren: a case-control study. *Thorax*, 50(1):74–78.

Mohan D (2004). *The road ahead - traffic injuries and fatalities in India*. Delhi, Indian Institute of Technology.

Mohan M, Sperduto RD, Angra SK, Milton RC, Mathur RL, Underwood BA, Jaffery N, Pandya CB, Chhabra VK, Vajpayee RB, Kalra VK, Sharma YR (1989). India-US case-control study of age-related cataracts. India-US case-control study group. *Archives of Ophthalmology*, 107(5):670–676. [Published erratum appears in *ibid* 107(9):1288].

Moreira ED Jr, de Souza VM, Sreenivasan M, Lopes NL, Barreto RB, de Carvalho LP (2003). Peridomestic risk factors for canine leishmaniasis in urban dwellings: new findings from a prospective study in Brazil. *American Journal of Tropical Medicine and Hygiene*, 69(4):393–397.

Motarjemi Y, Kaferstein F, Moy G, Quevedo F (1993). Contaminated weaning food: a major risk factor for diarrhoea and associated malnutrition. *Bulletin of the World Health Organization*, 71(1):79–92.

Muir DA (1988). Anopheline mosquitoes: vector reproduction, life cycle and biotope. In: Wernsdorfer WH, McGregor I., eds. *Malaria. Principles and practices of malariology*. New York, Churchill Livingstone.

National Heart, Lung and Blood Institute (2005). *COPD*. Bethesda, MD (http://www.nhlbi.nih.gov/health/dci/Diseases/Copd/Copd_Causes.html, accessed March 2005).

Needleman HL, McFarland C, Ness RB, Fienberg SE, Tobin MJ (2002). Bone lead levels in adjudicated delinquents. A case-control study. *Neurotoxicology and Teratology*, 24(6):711–717.

Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB (1996). Bone lead levels and delinquent behaviour. *Journal of the American Medical Association*, 275(5): 363–369.

Nessa K, Waris SA, Alam A, Huq M, Nahar S, Chawdhury FA, Monira S, Badal MU, Sultana J, Mahmud KF, Das J, Mitra DK, Sultan Z, Hossain N, Rahman M (2005). Sexually transmitted infections among brothel-based sex workers in Bangladesh: high prevalence of asymptomatic infection. *Sexually Transmitted Diseases*, 32(1):13–19.

