West Papua are known to be highly endemic regions for *T. solium* and hence the combined population of these provinces was used as the population at risk, with the remainder of Indonesia having zero risk.
- (7) There is no FAO data on pig populations in Sudan or South Sudan, the latter being predominantly non-Muslim and with little improved sanitation. However, extensive searches for information about pigs in Sudan revealed that the domestic pig population in both countries is negligible and hence there is virtually zero risk if cysticercosis.

Due to the absence of available data on all cysticercosis sequelae, only the frequency of NCC-associated epilepsy was estimated in this study.

Clinical Outcomes

Epilepsy-associated NCC [74]

Duration

No data.

Disability weight

- GBD2010 for epilepsy [58, 81–83]

Mortality

GBD2010 for epilepsy [58, 81–83]

Age distribution

GBD2010 for epilepsy [58, 81–83]

Sex distribution

GBD2010 for epilepsy [58, 81–83]

A4.28 Chlonorchiosis

Incidence

Incidence estimates and clinical sequelae for foodborne trematodiasis were mainly based on the results of two systematic review articles [77, 78]. The reviews identified available qualitative and quantitative information on prevalence, incidence, mortality and remission rates, sex- and age-distributions and the progression of foodborne trematodiasis into different sequelae. From these data, simplified disease models were developed and quantitative data summarized by meta-analyses. As information on incidence, remission, and duration of foodborne trematodiasis was particularly scant, zero remission was assumed and entered into the DisMod 3 software [304], together with the available prevalence and mortality estimates. DisMod 3 computed internally consistent and complete sets of sex-, age- and country-specific prevalence, incidence, remission, duration and mortality for foodborne trematodiasis and associated sequelae. However, unlike the original study, which computed incidence rates only for countries reporting national prevalence rates, and otherwise considered the incidence rate to be zero [77], the present study also imputed incidence rates for countries where no records of national prevalence or incidence rates were available, but at least one autochthonous human infection could be identified in the systematic review. Hierarchical random-effects models with incidence information from other countries as input data were applied in this additional imputation process [79].

Clinical Outcomes

Abdominal pelvic discomfort, carcinoma.

Duration

Lifelong due to low treatment coverage in affected populations, longevity of parasites in humans, high re-infection rates, supposedly high susceptibility of clinical cases, and irreversibility of pathology after several years of infection.

Disability weight

- Only for severe infections: 0.123.

Mortality

1% case fatality.

Age distribution

AGE DISTRIBUTION (YEARS)	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
G1Mean	5.0	8.1	11.5	13.4	22.7	12.4	13.9	8.1	2.9	1.1	0.6	0.2	0.0
G2 Mean	4.3	7.5	10.4	12.0	18.8	13.5	16.7	8.0	3.8	2.4	1.4	0.8	0.3
G3 Mean	4.1	7.2	9.1	13.0	32.9	18.6	12.9	1.6	0.3	0.1	0.1	0.1	0.1
G4Mean	6.8	8.8	11.2	12.4	23.5	12.2	13.4	7.6	2.6	0.9	0.5	0.2	0.0
G5Mean	5.1	8.2	11.4	13.2	22.6	12.5	14.0	8.0	2.9	1.1	0.6	0.3	0.1

Sex distribution

Male 65–68%.

A4.29 Fasciolosis

Incidence

Incidence estimates and clinical sequelae for foodborne trematodiasis were mainly based on the results of two systematic review articles [77, 78]. The reviews identified available qualitative and quantitative information on prevalence, incidence, mortality and remission rates, sex- and age-distributions and the progression of foodborne trematodiasis into different sequelae. From these data, simplified disease models were developed and quantitative data summarized by meta-analyses. As information on incidence, remission and duration of foodborne trematodiasis was particularly scant, zero remission was assumed, and entered into the DisMod 3 software [304], together with the available prevalence and mortality estimates. DisMod 3 computed internally consistent and complete sets of sex-, age- and country-specific prevalence, incidence, remission, duration and mortality for foodborne trematodiasis and associated sequelae. However, unlike the original study, which computed incidence rates only for countries reporting national prevalence rates and otherwise considered the incidence rate to be zero [77], the present study also imputed incidence rates for countries, where no records of national prevalence or incidence rates were available, but at least one autochthonous human infection could be identified in the systematic review. Hierarchical random-effects models with incidence information from other countries as input data were applied in this additional imputation process [79].

