World Health Report 2002 - Reducing Risks, Promoting Healthy Life

Methods Summaries for Risk Factors assessed in Chapter 4


Childhood and maternal under-nutrition

Underweight

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Weight-for-age was chosen as the index of child nutritional status for this analysis because it is the indicator most commonly used in developing countries, allowing for the inclusion of the largest number of studies. Although it does not distinguish between wasting and stunting, weight-for-age represents a combination of both aspects and has a high positive predictive value as an indicator for undernutrition in developing countries (WHO, 1995) The WHO Global Database on Child Growth and Malnutrition uses -2 standard deviations below the NCHS/WHO reference median as a cut-off point for underweight (WHO, 1997). The prevalence of underweight status among children 0-4 years of age was provided by the WHO Global Database on Child Growth and Malnutrition.

For estimates of mortality, Pelletier et al (1994) developed an approach to estimating these relations for all-cause mortality using published data. This analysis applies this approach to data
from ten cohort studies in which both weight-for-age category (< -3 SD, -3 to < -2 SD; -2 to < -1 SD; and > -1 SD) and cause of death information were obtained. All studies contributed information on weight-for-age and risk of diarrhoea, pneumonia and all-cause mortality, 6 studies also contributed information on deaths due to measles, and three studies also contributed information on deaths due to malaria or in some cases presumptive malaria as indicated by fever. By calculating the natural logarithm of the mortality rates by cause and anthropometric status in each country, and utilising weighted random effects models, the relation between weight-for-age and risk of dying was estimated. For the four causes of death, the relative risk for mild underweight (-2SD to <-1SD) ranged from 1.73 to 2.32. For children who were -3SD to < -2SD, the relative risks were 3.01 to 5.39 and for children < -3SD they were 5.22 to 12.50. Combining this information with data on the prevalence of each anthropometric category among children 0-4 years of age in each WHO region, allowed estimation of the burden of deaths by cause attributable to low-weight for age.

For estimates of morbidity, published literature were reviewed and meta-analyses conducted to examine the relationship between underweight status and risk of morbidity due to measles, malaria, pneumonia, and diarrhoea. The role of underweight status in pregnancy outcomes (neonatal/perinatal mortality, premature birth, low birth weight and congenital anomaly) and cognitive function was also reviewed. This analysis found that underweight status among pre-school-aged children was significantly associated with subsequent risk of morbidity from diarrhoea and pneumonia episodes, but not clinical malaria. There was no evidence that underweight status influenced susceptibility to measles infection. Underweight status appears to increase the risk of cognitive impairment and certain adverse pregnancy events, although summary risk estimates could not be calculated. Unlike the approach used for mortality, the morbidity estimates were a dichotomous comparison only (< -2 SD versus > -2 SD). Data on mild underweight status (-2 SD to –1 SD) was reviewed, and there was no relationship found for diarrhea morbidity, and inadequate data to evaluate other infections. The minimum risk distribution for weight-for-age among children was taken as the WHO Amr A distribution, for which 13.6% of children 0-4 years of age are in the category of -1 to -2 standard deviations and 2.3% are in the < -2 standard deviations group.
Iron deficiency

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Anaemia has been used as the hallmark of iron deficiency severe enough to affect tissue functions, and anaemia prevalence data were the only source of global prevalence data to estimate iron deficiency anaemia. Because of the limitations of available data, the current analysis estimates the risk associated with low haemoglobin, using haemoglobin as a continuous variable. However, iron deficiency is not the sole cause of anaemia in most populations and even within individuals anaemia may be caused by multiple factors. Based on evidence from iron supplementation trials, it was estimated that, on average, 50% of anaemia globally is caused by iron deficiency. Thus, the counterfactual scenario was a shift in the haemoglobin distribution sufficient to reduce the prevalence of anaemia by one half.

Comprehensive reviews were conducted of published literature linking iron deficiency to disability and death for four potential outcomes: child mortality, perinatal mortality, maternal mortality, and mild mental retardation. For all of these outcomes the best available data were prospective observational studies in which anaemia or haemoglobin concentration was the risk factor. Data on child mortality were not adequate for this task, although a true risk cannot be precluded. Summary relative risks for perinatal mortality, maternal mortality and mental retardation were estimated. Global anaemia prevalence data were obtained from the 1996 WHO Global Burden of Disease project, and converted to mean haemoglobin concentrations, assuming normal distribution and the mean of observed standard deviations from a large number of studies. To estimate the haemoglobin distribution if iron deficiency were corrected, it was assumed that the prevalence of anaemia would be reduced by 50% all age and sex groups. Using this theoretical minimum scenario for iron deficiency anemia, the average increase in haemoglobin concentration in world regions ranged from 0.1 to 1.3 g/dL in young children, and from 0.1 to 1.2 g/dL in adult women, with the greatest changes occurring in SEARO-D and EMRO-B and D.
**Vitamin A deficiency**

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Prevalence estimates of vitamin A deficiency among 0 – 4 year-old children and among 15 – 44 year-old pregnant women were calculated (based on low serum retinal concentration < 0.70 µmol/L) for each WHO region utilising data from a recent review. This review provided the first global set of comprehensive estimates for vitamin A deficiency, and was based on data from a variety of different data sources including WHO reports, a report published by the Micronutrient Initiative (MI/UNICEF/Tulane University 1998), published and unpublished papers and reports. The prevalence of vitamin A deficiency for the countries not included in that review was assumed to be zero. Disease endpoints included measles mortality, diarrhea mortality, malaria incidence and the risk of malaria mortality for children, and all-cause maternal mortality.

Data on the risk associations were drawn from a variety of randomized controlled intervention trials and prospective observational data. In children, the intervention trial data were used to estimate the risks associated with vitamin A deficiency by defining no receipt of vitamin A as the risk factor and calculating the inverse of the observed protective effect. The relative risk of all-cause maternal mortality was estimated from prospective observational data where night blindness during pregnancy was related to mortality during pregnancy and up to 6 weeks postpartum in Nepal. The burden of disease due to direct sequelae of vitamin A deficiency was from the GBD estimates.

**Zinc deficiency**

Laura E Caulfield (Johns Hopkins University), Robert E Black (Johns Hopkins University)

A systematic literature search was conducted to identify relevant studies investigating the role of zinc in human health. This search was limited to the results of randomized controlled trials conducted among paediatric populations in developing countries, and aimed to estimate the effects of zinc deficiency on the incidence of diarrhoea, pneumonia and malaria illness among children 0–4 years of age. The studies included were randomized trials of zinc supplementation.
for the prevention of diarrhoea, pneumonia, or malaria. In addition, there are numerous trials in which zinc supplements are evaluated as therapeutic agents, but these were not included. In all, nine studies contributed findings on zinc deficiency and risk of diarrhoea, five contributed findings on risk of pneumonia, and three contributed findings on risk of malaria. Results of the review indicated that zinc deficiency in young children increases their risk of diarrhoeal disease by 1.28 (95% CI 1.10–1.50), of pneumonia by 1.52 (95% CI 1.20–1.89) and of malaria by 1.56 (95% CI 1.29–1.89). These results were extended as best estimates of zinc deficiency affecting the risk of dying by these causes.

