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Database development

Over the 5-year duration of the GBD 2010, we sought to identify all published and unpublished data sources relevant to estimating causes of death for 187 countries from 1980 to 2010. Depending on the cause, various sources of data were used. We briefly outline in the following text the main types of data identified and how they were used. The appendix (p 2) provides a summary of the site-years of data identified by broad type of data system and the number of site-years by GBD region (the data presented in the appendix are mapped at the most detailed level for a given study; the aggregate levels are created by combining the detailed levels). Of the GBD regions, central sub-Saharan Africa had the most limited evidence base with data on only 27 causes from at least one country.

For vital registration with medical certification of causes of death, we identified 2798 site-years of data from 130 countries between 1980 and 2010. 3% of the site-years were coded with ICD 8, 44% with ICD 9, 40% with ICD 10, 12% with country-specific tabulations of ICD 8, ICD 9, and ICD 10, and 1% with non-ICD tabulations. Additionally, there is country to country variation in the detail used to report causes of death included in national reporting lists—namely, the basic tabulation list for ICD 9, the ICD 10 tabulation list, three-digit and four-digit detail, and special reporting lists. Overall, we identified 25 variants of cause of death reporting lists in use from 1980 to 2010 across all sources of vital registration.

The verbal autopsy data were collected through sample registration systems, demographic surveillance systems, or surveys. Verbal autopsy is a means for ascertaining the cause of death of individuals and the cause-specific mortality fractions in populations with incomplete vital registration systems. A trained interviewer uses a structured questionnaire to ask about the signs, symptoms, and demographic characteristics of a recently deceased individual from the next of kin. We identified 486 site-years of published and unpublished verbal autopsy data across 66 countries, of which 10% were nationally representative. Verbal autopsy data are highly heterogeneous: studies use different instruments, different cause lists from single causes to full ICD cause lists, different methods for assigning cause of death based on a completed verbal autopsy, different recall periods, and different age groups, quite apart from cultural differences in the interpretation of specific questions. The appendix (p 25) provides a full listing of the sources used for all verbal autopsy and non-vital registration data organised by country.

Population-based cancer registries provide an important source of data on incidence of cancers in various countries. We identified 2715 site-years of cancer registry data across 93 countries. Some registries also track cancer mortality and provide plausible data on the mortality-to-incidence ratio by age, sex, and site. Following the methods developed by Forouzanfar and colleagues,[31] we developed estimated mortality-to-incidence ratios for all major cancers by age, sex, and country. We estimated the log of the mortality-to-incidence ratio as a function of national income per head with random effects for country, year, and age. The estimated mortality-to-incidence ratios were used to map cancer registry data on incidence to expected deaths that have been incorporated into the database. Mortality-to-incidence ratios by country, age, and sex are available on request.

In most countries, police and crime reports are an important source of information for some types of injuries, notably road injuries and interpersonal violence. The police reports used in this analysis were obtained from published studies, national agencies, and institutional surveys such as the UN Crime Trends survey and the WHO Global Status Report on Road Safety Survey.[30,41] By comparing with other sources such as vital registration data, we assessed whether police reports were likely to be complete and cover the entire country. In total, we included in the analysis 1129 site-years of police reports from 122 countries from 1980 to 2010 that met our criteria.

We identified 32 site-years of burial and mortuary data in 11 countries from ministries of health, published reports, and mortuaries themselves. Because of known bias in the epidemiological composition of burial and mortuary data, we only used information on the fraction of injuries due to specific sub-causes from these sources. These proportionate fractions of injury deaths due to specific causes were transformed into fractions of all causes by multiplying by the fraction of all deaths due to injuries estimated from a model for all injuries.

Multiple demographic and health surveys, other surveys, and censuses provide data on the fraction of deaths in the reproductive age groups that are pregnancy-related. We identified 1557 survey years with sibling history data, and a further 52 household survey years or census years of data covering 61 countries. We also identified 56 surveys or censuses with information on injury mortality across 65 survey years or census years.

We identified eight countries with nationally representative maternal mortality surveillance systems covering 83 site-years and five GBD regions. Some surveillance systems were based on prospective verbal autopsy. Surveillance data on the number of maternal deaths, or the maternal mortality ratio multiplied by births, were converted into cause fractions by dividing by the total number of deaths estimated in the reproductive age groups.

Additionally, we included 21 site-years of data based on deaths in health facilities. However, we chose to only incorporate deaths due to injury from this source because of known bias. We adjusted data for bias using a revised version of the hospital adjustment method, which uses more data and is more consistent with the GBD cause list developed by Murray and colleagues in 2007.[42] This method attempts to correct for selection bias in the deaths that occur in hospital. Finally, we used only the fraction of injury deaths due to specific injuries from these sources and converted them to fractions of deaths

from all causes following the method described for burial and mortuary data.

Assessement and enhancement of data quality and comparability

We assessed and enhanced data quality following six steps outlined in more detail here:

Step 1 consists of the assessment of completeness of death recording in each source. In settings where a data source does not capture all deaths in a population, the cause composition of deaths captured might be different from those that are not. Murray and Lopez[43] postulated in the GBD 1990 that deaths recorded in countries with incomplete vital registration would more likely originate from wealthier sectors of populations for which the cause of death structure was skewed towards non-communicable rather than communicable diseases, communicable diseases being more common in those who cannot afford appropriate treatment. They proposed a correction based on the assumption that this inequality in death rates within a country was uniform across countries. This approach was used in subsequent GBD revisions and in some of the CHERG[19,44] analyses when making use of vital registration data.

There are reasons, however, to also be concerned that deaths recorded in systems with low coverage might be biased towards selected causes that are more likely to occur in hospital. Many vital registration systems begin with in-hospital deaths and progressively capture deaths in the community. Murray and colleagues[42] showed that the fraction of deaths in hospital was higher for acute causes for which death was not immediate but occurred over a matter of days such as for some maternal causes. Further, evidence on subnational mortality patterns[45] clearly shows that the assumption of uniform inequality is unlikely to be true; nor is the assumption that deaths are registered in order, from the richest to the poorest communities. For the GBD 2010, we assessed the completeness of vital registration or sample registration data over age 5 years using the most accurate variants of death distribution methods: synthetic extinct generations, the generalised growth balance method, and a hybrid of the two.[46] We assessed completeness for under age 5 years by comparing registration data with survey and census data on child mortality. More details on how the synthesis of these methods was done are provided by Wang and colleagues.[40] Completeness is often substantially different for child and adult deaths; in some regions such as Latin America, child completeness is usually lower than adult completeness, but other patterns are observed in Asia.[40] Completeness levels must also be interpreted with caution. Some systems, for example in Turkey, capture deaths relatively completely in selected administrative units only. That is, completeness of registration might be high but coverage is not.

For adults, few vital registration or sample registration datapoints exist with completeness lower than 70% in the database. Because completeness is often lower for deaths in children younger than 5 years compared with that in individuals older than 5 years, we investigated the effect of including data on causes of death with completeness lower than 70% (see appendix p 49 for more detail). We re-ran cause of death models for the major causes of death in children younger than 5 years in five different ways: excluding all data with completeness lower than 30%, lower than 40%, lower than 50%, lower than 60%, and lower than 70%. At the global level, the number of deaths estimated in 2010 for acute respiratory infections and diarrhoea, for example, differ by 0·9% and 1·2%, respectively, between models that include all data and those that exclude data where death registration for children younger than 5 years is less than 70% complete. The difference is slightly larger in 1980, for which including all data leads to higher numbers than excluding the incomplete data. Even in the 1980s at the regional or country level, the differences are small enough that we chose to use all available data. These sensitivity analyses suggest that, at least for major causes of child death, no consistent evidence of selection bias towards causes of death in richer populations exists.

