All experts comprising each panel were asked the same set of seed questions, and several sets of seed questions were used across panels. This allows some consistency checks to be performed between panels on performance and scoring outcomes. The number of questions varied from nine to twelve in total, depending on the panel. Experts were asked to provide a central judgment in terms of the median value, and a 90% credible interval for each question. Seed questions were administered by facilitators through one-to-one telephone interviews. The experts were not presented with the seed questions before the interview and they were asked to provide estimates based on their experience, knowledge and judgment, without referring to other sources of information.

4.6.6 Target questions

Target questions are the substantive questions of interest. In this study, for all identified hazards, we enquired about the percentage of all human disease cases caused by exposure through each of a number of indicated exposure routes. The point of exposure was chosen as the point of attribution, i.e. the experts were asked to distribute the disease burden on the sources that was the direct cause of human exposure. So, for example, someone with a norovirus infection might be exposed by eating food contaminated with the virus, although the food may have been contaminated by wastewater at an earlier stage.

Exposure routes varied between hazards, as indicated in Table 4. In order to reduce the time and effort burden of the elicitation on expert panelists, the hazard-based TFs decided which hazard exposure routes were relevant for present purposes. So, for example, human-to-human transmission was excluded as an exposure route for *Brucella* spp. However, the questionnaires did provide experts with an option to indicate additional routes of transmission if they disagreed with the TF's evaluation.

Experts were asked to complete a set of tables for each assigned hazard and subregion. Experts were provided with the tables at the end of the telephone interview during which the seed questions were asked, and the facilitator went through several target questions with the experts to ensure that they understood the task. For the target items (but not the seed questions), the experts were free to consult any information sources they felt appropriate in the four-week period they were given to return the target item spreadsheets.

As with the seed questions, the experts were asked to provide their 5th, 50th and 95th percentile values for each question. Technically, the median values of a joint distribution do not need to add up to 100%, but because we included a category "other", we asked about a joint distribution that logically spanned all possible exposure routes. Therefore, the experts' median values for source attribution percentages for a hazard should sum to a value close to 100%. In individual cases, where these sums were found to differ significantly (i.e. outside 100% ± 10%), the experts concerned were asked to review their responses.

4.6.7 Data analysis

Weights for individual experts were computed using the Classical Model formulation, using the EXCALIBUR software, by multiplying their calibration and informativeness scores, with the products then jointly normalized to sum to unity over all experts in the group. An expert was positively weighted only if his/her *p*-value was above a certain threshold, chosen to

optimize the combined score across all seed items. See [146, 149] for further details on the computation of expert performance weights.

The normalized experts' performance weights were used to construct the joint probability distribution complying with their assessments for individual target questions (i.e. attributable proportion of illness per pathway, subregion and hazard). In a final step, 10 000 random values from the marginal cumulative distributions were simulated using probability integral transform [34]. First, independent vectors of 10 000 random deviates from a Uniform (0,100) distribution, per exposure category within a hazard-subregion combination, were generated.

The quantiles corresponding to these random deviates were then obtained via linear interpolation. To ensure that the random attributional proportions summed to 100%, a "normalization" step was applied per iteration, in which each random value was divided by the sum of random values for each exposure pathway. The resulting 10 000 normalized random attributional proportions were then summarized by their median and a 95% uncertainty interval defined by the 2.5th and 97.5th percentiles. These final manipulations were performed in R 3.1.1 [150] using functions available in the 'FERG' package [151].