There are limited direct data on zinc deficiency in paediatric and other populations; however, the International Zinc Nutrition Consultative Group (IZiNCG) has developed a method for estimating the prevalence of inadequate zinc intakes based on the presence and bioavailability of zinc in each country’s food supply. Following this technique, the global prevalence of zinc deficiency was estimated as 31%, ranging from 6–73% across WHO mortality subregions. Evidence relating zinc deficiency to health outcomes in children more than five years of age and adult men and women is not available.

Other diet-related risk factors and physical activity

High blood pressure

Carlene Lawes (University of Auckland), Stephen Vander Hoorn (University of Auckland), Malcolm Law (St Bartholomew's and The Royal London School of Medicine), Paul Elliott (Imperial College School of Medicine), Stephen MacMahon (University of Sydney), Anthony Rodgers (University of Auckland)

Blood pressure will be defined as systolic blood pressure (SBP) in mmHg. SBP was chosen principally because data are widely available for this index of blood pressure and it appears to be at least as good a predictor of cardiovascular disease as other indices. Based on consistent direct, continuous associations in cohort studies and evidence from randomised clinical trials, the outcomes assessed were stroke, ischaemic heart disease,
hypertensive heart disease and other cardiac disease. Similar data suggested causal relationships with renal failure, but this could not be mapped to a Global Burden of Disease outcome.

Raw SBP data were obtained from studies after a systematic review of population-based surveys, and included data from about 230 surveys and over 650,000 participants. Sex-specific associations of SBP with age were estimated for each of the GBD regions separately based on country-weighted study estimates of mean values. There was moderate variation in the final age and sex specific estimates of mean blood pressure across the 14 regions, with the range between the highest and lowest age-specific mean systolic blood pressure levels across regions typically being about 20mmHg. Confidence intervals around mean SBP and standard deviations were calculated to reflect uncertainty. Trends in mean SBP over time were extrapolated from published epidemiological data.

A theoretical minimum distribution of SBP (i.e. one which would yield the lowest population risk of adverse health outcomes) was estimated at 115 SD 6mmHg (usual SBP) for all age, sex and region groups. The main basis for this estimate is the level of SBP down to which epidemiological relationships with cardiovascular disease outcomes are observed. This theoretical minimum is also consistent with the levels of SBP in populations with little or no cardiovascular disease. Furthermore, recent clinical trial data indicate reductions in stroke after lowering blood pressure by about 10mmHg SBP in those with mean SBP 125 mmHg.

Data on risk accumulation were obtained from the Asia Pacific Cohort Studies, an individual participant meta-analysis that combined data from 37 prospective observational cohorts involving over 425,000 individuals with 2-27 years of follow-up (mean 7 years), and in total over 3 million person-years of observation. Data from this meta-analysis were complemented by data from other overviews and large cohort studies. The main findings were direct, positive and continuous associations of usual SBP with the risks of all endpoints of interest. The risks were similar by sex and region, except there was possibly a stronger association of blood pressure with stroke in Asian compared to non-Asian populations, due partly to the higher proportion of haemorrhagic strokes. Overall, each 10mmHg lower usual SBP was associated with 37% (95% CI 35-38%) lower stroke risk and a 25% (95% CI 23-27%) lower risk of IHD. Each 10mmHg lower usual SBP was associated with 46% (95% CI 40-51%) and 18% (95% CI 15-20%) lower
risks of hypertensive disease and other cardiac disease. (Few data were available for the GBD endpoint ‘other cardiac disease’ and so given some uncertainty about causality and the varying composition for this endpoint around the world, the log of the relative risks were halved for this outcome.) There was attenuation of proportional associations with age for all these outcomes. 

Data on risk reversibility comes from meta-analyses of randomised controlled trials of blood pressure lowering. In total 23 trials were reviewed which included over 71,000 participants allocated a variety of blood pressure lowering agents or placebo. The mean overall SBP reduction was 10mmHg, and a total of 2,632 strokes and 3,693 IHD events were observed. Overall the trials confirmed the size of the reductions expected from epidemiological relationships in middle age. However the reductions in risk in old age were larger than expected from the epidemiology.

Observational epidemiological data were employed for risk accumulation and trial overviews for the time frame of risk reversibility. For example, in middle-age the epidemiology suggests a 10mmHg increase in SBP is associated with about 40% more stroke and 25% more IHD. The trials suggest that within 3-5 years of lowering SBP by 10mmHg most or all of this increased risk for stroke and hypertensive disease is reversed and approximately two thirds for IHD and other cardiovascular disease.

**High cholesterol**

Carlene Lawes (University of Auckland), Stephen Vander Hoorn (University of Auckland), Malcolm Law (St Bartholomew's and The Royal London School of Medicine), Stephen MacMahon (University of Sydney), Anthony Rodgers (University of Auckland)

Cholesterol was defined as total cholesterol in mmol/l, a continuous variable with mean and standard deviation (1 mmo/l =38.7 mg/dL). While there are different aetiological roles for
cholesterol subfractions, such as high and low density lipoprotein, the large majority of descriptive and epidemiological data are available only for total cholesterol levels.

Primary outcomes assessed were ischaemic heart disease and nonfatal stroke. Ischaemic heart disease was chosen on the basis of clear and consistent positive associations observed in cohort studies and evidence of reversibility in clinical trials of cholesterol lowering treatments. Cholesterol is positively associated with ischaemic stroke, but has a qualitatively different association with haemorrhagic stroke. As endpoints were mapped to the GBD classification system for disease, total stroke was used in analyses. However, adjustments to relative risks were made according to estimates of major stroke subtypes by age, sex and region. Cholesterol has been observed to be inversely associated with a number of other outcomes such as cancer and chronic respiratory disease. However, there is evidence that these associations are due to the effects of disease on cholesterol, rather than vice versa. Consequently these outcomes were not included.

Raw cholesterol data were obtained from studies after a systematic review of population-based surveys, and included data from about 160 surveys and almost 640,000 participants. Sex-specific associations of cholesterol with age were estimated for each of the GBD regions separately based on country-weighted study estimates of mean values. There was moderate variation in the final age and sex specific estimates of mean cholesterol across the 14 regions, with the range between the highest and lowest age-specific mean cholesterol levels across regions typically being about 2mmol/l.

A theoretical minimum distribution of cholesterol (i.e. one which would yield the lowest population risk of adverse health outcomes) was taken as 3.8 SD 1.0 mmol/l for all age, sex and region groups. The main basis for this estimate is the level of cholesterol down to which epidemiological relationships with cardiovascular disease outcomes are observed, and clinical trial data showing benefits from cholesterol lowering among those with below average cholesterol levels. This theoretical minimum is also consistent with the levels of cholesterol in populations with little or no cardiovascular disease.
Data on risk accumulation for ischaemic heart disease were included from an overview of the ten largest observational studies conducted in Western populations. This overview included data from 494,804 participants, followed for 7-23 years among whom 18,811 IHD events were observed. There was evidence of differences in the strength of the association by age but no difference between males and females. These data are closely similar in size and shape to the associations seen in an overview of 29 cohorts involving 353,065 participants from the Asia Pacific region. In this individual participant meta-analysis, 2,937 strokes were observed as well as 2,838 IHD events. Overall, each 0.6 mmol/l lower usual cholesterol was associated a 27% reduction in IHD. A 1 mmol/l lower cholesterol was associated with a 13% (6-19%) reduction in total stroke, predominantly due to the effect on ischaemic stroke.

