

THE LANCET

Neurology

Supplementary appendix

This appendix formed part of the original submission and has been peer reviewed. We post it as supplied by the authors.

Supplement to: GBD 2016 Epilepsy Collaborators. Global, regional, and national burden of epilepsy, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Neurol* 2019; published online Feb 14. [http://dx.doi.org/10.1016/S1474-4422\(18\)30454-X](http://dx.doi.org/10.1016/S1474-4422(18)30454-X).

Appendix

Summary of General Global Burden of Disease Study Methods

The Institute for Health Metrics and Evaluation with a growing collaboration of scientists produces annual updates of the Global Burden of Disease study. Estimates span the period from 1990 to the most recent completed year. By the time of the release of GBD 2016 in September 2017, there were over 2,700 collaborators in 132 countries who contributed to this global public good. Annual updates allow incorporation of new data and method improvements to ensure that the most up-to-date information is available to policy makers in a timely fashion to help make resource allocation decisions. In this analysis, we have aggregated results from GBD 2016 for 15 disease and injury outcomes that are generally cared for by neurological services. These include infectious conditions (tetanus, meningitis, encephalitis), stroke, brain and other nervous system cancers, traumatic brain injury, and spinal cord lesion which are classified outside the more narrowly defined category of neurological disorders in GBD (ie, Alzheimer's disease and other dementias, Parkinson's disease, multiple sclerosis, motor neuron disease, idiopathic epilepsy, migraine, tension-type headache, and a rest category of less common other neurological disorders). Compared to a previous analysis based on GBD 2015,¹ we were able to add the non-fatal outcomes of traumatic brain injury and spinal cord lesion, and medication overuse headache is no longer included as a separate cause but quantified as a consequence of the underlying headache types.

In the methods section of this overview paper we present a summary of the general methods of the global burden of disease. In the accompanying disease-specific papers we concentrate on methods that are specific to each disorder. The guiding principle of GBD is to assess health loss due to mortality and disability comprehensively, where we define disability as any departure from full health. In GBD 2016, estimates were made for 195 countries and territories, and 579 subnational locations, for 27 years starting from 1990, for 23 age groups and both sexes. Deaths were estimated for 264 diseases and injuries, while prevalence and incidence were estimated for 328 diseases and injuries. In order to allow meaningful comparisons between deaths and non-fatal disease outcomes as well as between diseases, the data on deaths and prevalence are summarised in a single indicator, the disability-adjusted life-year (DALY). DALYs are the sum of years of life lost (YLLs) and years lived with disability (YLDs). YLLs are estimated as the multiplication of counts of death and a standard, "ideal", remaining life expectancy at the age of death. The standard life expectancy is derived from the lowest observed mortality rates in any population in the world greater than 5 million.² YLDs are estimated as the product of prevalence of individual consequences of disease (or "sequelae") times a disability weight that quantifies the relative severity of a sequela as a number between zero (representing "full health") and 1 (representing death). Disability weights have been estimated in nine population surveys and an open-access internet survey in which respondents are asked to choose the "healthier"³ between random pairs of health states that are presented with a short description of the main features.

All-cause mortality rates are estimated from vital registration data in countries with complete coverage. For other countries, the probabilities of death before age 5 and between ages 15 and 60 are estimated from censuses and surveys asking mothers to provide a history of children ever born and those still alive, and surveys asking adults about siblings who are alive or have passed away. Using model life tables, these probabilities of death are transformed into age-specific death rates by location, year, and sex. GBD has collated a large database of cause of death data from vital registrations and verbal autopsy surveys in which relatives are asked a standard set of questions to ascertain the likely cause of death, supplemented with police and mortuary data for injury deaths in countries with no other data. For countries with vital registration data, the completeness is assessed with demographic methods based on comparing recorded deaths with population counts between two successive censuses. The cause of death information is provided in a large number of different classification systems based on versions of the International

Classification of Diseases or bespoke classifications in some countries. All data are mapped into the disease and injury categories of GBD. All classification systems contain codes that are less informative because they lack a specific diagnosis (eg, unspecified cancer) or refer to codes that cannot be underlying cause of death (eg, low back pain or senility) or are intermediate causes (eg, heart failure or sepsis). Such deaths are redistributed to more precise underlying causes of death.⁴ After these redistributions and corrections for under-registration, the data are analysed in CODEm (cause of death ensemble model), a highly systematised tool that runs many different models on the same data and chooses an ensemble of models that best reflects all the available input data. Models are chosen with variations in the statistical approach (“mixed effects” of spatiotemporal Gaussian Process Regression), in the unit of analysis (rates or cause fractions), and the choice of predictive covariates. The statistical performance of all models is tested by holding out 30% of the data and checking how well a model covers the data that were held out. To enforce consistency from CODEm, the sum of all cause-specific mortality rates is scaled to that of the all-cause mortality rates in each age, sex, location, and year category.