- Neumayr G, Propst A, Schwaighofer H, Judmaier G, Vogel W (1999). Lack of evidence for the heterosexual transmission of hepatitis C. *Quarterly Journal of Medicine*, 92(9):505–508.
- Nevin R (2000). How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy. *Environmental Research*, 83(1):1–22.
- Norris B, Wilson JR (2003). Preventing drowning through design – the contribution of human factors. *Injury Control and Safety Promotion*, 10(4):217–226.
- Nurminen M, Karjalainen A (2001). Epidemiologic estimate of the proportion of fatalities related to occupational factors in Finland. *Scandinavian Journal of Work and Environmental Health*, 27:161–213.
- Nurminen T (1995). Maternal pesticide exposure and pregnancy outcome. *Journal of Occupational and Environmental Medicine*, 37(8):935–940.
- Odense Municipality (Odense Kommune) (2004). Evaluering af Odense – Danmarks Nationale Cykleby. [*Evaluating Odense - Denmark's National Cycle City.*] Odense, Odense Municipality.
- Olsen O, Kristensen TS (1991). Impact of work environment on cardiovascular diseases in Denmark. *Journal of Epidemiology and Community Health*, 45:4–9.
- Olsson AR, Skogh T, Axelson O, Wingren G (2004). Occupations and exposures in the work environment as determinants for rheumatoid arthritis. *Occupational and Environmental Medicine*, 61(3):233–238.
- Passchier-Vermeer W, Passchier WF (2000). Noise exposure and public health. *Environmental Health Perspectives*, 108(Suppl. 1):123–131.
- Pavia M, Bianco A, Pileggi C, Angelillo IF (2003). Meta-analysis of residential exposure to radon gas and lung cancer. *Bulletin of the World Health Organization*, 81(10):732–738.
- Peden M, Scurfield R, Sleet D, Mohan D, Hyder AA, Jarawan E, Mathers C (2004). *World report on road traffic injury prevention*. World Health Organization, Geneva.
- Pelletier DL (1994). The relationship between child anthropometry and mortality in developing countries: implications for policy, programs and future research. *Journal of Nutrition*, 124:S2047–2081.
- Pope CA III, Burnett RT, Thun MJ, Calle E, Krewski D, Ito K, Thurston GD (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particle air pollution. *Journal of the American Medical Association*, 287:1132–1141.
- Prüss A, Mariotti SP (2000). Preventing trachoma through environmental sanitation: a review of the evidence base. *Bulletin of the World Health Organization*, 78(2):258–266.
- Prüss-Üstün A, Rapiti E, Hutin Y (2003). *Sharps injuries: Global burden of disease from sharps injuries to health-care workers*. Geneva, World Health Organization (Environmental burden of disease series No. 3. Also available online at http://www.who.int/quantifying_ehimpacts/publications/9241562463/en/).
- Prüss-Üstün A, Kay D, Fewtrell L, Bartram J (2004a). Unsafe water, sanitation and hygiene. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL eds. *Comparative quantification of health risks*. Geneva, World Health Organization.
- Prüss-Üstün A, Fewtrell LJ, Landrigan P, Ayuso-Mateos JL (2004b). Lead exposure. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL eds. *Comparative quantification of health risks*. Geneva, World Health Organization.
- Qin HL, Zhao XC, Zhou JH, Qiu J, Yang ZL, Jiang ZQ, Zhu BZ (2004). Effect of environment on extremely severe road traffic crashes: retrospective epidemic analysis during 2000–2001. *Chinese Journal of Traumatology*, 7(6): 323–329.
- Racioppi F, Eriksson L, Tingvall C, Villaveces A (2004). *Preventing road traffic injury: a public health perspective for Europe*. Copenhagen, WHO Regional Office for Europe.
- Ramsey JM, Cruz-Celis A, Salgado L, Espinosa L, Ordonez R, Lopez R, Schofield CJ, (2003). Efficacy of pyrethroid insecticides against domestic and peridomestic populations of *Triatoma pallidipennis* and *Triatoma barberi* (Reduviidae:Triatominae) vectors of Chagas disease in Mexico. *Journal of Medical Entomology*, 40(6):912–920.