An overview of 49 trials of cholesterol lowering indicated that risk reductions in ischaemic heart disease of 5%, 13%, 23% and 25% are achieved by 0-1, 1-2, 2-5 and >5 years respectively. After taking into account the size of the reduction in cholesterol, there was no clear difference in effects according to how the cholesterol reduction was achieved, including by diet, or one of several classes of drugs. This data indicate that in middle age, the risks associated with high cholesterol are reversed within a few years. An overview of cholesterol lowering trials also confirms a reduction in stroke.

For these analyses, observational epidemiological data were used for risk accumulation and trial overview data for the time frame of risk reversibility.

**High body mass index**

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Body mass index (BMI) was chosen as a simple measurement of body weight in relation to height. WHO criteria for both underweight and overweight in adults are set out using this index. Increases in BMI involve increments in both body fat and lean tissues, but the relationships of body weight to health are conventionally expressed in terms of BMI rather than body fat. Recently the same convention has been applied to children although the index is a less robust measure of body proportions in school aged children and adolescents. The relationship of body fat to BMI changes with age and current analyses suggest different proportions of body fat at equivalent BMIs in different ethnic groups.

It is widely acknowledged that there is an amplified risk of disease associated with increased weight particularly if the body fat is deposited within the abdomen, indicated by a high waist measurement. However there are insufficient waist measurement data available at this stage to allow a comprehensive analysis of sub-regional data on body fat distributions based on waist measurements and disease relationships.

There are many data sets with measurements of a population’s weights and heights, often obtained as part of general medical or economic surveys, but these are rarely published. Nevertheless several Ministries of Health have produced their own analyses in the formats requested and these are included where available. Where ministry data sets or published representative information is lacking, the present assessment has used earlier data published within each country. Often, however, no data are available so in deriving estimates of BMIs for the different age groups in each sub-region, there has been a requirement to extrapolate from data in other countries or regions. To avoid the use of misleading data, all information based on clinical or epidemiological studies of select groups within a population have been excluded. In addition, only those data obtained by actual measurement of heights and weights by trained observers have been included.

The disease outcomes relating to excessive weight were type 2 diabetes, the principal diseases of the cardiovascular system, i.e. ischaemic heart disease, strokes and hypertension, and osteoarthritis. Some cancers, i.e. post-menopausal breast, colon, endometrial and kidney cancers are also included as they are in part dependent on the rates of overweight and obesity
within a population. These relationships of BMI to disease appear to be causal in that weight loss in overweight and obese individuals reduces the risk of developing type 2 diabetes, but also leads to the remission of type 2 diabetes. Modest reductions in weight also lead to falls in blood pressure with greater effects in those with hypertension. Similarly, prospective studies now suggest that intentional weight loss leads to reductions in cardiovascular events and death rates. The problems of osteoarthritis in terms of its medical complications and therapeutic needs may also be reduced with weight loss. Preliminary evidence suggests that early intentional and maintained weight loss may reduce the risk at a later stage of developing some cancers, e.g. colon cancers.

Disease relationships were evident with increases in adult BMIs above 21 so the analyses of the burden of diseases linked to excessive BMIs were calculated from BMIs of 21. The analyses based on this continuous relationship therefore replaced the usual categorical analyses based on overweight and obesity rates in the different sub-regions. New analyses based on an individual participant data meta-analysis of 33 cohort studies within the Asia-Pacific were used to estimate the incremental risk associated with each BMI unit increase above BMI 21, by WHO age groups and sex. These relationships were consistent in size and shape with those published from North American and European cohorts. The relationship between BMI and the risk of type 2 diabetes was derived from unpublished and published data providing measured anthropometry and fasting blood sugar measurements, extracted from nationally representative studies, which allowed the required distinct age band relationships to be discriminated. Equivalent increments in the risks of co-morbidities associated with weight gain are assumed for all parts of the world.

**Low fruit and vegetable intake**

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The effect of eating fruit and vegetables in protecting against ischaemic heart disease, stroke, and lung, gastric, oesophageal, and colorectal cancers was estimated based on the results of a
systematic literature review and meta-analysis. Due to the limited information available on regional differences in risks, constant risk estimates were applied to all regions.

Risks were assessed compared to a theoretical ‘maximum’ intake of fruit and vegetable intake that would yield the lowest population risk of adverse health outcomes, of 600g/day in adults (equivalent to the highest national estimate of intake in countries where data on fruit and vegetable – disease relationships are available), 480 g/day in children aged 5-14 years, and 330 g/day in children aged 0-4 years.

The six outcomes assessed were ischaemic heart disease, stroke, and lung, gastric, oesophageal, and colorectal cancers. Systematic review and meta-analysis of published studies provided estimates of hazard size. Nationally representative individual level intake data on fruit and vegetable consumption by age and sex are uncommon. Fruit and vegetable intake data were obtained for 26 countries from 9 WHO regions from. The sample sizes ranged from 1,000-22,000. For 5 regions, no survey data were obtained. In these cases, data were combined on regional fruit and vegetable availability from FAO Food Balance Sheets with available survey information on age and sex distribution of intake from other regions.

Physical inactivity

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Physical inactivity was treated as a trichotomous variable. Level I exposure (‘inactive’) was defined as ‘doing no or very little physical activity at work, at home, for transport or in discretionary (leisure)-time’. Level 2 exposure (‘insufficiently active’) was 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.’ Level III (active) reflected levels of participation greater than Level
II. The definition of this risk factor is consistent with the focus of contemporary public health efforts.

The inclusion of activity across multiple domains is different from most, but not all, previous definitions. Multiple domains were included to better reflect the total levels of activity undertaken in diverse populations around the world. Three levels of activity were selected to avoid limiting the assessment of burden to only that associated with the highest risk, namely the most inactive (a dichotomous approach). However, unlike some other risk factors a more detailed continuous variable approach was not possible due to the lack of data. Furthermore, the lack of data prevented us from estimating a fourth category of ‘high level of activity.’ The consequence of limiting exposure to three levels is that the final estimates of burden are likely to underestimate the total attributable burden to inactivity.

Estimates of activity for all countries were sought via a comprehensive literature search and contact with key agencies and known researchers. Most data were available for discretionary-time activity, some data were found on occupational activity and little and no national data were available for transport- and domestic-related activity, respectively. For countries with no or missing data, statistical modeling techniques were used to predict domain specific estimates of levels of activity. Due to lack of data no attempt was made to predict activity around the home. Available data (where possible) and predicted estimates were used to calculate the level of ‘inactive’ and ‘insufficiently active’ for 145 countries and these data were aggregated and weighted to create estimates for 14 WHO epidemiological regions.