Table 4. Exposure routes included in the expert elicitation, per hazard

HAZARD	FOOD	ANIMAL CONTACT (DOMESTIC AND WILD)	HUMAN TO HUMAN CONTACT	WATER	SOIL	AIR	PAINT	COOKWARE, POTTERY OR GLASSWARE	TOYS	OTHER
DIARRHOEAL DISEASE										
Campylobacter spp.	x	x	x	x	x	na	N/A	N/A	N/A	x
Non-typhoid *Salmonella* spp.	x	x	x	x	x	N/A	N/A	N/A	N/A	x
Shiga toxin-producing *E. coli*	x	x	x	x	x	N/A	N/A	N/A	N/A	x
Brucella spp.	x	x	N/A	x	x	N/A	N/A	N/A	N/A	x
Shigella spp.	x	N/A	x	x	x	N/A	N/A	N/A	N/A	x
Enteropathogenic *E. coli*	x	x	x	x	N/A	N/A	N/A	N/A	N/A	x
Enterotoxigenic *E. coli*	x	x	x	x	N/A	N/A	N/A	N/A	N/A	x
Cryptosporidium spp.	x	x	x	x	N/A	N/A	N/A	N/A	N/A	x
Giardia spp.	x	x	x	x	N/A	N/A	N/A	N/A	N/A	x
Tyhoid *Salmonella* spp.	x	N/A	x	x	N/A	N/A	N/A	N/A	N/A	x
Vibrio cholerae	x	N/A	x	x	N/A	N/A	N/A	N/A	N/A	x
Entamoeba histolytica	x	N/A	x	x	N/A	N/A	N/A	N/A	N/A	x
Norovirus	x	N/A	x	x	N/A	N/A	N/A	N/A	N/A	x
Hepatitis A virus	x	N/A	x	x	N/A	N/A	N/A	N/A	N/A	x
PARASITIC DISEASE										
Toxoplasma gondii	x	x	N/A	x	x	N/A	N/A	N/A	N/A	x
Echinococcus granulosus	x	x	N/A	x	x	x	N/A	N/A	N/A	x
Echinococcus multilocularis	x	x	N/A	x	x	x	N/A	N/A	N/A	x
Ascaris spp.	x	x	x	x	x	N/A	N/A	N/A	N/A	x
CHEMICALS										
Lead	x	N/A	N/A	x	x	x	x	x	x	x

Notes: N/A = not applicable, meaning that these exposure routes were considered not possible or extremely unlikely by the respective FERG TF.

4.7 Computation

Different approaches can be taken for calculating DALYs, depending on whether the interest lies in quantifying the burden of a health outcome (such as diarrhoea), a hazard (e.g. a biological agent that may cause illness in humans such as *Salmonella enterica*), or a risk factor (e.g. an exposure that inceases the likelihood of illness such as unsafe water) [152]. Since FERG is concerned with the burden of FBDs, which are caused by a wide range of hazards (bacteria, viruses, protozoa, parasites, chemicals and toxins), a natural choice is the hazard-based approach. This approach

defines the burden of a specific foodborne hazard as that resulting from the health states, i.e. acute and chronic manifestations of disease, including death, that are causally related to the hazard transmitted through food, and which may become manifest at different time scales. This approach thus allows for a comprehensive estimate of the burden of disease due to a certain hazard, including sequelae, which may have a higher burden than acute illness alone [153- 155].

4.7.1 Disease models and epidemiological data

The starting point of the CTF workflow was the outline of disease models for each of the included hazards (as chosen by the hazard-based TFs), and the epidemiological data inputs that parameterized these disease models. To obtain this information, systematic reviews were commissioned and managed by three hazard-based TFs, i.e. the Enteric Diseases Task Force (EDTF), the Parasitic Diseases Task Force (PDTF), and the Chemicals and Toxins Task Force (CTTF).

The course of disease is characterized by various health states (e.g. acute or chronic phases; short-term or long-term sequelae), possibly having different severity levels. A disease model, also referred to as an outcome tree, is a schematic representation of the various health states associated with the concerned hazard, and the possible transitions between these states. A disease model for each hazard was defined by the members and commissioned experts of each hazard-based TF, considering relevant health outcomes identified in the respective reviews.

In the context of the CTF, disease models were defined as *computational* disease models, and not merely as *biological* disease models. While biological disease models merely reflect the natural history of disease, computational disease models also reflect the input parameters needed to calculate incidence and mortality of each of the relevant health states. As such, computational disease models are a combination of disease biology and data availability.