To assess completeness is feasible for vital registration and sample registration data but not for small-scale studies on verbal autopsy, which might not detect all deaths through household recall. In fact, household recall often yields a substantial undercount of deaths.[47,48] In the absence of evidence on the cause of death pattern in recalled versus not recalled deaths, we have made the simplifying assumption that verbal autopsy cause fractions are representative of the study population; the CHERG analyses of verbal autopsy data make the same assumption.[19,20]

Step 2 consists of mapping revisions and variants of the ICD (see appendix p 50 for more detail). Vital registration data and some verbal autopsy data for 1980–2010 are reported using several variants of the ICD 8, ICD 9, and ICD 10. We mapped these revisions to the GBD cause list in the appendix. This mapping provides the codes for the detailed list for ICD 9 and ICD 10, as well as the basic tabulation list for ICD 9 (BTL). We identified three national variants of ICD 9 BTL that we also mapped to the GBD cause list. Of note, there were 119 GBD causes not available in the BTL, such as pneumonia and diarrhoea aetiologies, some of the cancers, hepatitis by type, some of the cardiovascular causes, many of the mental and behavioural disorders, some musculoskeletal disorders, and some injury subtypes.

Step 3 relates to the redistribution of deaths assigned to garbage codes. Murray and Lopez[43] introduced the notion of "garbage codes" in the GBD 1990 and proposed methods to redistribute deaths assigned to garbage codes to probable underlying causes of death. Garbage codes are causes of death that should not be identified as underlying causes of death but have been entered as the underlying cause of death on death certificates. Classic examples of garbage codes include senility or cardiopulmonary arrest. In the GBD 1990, major garbage codes were identified and

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simple algorithms proposed to redistribute these proportionately to various causes (called "target codes") that were the likely underlying causes of death.[49] A similar approach was applied for the GBD 2000 and subsequent WHO updates. For the GBD 2010, we identified causes that should not be assigned as underlying cause of death at a much more detailed level.[50] In total, we identified 2759 garbage codes in ICD 10 detailed data, 3382 garbage codes in ICD 9 detailed data, and 85 garbage codes in the ICD 9 BTL, ranging from abdominal rigidity to yellow nail syndrome. Garbage codes have been identified at the most detailed level possible (eg, the fourth digit level for ICD 9 and ICD 10). For every garbage code, the potential underlying causes of death were identified on the basis of pathophysiology. For example, the target codes for peritonitis included acute gastric ulcers with perforation and acute tubulointerstitial nephritis; the target codes for disseminated intravascular coagulation included other septicaemia and premature separation of placenta. Moreover, redistribution proportionate to the number of deaths noted in the target codes cannot be reliably applied; for example, although many injuries exist, not all peritonitis deaths are likely due to injuries. Similarly, the probability of deaths due to a target cause being misclassified on death certificates as a garbage code is not equal. We have developed allocations of the garbage codes on the basis of the little published scientific literature, expert judgment, statistical analysis,[51] and in some cases proportionate allocation across target causes. The appendix (pp 71–103) provides a complete listing of the redistribution algorithms used, organised by garbage code. The extent of garbage coding in vital registration data varies widely across countries from a low of 5·5% in Finland to a high of 69·6% in Sri Lanka.

Step 4 consists of age splitting and age-sex splitting. Sources report data according to varying age groups; for consistency in the analysis, the GBD project defined and used a standardised set of 20 age groups throughout. Data reported for more aggregate age groups are split into estimates of age-specific deaths using the global observed pattern of relative risks of death for a cause by age and the local distribution of the population by age. Relative risks of death by age were computed for each cause using the entire pooled dataset on medically certified causes of death. In the few cases in which studies reported deaths for both sexes combined, a similar approach was used to allocate these deaths to age-sex groups. The appendix (p 104) provides more detail on the development of the age splitting model and the age-sex splitting model.

Step 5 consists of smoothing. For some causes in some countries, the number of deaths observed in a year is very low; zero, one, or two deaths might be noted in some years because of stochastic fluctuation. For models using the log of the death rate, either observations that record zero deaths are dropped or an arbitrary small number is substituted for zero observations; both approaches can lead to bias. This issue is exacerbated in modelling strategies that attempt to capture spatial and temporal correlation structure. In cases for which many years for a country-cause-age group did not report any deaths, we used a standardised smoothing algorithm, essentially a type of moving average, as described in the appendix (p 104).

Step 6 consists of outlier detection. Despite these efforts to enhance quality and comparability, the data from some sources seem completely implausible. Where these sources are one of many in a country for a given cause, they have little effect on the results. In some cases, however, time series estimation can be substantially affected by these outliers. We identified outliers that met the following criteria: large inconsistency with other data for the same cause in the same country at the same time; large inconsistency with other data for similar countries; or disproportionate effect on time series estimation. In these cases, the observation was excluded from subsequent analysis. The interpretation of large inconsistency or disproportionate effect varies by cause and was based on the consensus of the investigators.

Modelling of individual causes of death

We used six different modelling strategies for causes of death depending on the strength of the available data. The appendix (p 105) shows the modelling strategy used for each cause; in the table, "aggregation" means that the parent cause in the hierarchy is simply the sum of the causes under that rubric. In the following section, we provide additional detail on the different modelling strategies used. All of the strategies, however, were designed to generate uncertainty distributions for the cause-specific death rate by age, sex, country, and year. We attempted to capture uncertainty due to model parameter estimation, model specification, and fundamental uncertainty. For Cause of Death Ensemble Modelling (CODEm), the validity of uncertainty distributions were assessed. The uncertainty distribution for a cause for a given country, year, age, and sex group from the modelling process is propagated into computation of years of life lost because of premature mortality (YLLs) and various geographic and age-sex aggregates by sampling 1000 draws from the posterior distribution.

CODEm

For all major causes of death except for HIV/AIDS and measles, we used CODEm—133 causes in the cause list and three other special aggregates. CODEm was used to analyse maternal mortality, breast and cervical cancer mortality, and malaria mortality in published studies.[22,25,31] The logic and development of CODEm is reported in detail elsewhere.[38] In brief, CODEm develops models following three steps:

First, a large range of plausible statistical models are developed for each cause. Based on published studies, plausible relationships between covariates and the relevant cause are identified. Essentially all possible permutations of these selected covariates are tested. All models where the sign on the coefficient for a covariate is

in the direction expected based on the literature and the coefficient is statistically significant are retained. Where there are n covariates, this means testing 2^n models. Additionally, four families of statistical models are developed using covariates: mixed effects linear models of the log of the death rate, mixed effects linear models of the logit of the cause fraction, spatial-temporal Gaussian process regression (ST-GPR) models of the log of the death rate, and ST-GPR of the logit of the cause fraction. Finally, ensemble models, or blends of these various component models, are developed.

Second, the performance of all component models and ensembles is evaluated using out-of-sample predictive validity tests. 30% of the data is excluded from the initial model fits; half of that (15% of total) is used to evaluate component models and build ensembles. Out-of-sample predictive validity tests are based on comparing predictions for the remaining 15% of the data withheld from the model-building exercise with the actual observed data. Data are held out from the analysis using the pattern of missingness for each cause in the cause of death database. For example, if there are countries with no data, then the algorithm will exclude all data for some countries; if some countries only have data for children, then the algorithm will exclude all adult data for some countries. In this way, the out-of-sample predictive validity testing mimics the task required of a good cause of death model. The out-of-sample predictive validity testing is repeated until stable model results have been obtained. Tests of out-of-sample performance include the root-mean squared error of the log of the cause-specific death rate, the direction of the trend in the prediction compared to the data, and the validity of the 95% UI.