There is a large body of evidence supporting the independent, beneficial effects of regular physical activity. A review of the evidence on causality and the requirement for a GBD classification code led to the inclusion of 5 diseases; ischaemic heart disease, ischaemic stoke, breast cancer, colon cancer and type 2 diabetes mellitus. Although there is emerging consensus on the protective effects of physical activity in regards to preventing falls, osteoporosis and impaired mental health these disease endpoints did not meet inclusion criteria.
A search of literature from 1980 onwards revealed several quantitative and qualitative reviews of the association between physical inactivity and ischaemic heart disease (IHD) and stroke but no quantitative meta-analyses for breast cancer, colon cancer and type 2 diabetes. Given no available summary estimate of association for 3 endpoints, the differences between previous work on IHD and stroke, the focus of this work on physical activity not fitness and the use of a different referent group, a set of new meta-analyses for each health outcome were conducted.

Only data from studies using an outcome measure of physical activity were included, studies with measures of physical fitness were excluded. To address concerns regarding measurement error associated with physical activity, an adjustment factor was incorporated based on remeasurement studies. Log relative risks estimates were combined using an inverse-variance weighting scheme to derive summary estimates of relative risk for Level 1 (inactive) and Level 2 exposure (insufficiently active) for each health outcome. Attenuation for age categories of 70 years and above was incorporated to reflect the known decrease in relative risk in older age groups. These final age sex estimates of relative risk were applied uniformly across the 14 WHO epidemiological regions.

This work provides new estimates of the magnitude of risk associated with inactivity. The estimates of risk for ischemic heart disease are consistent with previous research given the differences in the study inclusion criteria and our treatment of the data. Although there were less data by which to compare our results on ischemic stroke, type 2 diabetes mellitus and breast and colon cancers, our estimates are similar to findings from recent qualitative reviews both in direction and magnitude. The heterogeneity across studies was notable and is a limitation to conducting meta-analyses in this field.

The final estimates of burden (deaths and DALY’s) attributable to physical inactivity are likely to underestimate the true magnitude for several reasons. First, estimating the current prevalence of inactivity by using only two levels of exposure (inactive and insufficiently active) the burden attributable to each increment of exposure is not counted nor is the risk associated with not doing high levels of total activity or high levels of vigorous-intensity activity. The estimates of risk included an adjustment for measurement error but some residual measurement error may remain.
The estimates of total burden are also restricted to 5 disease outcomes: ischemic heart disease, ischemic stroke, breast cancer, colon cancer and type 2 diabetes mellitus. Thus, the burden associated with other diseases remains unaccounted. Collectively these features explain the conservative magnitude of attributable burden. Future efforts would benefit from improved measures of exposure, and better quality and quantity of data on: prevalence; magnitude of association for omitted diseases endpoints; risk reversibility to improve estimates of current burden; and trend data to improve estimates of future burden attributable to inactive lifestyles.

**Sexual and reproductive health**

**Unsafe sex**

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Exposure to unsafe sex was theoretically defined as ‘vaginal sexual intercourse where one partner is infected with a sexually transmitted infection (STI) and the other is not’. This cannot be measured directly so a working definition of ‘risky sex’ was used to enable approximate estimates of conditional exposure to be calculated from available survey data. Risky sex is defined as ‘the proportion of people who, in the last year, had sex with a partner that they did not live with and who did not use a condom on the last occasion’. This definition was chosen as it is meaningful in all regions. However empirical data on the relationship between specific sexual behaviours and contracting an STI are scarce and cannot be generalised beyond the population in which they were estimated. Therefore it is not known how closely the proportion of people having risky sex corresponds to the proportion who are having unsafe sex in the different regions, or even in different age and sex subgroups.

To estimate the prevalence of risky sex nationally representative surveys were identified and the proportion having risky sex was calculated. Surveys used included Demographic and Health Surveys from Macro International, Reproductive Health Surveys from the Centers for Disease
Control, Knowledge, Attitudes, Behaviours and Practice Surveys carried out by organisations such as Population Services International, WHO Global Programme on AIDS and national governments. Infection with HIV was considered separately from the other STI. Given the definition of unsafe sex all STI, except HIV, were assumed to be attributable to unsafe sex, and therefore avoidable. HIV is a special case because it is fatal and the epidemic is explosive in many regions.

A literature search was carried out to find reported sexual risk factors for HIV infection and estimates of the risk relating to each factor. However, because HIV is transmitted from person to person, the relative risk attached to a particular behaviour changes with the prevalence of infection, and changes in prevalence also affect incidence of new infections. This means the standard CRA methodology could not be applied. Instead two different approaches were used.

UNAIDS data on the mode of transmission were used to calculate the proportion of prevalent infections in each region which had been sexually acquired. This was taken as the proportion of infections attributable to unsafe sex.

To estimate the avoidable burden of infections in high HIV prevalence regions a mathematical projection model was used (the Epidemic Projection Package (EPP) developed by the UNAIDS Epidemiology Reference Group). In other regions these estimates were based on the proportion of HIV infections prevalent in 2000 which had been sexually acquired: it was assumed that this distribution would remain constant in the future unless the level of unsafe sex changed. It was further assumed that any reduction in unsafe sex would lead to a directly proportional reduction in new HIV infections, and the number of avoidable infections was calculated from this. This is a tenable assumption where HIV prevalence is relatively low because any error introduced by ignoring the feedback between changes in prevalence and incidence will be small.

**Lack of contraception**

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The burden of disease attributable to non-use of contraception and use of ineffective methods includes obstetric complications and abortion-related morbidity and mortality associated with unintended pregnancies (unwanted and mistimed). To estimate this burden, a model was used for linking data on contraceptive use and fertility preferences to unwanted births and unsafe abortions as intermediate outcomes, which are then related to the maternal disease burden.

The health outcomes considered are the conditions associated with unsafe abortion and unwanted births. The abortion-related conditions are a separate subcategory (GBD endpoint 47) and the risk of abortion-related consequences is directly proportional to the risk of an unsafe abortion. The obstetric conditions linked to unwanted births are maternal haemorrhage, maternal sepsis, hypertensive disorders of pregnancy, obstructed labour and other maternal conditions. The burden of these obstetric complications attributable to non-use of contraception is proportional to the percentage of unwanted births among all births.

Contraceptive use reduces the risk of unintended conception but does not altogether eliminate it, and failure rates are higher for traditional methods than for modern methods. The categorical variable 'contraceptive use' has three levels of exposure: non-use, use of traditional methods and use of modern methods. Non-users experience the highest conception rates. The modern method category is used as reference category for calculating the relative risk of having an abortion and an unwanted birth.

Not all conceptions lead to an ‘avoidable’ burden, since many pregnancies are desired. We calculate how many unintended pregnancies are expected in one year by first estimating the proportion of women who will become pregnant and combining this with the probability that the pregnancy will be unwanted or mistimed, based on current reproductive intentions. The proportion of women becoming pregnant is derived from contraceptive failure rates among modern and traditional method users and biological expectations of the number of conceptions among non-users. Within the non-users, conception rates are applied to the fecund women only, excluding those who are not exposed to pregnancy for biological or behavioural reasons. Abortion probabilities are applied to determine how many of the mistimed and unwanted
pregnancies will end as abortions and unwanted births. Unwanted pregnancies will contribute to both abortion-related burden and the obstetric burden of maternal complications. Mistimed pregnancies only contribute to the abortion-related burden since preventing mistimed births by use of more effective contraception will not avert -only delay- any associated obstetric burden.