- Reznik BI, Minkov IP, Prudkii VI, Krivenkaia MN, Kildysheva AN, Grinfeld BN (1992). Environmental pollution and congenital abnormalities. *Gigiena i Sanitariya, [Hygiene and Sanitation,]* 7–8:6–9.
- Riedner G, Rusizoka M, Hoffmann O, Nichombe F, Lyamuya E, Mmbando D, Maboko L, Hay P, Todd J, Hayes R, Hoelscher M, Grosskurth H (2003). Baseline survey of sexually transmitted infections in a cohort of female bar workers in Mbeya Region, Tanzania. *Sexually Transmitted Infections*, 79(5):382–387.
- Ritz B, Yu F (1999). The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environmental Health Perspectives*, 107(1):17–25.
- Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA (2002). Ambient air pollution and risk of birth defects in Southern California. *American Journal of Epidemiology*, 155(1):17–25.
- Rojas-De-Arias A (2001). Chagas disease prevention through improved housing using an ecosystem approach to health. *Cadernos de Saude Publica, [Reports in Public Health]*, 17(Suppl.):89–97.
- Rom WN, Garay SM (2003). *Tuberculosis*. Philadelphia, Lippincott, Williams and Wilkins.
- Rosengren A, Hawken S, Ounpuu S, Sliwa K, Zubaid M, Almahmeed WA, Blackett KN, Sitthi-amorn C, Sato H, Yusuf S (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): case-control study. *Lancet*, 11;364(9438):953–962.
- Rossignol M, Leclerc A, Hilliquin P, Allaert FA, Rozenberg S, Valat JP, Avouac B, Coste P, Savarieau B, Fautrel B (2003). Primary osteoarthritis and occupations: a national cross sectional survey of 10 412 symptomatic patients. *Occupational and Environmental Medicine*, 60(11):882–886.
- Rozendaal JA (1997). *Vector control. Methods for use by individuals and communities*. Geneva, World Health Organization.
- Schoenthaler SI, Amos S, Doraz W, Kelly MA, Muedeking G, Wakefield J Jr (1997). The effects of randomized vitamin-mineral supplementation on violent and non-violent antisocial behavior among incarcerated juveniles. *Journal of Nutritional and Environmental Medicine*, 7: 343–352.
- Schrauzer GN (2002). Lithium: occurrence, dietary intakes, nutritional essentiality. *Journal of the American College of Nutrition (New York, NY)*, 21(1):14–21.
- Schroeder DG, Brown KH (1994). Nutritional status as a predictor of child survival: summarizing the association and quantifying its global impact. *Bulletin of the World Health Organization*, 72:569–579.
- Schwartz J (1995). Lead, blood pressure, and cardiovascular disease in men. *Archives of Environmental Health*, 50(1):31–37.
- Segarra-Obiol F, Lopez-Ibanez P, Perez Nicolas J (1983). Asbestosis and tuberculosis. *American Journal of Industrial Medicine*, 1983,4:755–757.
- Seidler A, Raum E, Arabin B, Hellenbrand W, Walter U, Schwartz FW (1999). Maternal occupational exposure to chemical substances and the risk of infants small-for-gestational-age. *American Journal of Industrial Medicine*, 36(1):213–222.
- Siemiatycki J, Richardson L, Straif K, Latreille B, Lakhani R, Campbell S, Rousseau MC, Boffetta P (2004). Listing occupational carcinogens. *Environmental Health Perspectives*, 112:1447–1459.
- Slovak K (2002). Gun violence and children: factors related to exposure and trauma. *Health and Social Work*, 27(2):104–112.
- Smith KR, Corvalán FC, Kjellström T (1999). How much ill health is attributable to environmental factors? *Epidemiology*, 10(5):573–584. Also available online at http://www.who.int/quantifying_ehimpacts/methods/en/smith.pdf, accessed March 2005.
- Smith KR, Mehta S, Maeusezahl-Feuz M (2004). Indoor air pollution from solid household fuels. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization.
- Smith KR, Samet JM, Romieu I, Bruce N (2000). Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax*, 55(6):518–532.
- Steenland K, Burnett C, Lalic N, Ward E, Hurrell J (2003). Dying for work: The magnitude of US mortality from selected causes of death associated with occupation. *American Journal of Industrial Medicine*, 43(5):461–482.