Clinical Outcomes

Abdominal pelvic discomfort.

Duration

Lifelong due to low treatment coverage in affected populations, longevity of parasites in humans, high re-infection rates, supposedly high susceptibility of clinical cases, and irreversibility of pathology after several years of infection

Disability weight

- 0.123

Mortality

Zero.

Age distribution

AGE DISTRIBUTION (YEARS)	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
G1 Mean	49.1	26.9	11.6	4.6	2.3	1.4	1.8	1.0	0.6	0.3	0.2	0.1	0.0
G2 Mean	19.5	33.1	29.5	10.4	4.3	1.4	1.0	0.4	0.2	0.1	0.0	0.0	0.0
G3 Mean	75.3	14.8	5.1	1.8	0.9	0.6	0.7	0.4	0.2	0.1	0.1	0.0	0.0
G4 Mean	21.9	52.6	16.1	1.3	0.5	0.7	1.5	1.3	1.1	0.8	0.7	0.7	0.7
G5 Mean	59.1	22.0	9.4	3.6	1.8	1.1	1.3	0.7	0.4	0.2	0.2	0.1	0.0

Sex distribution

Male = 49.5%

A4.30 Opisthorchosis

Incidence

Incidence estimates and clinical sequelae for foodborne trematodiasis were mainly based on the results of two systematic review articles [77, 78]. The reviews identified available qualitative and quantitative information on prevalence, incidence, mortality and remission rates, sex- and age-distributions and the progression of foodborne trematodiasis into different sequelae. From these data, simplified disease models were developed and quantitative data summarized by meta-analyses. As information on incidence, remission and duration of foodborne trematodiasis was particularly scant, zero remission was assumed and entered into the DisMod 3 software [304], together with the available prevalence and mortality estimates. DisMod 3 computed internally consistent and complete sets of sex-, age- and country-specific prevalence, incidence, remission, duration and mortality for foodborne trematodiasis and associated sequelae. However, unlike the original study, which computed incidence rates only for countries reporting national prevalence rates and otherwise considered the incidence rate to be zero [77], the present study also imputed incidence rates for countries, where no records of national prevalence or incidence rates, but at least one autochthonous human infection, could be identified in the systematic review. Hierarchical random-effects models with incidence information from other countries as input data were applied in this additional imputation process [79].

Clinical Outcomes

Abdominal pelvic discomfort, carcinoma.

Duration

Lifelong due to low treatment coverage in affected populations, longevity of parasites in humans, high re-infection rates, supposedly high susceptibility of clinical cases, and irreversibility of pathology after several years of infection

Disability weight

- 0.123

Mortality

Overall case fatality rate: 9.2%.

Age distribution

AGE DISTRIBUTION (YEARS)	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
G1 Mean	4.2	11.8	13.9	12.4	10.7	8.7	13.4	10.3	7.1	3.9	2.4	0.9	0.2
G2 Mean	4.3	13.4	15.8	15.4	12.7	8.5	10.9	8.1	5.5	2.3	2.1	0.9	0.2
G3 Mean	2.3	6.8	8.4	9.6	11.6	9.6	13.7	11.6	11.1	6.2	5.6	2.9	0.5
G4 Mean	4.2	11.7	13.8	12.4	10.7	8.7	13.4	10.3	7.2	4.0	2.4	1.0	0.2

Sex distribution

Male=55%

A4.31 Paragonimosis

Incidence

Incidence estimates and clinical sequelae for foodborne trematodiasis were mainly based on the results of two systematic review articles [77, 78]. The reviews identified available qualitative and quantitative information on prevalence, incidence, mortality and remission rates, sex- and age-distributions and the progression of foodborne trematodiasis into different sequelae. From these data, simplified disease models were developed and quantitative data summarized by meta-analyses. As information on incidence, remission and duration of foodborne trematodiasis was particularly scant, zero remission was assumed and entered into the DisMod 3 software [304], together with the available prevalence and mortality estimates. DisMod 3 computed internally consistent and complete sets of sex-, age- and country-specific prevalence, incidence, remission, duration and mortality for foodborne trematodiasis and associated sequelae. However, unlike the original study, which computed incidence rates only for countries reporting national prevalence rates and otherwise considered the incidence rate to be zero [77], the present study also imputed incidence rates for countries, where no records of national prevalence or incidence rates, but at least one autochthonous human infection, could be identified in the systematic review. Hierarchical random-effects models with incidence information from other countries as input data were applied in this additional imputation process [79].