As theoretical minimum exposure we have simulated the contraceptive distribution which would prevail if all women with a desire to either stop child bearing or postpone the next birth for at least another two years, adopt an effective modern method of contraception. All traditional method users and fecund non-users consist of women who want a birth in the next 2 years. At this theoretical minimum level, the relative risk of an unwanted birth and abortion becomes 0. Counterfactual levels of relative risk are calculated to take account of the changing distributions of fertility desires within each exposure category.

Regional levels of distribution of contraceptive use and the relative risk levels of abortions and unwanted births are derived by aggregating country estimates based on data from 58 Demographic and Health Surveys (DHSs). This source includes data on childbearing intentions and contraceptive use at the time of survey. Average method-specific and duration-specific failure rates were calculated from 18 DHS surveys countries with calendar data on contraceptive use. In each country, the method-duration-specific failure rates are combined with the method mix and data on duration of use of current methods. Abortion probabilities are derived from WHO estimates of incidence ratios (unsafe abortions per 100 live-births).

**Addictive substances**

**Tobacco**

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Comparable data on the prevalence of smoking are not widely available and often inaccurate, especially when age-specific data are required. More importantly, current prevalence of smoking is a poor proxy for cumulative exposure to the risks of smoking, which depend on several factors
including the age at which smoking began, duration of smoking, number of cigarettes smoked per day, and cigarette characteristics such as tar and nicotine content or the type of filter. To overcome this problem, the smoking impact ratio, SIR, is used as a marker for accumulated smoking risk (following the Peto-Lopez method). SIR is defined as population lung cancer mortality in excess of that of never-smokers, relative to excess lung cancer mortality levels for a known reference group of smokers. The reference population, which also provided estimates of hazard size, was from the American Cancer Society’s Cancer Prevention Study, Phase II (CPS-II).

Estimates of mortality and disease burden due to smoking were made for lung cancer, upper aerodigestive cancer, all other cancers, chronic obstructive pulmonary disease (COPD), other respiratory diseases, vascular diseases, and other medical causes. All diseases for which currently no plausible physio-biological causal mechanism is known were excluded from the analysis of the category “other medical causes”. No estimates were made for non-medical causes (injuries). For the analysis of morbidity, it was assumed that the effects of smoking on mortality are due to changes in incidence (versus case-fatality) and therefore the exposure-disease relations for mortality also applies to morbidity due to the same causes. Estimates were limited to ages 30 and above. Since there are no known significant health benefits from smoking, the theoretical minimum risk occurs in a population of life-long non-smokers. Relative risks were corrected for confounding and extrapolation to other regions using conservative fixed correction factors for groups of causes. Never-smoker lung cancer mortality rates were chosen based on the estimated use of coal in unvented stoves for domestic energy for populations in each Region. Quantitative analysis of uncertainty was conducted for 4 input parameters: population lung cancer mortality, non-smoker mortality rates, reference population smoker and nonsmoker lung cancer mortality, and relative risk estimates.

Alcohol

Jürgen Rehm (Addiction Research Institute and University of Toronto), Robin Room (Stockholm University), Maristela Monteiro (WHO), Gerhard Gmel (Swiss Institute for the Prevention of Alcohol and Other Drug Problems), Kathryn Graham (University of Western
A review of the epidemiological literature suggests that the health effects of alcohol can be broadly considered under three broad headings; diseases augmented by alcohol; injuries caused by alcohol and cardiovascular effects (either protective or harmful effects caused by binge drinking). Accordingly, the estimates were prepared taking into account volume of drinking, as measured by *average* per capita consumption, and *pattern* of drinking, as measured from survey data on drinks consumed on different drinking occasions. Estimates of average volume of drinking were made from published data on production, trade and sales, adjusted for estimates of illegally produced alcohol. These preliminary estimates were then further adjusted on the basis of survey data on alcohol consumption. This allowed the estimation of distribution of consumption across population groups, and particularly the proportion of abstainers. Data on patterns of drinking were systematically collected from over 90 countries on the basis of a standard questionnaire administered to key informants in countries. On the basis of these responses, a pattern score was calculated for each country ranging from 1 (least detrimental) to 4 (most detrimental). Survey data were used to distribute the population of each region by age and sex according to abstainers, and into 3 drinking categories according to average daily consumption, with different cutoffs for males (higher) and females.

Alcohol has been associated with effects on over 60 ICD categories. The evidence for each proposed outcome was systematically assessed for causality and only those disease outcomes where the evidence was sufficiently strong to suggest causality were retained in the analysis. Meta analyses were then conducted on the available data on risk relationships to estimate relative risks by average volume of consumption. For estimating the contribution of patterns of drinking to cardiovascular disease and injuries, multilevel hierarchical models were combined with cross sectional time series models. Sensitivity analyses suggests that this approach yielded more plausible results than pooled cross sectional time series models. These models were then used to estimate the effect of per capita consumption on cardiovascular disease at average drinking patterns for the world, and the estimated effects of different drinking patterns. In countries with the least detrimental pattern of drinking, alcohol consumption lowered cardiovascular risk by 17
to 18%. In populations with pattern 4 drinking, alcohol consumption had an overall negative effect on cardiovascular risk, and also for males in Pattern 3 populations.

The majority of alcohol attributed disease burden was estimated to occur from neuropsychiatric conditions (37%), primarily alcohol use disorder, and from unintentional injuries (30%). Another 12% was from intentional injuries, with injuries together accounting for 42% of total alcohol burden worldwide. Another 8% was due to alcohol causing cancer, (upper aero digestive system, breast, liver), and a further 9% from other noncommunicable diseases, primarily diabetes and liver cirrhosis. Overall, alcohol had a slight negative effect on CVD, with 4% of the global alcohol burden arising through this group of diseases.

Illicit drugs

Louisa Degenhardt (University of New South Wales), Wayne Hall (University of Queensland), Matthew Warner-Smith (University of New South Wales), Michael Lynskey (University of New South Wales)

Estimating mortality directly attributable to illicit drug use such as overdose death - the most tangible adverse heath effect of illicit drug use - is difficult because of variations in the quality and quantity of mortality data. As a result, it is necessary to make indirect estimates, involving estimates of the prevalence of illicit drug use. However, it is difficult to make even indirect estimates because the use of these drugs is illegal, stigmatised and hidden. Definitions of the variable of interest are difficult because of deficiencies in the data collected by countries on illicit drug use, and by disagreements over what constitutes “problematic” illicit drug use. The definition used here was long-term regular injecting use of opioids, amphetamines or cocaine.

Data on the prevalence of problematic illicit drug use were derived from a range of sources. A literature search was conducted of all studies that estimated the prevalence of problematic drug use. Other data sources included the United Nations Drug Control Program and European Monitoring Centre for Drugs and Drug Addiction. The existing sex ratios of drug use prevalence
were from developed countries. These ratios were adjusted based on the prevalence of other addictive substances (tobacco) for developing countries.