Non-fatal estimates are based on systematic reviews of published papers and unpublished documents, survey microdata, administrative records of health encounters, registries, and disease surveillance systems. Our Global Health Data Exchange (GHDx, <http://ghdx.healthdata.org/>) is the largest repository of health data globally. We first set a reference case definition and/or study method that best quantifies each disease or injury or consequence thereof. If there is evidence of a systematic bias in data that used different case definitions or methods compared to reference data we adjust those data points to reflect what its value would have been if measured as the reference. This is a necessary step if one wants to use all data pertaining to a particular quantity of interest rather than choosing a small subset of data of the highest quality only. DisMod-MR 2.1, a Bayesian meta-regression tool, is our main method of analyzing non-fatal data. It is designed as a geographical cascade where a first model is run on all the world’s data, which produces an initial global fit and estimates coefficients for predictor variables and the adjustments for alternative study characteristics. The global fit adjusted by the values of random effects for each of seven GBD super-regions, the coefficients on sex and country predictors, are passed down as data to a model for each super-region together with the input data for that geography. The same steps are repeated going from super-region to 21 region fits and then to 195 fits by country and where applicable a further level down to subnational units. Below the global fit, all models are run separately by sex and for six time periods: 1990, 1995, 2000, 2005, 2010, and 2016. During each fit all data on prevalence, incidence, remission (ie, cure rate) and mortality are forced to be internally consistent. For most diseases, the bulk of data on prevalence or incidence is at the disease level with fewer studies providing data on the proportions of cases of disease in each of the sequelae defined for the disease. The proportions in each sequela are pooled using DisMod-MR 2.1 or meta-analysis, or derived from analyses of patient-level datasets. The multiplication of prevalent cases for each disease sequela and the appropriate disability weight produces YLD estimates that do not yet take into account comorbidity. To correct for comorbidity, these data are used in a simulation to create hypothetical individuals in each age, sex, location, and year combination who experience no, one, or multiple sequelae simultaneously. We assume that disability weights are multiplicative rather than additive as this avoids assigning a combined disability weight value in any individual to exceed 1, ie, be worse than a “year lost due to death”. This comorbidity adjustment leads to an average scaling down of disease-specific YLDs ranging from about 2% in young children up to 17% in oldest ages.

All our estimates of causes of death are categorical: each death is assigned to a single underlying cause. This has the attractive property that all estimates add to 100%. For risks, we use a different, “counterfactual” approach, ie, answering the question: “what would the burden have been if the population had been exposed to a theoretical minimum level of exposure to a risk”. Thus, we need to define what level of exposure to a risk factor leads to the lowest amount of disease. We then analyse data

on the prevalence of exposure to a risk and derive relative risks for any risk-outcome pair for which we find sufficient evidence of a causal relationship. Prevalence of exposure is estimated in DisMod-MR 2.1, using spatiotemporal Gaussian Process Regression, or from satellite imagery in the case of ambient air pollution. Relative risk data are pooled using meta-analysis of cohort, case-control and/or intervention studies. For each risk and outcome pair, we evaluate the evidence and judge if the evidence falls into the categories of “convincing” or “probable” as defined by the World Cancer Research Fund.⁵ From the prevalence and relative risk results, population attributable fractions are estimated relative to the theoretical minimum risk exposure level (TMREL). When we aggregate estimates for clusters of risks, eg, metabolic or behavioural risks, we use a multiplicative function rather than simple addition and take into account how much of each risk is mediated through another risk. For instance, some of the risk of high body mass index is directly onto stroke as an outcome but much of its impact is mediated through high blood pressure, high cholesterol, or high fasting plasma glucose, and we would not want to double count the mediated effects when we estimate aggregates across risk factors.⁶

Uncertainty is propagated throughout all these calculations by creating 1,000 values for each prevalence, death, YLL, YLD, or DALY estimate and performing aggregations across causes and locations at the level of each of the 1,000 values for all intermediate steps in the calculation. The lower and upper bounds of the 95% uncertainty interval are the 25th and 975th values of the ordered 1,000 values. For all age-standardised rates, GBD uses a standard population calculated as the non-weighted average across all countries of the percentage of the population in each five-year age group for the years 2010 to 2035 from the United Nations Population Division’s World Population Prospects (2012 revision).^{7,8}

GBD uses a composite indicator or sociodemographic development, SDI, which reflects the geometric mean of normalised values of a location’s income per capita, the average years of schooling in the population 15 and over, and the total fertility rate. Countries and territories are grouped into five quintiles of high, high-middle, middle, low-middle, and low SDI based on their 2016 values.²

The fatal and non-fatal write-ups have been published before as part of the GBD papers on causes of death and non-fatal estimates published in the *Lancet* weekly (see references ^{2,4,6,7}).