- Stenstrom R, Bernard PA, Ben-Simhon H (1993). Exposure to environmental tobacco smoke as a risk factor for recurrent acute otitis media in children under the age of five years. *International Journal of Pediatric Otorhinolaryngology*, 27(2):127–136.
- Stretesky PB, Lynch MJ (2001). The relationship between lead exposure and homicide. *Archives of Pediatrics and Adolescent Medicine*, 155(5):579–582.
- Stretesky PB, Lynch MJ (2004). The relationship between lead and crime. *Journal of Health and Social Behavior*, 45(2):214–229.
- Sugihantono A, Slidell M, Syaifudin A, Pratjojo H, Utami IM, Sadjimin T, Mayer KH (2003). Syphilis and HIV prevalence among commercial sex workers in Central Java, Indonesia: risk-taking behavior and attitudes that may potentiate a wider epidemic. *AIDS Patient Care STDS*, 17(11):595–600.
- Sutter EE, Ballard RC (1983). Community participation in the control of trachoma in Gazankulu. *Social Science and Medicine*, 17:1813–1817.
- Takeda E, Robazzi ML, Lavrador MA (2001). Ocupacional de adquirir Tuberculose entre trabalhadores de enfermagem hospitalar. [Occupational risk of acquiring tuberculosis among hospital nursing personnel.] *Revista Brasileira de Enfermagem, [Brazilian Journal of Nursing]*, 54(3):456–465.
- Taha TE, Gray RH (1993). Agricultural pesticide exposure and perinatal mortality in central Sudan. *Bulletin of the World Health Organization*, 71(3-4):317–21.
- Tan XH, Wang SM, Xue NQ, Teng WT, Feng YQ (2004). Study on the risk factors and its interaction on Parkinson disease. *Chung-Hua Liu Hsing Ping Hsueh Tsa Chih, [Chinese Journal of Epidemiology]*, 25(6):527–530.
- Tatum AJ, Shapiro GG (2005). The effects of outdoor air pollution and tobacco smoke on asthma. *Immunology and Allergy Clinics of North America*, 25(1):15–30.
- Taylor HR, West SK, Rosenthal FS, Munoz B, Newland HS, Abbey H, Emmett EA (1988). Effect of ultraviolet radiation on cataract formation. *New England Journal of Medicine*, 319(22):1429–1433.
- Taylor R, Cumming R, Woodward A, Black M (2001). Passive smoking and lung cancer: a cumulative meta-analysis. *Australian and New Zealand Journal of Public Health*, 25(3):203–211.
- Tennant C (2001). Work-related stress and depressive disorders. *Journal of Psychosomatic Research*, 51(5):697–704.
- Transport Research Board (2005). *Does the built environment influence physical activity? Examining the evidence. Report 282*. Washington, DC (www.trb.org, accessed March 2005).
- Trapido AS, Mqoqi NP, Williams BG, White NW, Solomon A, Goode RH, Macheke CM, Davies AJ, Panter C (1998). Prevalence of occupational lung disease in a random sample of former mineworkers, Libode District, Eastern Cape Province, South Africa. *American Journal of Industrial Medicine*, 34:305–313.
- UNAIDS, WHO (2004). *AIDS epidemic update - December 2004*. Geneva, Joint United Nations Programme on HIV/AIDS.
- US Census Bureau, UNAIDS (2004). *HIV/AIDS surveillance data base*. Washington DC, US Census Bureau.
- Utzinger J, Tozan Y, Singer BH (2001). Efficacy and cost-effectiveness of environmental management for malaria control. *Tropical Medicine and International Health*, 6: 677–687.
- Valent F, Little D, Bertollini R, Nemer L, Barbone F, Tembulini G (2004). Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe. *Lancet* 363: 2032-2039.
- Vandelli C, Renzo F, Romano L, Tisminetzky S, De Palma M, Stroffolini T, Ventura E, Zanetti A (2004). Lack of evidence of sexual transmission of hepatitis C among monogamous couples: results of a 10-year prospective follow-up study. *American Journal of Gastroenterology*; 99(5):855–859.
- Vineis P, Airoidi L, Veglia P, Olgiati L, Pastorelli R, Autrup H, Dunning A, Garte S, Gormally E, Hainaut P, Malaveille C, Matullo G, Peluso M, Overvad K, Tjonneland A, Clavel-Chapelon F, Boeing H, Krogh V, Palli D, Panico S, Tumino R, Bueno-De-Mesquita B, Peeters P, Berglund G, Hallmans G, Saracci R, Riboli E (2005). Environmental tobacco smoke and risk of respiratory cancer and chronic obstructive pulmonary disease in former smokers and never smokers in the EPIC prospective study. *British Medical Journal*, 5:330(7486):265–266.