A search was also completed for cohort studies of drug users that had estimated mortality due to the individual causes of death (overdose, suicide, and trauma), and to all causes of death (updating previous systematic reviews). Data on the number of years of follow up were extracted from each study and a weighted average annual mortality rate was calculated for each of the four causes of death, and for their sum. An standardized mortality ratio (SMR) was also derived from previous estimates of the excess mortality from all causes attributable to illicit drugs. Direct estimates were made for some causes by applying an attributable fraction to estimates of total deaths for some causes. The median estimate of a range of estimates was used as the estimate for each WHO region. For HIV/AIDS, UNAIDS estimates for HIV incidence among drug users (based on prevalence surveys among high-risk groups) were used.

Available data on prevalence in countries with data were used to estimate the prevalence of problematic illicit drug use for WHO regions. These estimates were used in combination with pooled estimates of annual mortality to estimate the number of persons who died from each cause. The estimated all-cause mortality was derived by multiplying the rate of death among persons 15-54 years in countries for which this data was available by all-cause AF derived from the SMR.

**Risks in the environment**

**Unsafe water, sanitation, and hygiene**

Annette Prüss (WHO), David Kay (University of Wales), Lorna Fewtrell (University of Wales), Jamie Bartram (WHO)

For the most part studies examining the issue have been surveys, multi-country studies and intervention studies which have looked at changes in water supply, excreta disposal or hygiene practices and assessed the effects on diarrhoea morbidity or mortality rates (generally in young children), or case-control studies.
For the outcome infectious diarrhoea, typical exposure scenarios were defined according to water supply, sanitation infrastructure and more generally the level of faecal-oral pathogens in the environment. Exposure prevalence in terms of infrastructure was determined from the Global Water Supply and Sanitation Assessment 2000. This assessment is a synthesis of major international surveys and any national census reports presented by the countries and provides data for 89% of the global population. Considered parameters include access to improved water supply and to basic sanitation facilities. Furthermore, a low faecal-oral pathogen load in the environment was assumed if sanitation coverage exceeded 98%, which corresponds approximately to the situation in developed regions. Six risk scenarios were defined, with the lowest risk level corresponding to a situation where no disease transmission occurs through water, sanitation and hygiene. The lowest level was also chosen as the “counterfactual” or alternative exposure.

Relative risk estimates were based on reviews and large multi-country studies for high pathogen loads in the environment. The part of disease due to water, sanitation and hygiene in regions with low pathogen loads in the environment was based on a study analysing the pathogens involved in diarrhoeal diseases, supported by evidence from selected studies of high quality. The burdens from schistosomiasis, trachoma, ascariasis, trichuriasis and hookworm disease, which are totally due to this risk factor, were added to the resulting estimates for diarrhoea.

**Urban outdoor air pollution**

Ross Anderson (St George’s Hospital Medical School, University of London), Aaron Cohen (Health Effects Institute), Kersten Gutschmidt (WHO), Michal Krzyzanowski (WHO), Nino Künzli (University of Southern California), Bart Ostro (California Environmental Protection Agency), Kiran Dev Pandey (World Bank), Arden Pope (Brigham Young University), Isabelle Romieu (Instituto Nacional de Salud Publica, Mexico), Jonathan Samet (Johns Hopkins University), Kirk Smith (University of California, Berkeley)
The availability of measurements of outdoor concentrations of PM varies widely across the globe. In order to provide estimates for all 14 WHO regions, models developed by the World
Bank (http://www.worldbank.org/nipr/Atrium/mapping.html) were used to estimate annual average concentrations of inhalable particles (PM$_{10}$) for 3211 cities with populations greater than 100,000 and capital cities. The models estimate annual average levels from econometric data and the available PM measurements in residential areas. To allow the most appropriate epidemiologic studies to be used for burden estimation, the PM$_{10}$ estimates were converted to estimates of fine particles (PM$_{2.5}$) using available information on geographic variation in the ratio of PM$_{2.5}$ to PM$_{10}$. Data on the percentage of urban dwellers were used to derive population-weighted annual averages for each PM metric.

Three estimates of impact were calculated: mortality from cardio-pulmonary causes in adults, mortality from lung cancer, and mortality from acute respiratory infections in children 0-5. Attributable numbers of deaths and years-of-life lost for adults and children (<5 yr.) were estimated using risk coefficients from a large US cohort study of adults (Pope et al. 2002) and a meta-analytic summary estimate from 5 time-series studies of mortality in children, respectively. The estimates were calculated assuming that the risk of death increases linearly over a range of annual average concentrations of PM$_{2.5}$ (PM$_{10}$) between a reference (or counterfactual) level of 7.5 (15) and 50 (100) µg/m$^3$. The reference level corresponds to the low end of the observed concentrations in Pope et al. (2002). The uncertainty of the estimates was quantified by propagating the errors in the estimates of annual average concentration and the relative risks. Additional uncertainty due to assumptions about the shape of the concentration-response function, the choice of reference level, and the ratio of PM$_{2.5}$ to PM$_{10}$ was assessed in sensitivity analyses. An additional estimate of attributable deaths from all natural causes was calculated from time-series studies of daily mortality, based on results of a meta-analysis of the world literature.

**Indoor smoke from solid fuels**

Kirk R. Smith (University of California, Berkeley), Sumi Mehta (University of California, Berkeley), Mirjam Feuz (Federal Office of Public Health, Switzerland)

This assessment evaluates the effects of indoor combustion of solid fuels for cooking/heating. Exposure to solid fuel smoke was estimated by combining a number of national surveys of household fuel use into a model that predicts use according to independent variables such as
Relative risks from recent extensive reviews of epidemiological studies were combined in formal meta-analyses for the three most important disease endpoints: acute respiratory infections, chronic obstructive lung disease and lung cancer. To be conservative, reported results include no impacts from endpoints with moderate levels of evidence; tuberculosis, cataracts, and asthma.

Although solid fuel smoke is likely to be the major source of indoor air pollution worldwide, major related pollution sources not included in this analysis include cooking oil smoke and toxic elements in coal smoke. The current analysis focuses solely on the household environment, as the largest fraction of time spent indoors occurs at home, but other key indoor environments include schools, vehicles, and the workplace. The analysis also does not quantify several potentially important health outcomes, including adverse pregnancy outcomes and heart disease.

**Lead exposure**

Annette Prüss (WHO), Lorna Fewtrell (University of Wales), Philip Landrigan (Mount Sinai School of Medicine), José Luis Ayuso (Universidad Autonoma de Madrid)

The current assessment of burden addresses lead exposure from various pathways including the air, water, dust and food. Lead enters the body mainly by ingestion or inhalation, and results in a measurable body burden of lead. In this analysis, body burden, measured as blood lead level, served as exposure variable. Occupational exposures and exposures to “hot spots” were excluded except for their contribution to general population samples. Blood lead values from general population samples were compiled from more than 700 published studies, taking into account the likely effects of recent changes in blood lead levels mainly due to phasing out of gasoline. Recent literature suggests the theoretical minimal exposure should be considered as 0-1 µg/dl of blood lead.