1 GBD 2015 Neurological Disorders Collaborator Group. Global, regional, and national burden of neurological disorders during 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet Neurol* 2017; **16**: 877–97.

2 GBD 2016 Mortality Collaborators. Global, regional, and national under-5 mortality, adult mortality, age-specific mortality, and life expectancy, 1970–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Lond Engl* 2017; **390**: 1084–150.

3 Salomon JA, Haagsma JA, Davis A, *et al*. Disability weights for the Global Burden of Disease 2013 study. *Lancet Glob Health* 2015; **3**: e712–723.

4 GBD 2016 Causes of Death Collaborators. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Lond Engl* 2017; **390**: 1151–210.

5 American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research, 2007.

6 GBD 2016 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Lond Engl* 2017; **390**: 1345–422.

7 GBD 2013 Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet Lond Engl* 2015; **385**: 117–71.

8 United Nations Department of Economics and Social Affairs Population Division. World Population Prospects: The 2012 Revision. <http://esa.un.org/unpd/wpp/Documentation/publications.htm> (accessed Nov 4, 2014).

GATHER compliance table

GATHER checklist of information that should be included in reports of global health estimates, with description of compliance and location of information for GBD 2016 (Table 1).

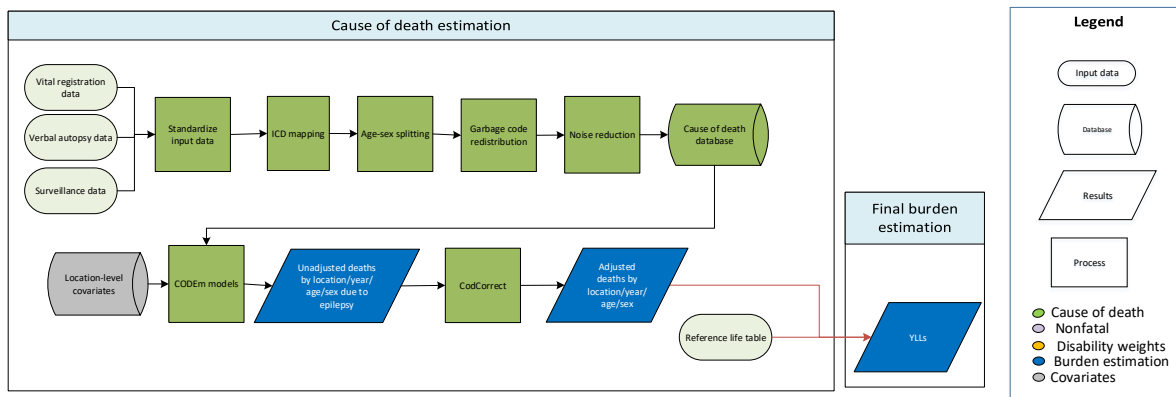
Table 1. GATHER check List

#	GATHER checklist item	Description of compliance	Reference
Objectives and funding			
1	Define the indicators, populations, and time periods for which estimates were made.	Narrative provided in paper and appendix describing indicators, definitions, and populations	Main text (Methods) and appendix
2	List the funding sources for the work.	Funding sources listed in paper	Summary (Funding)
Data Inputs			
<i>For all data inputs from multiple sources that are synthesised as part of the study:</i>			
3	Describe how the data were identified and how the data were accessed.	Narrative description of data seeking methods provided	Main text (Methods) and appendix
4	Specify the inclusion and exclusion criteria. Identify all ad-hoc exclusions.	Narrative about inclusion and exclusion criteria by data type provided; ad hoc exclusions in cause-specific write-ups	Main text (Methods) and appendix
5	Provide information on all included data sources and their main characteristics. For each data source used, report reference information or contact name/institution, population represented, data collection method, year(s) of data collection, sex and age range, diagnostic criteria or measurement method, and sample size, as relevant.	An interactive, online data source tool that provides metadata for data sources by component, geography, cause, risk, or impairment has been developed	Online data citation tools: http://ghdx.healthdata.org/gbd-2016
6	Identify and describe any categories of input data that have potentially important biases (e.g., based on characteristics listed in item 5).	Summary of known biases by cause included in appendix	Appendix
<i>For data inputs that contribute to the analysis but were not synthesised as part of the study:</i>			
7	Describe and give sources for any other data inputs.	Included in online data source tool	http://ghdx.healthdata.org/gbd-2016
<i>For all data inputs:</i>			
8	Provide all data inputs in a file format from which data can be efficiently extracted (e.g., a spreadsheet as opposed to a PDF), including all relevant meta-data listed in item 5. For any	Downloads of input data available through online tools, including data	Online data visualisation tools, data query tools, and