Third, on the basis of out-of-sample predictive validity, the best performing model or ensemble is selected. The rigorous evaluation of out-of-sample performance means that for every CODEm model, we generate objective data on the validity of the resulting UIs.

The appendix (p 112) summarises the performance of the CODEm models developed for 133 causes in the cause list for which we exclusively use CODEm and three special aggregates in the GBD 2010. For some causes, separate models were run for different age ranges when there was reason to believe that the relation between covariates and death rates might be different in different age ranges, for example, in children compared with adults. For every model, we show the in-sample root mean squared error of the log death rates (RMSE) and the out-of-sample performance in the 15% of data not used in the model building process. In all cases, the out-of-sample performance is worse (larger RMSE) than the in-sample performance. Of note, the gap between in-sample and out-of-sample performance varies substantially across causes—from mechanical forces (firearm) with the largest difference to leukaemia with the smallest. Out-of-sample performance also varies substantially across causes; kidney cancer has the largest RMSE in female individuals (2·039) and the smallest RMSE is for cardiovascular and circulatory disease in male individuals (0·555). More than 50% of the models the appendix (p 112) have an out-of-sample RMSE of less than 1. The next columns provide the assessment of how often the model predicts the trend from year to year observed in the data. Because of stochastic fluctuation in death rates, we do not expect a good model to predict the trend observed in the data 100% of the time. The gap between in-sample and out-of-sample trend test is less notable than the gap for the RMSE. The final assessment of model performance is the validity of the UIs; ideally, the 95% UI for a model would capture 95% of the data out-of-sample. Higher coverage suggests that UIs are too large and lower than 95% suggest UIs are too narrow. Coverage across the CODEm models ranges from 99·0% for "other neurological disorders" to a low of 84·2% for pneumoconiosis.

Negative binomial models

For 27 causes, the number of deaths recorded in the database was too low to generate stable estimates of out-of-sample predictive validity. For these causes, we developed negative binomial models using plausible covariates. These causes are identified in the appendix (p 105). For these negative binomial models, standard model building practice was followed, where plausible covariates were included in the model development and reverse stepwise procedures followed for covariate inclusion. Uncertainty distributions were estimated using both uncertainty in the regression betas for the covariates and from the gamma distribution of the negative binomial.

Fixed proportion models

In 27 causes where death is a rare event, we first modelled the parent cause in the GBD hierarchy using CODEm and then allocated deaths to specific causes using a fixed proportion. Proportions were computed using all available data in the database and were fixed over time, but, depending on data density, allowed to vary by region, age, or sex. Specifically, uterine fibroids, polycystic ovarian syndrome, endometriosis, genital prolapse, and other gynecological disorders varied by region and age for female individuals. Lower respiratory infections, upper respiratory infections, meningitis, and encephalitis varied by region and age. Thalassaemia, sickle-cell disease, glucose-6-phosphate dehydrogenase (G6PD) deficiency, and other haemoglobinopathies and haemolytic anaemias vary in proportion by country, age, and sex. Opioid, cocaine, amphetamine, and other drug use disorders varied by region and year. Finally, cellulitis, decubitus ulcer, other skin and subcutaneous diseases, abscess, impetigo, and other bacterial skin diseases all varied by age and sex.

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Diarrhoea, lower respiratory infection, meningitis, cirrhosis, maternal disorders, liver cancer, and chronic kidney disease disaggregated by subcause

The GBD 2010 cause list includes ten aetiologies for diarrhoea, five for lower respiratory infections, and four for meningitis. Additionally, we included a breakdown of maternal causes, cirrhosis, liver cancer, and chronic kidney disease by subcause. In most of these cases, published data are available on the cause or primary diagnosis for community, hospital, or registered cases, but not for deaths. For these causes, systematic reviews of the published data and careful review of statistical annuals such as renal registries have been undertaken. These studies or datapoints on aetiology were meta-analysed using the GBD Bayesian meta-regression method described elsewhere.[52] The meta-regression generated region-age-sex estimates with uncertainty of causal fractions for diarrhoea, lower respiratory infections, meningitis, chronic kidney disease, maternal disorders, cirrhosis, and liver cancer (appendix pp 121–129). These fractions were then applied to estimates of the parent cause, which were estimated using CODEm. In the cases of cirrhosis, liver cancer, maternal disorders, and chronic kidney disease, the studies or datasets on cause identified primary cause as assessed clinically; for diarrhoea, lower respiratory infections, and meningitis, cause was based on laboratory findings.

Natural history models

For a few selected causes, there is evidence that cause of death data systems do not capture sufficient information for one of two reasons. First, for some causes such as African trypanosomiasis, almost no deaths are recorded in vital registration or verbal autopsy studies, most likely because data have not been obtained in focal populations with substantial disease present. Second, there is systematic misclassification of deaths in cause of death data sources, particularly for congenital syphilis,[53,54] whooping cough,[55] measles,[56] and HIV/AIDS.[57] For these causes, natural history models have been used that begin with data on incidence or prevalence of disease and case-fatality rates (appendix pp 129–141). In the case of HIV/AIDS, a hybrid approach was used. For 36 countries, with complete and high quality vital registration systems, we used CODEm, in consultation with UNAIDS. For the remaining countries, we used the estimates with uncertainty by age and sex provided directly by UNAIDS based on their 2012 revision. In the case of Thailand and Panama, however, UNAIDS 2012 revision estimates are considerably higher than 2010 estimates and are inconsistent with the all-cause mortality evidence. For these two countries, we used the 2010 UNAIDS revision.

Mortality shock regressions

To estimate deaths directly due to natural disasters or collective violence, we used a different approach. First, we developed a variable for reported battle and disaster deaths per 10 000 using various databases for both disasters and collective violence; next, we estimated the empirical relation between under-5 mortality and mortality in adults ($_{45}q_{15}$) and this variable in settings where data were collected during these mortality shocks. As a final step, we used this empirical relation observed in periods of mortality shocks along with detailed data by age to allocate deaths due to natural disasters and collective violence by age. Details of this approach are outlined by Murray and colleagues.[58]

To develop the covariate on battle deaths during collective violence, we used data from the Armed Conflict Database from the International Institute for Strategic Studies (1997–2011), the Uppsala Conflict Data Program (UCDP)/PRIO Armed Conflict Dataset (1946–present), and available data from complete vital registration systems. In country-years where estimates are available from more than one source, priority is given to vital registration data if it gives higher estimated deaths. When vital registration data are not available, priority is given to the Uppsala Conflict Data Program (UCDP)/PRIO Armed Conflict Dataset since it has much longer and more consistent time series of estimates. The covariate for deaths due to natural disaster is based on the International Disaster Database (Centre for Research on the Epidemiology of Disasters).[59–61]

The relations between under-5 mortality and adult mortality and the disaster and collective violence

	All causes	Communicable, maternal, neonatal, and nutritional disorders	Non-communicable diseases	Injuries
1990 deaths (thousands)	46 511	15 859	26 560	4092
Deaths expected with 2010 population, 1990 population age structure, 1990 death rates (thousands)	61 307	23 295	32 647	5365
Deaths expected with 2010 population, 2010 population age structure, 1990 death rates (thousands)	70 316	21 513	43 062	5741
2010 deaths (thousands)	52 770	13 156	34 540	5073
Percentage change from 1990 due to population growth	31·8%	46·9%	22·9%	31·1%
Percentage change from 1990 due to population ageing	19·4%	−11·2%	39·2%	9·2%
Percentage change from 1990 due to change in death rates	−37·7%	−52·7%	−32·1%	−16·3%
Percentage change from 1990 to 2010	13·5%	−17·0%	30·0%	24·0%