Computational disease models may be represented as directed acyclic graphs, defined by parent and child nodes and directed edges (arrows) defining the relationships between nodes. In the CTF framework, parent nodes were either incidence, mortality, YLD or YLL rates, while child nodes were multiplicative elements, such as proportions or ratios (reflecting, e.g. the probability of developing a specific symptom following infection, or the proportion of illnesses attributable to the concerned hazard). A specific disease model "language" was developed to denote the relationship and contribution of the different nodes. Rectangles defined parent nodes, and rounded rectangles defined child nodes. Grey nodes did not contribute directly to the DALYs, green nodes contributed YLDs, and red nodes contributed YLLs. Nodes that contributed to the incidence of the index disease were identified by a thick border. Appendix 5 gives the disease models for all 36 FERG hazards.

In general, three main approaches can be distinguished for estimating the burden due to a specific hazard in food, i.e., categorical attribution, counterfactual analysis, and risk assessment. Table 5 gives an overview of the modelling strategy applied for each included hazard. As the choice of the modelling strategy was mainly driven by the type of data available, no sensitivity analyses could be performed to triangulate different modelling approaches.

Table 5. Modelling strategies for the hazards included in the WHO global burden of foodborne disease estimates

HAZARD	BURDEN ATTRIBUTION APPROACH	DISEASE MODEL APPROACH	IMPUTATION	FOODBORNE ATTRIBUTION
DIARRHOEAL DISEASE AGENTS				
Norovirus	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Campylobacter spp.	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Enteropathogenic *E. coli*	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Enterotoxigenic *E. coli*	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Shiga toxin-producing *E. coli*	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Non-typhoidal *S. enterica*	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Shigella spp.	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Vibrio cholerae	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Cryptosporidium spp.	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Entamoeba histolytica	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
Giardia spp.	Categorical attribution	Attribution	Modified CHERG approach [40]	Expert elicitation [156]
INVASIVE INFECTIOUS DISEASE AGENTS				
Hepatitis A virus	Categorical attribution	Direct: GBD2010 [3]	N/A (1)	Expert elicitation [156]
Brucella spp.	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
Listeria monocytogenes, perinatal	Categorical attribution	Transition	Random effects [79, 151]	100%
Listeria monocytogenes, acquired	Categorical attribution	Transition	Random effects [79, 151]	100%
Mycobacterium bovis	Categorical attribution	Attribution	N/A (1)	100%
Salmonella Paratyphi	Categorical attribution	Direct: GBD2010 [3]	N/A (1)	Expert elicitation [156]
Salmonella Typhi	Categorical attribution	Direct: GBD2010 [3]	N/A (1)	Expert elicitation [156]
Toxoplasma gondii, congenital	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
Toxoplasma gondii, acquired	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
ENTERIC INTOXICATIONS				
Bacillus cereus (2)	Categorical attribution	Direct	Uniform	100%
Clostridium botulinum (2)	Categorical attribution	Direct	Uniform	100%
Clostridium perfringens (2)	Categorical attribution	Direct	Uniform	100%

HAZARD	BURDEN ATTRIBUTION APPROACH	DISEASE MODEL APPROACH	IMPUTATION	FOODBORNE ATTRIBUTION
Staphylococcus aureus (2)	Categorical attribution	Direct	Uniform	100%
CESTODES				
Echinococcus granulosus, cases seeking treatment	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
Echinococcus granulosus, cases not seeking treatment	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
Echinococcus multilocularis	Categorical attribution	Transition	Random effects [79, 151]	Expert elicitation [156]
Taenia solium	Categorical attribution	Attribution	N/A (1)	100%
NEMATODES				
Ascaris spp.	Categorical attribution	Direct: GBD2010 [3]	N/A (1)	Expert elicitation [156]
Trichinella spp.	Categorical attribution	Direct	N/A (1)	100%
TREMATODES				
Clonorchis sinensis	Categorical attribution	Direct	Random effects [79, 151]	100%
Fasciola spp.	Categorical attribution	Direct	Random effects [79, 151]	100%
Intestinal flukes (3)	Categorical attribution	Direct	Random effects [79, 151]	100%
Opisthorchis spp.	Categorical attribution	Direct	Random effects [79, 151]	100%
Paragonimus spp.	Categorical attribution	Direct	Random effects [79, 151]	100%
ORGANIC POLLUTANTS				
Dioxin	Risk assessment	Direct	Random effects [79, 151]	100%
TOXINS AND ALLERGENS				
Aflatoxin	Counterfactual analysis	Attribution	Random effects [79, 151]	100%
Cyanide in cassava	Categorical attribution	Direct	Uniform	100%
Peanut allergens (2)	Categorical attribution	Direct	Uniform	100%

