degree of underestimation is difficult to determine as *konzo* occurs in rural areas, often under conditions of war, and the disease is not notifiable. The only reported calculation of underestimation was that of Tylleskar [91] in the DRC in 1994, when he estimated that at least twice as many cases may have occurred as those reported. The underestimation in the DRC is likely to be much greater more recently, due to war and displacement. It was therefore decided to account for the uncertainty in the underreporting by applying an expansion factor ranging uniformly from 1 to 10 to the observed cases. The mean annual incidence rate was therefore estimated as 0.9/100 000 (0.04 to 1.8/100 000). This estimate of the burden of *konzo* is restricted to the 5 African countries described above, and Angola. The decision to include Angola is based on a report to the World Congress on Neurology suggesting that cases have occurred in that country [92]. Although cassava consumption occurs in tropical areas throughout the world, the term *konzo* has only been used to describe cases in Africa. The incidence of *konzo* in other countries in Africa and other parts of the world is assumed to be zero.

The age of onset and gender distribution of these cases was assumed to be that observed by Tylleskar [90]. The *konzo* case-fatality ratio is approximately 21% based on four studies [90, 93– 95]. The age and gender distribution of fatal cases was assumed to be that of Tshala-Katumbay [93].

The onset of paraparesis in *konzo* is abrupt, usually within minutes or hours, with occasional progression during the first days of the illness. After that time, the paraparesis is non-progressive and permanent. As a result, duration is defined as lifelong for non-fatal cases. For fatal cases, it was assumed that death occurred one to seven years after onset, with a most likely value of three years after onset, following Banea *et al.* [94] and Tylleskar *et al.* [96].

There is no DW specifically for *konzo*. WHO defined three severity levels for *konzo*: (1) Mild = able to walk without support; (2) Moderate = uses one or two sticks or crutches to walk; and (3) Severe = not able to walk [89]. The GBD2010 DWs for mild, moderate, and severe motor impairment are 0.012, 0.076 and 0.377, respectively [82]. The distribution of *konzo* severity among 753 patients from nine different studies were mild (63%), moderate (27%) and severe (10%) [91, 93, 94, 96– 101]. This distribution and the DWs described above were used to assign a disability weight of 0.065 to *konzo*.

4.4.2 Peanut allergen

Prevalence data on peanut [*Arachis hypogaea*] allergy were used to make estimates of incidence, since allergy occurs early in life (<5 years) and is believed to be lifelong [102– 106]. All peanut allergy cases are assumed to be the result of eating peanuts or peanut products. In western countries, the prevalence of clinical peanut allergy in children is 0 to 1.8% of the population [102], corresponding to incidence rates of 0 to 22.6 per 100 000. Limited data exist on the mortality rate of peanut-induced anaphylaxis, but the majority of studies found similar rates, ranging from 0 to 0.006 deaths per 100 000 person-years [102]. Incidence was estimated only for the A level (high income) subregions; too few data exist to make estimates for other subregions [102]. Several studies have reported that 63– 66% of cases are male [102], but given the uncertainty in this number, the gender distribution was assumed to be equal for the burden of disease calculations. No DW exists for peanut allergy. Mullins *et al.* [103] reported that

52% of cases referred to a specialist allergy medical practice in Australia suffered from mild symptoms (skin and subcutaneous tissue involvement only), 42% from moderate symptoms (features suggestive of respiratory, cardiovascular or gastrointestinal involvement), and 6% from severe symptoms (cyanosis, hypotension, confusion, collapse, loss of consciousness, incontinence).
The DW for peanut allergy was assigned as a weighted average accounting for this severity distribution. GBD2010 DWs [82] for the health states defined in the category "Asthma: controlled" (DW=0.009) are considered applicable for mild and moderate cases (94%), and "Generic uncomplicated disease: anxiety about the diagnosis" (DW=0.054) for severe cases (6%), because anxiety is known to affect Quality of Life in food allergic patients [107], leading to a severity-weighted DW of 0.012 for clinically relevant peanut allergy. Unlike other childhood allergies, such as cow's milk and egg allergy, peanut allergy rarely resolves [108, 109].