Clinical Outcomes

Lifelong due to low treatment coverage in affected populations, longevity of parasites in humans, high re-infection rates, supposedly high susceptibility of clinical cases, and irreversibility of pathology after several years of infection.

Duration

Lifelong.

Disability weights

- Pulmonary: 0.132.
- Cerebral paragonimosis: 0.42.

Mortality

10% case fatality for cerebral cases only

Age distribution

AGE DISTRIBUTION (YEARS)	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
G1 Mean	8.7	11.5	12.3	10.1	9.2	6.7	12.6	12.2	7.9	4.6	2.8	1.2	0.2
G2 Mean	8.9	16.1	16.2	12.5	9.5	7.3	11.0	7.7	5.1	3.0	1.8	0.8	0.2
G3 Mean	2.6	9.0	11.4	11.5	10.9	9.8	16.5	12.5	8.1	4.2	2.4	0.9	0.2
G4 Mean	2.0	8.1	11.5	11.4	10.7	10.6	20.1	13.4	7.2	3.3	1.2	0.5	0.2
G5 Mean	8.6	11.7	12.4	10.2	9.2	6.7	12.6	12.0	7.8	4.5	2.8	1.2	0.2

Sex distribution

Male = 55.9%

A4.32 Intestinal flukes

Incidence

Incidence estimates and clinical sequelae for foodborne trematodiasis were mainly based on the results of two systematic review articles [77, 78]. The reviews identified available qualitative and quantitative information on prevalence, incidence, mortality and remission rates, sex- and age-distributions, and the progression of foodborne trematodiasis into different sequelae. From these data, simplified disease models were developed and quantitative data summarized by meta-analyses. As information on incidence, remission and duration of foodborne trematodiasis was particularly scant, zero remission was assumed and entered into the DisMod 3 software [304], together with the available prevalence and mortality estimates. DisMod 3 computed internally consistent and complete sets of sex-, age- and country-specific prevalence, incidence, remission, duration and mortality for foodborne trematodiasis and associated sequelae. However, unlike the original study, which computed incidence rates only for countries reporting national prevalence rates and otherwise considered the incidence rate to be zero [77], the present study also imputed incidence rates for countries, where no records of national prevalence or incidence rates, but at least one autochthonous human infection, could be identified in the systematic review. Hierarchical random-effects models with incidence information from other countries as input data were applied in this additional imputation process [79].

Clinical Outcomes

Abdominal pelvic discomfort.

Duration

Lifelong due to low treatment coverage in affected populations, longevity of parasites in humans, high re-infection rates, supposedly high susceptibility of clinical cases, and irreversibility of pathology after several years of infection.

Disability weight

- Heavy infections only: 0.123.

Mortality

Zero.

Age distribution

AGE DISTRIBUTION (YEARS)	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
G1 Mean	24.4	32.7	22.1	8.3	4.3	1.9	2.5	1.8	1.0	0.5	0.3	0.2	0.0
G2 Mean	18.3	38.4	24.2	9.8	4.7	1.9	1.3	0.5	0.3	0.2	0.1	0.1	0.0
G3 Mean	70.1	10.4	6.0	2.8	1.8	1.3	2.1	1.5	1.2	1.2	0.9	0.6	0.2
G4 Mean	56.4	26.4	9.1	3.1	1.6	0.9	1.1	0.6	0.4	0.2	0.1	0.1	0.0
G5 Mean	24.8	40.8	18.6	7.7	4.5	1.7	1.0	0.4	0.2	0.1	0.1	0.1	0.0
G6 Mean	46.4	27.7	13.3	4.8	2.5	1.3	1.6	1.0	0.6	0.4	0.2	0.1	0.0

Sex distribution

Male = 55%.

A4.33 Ascaris spp.

Incidence

Age-stratified prevalence was used to estimate the burden of disease in GBD2010 [81] and these data supplied by IHME were used to estimate incidence of ascariasis. The estimated numbers of prevalent cases were available for every country and each age group.