	data inputs that cannot be shared due to ethical or legal reasons, such as third-party ownership, provide a contact name or the name of the institution that retains the right to the data.	visualisation tools and data query tools; input data not available in tools will be made available upon request	the Global Health Data Exchange
Data analysis			
9	Provide a conceptual overview of the data analysis method. A diagram may be helpful.	Flow diagrams of the overall methodological processes, as well as cause-specific modelling processes, have been provided	Main text (Methods) and appendix
10	Provide a detailed description of all steps of the analysis, including mathematical formulae. This description should cover, as relevant, data cleaning, data pre-processing, data adjustments and weighting of data sources, and mathematical or statistical model(s).	Flow diagrams and corresponding methodological write-ups for each cause, as well as the databases and modelling processes, have been provided	Main text (Methods) and appendix
11	Describe how candidate models were evaluated and how the final model(s) were selected.	Provided in the methodological write-ups	Appendix
12	Provide the results of an evaluation of model performance, if done, as well as the results of any relevant sensitivity analysis.	Provided in the methodological write-ups	Appendix
13	Describe methods for calculating uncertainty of the estimates. State which sources of uncertainty were, and were not, accounted for in the uncertainty analysis.	Appendix	Appendix
14	State how analytic or statistical source code used to generate estimates can be accessed.	Appendix	http://ghdx.healthdata.org/gbd-2016-code
Results and Discussion			
15	Provide published estimates in a file format from which data can be efficiently extracted.	GBD 2016 results are available through online data visualisation tools, the Global Health Data Exchange, and the online data query tool	Main text, and online data tools (data visualisation tools, data query tools, and the Global Health Data Exchange)
16	Report a quantitative measure of the uncertainty of the estimates (e.g. uncertainty intervals).	Uncertainty intervals are provided with all results	Main text, appendix, and online data tools (data visualisation tools, data query tools, and the Global Health Data Exchange)
17	Interpret results in light of existing evidence. If updating a previous set of estimates, describe the reasons for changes in estimates.	Discussion of methodological changes between GBD rounds provided in the narrative of the manuscript and appendix	Main text (Methods and Discussion) and appendix
18	Discuss limitations of the estimates. Include a discussion of any modelling assumptions or data limitations that affect interpretation of the estimates.	Discussion of limitations provided in the narrative of the main paper, as well as in the methodological write-ups	Main text (Limitations) and appendix

Epilepsy mortality

Flowchart



Input data

Data used to estimate epilepsy mortality included vital registration (VR), verbal autopsy, and China mortality surveillance data from the cause of death (COD) database. Our outlier criteria were to exclude data points that (1) were implausibly high or low relative to global or regional patterns, (2) substantially conflicted with established age or temporal patterns, or (3) significantly conflicted with other data sources based from the same locations or locations with similar characteristics (ie, Socio-demographic Index).

Based on these criteria, we excluded ICD-9 BTL data for Sri Lanka, Fiji, and Kiribati as the estimates varied from year to year between zero and high values. We also excluded the Survey of Causes of Death Data and Medical Certification of Cause of Death Data for India, as these data types were not consistent with the Sample Registration System Data and would have led to discontinuities in our estimates over time.

Modelling strategy

The standard CODEm modelling approach was applied to estimate deaths due to epilepsy. Separate models were conducted for male and female mortality, and the age range for both models was 28 days–95+ years. For GBD 2016, the health systems access covariate was replaced with the healthcare access and quality index covariate. There were no other substantial changes for GBD 2016. The covariates used are displayed below (Table 2).