Table 1: **Decomposition analysis of the change of global death numbers (thousands) by level 1 causes from 1990 to 2010 into total population growth, population ageing, and changes in age-specific, sex-specific, and cause-specific death rates**

covariates were estimated using 43 empirical observations for disasters and 206 empirical observations for collective violence (only years with crude death rates from shocks of more than one per 10 000 were kept in this analysis). The relation was estimated for excess mortality from these data sources by first subtracting from observed mortality rates the expected death rates in shock years with the methods outlined by Murray and colleagues.[58] The coefficients from these regressions and the disaster and collective violence covariates were used to predict excess deaths from these two causes. Because these models take into account competing causes by estimating

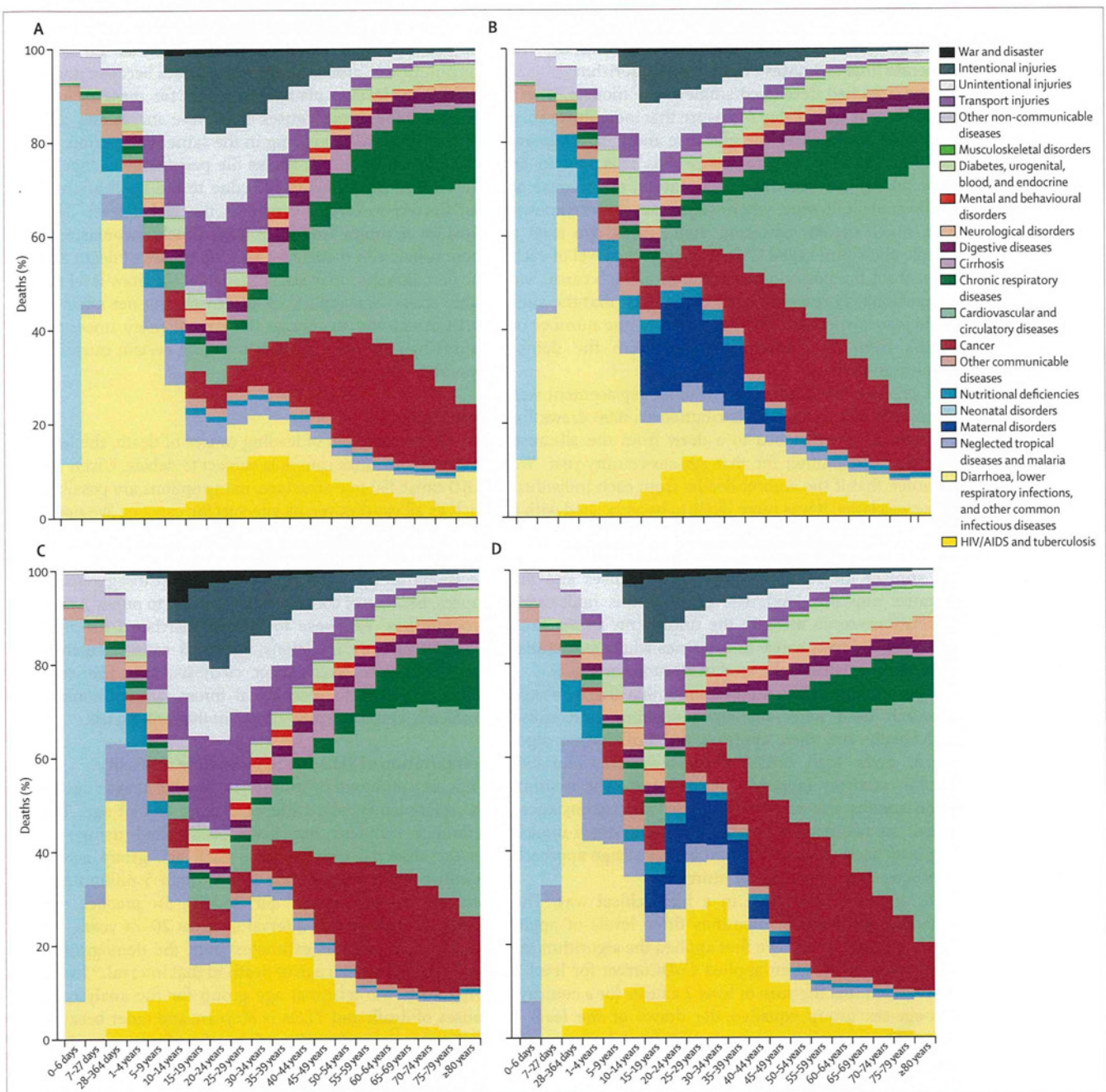

Figure 1: **Percentage of global deaths for female and male individuals in 1990 and 2010 by cause and age**
(A) Male individuals, 1990. (B) Female individuals, 1990. (C) Male individuals, 2010. (D) Female individuals, 2010. An interactive version of this figure is available online at http://healthmetricsandevaluation.org/gbd/visualizations/regional.

the relation between excess mortality and these covariates, we did not subject estimates for these two causes to the CoDCorrect algorithm described in the following text. The age pattern of mortality from these mortality shocks is based on the relative age pattern of mortality observed in the empirical data from functioning vital registration systems.

Combining results for individual causes of death to generate final estimates—CoDCorrect algorithm

Because we had developed single-cause models, it was imperative as a final step to ensure that individual cause estimates summed to the all-cause mortality estimate for every age-sex-country-year group. This had to be done taking into account uncertainty in every cause of death model outcome, where some causes were known with much greater precision than others. We used a simple algorithm called CoDCorrect; at the level of each draw from the posterior distribution of each cause, we proportionately rescaled every cause such that the sum of the cause-specific estimates equalled the number of deaths from all causes generated from the demographic analysis.[40]

In practice, a random draw without replacement was taken from the posterior distribution of 1000 draws for each cause and matched to a draw from the all-cause mortality distribution for that age-sex-country-year. We assumed that if the sum of deaths from each individual cause was large, it was more likely to be associated with a higher draw of the all-cause mortality level. To reflect this, we induced a rank order correlation of 1·0 between the sum of the random draws across causes and the all-cause mortality level. The effect of this rank order correlation was to increase the uncertainty in the final estimates for every cause in countries where substantial uncertainty existed in the level of all-cause mortality.

Repeated simulation studies show that the two-stage approach used here—namely, modelling each cause individually and then applying the CoDCorrect algorithm, gives high levels of cause-specific mortality fraction accuracy (appendix pp 146–148). These simulation studies also show that, under all circumstances tested, the two-stage approach to cause of death modelling is as good as or better than a single-stage approach as proposed by Salomon and Murray.[36]

We applied CoDCorrect in a hierarchical way. The appendix (pp 106–110) identifies three levels of application of CoDCorrect. We first applied the algorithm for level 1 causes. We then applied CoDCorrect for level 2 causes such that the sum of level 2 causes for a country-year-age-sex group equalled the draws of the level 1 cause. This cascade was repeated for level 3 causes. We chose levels for each cause based on consideration of the amount and quality of available data. For example, because there were substantially more data on all cardiovascular causes from verbal autopsy studies than for specific cardiovascular causes, we designated "all cardiovascular" as a level 1 cause for CoDCorrect. Another example of this approach is for the category "chronic respiratory diseases" where there was substantially more data for the aggregate cause than for chronic obstructive pulmonary disease (COPD), asthma, pneumoconiosis, and interstitial lung disease. Since we only wanted to group causes at level 2 or level 3 that were strongly related with common determinants, we did not use higher level aggregates such as "all non-communicable diseases" as level 1 causes because it was difficult to develop plausible models for these groups that included some causes that were increasing and others that were decreasing in the same time period.

The appendix (p 144) shows the percentage change in every cause of death for 2010 due to the application of CoDCorrect to level 1 causes at the global level. This provides a rough metric of how much inconsistency there is between models for specific causes of death and the demographic analysis. Although at the draw level the same scalar was applied to all causes, the net effect of CoDCorrect was to change the size of more uncertain causes by more than is done for more certain causes, a desirable property.