Prevalence at age t = P(t) Prevalence at age t+1 = P(t+1) Incidence = b = proportion of population infected between t and t= t+1 P(t+1) = b*(1-P(t))-m * P(t) As proportion acquiring new infections are at a rate of b*(1-P(t)) and proportion losing infections at rate -m * P(t) Therefore incidence (proportion) of new infections is given by b=(P(t+1)+m*p(t))/1-P(t) b = proportion that are infected in time t = 1 year m = proportion that lose their infection = death rate = 1/life expectancy Approximate life expectancy of Ascaris = 1 year Therefore b=(P(t+1)+P(t))/(1-P(t)) Incidence per 100 000 per year = b *100 000 It is well known [88] that the infection pressure or incidence varies with age with ascaris. In particular children have a higher incidence. But by using this step equation all that is required is the different prevalences (proportion infected) at age t and t+1 which can be calculated from the data provided by GBD2010. Assuming the duration for ascariasis and other manifestations (mild abdominopelvic discomfort and severe wasting) are of the same duration, then this can be used to estimate the incidence of all sequelae from the stratified prevalence data. There is some evidence that ascaris induces some degree of protective immunity. But this acts to decrease the abundance of infection rather than the prevalence and so can be discounted in this exercise.

Clinical Outcomes

The clinical outcomes were death in severe cases, severe wasting, pelvic abdominal disconfort and clinical ascariasis as described in GBD2010 [82]##

Duration

Duration of each incidence case was set at a mean of 1 year

Disability weight

- For severe wasting = 0.127
- For mild abdominal pelvic discomfort = 0.012
- For clinical ascariosis = 0.296

Mortality

Incidence of mortaility due to ascariasis used the GBD2010 mortality figures. In ascaris-endemic countries this ranged from a low of 0.000095 per 100 000 per annum in Dominica to a high of 0.159 per 100 000 per annum in Equitorial Guinea. Upper-income countries had zero mortality. Globally there were 0.031 deaths per 100 000

Age distribution

Fatalities, Ascariasis and mild abdomino pelvic problems <1 year, 9%; 1-4 years, 56.7%; 5-14 years, 16%; 15-24 years, 3%; 25-34 years 2.1%; >35 years, 13.2%.

Severe wasting: <1 year, 19%; 1-4 years, 81%.

Sex distribution

Male =55.1% (fatalities), Male = 50.1% (ascaris), Male=50.2% (mild abdominal pelvic discomfort),
Male= 51% (severe wasting).

A4.34 Trichinella

Incidence

Incidence was estimated from the results of a FERG-commisioned systematic review. Full details can be found in [71]

Clinical Outcomes

In the absence of data on the probability of occurrence of the major clinical symptoms of acute trichinellosis, it was assumed, as a worst case scenario, that all patients would develop diarrhoea, facial oedema, myalgia and fever/ headache [84].

Duration

Based on the systematic review by [71], disease duration ranged from 21.5 to 70 days. These values were divided by 365 to express the duration in years.

Disability weight

Because no specific DW for acute trichinellosis is available, DWs were derived for each of the outcomes separately. The four clinical symptoms were, respectively, matched to the GBD2010 health states:

- Diarrhoea: moderate - DW = 0.202.
- Disfigurement: level 2, with itch or pain - DW = 0.187.
- Musculoskeletal problems: generalized, moderate - DW = 0.292.
- Infectious disease: acute episode, severe (DW = 0.210) [82].

These four DWs were then aggregated using the multiplicative method, which defines the aggregated DW as $1-\prod_i(1-DW_i)=0.637$ [84].

Mortality

Mortality was estimated from the results of a FERG-commisioned systematic review. Full details can be found in [71]

Age distribution

According to [71], the majority of cases were between 20 and 50 years of age, with a median of 33.1. A generalized Beta distribution was fitted to the estimates to define the full distribution of cases from age 0 to 90 years [84].

Sex distribution

According to [71], 51% of cases were male.