4.4.3 Aflatoxin

Aflatoxins are secondary metabolites of the fungi *Aspergillus flavus* and *A. parasiticus*, and less frequently other *Aspergillus* species such as *A. nomius* [110]. These species are prevalent in food crops – particularly maize, peanuts (groundnuts), oilseeds and tree nuts – in tropical and subtropical regions worldwide [110]. It is believed that all aflatoxin exposure results from food consumption. A multiplicative model was assumed for the effects of aflatoxin exposure and hepatitis B virus (HBV) infection on hepatocellular carcinoma. Aflatoxin exposure by country is that described by Liu and Wu [110]. To account for differences in background rates between the study population from which the cancer potency factor was derived [111] and global populations, the population attributable fractions (PAFs) by country were estimated, and applied to HCC incidence and mortality based on information from WHO [112, 113].
A Bayesian log-normal random effects model [79] was used to extrapolate available PAFs to countries without data. Age-specific incidence estimates were derived from a study in China comparing age-specific incidence of HCC in Qidong, a city in China with high aflatoxin exposure, and Beijing, a city with low aflatoxin exposure [114]. The YLD and YLL envelopes for HCC available from WHO were multiplied by the proportion of the burden due to aflatoxin. Thus no DW was directly involved in the calculation.

4.4.4 Dioxin

Dioxins are mainly by-products of industrial processes, but can also result from natural phenomena, such as volcanic eruptions and forest fires. More than 90% of human exposure is through food, mainly meat and dairy products, fish and shellfish [115]. Due to the bio-accumulating and lipophilic characteristics of dioxins, daily dietary exposure leads to accumulation of these compounds in human body fat. In adults this accumulation is thought to reach a constant level (i.e. a steady state). Consequently, the dioxin body burden, rather than the daily exposure, is taken as the dose metric for chronic toxicity risk and the assessment of dioxins [116– 121]. In this context the dioxin concentration in breast milk fat directly reflects the concentration in body fat [121– 124].

Many national authorities have programmes in place to monitor dioxin in the food supply and breast milk [124– 126]. Dioxin-induced pre-natal and post-natal hypothyroidy and pre-natally induced reduced sperm production have been found to be the most sensitive

non-cancer toxic endpoints for dioxins. Estimates for dioxin-induced pre-natal and post-natal hypothyroidy and reduced fertility due to disturbed sperm formation were based on an exposure assessment, toxicity assessment and the comparison of both assessments [127, 128]. The exposure assessment is based on breast milk concentrations of dioxin from 50 countries [129]. The toxicity assessment utilizes the benchmark dose (BMD) approach [130– 132] in which the dose response of post-natal total thyroxine (TT; decrease of TT4 in adult blood), pre-natal thyroid stimulating hormone (TSH; increase in TSH in neonatal blood), and sperm production (reduced concentration of sperm cells) is analysed. The toxicity and exposure assessments are compared to derive the transgression of a dioxin-induced decrease in TT4, decrease in sperm cell count and increase in TSH across a physiological threshold indicating a disease status (i.e. incidence of hypothyroidy or impaired fertility). Additional details of these assessments may be found in Zeilmaker *et al.* [133]. The BMD analysis was performed on studies that served as the starting point for the derivation of a Tolerable Weekly Intake (TWI) [117– 120] or Reference Dose for dioxin (RfD) [121].

In a study of a mother-child cohort, Baccarelli *et al.* determined the relationship between maternal plasma dioxin concentration and TSH level [134]. A BMD analysis of these data resulted in a population distribution of the maternal body burden of dioxin corresponding to an increased TSH level of 5 µU/mL in offspring, a level not to be exceeded in 3% of newborns in iodine-replete populations [135].