Adverse health outcomes quantified as disease burden include lead-induced loss of IQ points resulting in mild mental retardation and increased blood pressure leading to cardiovascular diseases. Occurrence of anaemia and gastrointestinal effects was also quantified (Schwartz, NHANES II, ATSDR). The study used an exposure-based approach, estimating distribution of
blood lead levels in the population, measured in µg/dl. Account was made for regional variation in mental retardation caused by other known causes, such as iodine deficiency. In national assessments such exposures could be taken into account. Also, several impacts could not be considered in this analysis, e.g. delinquent behaviour, with potentially large indirect consequences on intentional injuries.

**Climate change**

Anthony McMichael (Australian National University), Diarmid Campbell-Lendrum (London School of Hygiene and Medicine), Sari Kovats (London School of Hygiene and Medicine), Sally Edwards (London School of Hygiene and Medicine), Paul Wilkinson (London School of Hygiene and Medicine), Frank Tanser (Medical Research Council, South Africa), David Le Sueur (Medical Research Council, South Africa), Michael Schlesinger (University of Illinois, Urbana-Champaign), Natasha Andronova (University of Illinois, Urbana-Champaign), Robert Nicholls (University of Middlesex), Theresa Wilson (University of Middlesex), Simon Hales (University of Otago)

Climate model simulations have been used to estimate the effects of past, present and future greenhouse gas emissions on future climate. Based on a range of alternative development scenarios and model parameterizations, the Intergovernmental Panel on Climate Change (IPCC) concluded that if no specific actions are taken to reduce greenhouse gas emissions, global temperatures will rise between 1.4 to 5.8°C from 1990 to 2100, the fastest rate experienced in the last 10,000 years. Predictions for precipitation and wind speed are less consistent, but also suggest significant changes.

Climate exhibits natural variability, and its effects on health are mediated by many other determinants. There are currently insufficient high-quality, long-term data on climate-sensitive diseases to provide a direct measurement of the health impact of anthropogenic climate change, particularly in the most vulnerable populations. Quantitative modelling is therefore the only practical route for estimating the health impacts of climate change. A limited number of
modelling studies have estimated health effects at the global scale, and not all of these directly estimate incidence or prevalence of GBD outcomes. Existing global models were adapted and new models generated, to fit a standardized framework. This compares the relative risk of cardiovascular and respiratory mortality in thermal extremes, deaths and injuries associated with floods, cases of diarrhoea, malaria, and dengue, and the prevalence of malnutrition, under 3 climate change scenarios (projected by IPCC-endorsed climate models), relative to climatic conditions in 1961-1990.

These estimates do not include possible effects of climate change on other infectious diseases, drought and famine, population displacement, destruction of health infrastructure in natural disasters, increased pollution and allergen levels, effects of plant pests and diseases on agriculture, and risk of conflict over natural resources.

**Selected occupational risks**

The percentage of the population exposed to the following selected occupational risk factors was estimated for each of the WHO regions: work-related risk factors leading to injuries and/or death; chemical or physical agents causing lung / trachea / bronchus cancer, leukemia, or mesothelioma; airborne particulates causing nonmalignant respiratory disease, such as silica, asbestos, coal dust, asthmagens, and agents leading to chronic obstructive pulmonary disease (COPD); ergonomic stressors leading to elevated rates of low back pain; elevated noise levels, which can cause noise-induced hearing loss; and needlestick injuries in health care workers, which may lead to infections with hepatitis B, hepatitis C, or HIV/AIDS. Workplace stress, which is linked with coronary heart disease, was qualitatively examined. Mortality was analysed for carcinogens, airborne particulates and injuries, while morbidity was analysed for noise, low back pain and infections due to needlesticks.

The kinds of jobs which people do and the industries in which they work are primary determinants of their exposures to occupational hazards. In general, the exposure assessments were based on the following factors: (1) economic sector and/or occupation in which a person
works, (2) the proportion of workers in that economic sector and/or occupation who have exposure to the risk factor, (3) whether those exposures were at low or high levels, (4) for risk factors with latent effects, adjustment for persons previously exposed but not currently employed, and (5) adjustment for the proportion of the population participating in the labor force.

The International Standard Industrial Classification (World Bank 2001) lists the major economic sectors and sub-sectors as agriculture, industry (mining, manufacturing, construction, utilities), or services (trade, transportation, finance, and community services). Similarly, occupations are categorized in the 1968 International Standard Classification Codes for Occupations (Source: http://www.icpsr.umich.edu/GSS/rnd1998/appendix/isco80a.htm) as Professional and Technical, Administrative and Managerial, Clerical, Sales, Service, Agriculture, or Production workers. Workers were assigned to economic sector (World Bank World Development Indicators 2001) and/or occupational category (The ILO Yearbook of Labor Statistics 1995) by region, gender, and age.

The analysis was based on the labor force aged 15 and above, i.e., all people aged 15 and above who supply labor for the production of goods and services during a specified period, including both the employed and the unemployed. Based on databases provided by the International Labour Organization, approximately two-thirds of all persons world-wide aged 15 and older (80% of males, 55% of females) participate in the labor force. Data limitations prevented the inclusion of child labor.

The proportion of workers with exposure to the various risk factors, and the levels of those exposures, were determined separately for each risk factor:

**Risk factors for injuries**

Marisol Concha (Asociacion Chilena de Seguridad, Chile), Deborah Imel Nelson (WHO), Marilyn Fingerhut (WHO), Annette Prüss (WHO), James Leigh (University of Sydney), Carlos Corvalan (WHO)
The number of workers exposed to hazardous energies was estimated by broad occupational categories for each region, gender, and age. Based on the types of chemicals, machinery, and other sources of energies used in the various occupations, workers in Professional, Administrative, and Clerical occupations have low levels of exposure; workers in Sales and Service have moderate levels of exposure; and Agricultural and Production workers have the highest levels of exposure to hazardous energies which may cause injury. The corresponding fatal injury rates were based on the most reliable data from countries within a region (or similar region).

**Carcinogens**

Tim Driscoll (University of Sydney), Deborah Imel Nelson (WHO), N. Kyle Steenland (National Institute for Occupational Safety and Health, USA), James Leigh (University of Sydney), Marisol Concha (Associacion Chilena de Seguridad, Chile), Annette Prüss (WHO), Marilyn Fingerhut (WHO), Carlos Corvalan (WHO)

Data on the proportion of the work force exposed to carcinogens by economic sector were obtained from the European Carcinogen Exposure (CAREX) database (Kauppinen et al 2000). Data on exposure to silica and benzene in China, Thailand, and Vietnam verified the applicability of CAREX to developing regions. These exposures were apportioned into low and high exposure levels based on published values for means, variation, and sampling distributions in several countries. The relative risks of mortality due to exposures to carcinogens, adjusted for level of exposure, were developed from values obtained from published peer-reviewed studies. For malignant mesothelioma, age-specific mortality rates were developed from values obtained from published peer-reviewed studies.