A4.35 Aflatoxin

Incidence

A population-attributable fraction (PAF) approach was used to estimate the incidence of aflatoxin-related hepatocellular carcinoma (HCC). We assumed a multiplicative model for the effects of aflatoxin exposure and hepatitis B virus (HBV) infection. The excess risk due to aflatoxin exposure is estimated as HCCa- = b * a for HBV-negative individuals and HCCa+ = b * h * a for HBV-positive individuals, where a = exposure to aflatoxin (ng/(kg bw * day)), b = aflatoxin cancer potency factor in HBV- individuals ((ng/(kg bw * day)-1) and h = relative risk for aflatoxin exposure in HBV+ individuals compared with HBV- individuals. We used potency factors as derived by JECFA [111]: b = 0.01 [0.002-0.03](ng/(kg bw * day)-1 and b * h = 0.30 [0.005-0.50](ng/(kg bw * day)-1. Uncertainty in the potency factors was modelled as a Gamma distribution with the most likely value as the mean and the range representing an approximate 95% confidence interval.

To account for differences in background rates between different populations, we estimated PAFs by country, and applied them to HCC incidence, based on [305]. We assumed PAFs for incidence and deaths were equal and calculated PAFs based on published studies on HCC

mortality. For the study population that was the basis of the JECFA potency estimates in Guangxi, China [306], the PAF was estimated as PAFa = ((1-p) * HCCa- + p * HCCa+)/HCC, where p = prevalence of HBV infection and HCC = total incidence of HCC by all causes. We used the HCC death rate for the Guangxi cohort, standardized to the global population (121.5 per 100 000) and calculated average exposure (607 ng/(kg bw * day) based on [[307], Table I]. HBV prevalence was 23% based on [306], resulting in PAFa = 0.383. Background death rate of HCC by all causes in the Guangxi population was calculated as HCC0,s = (HCCs-HCCa,s) / (1-ps + h * ps), with the subscript s referring to the study population; resulting in HCC0,s = 9.77 per 100 000.

To calculate attributable incidence in all countries, we estimated relative risks due to aflatoxin exposure as RRa,c = 1 + b * ac / HCC0,s, and PAFs per country as PAFa,c = (RRa,c-1) / RRa,c, with the subscript c indicating country. Attributable incidence was then calculated as HCCa,c = HCCc * PAFa,c. Aflatoxin exposure by country was based on [110], with uncertainty represented by a uniform distribution over the reported range. A Bayesian log-normal random effects model [79, 151] was used to extrapolate available PAFs to countries without data.

Clinical outcomes

Hepatocellular carcinoma.

Duration

Not applicable; population-attributable fractions as described above were directly applied to WHO YLD estimates [308].

Disability Weight

Not applicable; population-attributable fractions as described above were directly applied to WHO YLD estimates [308].

Mortality

Population-attributable fractions as described above were directly applied to WHO mortality estimates [308].

Age distribution

We compared age distributions of HCC in the populations of Beijing and Qidong from [114], and hypothesized that the main difference in HCC risk factors between these two cities is aflatoxin exposure, since every other risk factor is the same, and they are both predominantly Han (i.e. same ethnicity). Hence, the difference in age distributions was presumed to be the contribution of aflatoxin. This resulted in an average age at onset of 49.

Sex distribution

In absence of information on the sex distribution of aflatoxin-induced hepatocellular carcinoma, a 50:50 age distribution was assumed.

A4.36 Cyanide in cassava

Incidence

A total of 2376 konzo cases have been reported in 5 countries (Cameroon, Central African Republic, Democratic Republic of Congo, Mozambique and United Republic of Tanzania), corresponding to 149 cases per year for 122 million people [86]. Based on these cases and dividing the average annual number of case for each country by the corresponding country population gives an observed incidence of 0.043 to 0.179 per 100 000.

The degree of underestimation is difficult to estimate, as konzo occurs in remote rural areas, often under conditions of war, and the disease is not notifiable. The only previous calculation of underestimation was that of Tylleskar [90] in the DRC in 1994, when he estimated that there may have been at least twice as many cases as those reported. The underestimation in the DRC is now likely to be much greater, due to war and displacement. It was decided to account for the uncertainty in the underreporting by applying an expansion factor ranging from 1 to 10 to the observed cases. Therefore, the annual total of new cases would range from 149 to 1490 in the 5 countries and the mean annual incidence rate would be 0.9 per 100 000 (0.04 to 1.8 per 100 000).