Table 2. List of covariates

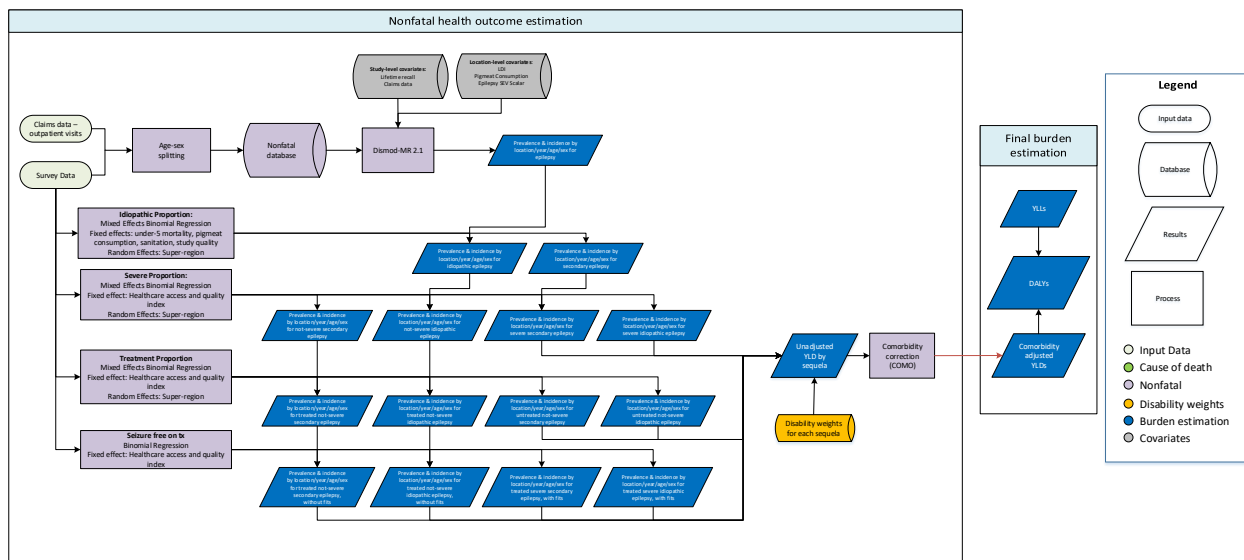
Level	Covariate	Direction
1	pig meat consumption (kcal per capita)	+
	pigs (per capita)	+
	SEV scalar: epilepsy	+

	mean systolic blood pressure (mmHg)	+
2	health access and quality index	-
	mean body mass index	+
	mean serum total cholesterol (mmol/L)	+
3	cumulative cigarettes (10 years)	+
	cumulative cigarettes (5 years)	+
	education (years per capita)	-
	log LDI (per capita)	-
	Socio-demographic Index	-

Epilepsy Impairment

Flowchart

Epilepsy



Case definition

Since GBD 2013, we have used the following definitions from the “Guidelines for Epidemiologic Studies on Epilepsy”: 1) Epilepsy: a condition characterized by recurrent (two or more) epileptic seizures, unprovoked by any immediate identified cause, and 2) “Active” epilepsy: a prevalent case of active epilepsy is defined as a person with epilepsy who has had at least one epileptic seizure in the previous five years, regardless of antiepileptic drug (AED) treatment. We also use the following ICD-10 codes for epilepsy: G40 (Neuro, epilepsy, total) and G41 (Neuro, epilepsy, status epilepticus). We defined severe epilepsy as having seizures one or more times per month.

Input data

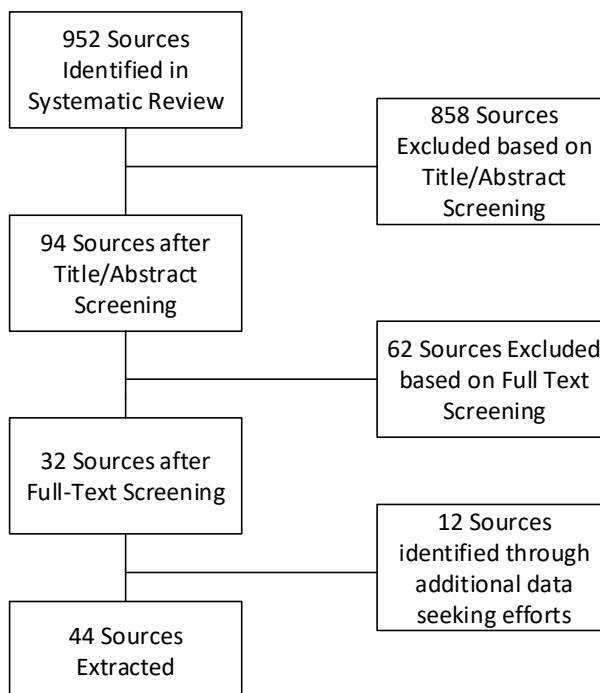
Model inputs

For GBD 2016, we conducted a systematic review covering 10/1/2014 to 10/17/2016 using the following search string:

("2014/10/01"[PDAT] : "2016"[PDAT]) AND ("epilepsy"[MeSH Terms] OR "epilepsy, partial, motor"[MeSH Terms] OR "epilepsy, benign neonatal"[MeSH Terms] OR "epilepsy, reflex"[MeSH Terms] OR "myoclonic epilepsy, juvenile"[MeSH Terms] OR "epilepsy, frontal lobe"[MeSH Terms] OR "epilepsy, complex

partial"[MeSH Terms] OR "epilepsy, post-traumatic"[MeSH Terms] OR "epilepsy, temporal lobe"[MeSH Terms] OR "epilepsy, absence"[MeSH Terms] OR "epilepsy, tonic-clonic"[MeSH Terms] OR "epilepsies, myoclonic"[MeSH Terms] OR "epilepsies, partial"[MeSH Terms] OR epilepsy[Title/Abstract]) AND (incidence[Title/Abstract] OR prevalence[Title/Abstract]) NOT(animals[MeSH] NOT humans[MeSH]).

Of 952 hits, 32 were marked for extraction. Additional data-seeking efforts also led to the addition of 12 more sources on epilepsy prevalence in India subnationals. A flow chart documenting the review is displayed below.



We included representative, population-based surveys that reported of prevalence, incidence, remission rate, excess mortality rate, relative risk of mortality, standardised mortality ratio, or with-condition mortality rate. We excluded studies with no clearly defined sample (eg, among clinic attenders or patient organisation members with non-specific or non-representative catchment area). The table below (Table 3) details the model inputs used to estimate the epilepsy impairment.

Table 3. Model inputs used to estimate epilepsy impairment

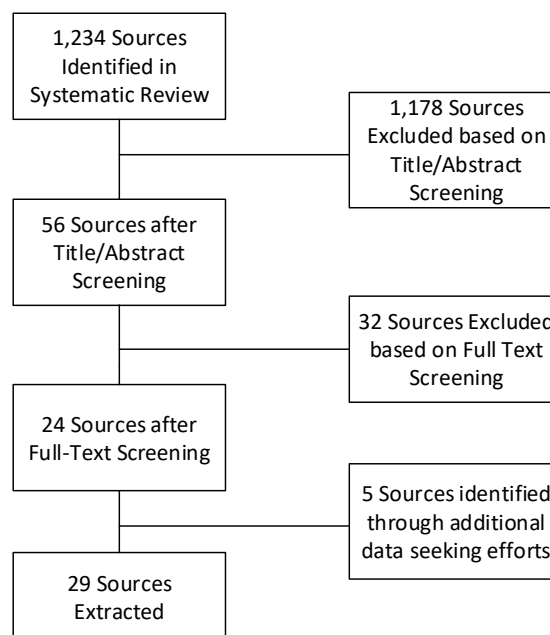
	Prevalence	Incidence	Mortality risk
Studies	317	81	23
Countries/subnationals	192	51	23
GBD world regions	20	15	10

The inputs for the regressions used to split the epilepsy impairment envelope were also updated for GBD 2016. These regressions are used to determine the proportion of epilepsy that is primary or idiopathic, the proportion of epilepsy that is severe (one or more seizures per month), the proportion of epilepsy that is untreated (the treatment gap), and the proportion of treated epilepsy that is treated without seizures (no seizures reported in the preceding year).

For GBD 2016, a new systematic review was conducted covering 1/1/2006 to 10/17/2016 using the search term:

("2006"[PDAT] : "2016"[PDAT]) AND ("epilepsy"[MeSH Terms] OR "epilepsy, partial, motor"[MeSH Terms] OR "epilepsy, benign neonatal"[MeSH Terms] OR "epilepsy, reflex"[MeSH Terms] OR "myoclonic epilepsy, juvenile"[MeSH Terms] OR "epilepsy, frontal lobe"[MeSH Terms] OR "epilepsy, complex partial"[MeSH Terms] OR "epilepsy, post-traumatic"[MeSH Terms] OR "epilepsy, temporal lobe"[MeSH Terms] OR "epilepsy, absence"[MeSH Terms] OR "epilepsy, tonic-clonic"[MeSH Terms] OR "epilepsies, myoclonic"[MeSH Terms] OR "epilepsies, partial"[MeSH Terms] OR epilepsy[Title/Abstract]) AND (incidence[Title/Abstract] OR prevalence[Title/Abstract] OR epidemiology[Title/Abstract]) AND (sever*[Title/Abstract] OR treated[Title/Abstract] OR "drug resistant epilepsy"[MeSH] OR "treatment resistant"[Title/Abstract] OR proportion[Title/Abstract] OR "clinical characteristics"[Title/Abstracts] OR "treatment gap"[Title/Abstract]) NOT(animals[MeSH] NOT humans[MeSH])

Of 1,234 hits, 37 were marked for extraction. Additional data-seeking efforts also led to the addition of five more sources on the treatment gap in India subnationals. A flow chart documenting the review is displayed below.