Following administration of an acute oral dose to pregnant Long Evans rats on day 15 of gestation, Gray *et al.* measured the reduction in cauda epididymis sperm count in male offspring [136]. The resulting dose response data were used to calculate a BMD lower confidence limit (BMDL) and upper confidence limit (BMDU) dioxin body burden for various levels of reduction in sperm count. A WHO reference cut-off value for impaired fertility of 20 ×106 sperm cells/mL was used to link toxicity (sperm count reduction) to a disease status (impaired fertility) (i.e. the calculation of the probability of a male being born with dioxin-impaired fertility) [137].

A BMD analysis of a National Toxicology Program (NTP) two-year feeding study in rats was used to make estimates of dioxin-induced thyroid toxicity. The NTP study administered 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) [138] and 2,3,4,7,8-pentachlorodibenzofuran [139] for periods of 14, 31 and 53 weeks. The concentrations were converted to Toxic Equivalent Quotients [140] to enable a combined analysis of both congeners. BMDL and BMDU body burdens for reduction in TT4 were calculated for each of the exposure periods. A distribution of TT4 in human blood has been reported by Aoki *et al.* [135]. The 5th percentile of this distribution (65 nmol/L) was used as the cut-off for overt clinical hypothyroidism in adults.

The results of the BMD analyses and the breast milk concentrations for 50 countries were compared, taking account of possible differences between experimental animals and humans and among individual humans [127, 128]. This comparison provided country-specific estimates of the incidence of dioxin induced pre-natal and post-natal hypothyroidy and impaired fertility. The estimates were extrapolated to other countries for which no breast milk concentrations were available, by means of Bayesian random effects modelling [79].

4.5 Outcomes and disability weights

DALYs incorporate the severity of health states through the DW, reflecting the corresponding relative reduction in healthy life on a scale from zero to one. Table 1 lists the DWs used for the health states associated with each hazard. Further details are given in Appendix 5 – Structuring of the health states into disease models for computation, and Appendix 6 – Sources and derivation of DWs.

DWs for several health states have been derived for the GBD studies and for various national burden of disease studies [141]. To ensure comparability, the CTF adopted the DWs that were used for WHO's Global Health Estimates [4]. These DWs were based on those derived for the GBD 2010 study [82], but with an alternative value for primary infertility (i.e. 0.056 instead of 0.011). The latter revision was motiviated by an analysis showing that the GBD 2010 weights undervalued the health states associated with fertility [4]. For dioxin-induced hypothyroidy, the GBD2013 DW for hypothyroidy was adopted, as this health state was not included in the GBD2010 DW study [142].

Several FBDs present with unique clinical signs, for which no DWs have been derived. Acute trichinellosis, for instance, typically presents with myalgia and facial oedema, for which no specific DWs are available [84]. When DWs were missing, proxy health states were selected by a medical expert and DW expert in the CTF and confirmed by disease experts in the hazard-specific TFs.
In other instances, DWs were available for severity levels that were not explicitly considered in the disease models.
For diarrhoea, for instance, DWs were available for mild, moderate and severe diarrhoea, although the disease models only included diarrhoea as such. In those cases, weighted averages were calculated based on published reviews of severity distributions, avoiding an over- or under-estimation of YLDs that would occur if only one DW would have been selected.

Table 1. FERG hazards, causally related health states and corresponding disability weights (DWs). Details on the derivation of the DWs are provided in Appendix 4.

