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Supplementary appendix

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Supplement to: GBD 2015 SDG Collaborators. Measuring the health-related Sustainable Development Goals in 188 countries: a baseline analysis from the Global Burden of Disease Study 2015. *Lancet* 2016; published online Sept 21. http://dx.doi.org/10.1016/S0140-6736(16)31467-2.

Methods Appendix to Measuring the health-related Sustainable Development Goals in 188 countries: An analysis from the Global Burden of Disease Study 2015

This methods appendix provides further methodological detail for the health-related Sustainable Development Goals (SDGs). The appendix is organized into broad sections following the structure of the main paper.

The supplementary results offer additional results tables and figures, as well as more detailed methodological figures.

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Preamble

This appendix provides methodological detail, supplemental figures and tables, and more detailed results for the health-related Sustainable Development Goals (SDGs). The appendix is organized into broad sections following the structure of the main paper. This study complies with the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER) recommendations, and this appendix is more comprehensive and encyclopedic than previous Global Burden of Disease (GBD) appendices. It includes detailed tables, figures, indicator modeling write-ups and flowcharts, and information on data sourcing in an effort to maximize transparency in our estimation processes and provide a comprehensive account of analytical steps. Components of this document are the same as described in earlier GBD 2015 Capstone appendices but much more of this appendix are new text for the SDG Capstone. We intend this to be a living document, to be updated with each annual iteration of the Global Burden of Disease and in accordance with the 15 year timeline of the SDG cycle until their conclusion in 2030.

GATHER statement

This study complies with the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER) recommendations. We have documented the steps involved in our analytical procedures and detailed the data sources used in compliance with the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER). For additional GATHER reporting, please refer to Appendix Table 4 in Section 3.

Methods Appendix Table 1. GATHER checklist of information that should be included in reports of global health estimates, with description of compliance and location of information for SDG Capstone

#	GATHER checklist item	Description of compliance	Reference			
Object	Objectives and funding					
1	Define the indicators, populations, and time periods for which	Narrative provided in paper and	Summary; Main text; Appendix Part 1.			
	estimates were made.	appendix describing indicators,	Sections 1-3; Supplementary Results			
		definitions, and populations.				
2	List the funding sources for the work.	Funding sources listed in paper.	Main text			
Data II	nputs					
For all	data inputs from multiple sources that are synthesized as part of the	study:				
3	Describe how the data were identified and how the data were	Narrative description of data seeking	Appendix Part 1. Sections 1-3			
	accessed.	methodology provided.				
4	Specify the inclusion and exclusion criteria. Identify all ad-hoc	Narrative about inclusion and exclusion	Appendix Part 1. Sections 1-3			
	exclusions.	criteria by data type provided.				
5	Provide information on all included data sources and their main	List of all data sources provided in	Appendix Part 3. Section 1.			
	characteristics. For each data source used, report reference	submission materials; interactive, online				
	information or contact name/institution, population represented,	data source tool that provides metadata	http://ghdx.healthdata.org/			
	data collection method, year(s) of data collection, sex and age	for data sources by component,				
	range, diagnostic criteria or measurement method, and sample	geography, cause, risk, or impairment has	There is a forthcoming custom data			
	size, as relevant.	been developed.	source tool with additional information			
			on data sourcing for GBD and SDG			
			capstone publications.			
6	Identify and describe any categories of input data that have	Summary of known biases by cause	Appendix Part 1. Section 3			
	potentially important biases (e.g., based on characteristics listed	included in methodological appendix.				
	in item 5).					
For da	ta inputs that contribute to the analysis but were not synthesized as p	part of the study:				
7	Describe and give sources for any other data inputs.	Included in list of all data sources	http://ghdx.healthdata.org/			
		provided in submission materials, as well				
		as online data source tool.	There is a forthcoming custom data			
			source tool with additional information			
			on data sourcing for GBD and SDG			
			capstone publications.			
For all	data inputs:					
8	Provide all data inputs in a file format from which data can be	Downloads of input data will be available	Online data tools			
	efficiently extracted (e.g., a spreadsheet as opposed to a PDF),	through online tools, including data	http://www.healthdata.org/results/data-			
	including all relevant meta-data listed in item 5. For any data	visualization tools and data query tools.	visualizations;			
	inputs that cannot be shared due to ethical or legal reasons, such	Input data not available in tools will be	http://ghdx.healthdata.org/;			
		made available upon request.	http://ghdx.healthdata.org/gbd-data-tool			