Notes: (1) N/A = not applicable as no imputation had to be performed because data were used that had already been imputed. (2) Excluded from global burden assessments. (3) Includes *Echinostoma* spp., *Fasciolopsis buski*, *Heterophyes* spp., *Metagonimus* spp. and other foodborne intestinal trematode species.

Categorical attribution can be used when a foodborne hazard results in an outcome (death or a specific syndrome) that is identifiable as caused by the hazard in individual cases [157]. Following the typology of Devleesschauwer et al. [152], the burden due to a specific hazard can then be calculated using an attributional model (in which the incidence of the symptom is multiplied with the attributable proportion for a given hazard) or a transitional model (in which the incidence of the hazard is multiplied with the probability of developing a given symptom). Categorical attribution was applicable for all viral, bacterial and parasitic hazards, and for cyanide in cassava and peanut allergens, and was therefore the standard method used by FERG. Appendix 5 shows the computational disease model for *Mycobacterium bovis*, which is characteristic for the attributional models. In this model, the overall incidence and mortality of tuberculosis is multiplied with the proportion attributable to *M. bovis*, resulting in the incidence and mortality of *M. bovis* tuberculosis. Appendix 5 shows the computational disease model for *Echinococcus granulosus*, which is characteristic for the transitional models. In this model, the overall incidence of infection by this parasite was multiplied with child nodes reflecting the probability of developing the concerned health states, resulting in the incidences of the specific health states.

When the hazard elevates the risk of a disease or disability outcome that occurs in the population from other causes as well, causal attribution can only be made statistically, and not on an individual basis. This is the case for many chemicals, including aflatoxin and dioxin. Aflatoxin for instance may increase the risk of hepatocellular carcinoma, but it is not possible to specify that a specific liver cancer case was caused by aflatoxin. In this situation, the standard approach for calculating the burden of environmental exposures is to use a *counterfactual analysis* in which the current disease outcomes with current exposure are compared with the disease outcomes under an alternative exposure (a min. risk exposure which could be zero, or some accepted background level) [158]. This allows calculation of a population attributable fraction (PAF) that can be applied to the all-cause burden estimates for the relevant disease outcome (the so-called burden envelope), leading to a special case of the attributional model [152]. In the context of FERG, counterfactual analysis was used to estimate the burden of aflatoxin-related hepatocellular carcinoma.

In addition to categorical attribution and counterfactual analysis, which can be considered top-down approaches, FBD burden can also be estimated by a *risk assessment* approach, which can be considered a bottom-up approach. In this approach, the incidences of the specific health states (e.g. impaired male fertility due to prenatal dioxin exposure) are estimated by combining exposure and dose-response data. The dose-response model may for instance define the probability of illness at a given exposure level, which can then be translated into an estimate of the number of incident or prevalent cases expected to occur in the exposed population [158, 191]. As this approach does not involve burden attribution, it does not necessarily ensure consistency with existing health statistics. However, risk assessment may be a valid alternative when no burden envelopes exist or when it can be demonstrated that the estimated excess risk is additive to the background risk. In the context of FERG, risk assessment was used to estimate the burden of dioxin-related hypothyroidy and impaired fertility.

4.7.2 CTF database template

A database template was developed in Excel™ [7] to collect in a standardized way the data resulting from the systematic reviews. The structure of the database was based on the disease models, with one sheet per node. Three generic sheets were defined: (1) a "RATE" sheet, for rates by country; (2) a "PROB-local" sheet, for proportions or ratios by country; and (3) a "PROB-global" sheet, for a single proportion or ratio that applied to all countries.