Ranking lists

For the presentation of leading causes of death, the level at which one ranks causes is subject to debate. Given the GBD cause list tree structure, many options are possible, such as all cancers versus site-specific cancers. We opted to produce tables of rankings using the level of disaggregation that seemed most relevant for public health decision making. Although we report more disaggregated causes, because of considerations related to public health programmes, we chose to include diarrhoeal diseases, lower respiratory infections, maternal causes, cerebrovascular disease, liver cancer, cirrhosis, drug use, road injury, exposure to mechanical forces, animal contact, homicide, and congenital causes in the ranking list.

Computation of YLLs due to premature mortality

YLLs are computed by multiplying deaths at each age by the reference standard life expectancy at that age. The reference standard has been constructed using the lowest observed death rate in each age group across countries with a population greater than 5 million (see Murray and colleagues[39] for details). In practice, for deaths in a given age-interval such as 20–24 years, we used country-specific estimates from the demographic analysis of the mean age of death in that interval.[40] In the GBD 2010, the terminal age group for the analysis of causes of death and YLDs is 80 years and older because of the scarcity and quality of data for older age groups. Because the all-cause mortality analysis was undertaken, however, for more detailed age groups up to age 110 years, we were able to take into account the mean age of death over 80 years in every country-year-sex group in computing YLLs.

	All ages deaths (thousands)			Age-standardised death rates (per 100 000)		
	1990	2010	%Δ	1990	2010	%Δ
All causes	**46 511·2 (45 497·4–47 726·2)**	**52 769·7 (50 877·7–53 917·2)**	**13·5%**	**999·1 (979·2–1022·0)**	**784·5 (756·3–801·6)**	**–21·5**
Communicable, maternal, neonatal, and nutritional disorders	15 859·2 (15 065·8–16 842·5)	13 156·4 (12 377·2–13 807·6)	–17·0%	271·1 (258·4–287·2)	189·8 (178·6–199·2)	–30·0
HIV/AIDS and tuberculosis	1770·3 (1600·2–2032·7)	2661·4 (2358·1–2895·7)	50·3%	39·3 (35·4–45·2)	39·4 (34·8–42·9)	0·2
Tuberculosis	1471·5 (1318·5–1716·1)	1196·0 (923·7–1376·8)	–18·7%	33·3 (29·8–38·7)	18·0 (13·9–20·7)	–46·0
HIV/AIDS	298·8 (242·0–378·5)	1465·4 (1334·2–1606·0)	390·4%	6·0 (4·8–7·7)	21·4 (19·4–23·5)	258·4
HIV disease resulting in mycobacterial infection	53·8 (42·4–70·0)	256·9 (231·9–284·1)	377·2%	1·1 (0·8–1·4)	3·7 (3·4–4·2)	254·4
HIV disease resulting in other specified or unspecified diseases	245·0 (197·7–312·6)	1208·4 (1091·6–1333·9)	393·3%	4·9 (3·9–6·3)	17·6 (15·9–19·5)	259·3
Diarrhoea, lower respiratory infections, meningitis, and other common infectious diseases	7772·1 (7136·0–8769·2)	5276·9 (4742·2–5790·4)	–32·1%	131·9 (122·4–146·5)	76·4 (68·6–83·7)	–42·1
Diarrhoeal diseases	2487·4 (2306·8–2661·9)	1445·8 (1278·9–1607·0)	–41·9%	41·0 (38·3–43·6)	20·9 (18·5–23·3)	–49·0
Cholera	120·9 (96·7–149·1)	58·1 (44·2–74·3)	–52·0%	1·8 (1·4–2·2)	0·8 (0·6–1·0)	–54·5
Other salmonella infections	134·7 (107·5–162·4)	81·3 (61·8–101·7)	–39·6%	2·3 (1·8–2·7)	1·2 (0·9–1·5)	–48·2
Shigellosis	194·0 (161·5–227·4)	122·8 (97·4–149·6)	–36·7%	3·3 (2·8–3·9)	1·8 (1·4–2·2)	–46·5
Enteropathogenic *E coli* infection	205·5 (163·0–250·2)	88·7 (66·8–112·8)	–56·8%	3·0 (2·4–3·6)	1·2 (0·9–1·6)	–58·2
Enterotoxigenic *E coli* infection	184·0 (155·6–218·2)	120·8 (95·7–147·6)	–34·4%	3·3 (2·7–3·9)	1·8 (1·4–2·2)	–45·8