HAZARD	HEALTH STATE	DW
DIARRHOEAL DISEASE AGENTS		
Norovirus	Diarrhoeal disease	0.074
Campylobacter spp.	Diarrhoeal disease	0.101
	Guillain-Barré syndrome	0.445
Enteropathogenic E. coli	Diarrhoeal disease	0.074
Enterotoxigenic *E. coli*	Diarrhoeal disease	0.074
Shiga toxin-producing *E. coli*	Diarrhoeal disease	0.091
	Haemolytic uraemic syndrome	0.210
	End-stage renal disease	0.573
Non-typhoidal *S. enterica*	Diarrhoeal disease	0.101
	Invasive salmonellosis	0.210
Shigella spp.	Diarrhoeal disease	0.101
Vibrio cholerae	Diarrhoeal disease	0.194
Cryptosporidium spp.	Diarrhoeal disease	0.074
Entamoeba histolytica	Diarrhoeal disease	0.074
Giardia spp.	Diarrhoeal disease	0.074
INVASIVE INFECTIOUS DISEASE AGENTS		
Hepatitis A virus	Hepatitis	0.108
Brucella spp.	Acute brucellosis	0.108
	Chronic brucellosis	0.079
	Orchitis	0.097
Listeria monocytogenes, perinatal	Sepsis	0.210
	Central nervous system infection	0.426
	Neurological sequelae	0.292
Listeria monocytogenes, acquired	Sepsis	0.210
	Central nervous system infection	0.426
	Neurological sequelae	0.292
Mycobacterium bovis	Tuberculosis	0.331
Salmonella Paratyphi	Paratyphoid fever	0.210
	Liver abscesses and cysts	0.254
Salmonella Typhi	Typhoid fever	0.210
	Liver abscesses and cysts	0.254
Toxoplasma gondii, congenital	Intracranial calcification	0.010
	Hydrocephalus	0.360
	Chorioretinitis, early in life	0.033
	Chorioretinitis, later in life	0.033
	CNS abnormalities	0.360
Toxoplasma gondii, acquired	Chorioretinitis, mild	0.004
	Chorioretinitis, moderate	0.033
	Chorioretinitis, severe	0.191
	Acute illness	0.053
	Post-acute illness	0.254
ENTERIC INTOXICATIONS		
Bacillus cereus [1]	Acute intoxication	0.061
Clostridium botulinum [1]	Moderate/mild botulism	0.198
	Severe botulism	0.445

HAZARD	HEALTH STATE	DW
Clostridium perfringens (1)	Acute intoxication	0.061
Staphylococcus aureus (1)	Acute intoxication	0.061
CESTODES		
Echinococcus granulosus, cases seeking treatment	Pulmonary cystic echinococcosis	0.192
	Hepatic cystic echinococcosis	0.123
	CNS cystic echinococcosis	0.221
Echinococcus granulosus, cases not seeking treatment	Pulmonary cystic echinococcosis	0.015
	Hepatic cystic echinococcosis	0.012
	CNS cystic echinococcosis	0.054
Echinococcus multilocularis	Alveolar echinococcosis	0.123
Taenia solium	Epilepsy: treated, seizure free	0.072
	Epilepsy: treated, with recent seizures	0.319
	Epilepsy: severe	0.657
	Epilepsy: untreated	0.420
NEMATODES		
Ascaris spp.	Ascariasis infestation	0.030
	Mild abdominopelvic problems due to ascariasis	0.012
	Severe wasting due to ascariasis	0.127
Trichinella spp.	Acute clinical trichinellosis	0.637
Trematodes		
Clonorchis sinensis	Abdominopelvic problems due to heavy clonorchiosis	0.123
Fasciola spp.	Abdominopelvic problems due to heavy fasciolosis	0.123
Intestinal flukes (2)	Abdominopelvic problems due to heavy intestinal fluke infections	0.123
Opisthorchis spp.	Abdominopelvic problems due to heavy opisthorchiosis	0.123
Paragonimus spp.	Central nervous system problems due to heavy paragonimosis	0.420
	Pulmonary problems due to heavy paragonimosis	0.132
ORGANIC POLLUTANTS		
Dioxin	Infertility	0.056
	Hypothyroidy due to pre-natal exposure	0.019
	Hypothyroidy due to post-natal exposure	0.019
TOXINS AND ALLERGENS		
Aflatoxin	Hepatocellular carcinoma: diagnosis and primary therapy	0.294
	Hepatocellular carcinoma: metastatic	0.484
	Hepatocellular carcinoma: terminal phase with medication	0.508
	Hepatocellular carcinoma: terminal phase without medication	0.519
Cyanide in cassava	*Konzo*	0.065
Peanut allergens (1)	Living with peanut-induced allergy	0.012