Each sheet consisted of four tables for entering: (1) the rate or proportion/ratio data; (2) the age distribution; (3) the sex distribution; and (4) if applicable, the duration. Using a drop-down menu, different formats could be selected for entering the input parameters, including a mean and 95% confidence interval; minimum, most likely and maximum percentiles; the shape and rate of a Gamma distribution (for rates); and the shape parameters of a Beta distribution (for proportions). Gamma and Beta distributions were chosen because their domains correspond to that of rates and proportions, respectively, and because their parameters have an intuitive interpretation (i.e., number of cases and sample size, respectively, number of positives and number of negatives). Likewise, different levels of stratification could be selected for the duration parameters (i.e. none, by age only, by sex only, by age and sex). Age distribution, sex distribution and duration were allowed to vary by country, by defining different "groups" and assigning countries to "groups".

4.7.3 Imputation

Extrapolation or imputation models may be needed when literature searches cannot provide essential epidemiological data such as incidence or mortality rates [159]. These models estimate parameters based on data of neighboring regions or other time periods. The external data used must thus be representative of the selected population, region and time. The CTF developed, tested and evaluated several possible approaches to impute missing incidence data at the country level [79]. This exercise identified several pitfalls in the use of explanatory covariates, such as the potential for overfitting and the arbitrariness in the selection of covariates. Therefore, and further motivated by a strive for parsimony and transparency, we decided to use a log-Normal random effects model as the default model for imputing missing country-level incidence data. We used the subregions as defined in Appendix 2 as the random effect or cluster variable. This model assumes that the log-transformed incidence rate in country j belonging to subregion i arises from a Normal distribution with subregion specific mean μ_i and a within-region (= between-country) variance σ_w^2. Each subregion specific mean μ_i is in turn assumed to arise from a Normal distribution with mean μ_0 and a between-region variance σ_b^2:

$$\log(\theta_{ij}) \sim Normal(\mu_i, \sigma_w^2)$$

$$\mu_i \sim Normal(\mu_0, \sigma_b^2)$$

After fitting this hierarchical random effects model to the available data, incidence values for countries with no data were imputed based on the resulting posterior predictive distributions. In other words, we represented missing incidence data by log-Normal distributions based on the fitted mean and variance parameters. For countries in a subregion where none of the countries had data, the log-incidence was imputed as multiple random draws

[7] Microsoft Corp., Redmond, Washington, USA

from a Normal distribution with mean equal to the fitted global intercept μ_0 and variance equal to the sum of the fitted between-region variance σ_b^2 and the fitted within-region variance σ_w^2 (thus imputing the log-incidence as that of a "random" country within a "random" subregion, with the uncertainty interval describing the variability between and within subregions):

$$\log(\theta_{ij}^*) \sim Normal(\mu_0, \sigma_b^2 + \sigma_w^2)$$

For countries in a subregion where at least one of the other countries had data, the log-incidence was imputed as multiple random draws from a Normal distribution with mean equal to the fitted region-specific intercept μ_i and variance equal to the fitted within-region variance σ_w^2 (thus imputing the log-incidence as that of a "random" country within the concerned subregion, with the uncertainty interval describing the variability within subregions):

$$\log(\theta_{ij}^*) \sim Normal(\mu_i, \sigma_w^2)$$

When countries were considered free from exposure through the food chain, they were excluded from the imputation model and thus did not contribute to the subregional estimates. This was the case for *Brucella* spp., as discussed in [168], and foodborne trematodes and *Echinococcus* spp., as discussed in [261]. For countries with available incidence data, no imputation was performed. The incidence data used in the probabilistic burden assessments were thus a combination of actual data and imputed estimates. No additional step had to be included to correct incidence data for potential underreporting, as this was already captured by the previous steps of the framework. Indeed, for the hazards that used an attributional model, disease envelopes were used that had already been corrected for underreporting, while for other hazards we directly drew on GBD 2010 estimates (Table 5). For the remaining hazards, either epidemiological data were used that did not need (further) correction, or the underreporting factor was included in the disease model (which was the case for *Trichinella* spp. and cyanide in cassava).

For aflatoxin, the same random effects model was used to extrapolate PAFs, but now using logit-transformed instead of log-transformed values.