	as third-party ownership, provide a contact name or the name of		
Data a	the institution that retains the right to the data. nalysis		
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; Appendix Part 1. Section 3
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 and modelling processes have been provided.	Appendix Part 1. Section 3
11	Describe how candidate models were evaluated and how the final model(s) were selected.	Provided in the methodological write- ups.	Appendix Part 1. Section 3
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 Part 2
13	Describe methods for calculating uncertainty of the estimates. State which sources of uncertainty were, and were not, accounted for in the uncertainty analysis.	Provided in the methodological write- ups.	Appendix Part 1. Section 3
14	State how analytic or statistical source code used to generate estimates can be accessed.	Access statement provided.	This will be available in an online repository that will be released upon publication of GBD 2015 Capstones.
Result	s and Discussion		
15	Provide published estimates in a file format from which data can be efficiently extracted.	GBD 2015 results will be made available through online data visualization tools, the Global Health Data Exchange, and the online data query tool (these tools are already available for GBD 2013 results).	Supplementary Results
16	Report a quantitative measure of the uncertainty of the estimates (e.g. uncertainty intervals).	Uncertainty intervals are provided with all results.	Main text; Supplementary Results
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 SDG rounds provided in the narrative of the paper and appendix.	Main text; Appendix Part 1. Section XX
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 in the appendix.	Main text; Appendix Part 1. Section 3

Part 1. Health-related SDG indicators

Section 1. Sustainable Development Goals overview

In September 2015, the United Nations (UN) General Assembly established the Sustainable Development Goals (SDGs). The SDGs substantially broaden the development agenda beyond the MDGs and are expected to frame UN member state policies over the next 15 years. The SDGs specify 17 universal goals, 169 targets, and 230 indicators leading up to 2030. The SDGs substantially broaden the development agenda beyond the MDGs and are expected to frame UN member state policies over the next 15 years. We provide an analysis of 33 out of the 47 health-related SDG indicators based on data used and generated by the Global Burden of Diseases, Injuries and Risk Factors Study 2015 (GBD 2015).

Section 2. Health-related SDGs

Health is a core dimension of the SDGs; the third SDG aims to "ensure healthy lives and promote wellbeing for all at all ages." Health-related indicators are also present among ten of the other 16 goals. Across these 11 goals, there are 28 health-related targets with a total of 47 health-related indicators.

Of the 47 health-related indicators included as part of the SDGs, estimates for 33 indicators, using consistent approaches built on systematic efforts to compile all available data, are included as part of the GBD study. In this paper, while acknowledging the continued debate about the structure and choices of SDG indicators, we use the GBD study to provide an assessment of the current status of these 33 health-related SDG indicators, develop and compute a summary indicator of the health-related SDG indicators, document historical trends, identify high achievers to inform roadmaps and provide a basis for future monitoring of the health-related SDG indicators.

The GBD study is an annual effort to measure the health of populations at national, and selected subnational levels, from 1990 to the most recent year of 2015. The GBD study produces estimates of mortality and morbidity by cause, age and sex as well as that attributable to a selected set of major risk factors. Many of the 47 health-related SDG indicators selected by the Inter-Agency and Expert Group on Sustainable Development Goal Indicators (IAEG-SDGs are produced as part of the GBD. Elsewhere in this appendix, we outline the 10 SDGs, corresponding 21 health-related targets, and 33 health-related indicators included in this iteration of the GBD and SDG reporting. Part 1. Section 3 of this appendix also further outlines the definition of each indicator used in analysis, as well as the estimation method and data sources.

Direct outputs of the GBD study that are health-related SDG indicators include mortality rates disaggregated by age (under-5 and neonatal) and cause (maternal, cardiovascular diseases, cancers, diabetes, chronic respiratory diseases, road traffic injuries, self-harm, unintentional poisonings, exposure to forces of nature, interpersonal violence, and collective violence and legal intervention) as well as measures of disease incidence (HIV/AIDS, malaria, tuberculosis [TB], hepatitis B) and prevalence (neglected tropical diseases [NTDs]). The GBD risk factor analysis includes measurement of exposure prevalence included as health-related SDG indicators (under-5 stunting, wasting and overweight; tobacco smoking; harmful alcohol use; intimate partner violence; unsafe water, sanitation, and hygiene [WaSH]; household air pollution; and ambient particulate matter) as well as deaths or disease burden attributable to risk factors selected as health-related SDG indicators (WaSH, household and ambient air pollution, and occupational risks).