- Welsh BC, Farrington DP (2004). Surveillance for crime prevention in public space: results and policy choices in Britain and America. *Criminology and Public Policy*, 3(3):497–526.
- West S (1992). Does smoke get in your eyes? *Journal of the American Academy of Medicine*, 268:1025–1026.
- West SK, Duncan DD, Munoz B, Rubin GS, Fried LP, Bandeen-Roche K, Schein OD (1998). Sunlight exposure and risk of lens opacities in a population-based study: the Salisbury Eye Evaluation project. *Journal of the American Medical Association*, 280(8):714–718.
- WHO (1982). *Manual on environmental management for mosquito control*. Geneva, World Health Organization.
- WHO (1992). *ICD-10 – International statistical classification of diseases and related health problems*, Tenth revision, Volume 1. Geneva, World Health Organization.
- WHO (1995). *Physical Status: The use and interpretation of anthropometry. Report of a WHO Expert Committee (Technical Report Series 854)*, Geneva, World Health Organization, pp. 161–262.
- WHO (1997). *Health and environment in sustainable development*. Geneva, World Health Organization.
- WHO (2000). Growth patterns of breastfed infants in seven countries. *Acta Paediatrica*, 89:215–222.
- WHO (2002). *World Health Report 2002 – Reducing risks, promoting healthy life*. Geneva, World Health Organization (<http://www.who.int/whr/2002/>, accessed March 2005).
- WHO (2003). *Guidelines for safe recreational water environments. Volume 1: Coastal and freshwaters*. Geneva, World Health Organization.
- WHO (2004a). *World Health Report 2004 – changing history*. Geneva, World Health Organization.
- WHO (2004b). *Evaluation of the costs and benefits of water and sanitation improvements at the global level*. Geneva, World Health Organization.
- WHO (2005a). *Water-related diseases: Malnutrition*. Geneva, World Health Organization (http://www.who.int/water_sanitation_health/diseases/malnutrition/en/, accessed June 2005).
- WHO (2005b). *Ecosystems and human health – health synthesis*. Geneva, World Health Organization.
- WHO (2006a). *Nutrients in drinking water: Potential health consequences of long-term consumption of demineralized, remineralized and altered mineral content drinking water*. Report of an expert meeting. Geneva, World Health Organization (in press).
- WHO (2006b). *Fuel for life, household energy and health*. Geneva, World Health Organization (in press).
- WHO Regional Office for Europe (2004). *How can injuries in children and older people be prevented?* Geneva, World Health Organization, Health Evidence Network (<http://www.who.dk/Document/E84938.pdf>, accessed April 2004).
- WHO, UNICEF (2004). *Meeting the MDG drinking water and sanitation target: A mid-term assessment of progress*. Geneva, World Health Organization.
- WHO, UNICEF (2005). *Water for life. Making it happen*. Geneva, World Health Organization.
- Wilhelm K, Kovess V, Rios-Seidel C, Finch A (2004). Work and mental health. *Social Psychiatry and Psychiatric Epidemiology*, 39(11):866–873.
- Wilkinson D, Gunnell D (2000). Comparison of trends in method-specific suicide rates in Australia and England and Wales, 1968–97. *Australian and New Zealand Journal of Public Health (Canberra)*, 24(2):153–157.
- Williams BG, Campbell CM, Mqoqi NP, Kleinschmidt I (1998). Occupational health, occupational illness: tuberculosis, silicosis and HIV on the South African mines. In: Parker JE, ed. *Occupational lung disease: An international perspective*. London, Chapman and Hall Medical, pp. 95–103.
- Williams MD, Sandler AB (2001). The epidemiology of lung cancer. *Cancer Treatment and Research*, 105:31–52.

Wilson MD, Cheke RA, Flasse SP, Grist S, Osei-Ateweneboana MY, Tetteh-Kumah A, Fiasorgbor GK, Jolliffe FR, Boakye DA, Hougard JM, Yameogo L, Post RJ (2002). Deforestation and the spatio-temporal distribution of savannah and forest members of the *Simulium damnosum* complex in southern Ghana and south-western Togo. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 96(6):632–639.

Xu X, Niu T, Christian D (1996). Occupational and environmental risk factors for asthma in rural communities in China. *International Journal of Occupational and Environmental Health*, 2:172–176.

Xueref S, Holianjavony J, Daniel R, Kerouedan D, Fabry J, Vanhems P (2003). The absence of HIV seropositivity contrasts with a high prevalence of markers of sexually transmitted infections among registered female sex workers in Toliary, Madagascar. *Tropical Medicine and International Health*, 8(1):60–66.

Yadon ZE, Rodrigues LC, Davies CR, Quigley MA (2003). Indoor and peridomestic transmission of American cutaneous leishmaniasis in north-western Argentina: a retrospective case-control study. *American Journal of Tropical Medicine and Hygiene*, 68(5):519–526.

Yoshimura N, Nishioka S, Kinoshita H, Hori N, Nishioka T, Ryujin M, Mantani Y, Miyake M, Coggon D, Cooper C (2004). Risk factors for knee osteoarthritis in Japanese women: heavy weight, previous joint injuries, and occupational activities. *Journal of Rheumatology*, 31(1):157–162.

Zachariah R, Spielmann MP, Harries AD, Salaniponi FM (2002). Moderate to severe malnutrition in patients with tuberculosis is a risk factor associated with early death. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 96:291–294.

Zhang J, Cai WW, Lee DJ (1992). Occupational hazards and pregnancy outcomes. *American Journal of Industrial Medicine*, 21(3):397–408.

Zodpey SP, Ughade SN (1999). Exposure to cheaper cooking fuels and risk of age-related cataract in women. *Indian Journal of Occupational and Environmental Medicine*, 3(4):159–161.