Notes: (1) Excluded from global burden assessments. (2) Includes *Echinostoma* spp., *Fasciolopsis buski*, *Heterophyes* spp., *Metagonimus* spp. and other foodborne intestinal trematode species.

4.6 Attribution

Overall, the study was designed to provide estimates of the proportion of illness acquired through different major routes of exposure. Major exposure routes considered were: food, environmental (water, soil, air), human-to-human transmission, direct animal contact, and a variety of potential lead exposure sources. Exposure route attribution estimates were developed for 19 individual hazards for each of the fourteen subregions (Table 2). Three hazard-based TFs within FERG (EDTF, PDTF and CTTF) identified, from their prioritized lists of hazards, those to be included in the expert elicitation.

Certain hazards were considered 100% foodborne, i.e. *Listeria monocytogenes*, *Mycobacterium bovis*, all foodborne trematodes, *Taenia solium*, *Trichinella* spp., cyanide in cassava and peanut allergens. For aflatoxin, inorganic arsenic, cadmium, dioxin and methyl mercury, CTTF determined that adequate data on foodborne exposure existed to allow use of a risk assessment approach for estimating the foodborne disease burden, thus negating the need for attribution. The remaining hazards were included in the structured expert elicitation (Table 1).

Fish-borne trematodes and *Trichinella* spp. were assumed to be 100% foodborne, based on the nature of their life cycle. In addition, *Fasciola* spp. were assumed to be 100% foodborne, although there may be small opportunities for waterborne transmission [77, 143]. *Taenia solium* cysticercosis was assumed to be 100% foodborne, but indirectly. In other words, the *T. solium* life cycle cannot persist without foodborne transmission of the parasite between pigs and humans. Humans become infected by the adult stage of *T. solium* by eating pork, resulting in intestinal taeniosis. However individuals who have *T. solium* taeniosis infect themselves or others by eggs excreted in their faeces, which are then ingested, often through food contamination, resulting in cysticercosis. In the complete absence of pork consumption, there would be no *T. solium* taeniosis and hence no cysticercosis.

The regions selected for this study were based on mortality. Six general regions: Africa (AFR), the Americas (AMR), the Eastern Mediterranean (EMR), Europe (EUR), South-East Asia (SEAR) and the Western Pacific (WP) were then divided into subregions on the basis of child and adult mortality, where Stratum A = very low child and adult mortality; Stratum B = low child mortality and very low adult mortality; Stratum C = low child mortality and high adult mortality; Stratum D = high child and adult mortality; and Stratum E = high child mortality and very high adult mortality [5, 144].

4.6.1 Identification of experts

An iterative peer nomination process based on a social network sampling technique called "snowball sampling" was used to identify a pool of potential expert participants for this study. The first points of contact were identified through FERG members and other networks (e.g. Global Foodborne Infections Network – GFN; Global Environment Monitoring System – GEMS; International Network of Food Safety Authorities – INFOSAN; Joint FAO/WHO Expert meeting on Microbial Risk Assessment – JEMRA; Joint FAO/WHO Expert Committee on Food Additives – JECFA; European Food Safety Authority – EFSA scientific panels; and WHO regional food safety advisors). These persons were asked to use their professional networks and recognized expertise in relevant areas to nominate additional experts. Since the purpose of this process was to identify an adequately large pool of appropriate experts, rather than to identify the entire expert network, the process of referral continued until an adequate size pool was identified to fill panels of typically 8 to 12 experts per panel.

Table 2. Foodborne hazards, and structure of the expert panels.