**Airborne particulates**

Tim Driscoll (University of Sydney), N. Kyle Steenland (National Institute for Occupational Safety and Health, USA), Deborah Imel Nelson (WHO), James Leigh (University of Sydney), Marisol Concha (Associacion Chilena de Seguridad, Chile), Marilyn Fingerhut (WHO)
Exposures to silica and asbestos were assessed using the same methods for carcinogens. The number of workers exposed to coal dust was based on national production of coal. Age-specific mortality rates were developed from values obtained from published peer-reviewed studies. For asthma and COPD, three large community studies provided estimates of relative risks by economic activity categories.

**Stressors**

Annette Prüss (WHO), Laura Punnett (University of Massachusetts, Lowell), SangWoo Tak (University of Massachusetts, Lowell), Deborah Imel Nelson (WHO), Marilyn Fingerhut (WHO), James Leigh (University of Sydney), Sharonne Phillips (Occupational Ergonomics Pty Limited, Australia)

For low back pain, occupation was used as an indicator of exposure to ergonomic stressors leading to low back pain. The number of workers exposed to ergonomic stressors was estimated on the basis of employment in occupations with elevated rates of low back pain. For example, the highest rates of low back pain occur in farmers; moderate rates occur in operators and service workers; and the lowest rates occur in professional and administrative workers. The relative risks of low back pain resulting from exposure to ergonomic stressors were obtained from published peer-reviewed studies.

**Noise**

Deborah Imel Nelson (WHO), Marisol Concha (Asociacion Chilena de Seguridad, Chile), Marilyn Fingerhut (WHO)

The number of workers exposed to elevated noise levels was estimated on the basis of employment by occupation and economic sector, and published data on exposure to noise in various occupations and economic sectors in the United States (NIOSH 1998). The applicability of these values to regions outside the United States was evaluated by literature survey. The relative risks of hearing loss resulting from exposure to noise were developed from values obtained from published peer-reviewed studies.
Other selected risks

Unsafe health care injections

Anja M. Hauri (WHO), Gregory L. Armstrong (US Centres for Disease Control and Prevention), Yvan J. F. Hutin (WHO)

To estimate the fraction of new cases of hepatitis B virus infection, hepatitis C virus infection and HIV infections attributable to health care injections, mass action models were adapted. The estimates were based on the relative risk of infection associated with receiving at least one contaminated injection and the proportion of the population exposed. These two quantities were indirectly estimated on the basis of the annual number of injections per person, the proportion of injections administered with equipment reused in the absence of sterilization, the virus-specific transmission potential and data on prevalence, incidence and immunity levels to HBV, HCV and HIV infections. Data sources included published studies and unpublished WHO reports. Studies were reviewed using a standardized decision-making algorithm based upon the quality of the data to generate region-specific estimates of the annual number of injections per person and of the proportion of injections reused in the absence of sterilization. When more than one source of information regarding injection frequency or reuse of equipment was available for one stratum, all were used to compute an estimate. The fraction of new HBV, HCV and HIV infections attributable to contaminated injections was modelled on using these parameters and the values of the attributable fractions were compared with attributable fractions obtained in case-control and cohort studies that examined the association between receiving injections and the risk of infection. To estimate the future burden of diseases caused by contaminated injections in 2000, we constructed theoretical cohorts of infected persons that were until 2030 for the occurrence of death associated with HBV, HCV and HIV infection according to the natural history of these infections. Background mortality was taken into account as most deaths associated with injection-associated infections would occur in the future.
Childhood sexual abuse

Gavin Andrews (University of New South Wales), Justine Corry (University of New South Wales), Cathy Issakidis (University of New South Wales), Tim Slade (University of New South Wales), Heather Swanston (University of New South Wales)

Child sexual abuse (CSA) typically includes unwanted and inappropriate sexual solicitation of, or exposure to a child by an older person, genital touching or fondling, or penetration in terms of oral, anal or vaginal intercourse or attempted intercourse. Severity of CSA was defined according to type of abuse. Accordingly, the three levels of exposure used in the analysis were non-contact, contact and intercourse forms of abuse. Ninety-three prevalence estimates for males and 143 for females were available for 12 of the 14 WHO regions. Prevalence rates were adjusted to account for methodological differences between studies. For example, college student and general practice attendee samples were associated with higher rates in males and so prevalence rates in these male samples were adjusted to reflect community rates. The prevalence of CSA was found to be higher in females compared to males. Due to the paucity of prevalence estimates in regions other than AMR A, EUR A and WPR A firm conclusions could not be drawn about differences in prevalence rates between regions. However, the pattern of results suggested that a high prevalence of CSA is found in the AFR E and SEAR D regions.

Estimates of the relative risk of developing five mental disorders and attempting suicide given exposure to CSA were available from 12 countries. All of the countries from which the relative risk estimates were derived are considered developed countries and they represent three of the 14 WHO regions. After controlling for the confounding factors of family dysfunction and other forms of abuse, contact and intercourse but not non-contact CSA increases the risk of all five mental disorders as well as the risk of attempting suicide. For contact abuse relative risks ranged from 1.3 for drug abuse/dependence to 3.0 for posttraumatic stress disorder. For intercourse types of abuse the relative risks ranged from 1.9 for alcohol abuse/dependence to 4.5 for posttraumatic stress disorder. The relative risks increased as the intrusiveness of the abuse increased. However, these estimates are made uncertain by lack of knowledge on the impact of cultural differences on the prevalence of childhood sexual abuse and on its relationship with mental disorders. To some extent these are culturally determined as they rely on normative
judgements about the age at which an individual becomes an adult as well as acceptable and unacceptable sexual behaviour.

*Risks to health and poverty*

Tony Blakely (University of Otago), Simon Hales (University of Otago), Charlotte Kieft (University of Otago), Nick Wilson (University of Otago), Alistair Woodward (University of Otago)

The percentage of the population living on less than US$1, between US$1 and US$2, and greater than US$2 was estimated for each of the 14 WHO regions using World Bank sponsored estimates of poverty by countries. Data on prevalence were obtained from the groups of scientists assessing risk factors for the World Health Report. The distribution of ten risk factors with absolute poverty was determined by an indirect method using asset scores calculated from the Demographic and Health Survey (DHS) data and income from the LSMS and China data. First, the joint distribution of the asset score or income variable with the risk factor was determined for each WHO region. Second, the percentage estimates of poverty by WHO region were overlaid upon the ranked asset scores and income variables. For example, if 20% of people in a WHO region were estimated to be living on less than $1 per day, then the prevalence of each factor among these impoverished people was assumed to be that observed for the 20% of people with lowest asset or income scores. Third, the relative risks of each risk factor by level of poverty were estimated based on this overlay. Finally, the proportion of each risk factor attributable to poverty for each WHO region was estimated for cut-offs of $1 and $2 per day (i.e. population impact fractions). For each cut-off, two counterfactual scenarios were specified: (i) everyone beneath $2 was ‘redistributed’ by income above $2 per day in the same proportionate manner as that observed; (ii) everyone beneath $2 was ‘shifted’ to an income of exactly $2 per day. In parallel with these quantitative analyses, selective literature reviews were conducted for some risk factors.