ACKNOWLEDGEMENTS

We are very grateful to the experts who provided estimates of the attributable fractions for the diseases and risk factors, as well as to those who provided non-quantitative opinions:

- B.E. Ainsworth, San Diego State University, San Diego, USA.
- A. Aitio, World Health Organization, Geneva, Switzerland.
- G. Andrews, University of New South Wales, School of Psychiatry, Sydney, Australia.
- T. Armstrong, World Health Organization, Geneva, Switzerland.
- S. Ault, Pan American Health Organization, Brasilia, Brazil.
- L. Ayuso-Mateos, Hospital Universitario de la Princesa, Madrid, Spain.
- G.A. Baker, Clinical Science Centre for Research and Education, Liverpool, UK.
- K. Balakrishan, Sri Ramachandra Medical College and Research Institute, Chennai, India.
- J. Bartram, World Health Organization, Geneva, Switzerland.
- R. Beaglehole, World Health Organization, Geneva, Switzerland.
- J. Bertolote, World Health Organization, Geneva, Switzerland.
- L. Blanc, World Health Organization, Geneva, Switzerland.
- X. Bonnefoy, WHO European Centre for Environment and Health, Bonn, Germany.
- R. Bos, World Health Organization, Geneva, Switzerland.
- C.M. Branche, Centers for Disease Control and Prevention, Atlanta, GA, USA.
- C. Brewster, International Life Saving Federation, San Diego, CA, USA.
- N. Broutet, World Health Organization, Geneva, Switzerland.
- R.C. Brownson, Saint Louis University School of Public Health, St. Louis, MO, USA.
- N. Bruce, University of Liverpool, Liverpool, UK.
- R. Butchart, World Health Organization, Geneva, Switzerland.
- D. Campbell-Lendrum, World Health Organization, Geneva, Switzerland.
- J.M. Colford, University of California, Berkeley, CA USA.
- A. Correa, Centers for Disease Control and Prevention, Atlanta, GA, USA.
- J.R. Coura, Instituto Oswaldo Cruz, Rio de Janeiro, Brazil.
- C.L. Craig, Canadian Fitness and Lifestyle Research Institute, Ottawa, Canada.
- B. Cugier, Federal Institute for Occupational Safety and Health, Berlin, Germany.
- R. Dales, University of Ottawa, The Ottawa Hospital, Ottawa, Canada.
- I. de Bourdeaudhuij, Ghent University, Ghent, Belgium.
- M. de Onis, World Health Organization, Geneva, Switzerland.
- C. Dora, World Health Organization, Geneva, Switzerland.
- T. Farley, World Health Organization, Geneva, Switzerland.
- D. Farrington, University of Cambridge, Cambridge, UK.
- A.O. Filho, Universidade Federal do Rio De Janeiro, Rio de Janeiro, Brazil.
- E. Fondjo, Organisation de Coordination pour la lutte contre les Endémies en Afrique, Yaoundé, Cameroun.
- S. Forjuoh, Health Science Center, Texas A and M University, Temple, TX, USA.
- B. Gesch, University of Oxford, University Laboratory of Physiology, Oxford, UK.
- B. Giles-Corti, University of Western Australia, Crawley, Australia.
- O. Girardin, Centre Suisse de Recherches Scientifiques en Côte d'Ivoire (CSRS), Abidjan, Côte d'Ivoire.
- S.L. Hinde, Australian National University, Canberra, Australia.
- R. Hughes, University of Queensland, Brisbane, Australia.
- R. Jenkins, Institute of Psychiatry, King's College, London, UK.
- B.H. Kay, Royal Brisbane Hospital, Brisbane, Australia.
- D. Kay, University of Wales, Aberystwyth, UK.
- J. Keiser, Swiss Tropical Institute, Basel, Switzerland.
- R. Kessler, Harvard Medical School, Boston, MA, USA.
- N. Khaltsev, World Health Organization, Geneva, Switzerland.
- G. Killeen, Ifakara Health Research and Development Centre, Ifakara, Tanzania.
- T. Kjellstrom, Australian National University, Canberra, Australia.
- O. Kobusingye, WHO Regional Office for Africa, Brazzaville, Congo.
- M. Kramer, McGill University, Faculty of Medicine, Montreal, Canada.
- F. Laihad, Ministry of Health, Jakarta, Indonesia.
- P. Landsbergis, Mount Sinai Medical Center, New York, NY, USA.
- D. Lavanchy, World Health Organization, Geneva, Switzerland.
- A. Leenars, Ontario, Canada.
- Y. Li, School of Public Health, Fudan University, Shanghai, China.
- F. Liebers, Federal Institute for Occupational Safety and Health, Berlin, Germany.
- S.W. Lindsay, University of Durham, Durham, UK.