HAZARD GROUPS	HAZARDS	PANEL STRUCTURE[a]	NO. OF PANELS
DIARRHEAL DISEASE			
Bacteria	*Campylobacter* spp., enteropathogenic *Escherischia coli* (EPEC), enterotoxigenic *E. coli* (ETEC), Shiga-toxin producing *E. coli* (STEC), non-typhoidal *Salmonella* spp., *Shigella* spp., *Vibrio cholerae*	Sub regional	7
Virus	Norovirus	Sub regional	1
Intestinal protozoa	*Cryptosporidium* spp., *Entamoeba histolytica, Giardia* spp.	Global	3
OTHER INFECTIOUS DISEASE			
Bacteria	*Brucella* spp., *Salmonella* Typhi	Global Sub regional	2
Virus	Hepatitis A virus	Global	1
Protozoa	*Toxoplasma gondii*	Global	1
Helminths	*Ascaris* spp., *Echinococcus granulosus, Echinococcus multilocularis*	Global	3
CHEMICALS			
Lead		Global	1
Total			**19**

[a] Experts on a global panel were asked to provide estimates for all 14 sub regions, whereas experts on a sub-regional panel could choose a set of sub regions depending on their expertise.

4.6.2 Selection of experts

In collaboration with the FERG hazard-based TFs, SATF defined a set of criteria for inclusion of experts. These criteria considered each expert's background (education, and current and past positions), years of experience within the field, and geographic coverage of expert within the panel. The WHO invited nominated experts to participate, and the experts were asked to complete a declaration of interests (DOI), and an expert sheet providing information on their research/working area, highest education, current position, geographical experience, and years of experience. The experts were asked also to indicate for which panel(s) they believed themselves to be best suited for. The experts were not offered any compensation for their participation. The chairs of the three hazard-based TFs and the SATF reviewed the expert's information and CVs, and a final selection was made. FERG TF chairs and members of the SATF were not eligible for the study. DOIs were evaluated by WHO.

Given the broad nature of the attribution task, care was taken to include a suitably wide range of scientific backgrounds and professional experience, and to ensure adequate geographical representation. Frequently, expert elicitations use publication record as the measure of recognized expertise [145]. However, for this study, restricting expert selection to choices based solely on publication records would have eliminated important groups of experts, in particular public-health field workers and food-safety professionals in developing countries.

4.6.3 Expert panels

The panels for *Brucella* spp., hepatitis A virus, parasitic diseases including intestinal protozoa, and lead were structured as global panels, meaning that all experts in those panels were asked to provide estimates for all fourteen subregions.
The panels for the eight bacterial and viral pathogens and *Salmonella* Typhi were structured as subregional panels. The experts on subregional panels were free to decide the subregions for which they provided their judgments. Experts participating in panels addressing more than one hazard could also choose to provide estimates only for those hazards for which they felt they had adequate expertise.

4.6.4 Analytical method

The study used Cooke's [145– 147] "Classical Model" for expert elicitation. This approach uses "calibration" or "seed" questions to develop performance weights used in aggregating experts' judgments. The paradigmatic seed question is one for which the true value is not known at the time the experts answer the question, but will be known or is expected to become known *post hoc*. So the experts are not expected to know these values, but should be able to capture a majority of them reliably by defining suitable credible intervals.

Analysis of the experts' performance on the seed variables has two main purposes: 1), to evaluate the expert's *statistical accuracy* when assigning values to probability outcomes against the seed values (i.e. how reliably the expert's credible interval responses capture the true values of the seed variables, statistically), and 2), to evaluate the expert's *informativeness* when providing uncertainty distributions over the seed variables (i.e. how concentrated (narrow) are the distributions provided). Experts are thus scored with regard to statistical accuracy (calibration score) and informativeness (information score). The statistical accuracy is measured as the p-value at which one would falsely reject the hypothesis that the expert's probability assessments were statistically accurate, and informativeness is measured as Shannon relative information with respect to a user-supplied background measure. Informativeness scores are not absolute, but relative to a set of experts assessing the same variables. The calculated calibration and information scores are used to aggregate experts' judgments on target variables. The same measures can be applied to any combination of the experts' assessments to implement criteria for aggregating the assessments.