We restricted our estimates of konzo disease to the 5 African countries in which the disease has been reported, together with Angola, based on a report to the World Congress on Neurology suggesting that cases have occurred in that country [92]. The incidence of konzo disease was assumed to be null in other countries around the world.

Clinical outcomes

Konzo disease is a paraparesis occurring in populations exposed to cyanogenic glycoside in a context of bitter cassava consumption associated with a low intake of protein-rich food.

Duration

The onset of paraparesis 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 was defined as lifelong for non-fatal cases. For fatal cases, it was assumed that death occurred one to seven years after onset, with an average of three years after onset, following [93].

Disability Weight

No specific DW exists for konzo paraparesis. WHO [89] defined three severity levels for konzo:

1. Mild = Able to walk without support

2. Moderate = uses one or two sticks or crutches to walk

3. Severe = not being able to walk

These three severity levels can be matched with the GBD2010 health states:

- Motor impairment, mild: DW = 0.012.
- Motor impairment, moderate: DW = 0.076.
- Motor impairment, severe: DW = 0.377 [82].

Information on the distribution of konzo severity levels is available from 9 studies [86][1]. Out of a total of 753 cases, 476 (63%) were mild, 203 (27%) were moderate and 74 (10%) were severe.

The resulting weighted DW equalled 0.065.

Mortality

Information on case fatality was provided in 4 studies [94–96, 309]. Out of a total of 340 cases, 73 deaths were observed, yielding an average case fatality ratio of 21%.

Age distribution

The age and sex distribution observed by [90] was generalized to the whole konzo affected population. The age distribution for fatal cases was adapted from [309].

Sex distribution

The age and sex distribution observed by [90] was generalized to the whole konzo affected population. The sex distribution for fatal cases was adapted from [309].

A4.37 Dioxin

Incidence

Incidence rates were generated for 50 countries and specified as lower and upper bounds (hypothyroidy-postnatal & male infertility) or point estimates (hypothyroidy-prenatal). Incidence rates for the remaining 144 countries were imputed using a Bayesian log-normal random effects model [151].

Clinical outcomes

Hypothyroidy due to prenatal exposure; hypothyroidy due to postnatal exposure; or male infertility due to prenatal exposure.

Duration

Hypothyroidy was assumed to be lifelong; the male infertility impact was assumed to be present in the 20–44 age group, in accordance with [83].

Disability Weights

- Hypothyroidy due to prenatal exposure: 0.019; corresponding to GBD 2013 health state Hypothyroidy [142]. Note that no corresponding DW was available in GBD2010 or WHO Global Health Estimates (GHE).
- Hypothyroidy due to postnatal exposure: 0.019; corresponding to GBD 2013 health state Hypothyroidy [142]. Note that no corresponding DW was available in GBD2010 or WHO GHE.
- Male infertility: 0.056; corresponding to WHO GHE health state Infertility: primary [310]. Note that this is higher than the corresponding GBD2010 health state.

Mortality

No mortality was assumed.

Age distribution

Hypothyroidy due to prenatal exposure: Onset = birth.

Hypothyroidy due to postnatal exposure: Onset = 20 years.

Male infertility: Onset = 20 years.

Sex distribution

In absence of information on the sex distribution of dioxin-induced hypothyroidy, a 50:50 age distribution was assumed.

For male infertility, the entire burden was assigned to males.

A4.38 Peanut allergens

Incidence

Data on clinically confirmed peanut [*Arachis hypogaea*] allergy in children were available from six countries (Canada, Denmark, Iceland, Sweden, Turkey and UK). Average incidences ranged from 0 to 22.6 per 100 000 [102]. To reflect this uncertainty, the incidence rate of clinical peanut allergy in subregion "A" countries was modelled as a Uniform distribution ranging from 0/100 000 to 22.6/100 000. Given the lack of data, no estimates were generated for other countries.

Clinical outcomes

The symptoms of peanut allergy vary from mild to severe, from swollen lips, shortness of breath, to an anaphylactic shock, which is potentially fatal. However, because of the very short duration of acute peanut allergy, we decided not to include acute peanut allergy in the burden assessment. The considered clinical outcome was therefore living with peanut allergy and the anxiety of a possible allergic reaction.