- A. Luttmann, Institute for Occupational Physiology at the University of Dortmund, Germany.
- R. Lucas, Australian National University, Canberra, Australia.
- S. Mendis, World Health Organization, Geneva, Switzerland.
- M. Merialdi, World Health Organization, Geneva, Switzerland.
- A. Mnzava, WHO Regional Office for the Eastern Mediterranean, Cairo, Egypt.
- D. Mohan, Indian Institute of Technology, New Delhi, India.
- A.J. McMichael, Australian National University, Canberra, Australia.
- J. Mercy, Centers for Disease Control, Atlanta, GA, USA.
- A.B. Miller, Toronto, Canada.
- D. Molyneux, Liverpool School of Hygiene and Tropical Medicine, Liverpool, UK.
- M. Nathan, World Health Organization, Geneva, Switzerland.
- F. Ndowa, World Health Organization, Geneva, Switzerland.
- L. Onyon, World Health Organization, Geneva, Switzerland.
- Y. Rubio-Palis, Instituto de Altos Estudios de Salud Pública "Dr. Arnoldo Gabaldon", Maracay, Venezuela.
- K. Palmer, WHO Western Pacific Regional Office, Manila, Philippines.
- L.R. Panganiban, University of the Philippines, Manila, Philippines.
- R. Pararajasegaram, World Health Organization, Geneva, Switzerland.
- P. Pisani, WHO International Agency for Research on Cancer (IARC), Lyon, France.
- G.P. Pokharel, World Health Organization, Geneva, Switzerland.
- V. Poznyak, World Health Organization, Geneva, Switzerland.
- A. Prata, Faculdade de Medicina do Trifngulo, Mineiro, Brazil.
- J. Pronczuk, World Health Organization, Geneva, Switzerland.
- F. Racioppi, WHO European Centre for Environment and Health, Rome, Italy.
- F. Rahman, Institute of Child and Mother Health, Dhaka, Bangladesh.
- E. Robert, Institut Européen des Génomutations, Lyon, France.
- W.H.J. Rogmans, Consumer Safety Institute, Amsterdam, The Netherlands.
- I. Romieu, Instituto Nacional de Salud Publica, Cuernavaca Morelos, Mexico.
- H. Rutter, Government Office for the South East, Guildford, UK.
- H.P.S. Sachdev, Vasant Vihar, New Dehli, India.
- G. Schmid, World Health Organization, Geneva, Switzerland.
- V.P. Sharma, Malaria Research Centre, Delhi, India.
- G.M. Shaw, March of Dimes Birth Defects Foundation, Berkeley, CA, USA.
- A.C. Silveira, Pan American Health Organization, Brasilia, Brazil.
- K. Smith, University of California, Berkeley, CA, USA.
- A. Spielmann, Harvard School of Public Health, Boston, MA, USA.
- K. Steenland, Rollins School of Public Health, Emory University, Atlanta, GA, USA.
- K. Straif, WHO International Agency for Research on Cancer (IARC), Lyon, France.
- D. Sutherland, World Health Organization, Geneva, Switzerland.
- S. Tarlo, University of Toronto, The Toronto Western Hospital, Toronto, Canada.
- H. Taylor, University of Melbourne, Melbourne, Australia.
- W.A. Temple, University of Otago, Dunedin, New Zealand.
- J. Tempowski, World Health Organization, Geneva, Switzerland.
- T. To, University of Toronto, Hospital for Sick Children, Toronto, Canada.
- Y. Touré, World Health Organization, Geneva, Switzerland.
- T. Ukety, World Health Organization, Geneva, Switzerland.
- B. Üstün, World Health Organization, Geneva, Switzerland.
- J. Utzinger, Swiss Tropical Institute, Basel, Switzerland
- P. Van Damme, University of Antwerp, Antwerp, Belgium.
- G. Viegli, University of Pisa, Pisa, Italy.
- M. Weber, World Health Organization, Geneva, Switzerland.
- WHO/UNAIDS Working Group on Global HIV/AIDS and STI Surveillance, Geneva, Switzerland.
- S. Wiersma, World Health Organization, Geneva, Switzerland.
- B. Williams, World Health Organization, Geneva, Switzerland.
- A. Wolf, Harvard Medical School, Boston, MA, USA.
- T. Woodruff, Environmental Protection Agency, San Francisco, CA, USA.
- A. Woodward, University of Auckland, Auckland, New Zealand.
- A. Wooler, Royal National Lifeboat Institution, Saltash, UK.
- D. Zalk, International Occupational Hygiene Association, Derby, UK.
- A. Zanetti, University of Milan, Milan, Italy.
- J. Zupan, World Health Organization, Geneva, Switzerland.