Severity splits & disability weights

To adjust for differences in methodological quality, all prevalence studies included in GBD were scored according to a modified version (dichotomised variables) of published methodological quality criteria for epilepsy epidemiological studies, taking into account the representativeness of the population of interest (representative of country or community versus selected population), quality of sampling (random sample of the population of interest versus not random sample), recall period (one-year prevalence versus other recall period), participation rate ($\geq 70\%$ versus $<70\%$), survey method (face to face with epilepsy expert or trained interviewer versus other), validation of diagnostic instrument (sensitivity or specificity $\geq 70\%$ versus $< 70\%$ or no validation). In DisMod-MR these methodological variables were evaluated for a systematic difference and corrected accordingly.

Table 4 illustrates the severity levels, descriptions, and disability weights associated with epilepsy. These are calculated using regressions from literature (ie, frequency of seizures).

Table 4. Severity levels, descriptions, and disability weights associated with epilepsy

Severity level	Lay description	Disability weights (95% CI)
severe (seizures \geq once per month)	This person has sudden seizures one or more times each month, with violent muscle contractions and stiffness, loss of consciousness, and loss of urine or bowel control. Between seizures the person has memory loss and difficulty concentrating.	0.552 (0.375–0.71)
less severe (seizures $<$ once per month)	This person has sudden seizures two to five times a year, with violent muscle contractions and stiffness, loss of consciousness, and loss of urine or bowel control.	0.263 (0.173–0.367)
Treated without fits	This person has a chronic disease that requires medication every day and causes some worry but minimal interference with daily activities.	0.049 (0.031–0.072)

Modelling strategy

We modelled the prevalence of epilepsy in two steps: first, we created an epilepsy impairment envelope. Second, we split the envelope into primary (or idiopathic) and secondary epilepsies. Each of these were subdivided into “severe” (on average one or more fits per month) and “non-severe.” Non-severe cases were subdivided into “treated” and “un-treated.” Finally, “treated” cases were divided into “treated cases with fits” (between one and 11 fits on average in the preceding year) and “treated cases without fits” (no fits reported in the preceding year).

In the first step, we used the DisMod-MR tool for the epilepsy impairment envelope to model a consistent fit between incidence, prevalence, remission, and standardised mortality ratio data while using meta-regression to correct data points with non-reference study quality characteristics. We found no systematic bias for the covariate “non-standard case definition” indicating studies that did not define “active epilepsy” and additionally the covariate was not significant as a “z-cov”, which acts as a multiplier applied to the standard error and thus results in these data points being given less weight in the analysis than the “reference” data points. Therefore, we excluded this covariate from the model. We also included data on lifetime prevalence and therefore added a covariate on lifetime prevalence data points. We also included country-level covariates on prevalence for the SEV epilepsy scalar, which summarises the epilepsy risk exposure level for each country, and pig meat consumption per capita, which is used as a proxy for the level of neurocysticercosis, a common cause of secondary epilepsy. We included cause-specific mortality rate (CSMR) results from the epilepsy mortality model as input data to the DisMod model. Where age-specific prevalence data were available, we calculated excess mortality rate (EMR) from prevalence and CSMR. We included the log of the lag distributed income (LDI) as a covariate on EMR to account for lower mortality in developed countries. We included Bayesian priors on remission to account for the scarcity of remission data. We set bounds on remission from 0 to 0.25 from age 0 to 60 and 0 to 0.05 from age 61 to 100. Table 5 indicates the covariates used in the estimation process, as well as parameters, betas, and exponentiated betas.

Table 5. Covariates used in the estimation process

Measure	Variable Name	Beta	Exponentiated
prevalence	Recall Lifetime	0.18 (0.15 – 0.22)	1.20 (1.17–1.24)
prevalence	All MarketScan, year 2000	-0.89 (-0.94 – -0.83)	0.41 (0.39–0.43)
prevalence	All MarketScan, year 2010	-0.43 (-0.47 – -0.37)	0.65 (0.62–0.69)
prevalence	All MarketScan, year 2012	-0.35 (-0.41 – -0.30)	0.70 (0.67–0.74)
prevalence	Pig Meat Consumption (kg per capita)	0.0054 (0.00012 – 0.015)	1.01 (1.00–1.02)
prevalence	Log-transformed age-standardized SEV scalar for epilepsy	0.79 (0.75-0.90)	2.21 (2.12-2.46)
excess mortality rate	LDI (I\$ per capita)	-0.55 (-1 – -0.1)	0.58 (1.37–0.90)

In the second step, we used a mixed-effects generalized linear model (binomial family) to predict the proportion of idiopathic epilepsy, the proportion of severe epilepsy, the proportion of treated epilepsy, and the proportion of epilepsy that is treated without fits.