Campylobacter enteritis	210·8 (171·2–253·6)	109·7 (81·8–137·2)	–48·0%	3·3 (2·7–4·0)	1·6 (1·2–2·0)	–52·5
Amoebiasis	67·7 (53·2–82·8)	55·5 (40·6–73·8)	–18·1%	1·4 (1·1–1·7)	0·8 (0·6–1·1)	–39·0
Cryptosporidiosis	222·6 (181·5–264·7)	99·8 (76·1–125·0)	–55·2%	3·2 (2·6–3·8)	1·4 (1·1–1·8)	–56·6
Rotaviral enteritis	523·3 (433·5–605·7)	250·9 (191·5–308·2)	–52·1%	7·9 (6·5–9·2)	3·6 (2·7–4·4)	–54·9
Other diarrhoeal diseases	623·9 (466·5–814·3)	458·3 (339·1–603·9)	–26·5%	11·6 (8·8–14·8)	6·8 (5·0–8·9)	–41·6
Typhoid and paratyphoid fevers	136·5 (16·5–254·7)	190·2 (23·8–359·1)	39·4%	2·4 (0·3–4·4)	2·7 (0·3–5·1)	15·5
Lower respiratory infections	3415·4 (3109·5–3650·9)	2814·4 (2487·8–3033·0)	–17·6%	62·3 (57·0–67·2)	41·0 (36·3–44·2)	–34·1
Influenza	574·6 (519·3–625·8)	507·9 (444·2–553·8)	–11·6%	10·9 (10·0–11·8)	7·5 (6·5–8·1)	–31·8
Pneumococcal pneumonia	858·4 (778·5–932·3)	827·3 (718·4–899·5)	–3·6%	17·0 (15·5–18·6)	12·1 (10·5–13·2)	–28·7
H influenzae type B pneumonia	606·9 (541·5–669·6)	379·9 (337·1–420·5)	–37·4%	9·8 (8·9–10·8)	5·5 (4·8–6·0)	–44·4
Respiratory syncytial virus pneumonia	534·8 (463·4–608·4)	253·5 (215·0–296·6)	–52·6%	7·6 (6·6–8·6)	3·5 (3·0–4·1)	–53·3
Other lower respiratory infections	840·6 (747·9–926·9)	845·8 (734·1–927·6)	0·6%	16·9 (15·1–18·6)	12·4 (10·8–13·6)	–26·5
Upper respiratory infections	4·0 (3·6–4·2)	3·0 (2·7–3·4)	–23·6%	0·1 (0·1–0·1)	<0·05 (0·0–0·05)	–36·2
Otitis media	5·2 (0·0–61·0)	3·5 (0·0–39·8)	–33·5%	0·1 (0·0–1·0)	<0·05 (0·0–0·6)	–42·3
Meningitis	492·2 (444·1–583·3)	422·9 (360·2–471·7)	–14·1%	8·1 (7·4–9·4)	6·1 (5·1–6·7)	–25·0
Pneumococcal meningitis	124·9 (111·8–149·3)	118·4 (98·4–132·0)	–5·2%	2·1 (1·9–2·5)	1·7 (1·4–1·9)	–19·5
H influenzae type B meningitis	118·9 (103·2–148·5)	83·0 (70·6–97·0)	–30·2%	1·8 (1·5–2·2)	1·2 (1·0–1·4)	–33·9
Meningococcal infection	77·1 (68·8–92·7)	75·0 (61·8–85·0)	–2·6%	1·3 (1·2–1·5)	1·1 (0·9–1·2)	–16·5
Other meningitis	171·3 (153·2–199·2)	146·4 (119·8–164·4)	–14·6%	2·9 (2·6–3·3)	2·1 (1·7–2·4)	–27·4
Encephalitis	143·5 (126·7–168·1)	119·3 (98·0–137·1)	–16·9%	2·4 (2·1–2·8)	1·7 (1·4–2·0)	–28·3
Diphtheria	6·3 (0·0–53·0)	2·9 (0·0–24·9)	–53·5%	0·1 (0·0–0·8)	<0·05 (0·0–0·3)	–55·2
Whooping cough	166·5 (0·6–815·7)	81·4 (0·3–399·0)	–51·1%	2·3 (0·0–11·4)	1·1 (0·0–5·5)	–51·6
Tetanus	272·8 (163·4–456·1)	61·3 (31·0–114·0)	–77·5%	4·1 (2·4–7·6)	0·9 (0·4–1·6)	–78·8
Measles	631·2 (188·2–1492·6)	125·4 (41·3–295·5)	–80·1%	9·0 (2·7–21·3)	1·7 (0·6–4·1)	–80·6
Varicella	11·2 (0·0–75·0)	6·8 (0·0–46·4)	–38·9%	0·2 (0·0–1·3)	0·1 (0·0–0·7)	–50·8
Neglected tropical diseases and malaria	1210·6 (1014·1–1485·4)	1321·8 (1055·6–1677·6)	9·2%	21·0 (17·5–25·9)	18·9 (15·1–23·9)	–10·0
Malaria	975·7 (781·2–1239·5)	1169·5 (916·5–1526·9)	19·9%	16·6 (13·4–21·3)	16·7 (13·0–21·7)	0·5
Chagas disease	9·3 (4·6–19·9)	10·3 (5·1–28·6)	10·8%	0·2 (0·1–0·5)	0·2 (0·1–0·4)	–30·4
Leishmaniasis	87·2 (50·6–138·4)	51·6 (33·2–76·1)	–40·9%	1·5 (0·9–2·4)	0·7 (0·5–1·1)	–51·3
African trypanosomiasis	33·5 (9·9–72·7)	9·1 (1·1–29·0)	–72·8%	0·6 (0·2–1·4)	0·1 (0·0–0·4)	–79·2
Schistosomiasis	10·5 (0·0–62·9)	11·7 (0·0–69·8)	10·9%	0·2 (0·0–1·5)	0·2 (0·0–1·1)	–28·6
Cysticercosis	0·7 (0·0–2·8)	1·2 (0·0–4·3)	58·5%	<0·05 (0·0–0·1)	<0·05 (0·0–0·1)	7·3
Echinococcosis	2·0 (0·0–7·7)	1·2 (0·0–4·7)	–41·2%	<0·05 (0·0–0·2)	<0·05 (0·0–0·1)	–62·2
Dengue	11·4 (3·7–23·5)	14·7 (6·1–24·3)	28·9%	0·2 (0·1–0·4)	0·2 (0·1–0·4)	3·2
Rabies	54·1 (32·4–103·4)	26·4 (15·2–45·2)	–51·2%	1·0 (0·6–1·9)	0·4 (0·2–0·7)	–61·7