The model was implemented in a Bayesian framework, using independent Normal(0, 1e5) priors for μ_0 *and* μ_i; a Uniform(0, 10) prior for σ_w; and a Folded-t(1) prior for σ_b, as suggested by Gelman [160]. Sensitivity analyses using Gamma priors for the variance parameters did not yield meaningful differences. The model was run in JAGS [161] through the 'rjags' package in R [162]. After a burn-in of 5000 iterations, another 5000 iterations were retained for inference. Two chains were run, and convergence was ascertained through density and trace plots, and the multivariate potential scale reduction factor (or Brooks-Gelman-Rubin diagnostic).

A crucial assumption made by this imputation model is that missing data were considered "missing at random" (MAR), meaning that missingness was independent of the unobserved data, given the observed data [163, 164]. In our case, this assumption implied that, within each subregion, countries with data provided unbiased information on those without data, and that, across subregions, subregions with data provided unbiased information on those without data. However, for five hazards (*Bacillus cereus, Clostridium perfringens, Clostridium botulinum, Staphylococcus aureus* and peanut allergens), only data from high-

income subregions, i.e. subregions A or B, could be retrieved. In those instances, the assumption of MAR was clearly violated, and it was decided not to extrapolate those data to the rest of the world. As a result, those hazards were excluded from the global burden of disease estimates.

Table 5 shows which imputation strategy was used for each of the included hazards. For the four intoxications, peanut allergens and cyanide in cassava, the default random effects model was not used because of the limited number of data points. Instead, the burden for each concerned country was imputed as draws from a Uniform distribution defined by the lowest and highest globally observed incidence or mortality rates. To ensure consistency with results of the Child Health Epidemiology Reference Group (CHERG), alternative imputation approaches were applied for estimating aetiological fractions for the eleven diarrhoeal agents [40, 50, 168]. For seven other hazards, no imputation had to be performed because data were used that had already been imputed. This was the case for hepatitis A virus, *Salmonella* Typhi, *Salmonella* Paratyphi, *Ascaris* spp. and *Taenia solium*, for which GBD2010 data were used, and for *Mycobacterium bovis* and *Trichinella* spp., for which other published data were used [84, 165].

4.7.4 Probabilistic burden assessment

For each hazard, incidence, mortality, YLD, YLL and DALY rates were calculated for 11 age groups (<1; 1-4; 5-14; 15-24; 25-34; 35-44; 45-54; 55-64; 65-74; 75-84; ≥85) and both sexes. When necessary, age and sex specific rates were obtained by multiplying the overall rates with outcome specific age and sex distributions. The reference year for the calculation of absolute numbers was 2010, with population estimates obtained from the 2012 revision of the United Nations World Population Prospects [166]. All estimates were generated per country, and subsequently aggregated per subregion, per region, and globally (Appendix 2).

The duration component of the YLDs is defined as the average observed duration until remission or death. For calculating YLDs when duration was lifelong, we therefore used the country-specific life expectancy (LE) [166] as duration. The time component of the YLLs, on the other hand, is defined as the ideal residual life expectancy a person would have if the world would be free from disease and provide maximal access to health care. We used the highest projected LE for 2050 as normative LE for calculating YLLs [4]. This LE table has a LE at birth of 92, higher than that of the LE tables used in the GBD studies, which were based on current death rates [1, 6]. Since even for the lowest observed death rates there are a proportion of deaths which are preventable or avertable, the highest projected LE for the year 2050 was deemed to better represent the maximum life span of an individual in good health, while acknowledging that it may still not represent the ultimate achievable human life span [166].

In line with current global burden of disease assessments, no age weighting or time discounting was applied [4, 6]. HIV infected invasive salmonellosis cases and deaths, and HIV infected *M. bovis* deaths, were excluded from the burden estimates. No further corrections were made for possible co-morbidities.

Parameter uncertainty was taken into account by performing the burden assessments in a probabilistic framework. Ten thousand Monte Carlo (parametric bootstrap) simulations of the input parameters were generated to calculate 10,000 estimates of incidence, mortality, YLD, YLL and DALY rates. These 10,000