As noted in the main text, for selected indicators proposed by the IAEG-SDGs, we made modifications to the definition for clarity and/or based on the definition used in GBD. For example, Indicator 2.2.2 proposes to measure of malnutrition that combined prevalence of wasting and overweight among children under 5. As childhood wasting and overweight have very different determinants, we have selected to report them separately. For childhood overweight, we report prevalence among children aged 2 to 4 years, the definition used in GBD based on thresholds set by the International Obesity Task Force (IOTF).

Further details on the estimation and data sources used for all indicators, compliant with Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER), are included in Appendix Part 1. Section 3. Indicator-specific estimation and Appendix Part 4. Section 1. Comprehensive citation list.

Section 3. Indicator-specific estimation

The indicator-specific modeling write-ups follow the order of the SDG goals, targets and indicators proposed by the United Nations. In some cases, multiple indicators were addressed in a single write-up, for example natural disaster related-indicators (1.5.1, 11.5.1, and 13.1.2) are included in a single write-up along with war mortality (16.1.2).

The organization of this section is as follows:

Natural disasters (1.5.1, 11.5.1, 13.1.2)
Stunting (2.2.1)
Wasting (2.2.2a)
Overweight (2.2.2b)
Maternal mortality ratio (3.1.1.)
Skilled birth attendance (3.1.2, plus UHC [3.8.1])
Under-5 and neonatal mortality (3.2.1 & 3.3.2)
HIV incidence (3.3.1)
TB incidence (3.3.2)
Malaria incidence (3.3.3)
Hepatitis B incidence (3.3.4)
NTDs prevalence (3.3.5) – includes 14 individual NTDs
NCD mortality (3.4.1) - includes cardiovascular diseases, cancers, diabetes, and chronic respiratory diseases
Self-harm mortality (3.4.2)
Alcohol use (SEV) (3.5.2)
Road injury mortality (3.6.1)
Family planning need met with modern contraception (3.7.1)
Adolescent birth rates (3.7.2)

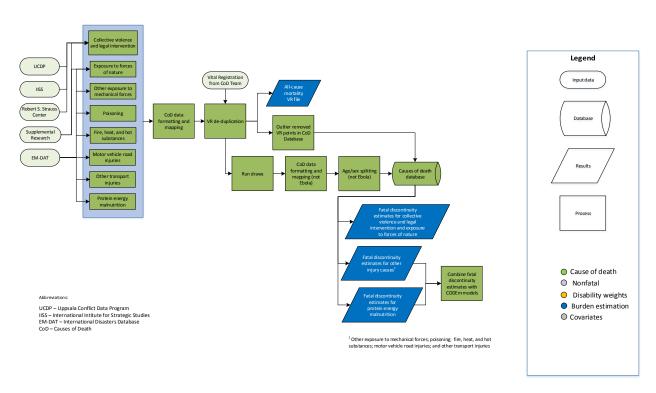
UHC – Universal Health Coverage: TB case detection, immunization, ANC1 and ANC4, skilled birth attendance, in-facility delivery rate, ART coverage, ITN coverage, and family planning need met with modern contraception (see also 3.7.1) (3.8.1) UHC - TB case detection (3.8.1) UHC - Immunization (3.8.1) UHC - ANC1 and ANC4 (3.8.1) UHC - In-facility delivery rate (3.8.1) UHC - ART coverage (3.8.1) UHC – Family planning need met with modern contraception (3.8.1, also 3.7.1) UHC - ITN coverage Deaths attributable to household air pollution and ambient air pollution (3.9.1) Deaths attributable to WaSH (3.9.2) Unintentional poisonings mortality (3.9.3) Smoking prevalence (3.a.1) Prevalence of intimate partner violence (5.2.1) Water (SEV) (6.1.1.) Sanitation (SEV) (6.2.1a) Hygiene (SEV) (6.2.1b) Household air pollution (SEV) (7.1.2) DALY rates attributable to occupational risks (8.8.1) Population-weighted PM2.5 (fine particulate matter) (11.6.2) Interpersonal violence mortality (16.1.1)

War mortality (16.1.2)

Fatal Discontinuities SDG Capstone Appendix:

War mortality; natural disasters; and other fatal discontinuities

Flowchart



Input Data and Methodological Summary

Indicator definition

This modeling strategy encompasses indicators associated with natural disasters: 1.5.1, 11.5.1, 13.1.2; and war mortality: 16.1.2.

Indicator 1.5.1

As a component of SDG Goal 1. End poverty in all its forms everywhere, SDG Target 1.5., by 2030, build the resilience of the poor and those in vulnerable situations and reduce their exposure and vulnerability to climate-related extreme events and other economic, social and environmental shocks and disasters, is measured using SDG Health Index Indicator 1.5.1, number of deaths due to exposure to forces of nature per 100,000.