The Cooke Classical Model provides a rigorous, quantitative means for estimating model parameters and their uncertainties and is the only elicitation procedure that has objective empirical control on expert scoring. Moreover, it allows formal optimization of aggregated uncertainty distributions in terms of statistical accuracy and informativeness [146]. The expert judgment processing software EXCALIBUR (http://www.lighttwist.net/wp/excalibur) also allows direct comparison of the results that would be obtained from unweighted aggregation of expert judgments versus those produced by weighted linear pooling (or other combination schemes).

4.6.5 Seed questions

It is not always possible to develop seed questions that are in the paradigmatic form of asking about a future event or measurement that has not been made, but could be made, in principle. The essential feature of a viable seed question is that the expert is not expected to know the exact value but, if they are a subject-

matter expert, should be able to define a narrow uncertainty range that captures the value. Therefore, an alternative is to ask about selected data or values in the topic domain, about which the expert will not have perfect knowledge, nor access to realization values at the time they are answering the seed questions, but for which the values are known to the analyst. Such "retrospective" questions are frequently used in expert elicitations applying the Cooke Classical Model (see e.g. [31, 148]).

In the present case, the seed questions formulated were a mixture of retrospective and prospective seed variables. It is possible that expert uncertainty judgments vary by subject matter domain. In this study, the possibility of such biases relevant to foodborne illness source attribution was of concern. Therefore, the seed questions were designed to focus on questions that are substantively related to foodborne illness source attribution. Further, to account for the wide range of scientific backgrounds and experiences, seed questions covered a range of substantive topics relevant to source attribution. Five main categories of seed questions were identified for the panels on biological hazards (diarrhoeal pathogens and parasites): (1) dietary patterns and food supply; (2) under 5 years mortality rate; (3) access to improved water and sanitation; (4) disease surveillance; and (5) systematic reviews related to these and other scientific topics relevant to source attribution. For the panel on lead, questions were categorized as: (1) mean blood levels; (2) dietary exposure; and (3) dietary patterns and food supply. Examples of seed questions are presented in Table 3.

Table 3. Examples of calibration seed questions

TOPIC	HAZARD	QUESTION
Dietary patterns and food supply	All microbial hazards	Among all subregions in 2010, what was the proportion of regional vegetable supply (tonne) that was imported rather than produced domestically in the subregion with the highest such percentage?
Under 5 mortality rate	*Brucella* spp., *Echinococcus* spp., intestinal protozoa, diarrhoeal pathogens	Based on WHO estimates, think of the country in the African Region that had the largest percentage point decrease from 2000 to 2010 in all-cause <5 mortality due to diarrhoea. What was that percentage point decrease?
Disease surveillance	*Ascaris* spp., *Echinococcus* spp., intestinal protozoa, hepatitis A virus, diarrhoeal pathogens (developed subregions only)	What will be the rate per 100 000 population of laboratory-confirmed human cases of *Campylobacter*iosis in 2012 in all EU member states as reported in EFSA's annual report?
Systematic review	All microbial hazards	Fewtrell *et al.* (2005) conducted a systematic review and meta-analysis to compare the evidence of relative effectiveness of improvements in drinking water, sanitation facilities and hygiene practices in less developed countries in reducing diarrhoeal illness. The meta-analysis of 5 studies was used to estimate the relative risk of diarrhoeal illness with and without multiple interventions. What was the estimated relative risk?
Mean blood level	Lead	What was the geometric mean blood lead concentration for all participants ages 1 year and older in the 2007- 2008 U.S. NHANES survey? Please express your answer as positive micrograms per deciliter (μg/dL).