In addition, we would like to express our thanks to: Alan Hubbard, University of California, Berkeley, CA, USA; Jürgen Rehm, Centre for Addiction and Mental Health, Toronto, Canada; Colin Mathers, Ian Scott, and Fiona Gore World Health Organization, Geneva, Switzerland.

CREDITS

Cover photo credits:

From top, left to right:

Chest exam/WHO/TBP/Davenport

Women in Cape Verde. Photo credit: Wolfgang Schmidt/Still Pictures

Children at beach, Phillipines/Jorgen Schytte/Still Pictures

Cyclists at rush hour, Shanghai/Julio Etchart/Still Pictures

Teens in Miami, USA. Photo credit: © Jeff Greenberg/Peter Arnold, Inc.

Background photos:

pgs. 6-7: Air pollution exposure in China. Photo credit: UNEP/Shihua Zhao/TopFoto.co.uk

p.17: Children at the beach in the Phillipines. Photo credit: Jorgen Schytte/Still Pictures

p.18: Children in forest in Thailand. Photo credit: Pramkaew/UNEP/Still Pictures

p. 20: Toxic smoke from a fire at a European chemical plant fills the sky while a farmer tends his fields. Photo credit: Z. Jovanovic/UNEP/Still pictures

p. 24: Metal worker in San Jose, Costa Rica. Photo Credit: Mark Edwards/Still Pictures

p. 26: Sand dune threatens to engulf desert oasis in the Sahel region of Africa. Photo credit: Voltchev/UNEP/Still Pictures

p. 32: Woman collects water from a river in Bengladesh. Photo Credit: Mark Edwards/Still Pictures

p. 57: Women transplant rice in an irrigated paddyfield in India. Photo credit: Joerg Boethling/Still Pictures

p. 58 : Desert Landscape, China. Photo credit: W. WU/UNEP

p. 63: Women and girls carry water from the village pump in Mozambique. Photo credit: Jorgen Schytte/Still Pictures

p. 64: Street life in Copenhagen, Denmark. Photo Credit: Jorgen Schytte/Still Pictures

p. 71: Pumping water from a well covered to protect from debris, Mozambique. Photo credit: Jorgen Schytte/Still Pictures

p. 72: Tour of waste water treatment plant, Donner Lake, California. Ray Pfortner/Peter Arnold, Inc.

p. 74: Children play in water in the Phillipines. Henning Christoph/Still Pictures

p. 81: Children play arcade games. Mark Edwards/Still Pictures

Graphic design :  www.paprika-annecy.com

Communications: Nada Osseiran

Editorial production: Elaine Fletcher

Text editing: Kevin Farrell

Administrative support: Eileen Brown and Eileen Tawffik

How much disease could be prevented through better management of our environment? The environment influences our health in many ways – through exposures to physical, chemical and biological risk factors, and through related changes in our behaviour in response to those factors. To answer this question, the available scientific evidence was summarized and more than 100 experts were consulted for their estimates of how much environmental risk factors contribute to the disease burden of 85 diseases. This report summarizes the results globally, by 14 regions worldwide, and separately for children.

The evidence shows that environmental risk factors play a role in more than 80% of the diseases regularly reported by the World Health Organization. Globally, nearly one quarter of all deaths and of the total disease burden can be attributed to the environment. In children, however, environmental risk factors can account for slightly more than one-third of the disease burden. These findings have important policy implications, because the environmental risk factors that were studied largely can be modified by established, cost-effective interventions. The interventions promote equity by benefiting everyone in the society, while addressing the needs of those most at risk.

ISBN 92 4 159382 2



9 789241 593823