(Continues on next page)

	All ages deaths (thousands)			Age-standardised death rates (per 100 000)		
	1990	2010	%Δ	1990	2010	%Δ
(Continued from previous page)						
Intestinal nematode infections	3·4 (0·0–16·4)	2·7 (0·0–13·0)	−21·7%	0·1 (0·0–0·2)	<0·05 (0·0–0·2)	−27·3
Ascariasis	3·4 (0·0–16·4)	2·7 (0·0–13·0)	−21·7%	0·1 (0·0–0·2)	<0·05 (0·0–0·2)	−27·3
Other neglected tropical diseases	22·9 (14·3–29·5)	23·7 (16·6–30·9)	3·4%	0·5 (0·3–0·6)	0·3 (0·2–0·5)	−23·6
Maternal disorders	358·6 (297·7–429·4)	254·7 (203·8–303·3)	−29·0%	6·9 (5·7–8·3)	3·7 (2·9–4·4)	−47·2
Maternal haemorrhage	84·8 (69·0–101·7)	58·3 (46·2–68·7)	−31·2%	1·7 (1·4–2·0)	0·8 (0·7–1·0)	−49·8
Maternal sepsis	33·8 (28·0–41·6)	21·9 (17·6–26·7)	−35·3%	0·6 (0·5–0·8)	0·3 (0·2–0·4)	−50·8
Hypertensive disorders of pregnancy	69·8 (57·6–85·0)	47·1 (37·7–57·2)	−32·5%	1·3 (1·1–1·6)	0·7 (0·5–0·8)	−48·8
Obstructed labour	19·1 (15·6–23·8)	10·9 (8·6–13·5)	−43·0%	0·4 (0·3–0·5)	0·2 (0·1–0·2)	−57·3
Abortion	56·1 (46·4–68·7)	37·1 (29·8–45·1)	−33·8%	1·1 (0·9–1·3)	0·5 (0·4–0·6)	−50·3
Other maternal disorders	95·1 (78·4–112·8)	79·4 (63·0–92·4)	−16·6%	1·9 (1·5–2·2)	1·1 (0·9–1·3)	−38·7
Neonatal disorders	3081·1 (2684·2–3393·8)	2236·4 (2014·6–2470·1)	−27·4%	42·4 (36·9–46·7)	31·0 (27·9–34·3)	−26·8
Preterm birth complications	1204·1 (998·1–1376·8)	859·7 (731·6–990·1)	−28·6%	16·6 (13·7–18·9)	11·9 (10·1–13·7)	−28·0
Neonatal encephalopathy (birth asphyxia/trauma)	638·1 (516·7–798·1)	511·4 (402·2–619·4)	−19·9%	8·8 (7·1–11·0)	7·1 (5·6–8·6)	−19·2
Sepsis and other infectious disorders of the newborn baby	534·6 (292·0–817·1)	513·7 (317·6–841·0)	−3·9%	7·4 (4·0–11·2)	7·1 (4·4–11·7)	−3·1
Other neonatal disorders	704·3 (529·1–860·3)	351·7 (293·5–429·8)	−50·1%	9·7 (7·3–11·8)	4·9 (4·1–6·0)	−49·7
Nutritional deficiencies	976·9 (854·4–1155·7)	684·1 (546·0–790·1)	−30·0%	17·3 (15·1–20·4)	9·9 (7·9–11·5)	−42·8
Protein-energy malnutrition	883·0 (726·7–1052·6)	599·8 (459·4–701·9)	−32·1%	15·4 (12·6–18·3)	8·7 (6·6–10·1)	−43·7
Iodine deficiency	2·0 (1·7–2·4)	3·4 (2·4–3·8)	67·7%	<0·05 (0·0–0·1)	<0·05 (0·0–0·1)	17·5
Iron-deficiency anaemia	80·8 (66·5–97·8)	69·4 (51·6–78·9)	−14·1%	1·6 (1·3–2·0)	1·0 (0·8–1·2)	−37·3
Other nutritional deficiencies	11·1 (9·6–14·0)	11·5 (8·0–12·8)	3·4%	0·2 (0·2–0·3)	0·2 (0·1–0·2)	−31·4
Other communicable, maternal, neonatal, and nutritional disorders	689·5 (569·9–815·1)	721·2 (626·8–830·4)	4·6%	12·3 (10·4–14·2)	10·6 (9·2–12·1)	−14·3
Sexually transmitted diseases excluding HIV	209·4 (130·0–324·3)	118·3 (71·6–187·7)	−43·5%	3·0 (1·9–4·6)	1·6 (1·0–2·6)	−45·6
Syphilis	202·9 (121·9–315·8)	113·3 (66·9–181·7)	−44·1%	2·9 (1·8–4·4)	1·6 (0·9–2·5)	−45·4
Sexually transmitted chlamydial diseases	1·5 (0·8–2·0)	1·2 (0·8–1·8)	−23·7%	<0·05 (0·0–0·05)	<0·05 (0·0–0·05)	−49·1
Gonococcal infection	1·1 (0·6–1·5)	0·9 (0·6–1·3)	−23·6%	<0·05 (0·0–0·05)	<0·05 (0·0–0·05)	−49·0
Other sexually transmitted diseases	3·8 (2·0–5·0)	2·9 (2·0–4·5)	−23·6%	0·1 (0·0–0·1)	<0·05 (0·0–0·01)	−49·0
Hepatitis	210·2 (200·3–221·1)	307·7 (268·2–356·5)	46·4%	4·4 (4·2–4·6)	4·6 (4·0–5·3)	4·6
Acute hepatitis A	99·0 (56·5–154·2)	102·8 (51·2–228·1)	3·9%	2·1 (1·1–3·3)	1·5 (0·8–3·4)	−25·1
Acute hepatitis B	68·6 (46·7–84·4)	132·2 (91·1–169·7)	92·7%	1·5 (1·1–1·9)	2·0 (1·4–2·6)	29·2
Acute hepatitis C	8·1 (4·9–11·6)	16·0 (11·6–21·4)	97·1%	0·2 (0·1–0·3)	0·2 (0·2–0·3)	25·6
Acute hepatitis E	34·5 (19·6–55·0)	56·6 (23·3–113·3)	64·2%	0·6 (0·3–0·9)	0·8 (0·3–1·6)	36·1
Other infectious diseases	269·9 (192·2–320·5)	295·2 (205·6–362·1)	9·4%	4·9 (3·5–5·7)	4·3 (3·0–5·3)	−11·8
Non-communicable diseases	26 560·3 (25 843·4–27 249·3)	34 539·9 (33 164·7–35 313·0)	30·0%	645·9 (629·9–662·9)	520·4 (499·5–532·0)	−19·4
Neoplasms	5779·1 (5415·9–6201·9)	7977·9 (7337·1–8403·8)	38·0%	140·8 (131·9–151·4)	121·4 (111·6–127·9)	−13·8
Oesophageal cancer	344·7 (279·7–428·8)	395·2 (298·4–482·1)	14·7%	8·5 (6·9–10·6)	6·1 (4·6–7·4)	−28·7
Stomach cancer	774·1 (602·8–1014·2)	754·9 (571·9–990·4)	−2·5%	19·0 (14·8–25·0)	11·5 (8·7–15·1)	−39·5
Liver cancer	463·0 (386·5–526·8)	752·1 (643·6–880·3)	62·4%	11·2 (9·4–12·8)	11·5 (9·8–13·4)	2·3
Liver cancer secondary to hepatitis B	210·2 (176·9–239·4)	341·4 (290·1–402·6)	62·4%	5·1 (4·3–5·8)	5·2 (4·4–6·1)	2·6
Liver cancer secondary to hepatitis C	113·0 (96·6–129·3)	195·7 (165·2–222·0)	73·3%	2·8 (2·4–3·2)	3·0 (2·5–3·4)	7·6
Liver cancer secondary to alcohol use	93·4 (78·6–106·4)	149·0 (127·3–172·6)	59·5%	2·3 (1·9–2·6)	2·3 (1·9–2·6)	−0·1
Other liver cancer	46·5 (38·2–52·6)	66·0 (57·2–77·3)	42·0%	1·1 (0·9–1·2)	1·0 (0·9–1·2)	−7·7
Larynx cancer	81·9 (43·5–133·4)	98·3 (52·8–159·2)	20·1%	2·0 (1·1–3·3)	1·5 (0·8–2·4)	−25·1
Trachea, bronchus, and lung cancers	1036·3 (825·8–1314·3)	1527·1 (1126·3–1779·4)	47·4%	25·5 (20·4–32·4)	23·4 (17·3–27·3)	−8·3
Breast cancer	319·1 (310·1–337·0)	438·7 (420·1–461·9)	37·5%	7·8 (7·6–8·3)	6·6 (6·4–7·0)	−15·3
Cervical cancer	192·3 (120·5–264·4)	225·4 (145·2–311·5)	17·3%	4·7 (2·9–6·4)	3·4 (2·2–4·7)	−26·9
Uterine cancer	45·2 (25·3–79·4)	58·6 (27·5–87·8)	29·7%	1·1 (0·6–2·0)	0·9 (0·4–1·3)	−20·2
Prostate cancer	155·6 (88·8–239·6)	256·0 (141·1–404·4)	64·5%	4·0 (2·3–6·1)	3·8 (2·1–6·1)	−3·1
Colon and rectum cancers	490·5 (417·2–547·3)	714·6 (627·9–822·6)	45·7%	12·2 (10·4–13·6)	10·8 (9·5–12·5)	−10·9