Because not all of the data on the proportion of idiopathic epilepsy use optimal case-finding methods (using CT scans or MRIs in addition to EEGs in order to diagnose secondary epilepsy), for GBD 2016 we decided to add a covariate to crosswalk studies with non-optimal case-finding methods to those with adequate methods. The regression for the proportion of epilepsy that is idiopathic therefore has fixed effects on this study quality covariate as well as the under-5 mortality rate, the log of pig meat consumption (per capita), and the proportion of a country with access to proper sanitation, as well as a random effect on super-region.

We used similar models to predict the proportion of severe epilepsy and treatment gap based on the reported proportions extracted from the systematic review. To predict the proportion of severe epilepsy and the treatment gap, we used mixed-effects models with a fixed effect on the log of HAQ Index and a random effect on super-region.

For GBD 2015, a meta-analysis was used to generate two different pooled estimates for proportion of treated epilepsy that is seizure-free in developing and developed countries, as there were not enough data to run a regression. However, for GBD 2016 the expanded dataset allowed for the implementation of a generalised linear model (binomial family) to generate predictions for the proportion of treated epilepsy that is seizure-free. We used a fixed effect on the log of HAQ Index.

We tested a fixed effect on Socio-demographic Index (SDI) and random effects on region and country in different models, but they did not improve the model. We generated 1,000 draws of country-specific estimates for each year between 1980 and 2016 for each of the models. Table 6 shows the betas from these regressions.

Table 6. Estimated parameters (beta) with standard errors from regression models

Regression	covariate	beta	SE
Idiopathic	Under-5 mortality	-65.82	7.50
Idiopathic	Pig meat consumption	-0.12	0.02
Idiopathic	Sanitation	0.45	0.16

Idiopathic	Study quality	0.88	0.07
Severe	HAQ Index	-2.15	0.24
Treatment gap	HAQ Index	-3.17	0.18
Treated without fits	HAQ Index	3.65	0.21

Definition of GBD super-regions and regions

Table 7. GBD Super-regions and Regions

Super-region	Region_name
East Asia, Southeast Asia and Oceania	East Asia
	Southeast Asia
	Oceania
Central Asia, Central Europe, Eastern Europe	Central Asia
	Central Europe
	Eastern Europe
High-income	High-income Asia Pacific
	Australasia
	Western Europe
	High-income North America
	Southern Latin America
Latin America & Caribbean	Caribbean
	Andean Latin America
	Central Latin America
	Tropical Latin America
North Africa and Middle East	North Africa and Middle East
South Asia	South Asia
Sub-Saharan Africa	Central sub-Saharan Africa
	Eastern sub-Saharan Africa
	Southern sub-Saharan Africa
	Western sub-Saharan Africa

Count of data sources used in nonfatal modelling for epilepsy by 21 regions in 2016

Table 8. Count of data sources used in nonfatal modelling for epilepsy by 21 regions in 2016

Region_name	Incidence	Prevalence	Remission	Mortality	Hospital_claims
East Asia	3	40	0	4	0
Southeast Asia	2	5	0	0	0
Oceania	0	0	0	0	0
Central Asia	0	2	0	0	0
Central Europe	2	4	0	0	0
Eastern Europe	2	4	0	1	0
High-income Asia Pacific	2	6	0	0	0
Australasia	0	2	0	1	0
Western Europe	42	75	0	10	0
Southern Latin America	1	6	1	1	0
High-income North America	7	16	1	4	3
Caribbean	0	2	0	0	0
Andean Latin America	3	9	1	1	0
Central Latin America	0	12	0	0	0
Tropical Latin America	1	8	0	0	0
North Africa and Middle East	5	23	0	0	0
South Asia	4	36	0	2	0
Central sub-Saharan Africa	0	2	0	0	0
Eastern sub-Saharan Africa	4	25	0	1	0
Southern sub-Saharan Africa	1	5	0	0	0
Western sub-Saharan Africa	3	37	0	3	0
Total	82	319	3	28	3

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Treatment Gap Regression

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