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	All ages deaths (thousands)			Age-standardised death rates (per 100 000)		
	1990	2010	%Δ	1990	2010	%Δ
(Continued from previous page)						
Mouth cancer	81·9 (68·6–88·3)	123·9 (104·2–136·3)	51·2%	2·0 (1·7–2·2)	1·9 (1·6–2·1)	−5·9
Nasopharynx cancer	45·2 (29·9–59·6)	64·9 (42·3–83·3)	43·6%	1·1 (0·7–1·4)	1·0 (0·6–1·3)	−8·2
Cancer of other part of pharynx and oropharynx	74·0 (43·8–90·9)	102·4 (59·5–128·5)	38·3%	1·8 (1·1–2·2)	1·6 (0·9–2·0)	−12·9
Gallbladder and biliary tract cancer	97·4 (66·1–136·0)	151·7 (100·4–206·8)	55·7%	2·4 (1·6–3·4)	2·3 (1·5–3·1)	−4·7
Pancreatic cancer	200·0 (154·1–261·5)	310·2 (231·7–393·1)	55·1%	5·0 (3·8–6·5)	4·7 (3·5–6·0)	−4·8
Malignant melanoma of skin	31·0 (20·3–46·6)	49·1 (29·9–69·5)	58·4%	0·8 (0·5–1·1)	0·7 (0·5–1·1)	−1·5
Non-melanoma skin cancer	20·5 (12·5–32·7)	30·6 (17·5–46·3)	49·6%	0·5 (0·3–0·8)	0·5 (0·3–0·7)	−10·7
Ovarian cancer	113·6 (82·9–138·8)	160·5 (115·9–200·6)	41·2%	2·8 (2·0–3·4)	2·4 (1·8–3·1)	−12·1
Testicular cancer	6·5 (3·8–8·3)	7·7 (4·8–10·0)	18·6%	0·1 (0·1–0·2)	0·1 (0·1–0·1)	−18·9
Kidney and other urinary organ cancers	85·1 (62·0–112·9)	162·1 (125·5–219·8)	90·6%	2·1 (1·5–2·7)	2·5 (1·9–3·3)	19·4
Bladder cancer	123·4 (100·2–148·5)	170·7 (131·1–201·2)	38·3%	3·1 (2·5–3·7)	2·6 (2·0–3·0)	−16·3
Brain and nervous system cancers	131·5 (88·7–188·3)	195·5 (115·1–239·3)	48·7%	3·0 (2·1–4·4)	3·0 (1·7–3·6)	−2·5
Thyroid cancer	24·0 (18·0–29·9)	36·0 (26·4–43·2)	50·2%	0·6 (0·4–0·7)	0·5 (0·4–0·7)	−6·7
Hodgkin's disease	18·9 (11·8–26·2)	17·7 (11·6–25·5)	−6·0%	0·4 (0·3–0·6)	0·3 (0·2–0·4)	−36·7
Non-Hodgkin lymphoma	143·2 (119·4–158·9)	210·0 (166·0–228·5)	46·7%	3·3 (2·8–3·7)	3·2 (2·5–3·4)	−5·0
Multiple myeloma	49·3 (34·5–71·2)	74·1 (48·9–102·2)	50·4%	1·2 (0·9–1·8)	1·1 (0·7–1·6)	−7·5
Leukaemia	218·3 (175·7–269·2)	281·3 (219·6–328·0)	28·9%	4·7 (3·8–5·9)	4·2 (3·3–4·9)	−11·5
Other neoplasms	412·7 (319·5–521·9)	608·4 (441·2–737·3)	47·4%	9·8 (7·6–12·4)	9·2 (6·7–11·2)	−5·7
Cardiovascular and circulatory diseases	11 903·7 (11 329·4–12 589·3)	15 616·1 (14 542·2–16 315·1)	31·2%	298·1 (283·9–314·9)	234·8 (218·7–245·2)	−21·2
Rheumatic heart disease	462·6 (431·5–517·7)	345·1 (305·8–374·3)	−25·4%	11·1 (10·3–12·4)	5·2 (4·6–5·6)	−53·1
Ischaemic heart disease	5211·8 (5014·5–5643·9)	7029·3 (6577·2–7431·1)	34·9%	131·3 (126·4–142·2)	105·7 (98·8–111·9)	−19·5
Cerebrovascular disease	4660·4 (4436·1–5154·9)	5874·2 (5304·7–6280·1)	26·0%	105·7 (98·8–111·9)	88·4 (79·8–94·4)	−24·6
Ischaemic stroke	2241·1 (2088·0–2494·9)	2835·4 (2657·0–3262·8)	26·5%	57·6 (53·7–64·0)	42·3 (39·6–48·7)	−26·6
Haemorrhagic and other non-ischaemic stroke	2419·4 (2050·9–2827·9)	3038·8 (2643·4–3496·9)	25·6%	59·7 (50·6–69·7)	46·1 (40·1–53·1)	−22·7
Hypertensive heart disease	590·7 (481·0–740·6)	873·2 (715·5–1074·1)	47·8%	14·9 (12·1–18·6)	13·1 (10·8–16·2)	−11·5
Cardiomyopathy and myocarditis	286·8 (250·5–316·8)	403·9 (361·5–450·4)	40·8%	6·7 (5·9–7·4)	6·1 (5·4–6·8)	−9·8
Atrial fibrillation and flutter	34·4 (27·9–43·1)	114·7 (92·7–144·7)	233·9%	0·9 (0·7–1·1)	1·7 (1·4–2·1)	89·6
Aortic aneurysm	131·9 (94·6–173·3)	191·7 (140·3–249·2)	45·3%	3·3 (2·4–4·3)	2·9 (2·1–3·8)	−12·7
Peripheral vascular disease	18·6 (12·2–28·7)	49·8 (32·9–74·8)	167·0%	0·5 (0·3–0·7)	0·7 (0·5–1·1)	53·3
Endocarditis	35·8 (30·0–44·4)	48·3 (39·3–55·4)	34·8%	0·8 (0·7–1·0)	0·7 (0·6–0·8)	−8·0
Other cardiovascular and circulatory diseases	470·6 (446·3–489·9)	685·9 (664·0–705·3)	45·7%	11·5 (11·0–11·9)	10·3 (9·9–10·5)	−10·9
Chronic respiratory diseases	3986·3 (3914·3–4063·8)	3776·3 (3648·2–3934·1)	−5·3%	98·2 (96·4–100·1)	57·0 (55·1–59·4)	−41·9
Chronic obstructive pulmonary disease	3099·0 (2914·2–3338·6)	2899·9 (2669·3–3245·8)	−6·4%	77·4 (72·8–83·3)	43·8 (40·4–49·1)	−43·3
Pneumoconiosis	167·0 (86·3–295·2)	124·7 (78·3–196·9)	−25·3%	4·2 (2·2–7·3)	1·9 (1·2–3·0)	−54·8
Asthma	380·2 (273·8–589·6)	345·7 (282·6–529·1)	−9·1%	9·0 (6·6–13·9)	5·2 (4·3–8·0)	−42·1
Interstitial lung disease and pulmonary sarcoidosis	65·0 (44·5–89·8)	115·1 (76·7–152·0)	77·2%	1·6 (1·1–2·2)	1·7 (1·2–2·3)	9·1
Other chronic respiratory diseases	275·2 (200·8–375·8)	290·8 (226·8–356·5)	5·7%	6·0 (4·4–8·1)	4·3 (3·4–5·3)	−28·3
Cirrhosis of the liver	777·8 (663·1–867·9)	1030·8 (868·8–1160·5)	32·5%	18·6 (15·8–20·7)	15·6 (13·2–17·6)	−15·8
Cirrhosis of the liver secondary to hepatitis B	241·7 (198·5–270·5)	312·4 (270·8–378·3)	29·3%	5·8 (4·8–6·5)	4·8 (4·1–5·8)	−18·5
Cirrhosis of the liver secondary to hepatitis C	211·9 (181·1–240·7)	287·4 (245·4–330·5)	35·6%	5·2 (4·4–5·9)	4·4 (3·7–5·0)	−15·3
Cirrhosis of the liver secondary to alcohol use	206·1 (168·6–245·3)	282·8 (225·6–335·0)	37·2%	5·0 (4·1–5·9)	4·3 (3·4–5·1)	−13·9
Other cirrhosis of the liver	118·2 (101·4–136·7)	148·2 (126·6–173·0)	25·4%	2·6 (2·2–3·0)	2·2 (1·9–2·6)	−14·4
Digestive diseases (except cirrhosis)	973·1 (877·1–1063·5)	1111·7 (999·5–1210·0)	14·2%	22·9 (20·7–25·0)	16·7 (15·0–18·1)	−27·2
Peptic ulcer disease	319·3 (265·9–338·8)	246·3 (215·0–282·2)	−22·9%	7·5 (6·3–8·0)	3·7 (3·2–4·2)	−50·9
Gastritis and duodenitis	15·6 (11·3–21·1)	14·3 (11·0–18·2)	−8·7%	0·4 (0·3–0·5)	0·2 (0·2–0·3)	−42·1
Appendicitis	39·5 (27·2–57·0)	34·8 (22·0–46·9)	−12·0%	0·8 (0·6–1·2)	0·5 (0·3–0·7)	−38·1
Paralytic ileus and intestinal obstruction without hernia	121·0 (78·7–141·1)	148·1 (112·1–192·2)	22·4%	2·8 (1·8–3·2)	2·2 (1·7–2·9)	−20·9
Inguinal or femoral hernia	23·3 (22·8–23·7)	17·1 (16·7–17·3)	−26·7%	0·5 (0·5–0·6)	0·3 (0·2–0·3)	−53·4
Non-infective inflammatory bowel disease	29·5 (16·8–37·7)	34·0 (23·6–39·7)	15·1%	0·6 (0·4–0·8)	0·5 (0·3–0·6)	−20·3
Vascular disorders of intestine	51·4 (28·9–104·6)	73·4 (41·2–150·0)	42·9%	1·3 (0·7–2·6)	1·1 (0·6–2·3)	−15·2

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