Indicator 11.5.1

As a component of SDG Goal 11. Make cities and human settlements inclusive, safe, resilient and sustainable, SDG Target 11.5, by 2030, significantly reduce the number of deaths and the number of people affected and substantially decrease the direct economic losses relative to global gross domestic product caused by disasters, including water-related disasters, with a focus on protecting the poor and

people in vulnerable situations, is measured using SDG Health Index Indicator 11.5.1, Number of deaths, missing persons and persons affected by disaster per 100,000 people.

Indicator 13.1.2

As a component of SDG Goal 13. Take urgent action to combat climate change and its impacts, SDG Target 13.1, strengthen resilience and adaptive capacity to climate-related hazards and natural disasters in all countries, is measured using SDG Health Index Indicator 13.1.2, number of deaths due to exposure to forces of nature per 100,000 people.

Indicator 16.1.2

As a component of SDG Goal 16. Promote peaceful and inclusive societies for sustainable development, provide access to justice for all and build effective, accountable and inclusive institutions at all levels, SDG Target 16.1, significantly reduce all forms of violence and related death rates everywhere, is measured using SDG Health Index Indicator 16.1.2, number of deaths due to collective violence and legal intervention (war) per 100,000.

Input data

Collective Violence and Legal Intervention

Data for collective violence and legal intervention comes from the Uppsala Conflict Data Program (UCDP), International Institute for Strategic Studies (IISS), and Robert S. Strauss Center for International Security and Law. The table below provides details about the various datasets we utilized from these sources, the dates they were last accessed, and the years for which we used the data provided.

Data source name	Date	Years of data	Type of data included
	accessed	accessed	
Uppsala Conflict Data Program ¹			
Battles	9/23/15	1989-2015	Armed conflict: incompatibility that concerns government and/or territory over which the use of armed force between the military forces of two parties, of which at least one is the government of a state, has resulted in at least 25 battle-related deaths each year
Non-state	11/4/15	1989-2015	The use of armed force between two organized armed groups, neither of which is the government of a state, which results in at least 25 battle-related deaths each year
One-sided	11/3/15	1989-2015	The use of armed force by the government of a state or by a formally organized group against civilians which results in at least 25 deaths in a year
Africa Georeferenced Event Dataset	11/4/15	1989-2008	UCDP battles; non-state; one-sided for African countries
PRIO Battles Deaths Dataset	9/23/15	1970-1989	Armed conflict (civil wars, etc.)
International Institute for Strategic S	Studies		
Armed Conflict Dataset	9/25/15	1997-Present	Insurgency, Inter-state, Intra-state
Robert S. Strauss Center For Interna	tional Security	And Law	
Armed Conflict Location and Event Dataset (ACLED)	9/15/15	1997-2015	Actions of opposition groups, governments, and militias across Africa, specifying the exact location and date of battle events, transfers of military control, headquarter establishment, civilian violence, and rioting
Social Conflict Analysis Database (SCAD)	9/15/15	1990-2015	Protests, riots, strikes, inter-communal conflict, government violence against civilians, and other forms of social conflict (covers Africa and Latin America)

Supplemental online research was conducted for recent conflicts where the databases above were not up-to-date. Where there was large variance in death estimates in recent years, we averaged estimates from all sources at the country-year level.

For country-years where multiple sources provided estimates, we prioritized sources in the following order: (1) country vital registration (VR) data, if death estimates were highest of all sources; (2) UCDP; (3) IISS; (4) country VR if death estimates were not the highest of all sources; (5) Strauss Center; (6) online supplemental research; and (7) combined average country data where applicable.

Exposure to Forces of Nature and Other Injury Causes

Data for disaster events which caused greater than 50 deaths due to exposure to forces of nature; poisonings; fire, heat, and hot substances; motor vehicle road injuries; other transport injuries; and other exposure to mechanical forces came from the Centre for Research on the Epidemiology of Disasters' International Disaster Database (EM-DAT). Data from EM-DAT were last accessed January 3, 2016. Supplemental online research was conducted for events where EM-DAT was not up-to-date.

For country-years where multiple sources provided estimates, we prioritized sources in the following order: (1) country VR data, if death estimates were highest of all sources; (2) EM-DAT; (3) country VR if death estimates were not the highest of all sources; (4) online supplemental research. Exceptions were made where it was clear that vital registration systems had been compromised by the event being measured. In those cases, such as in the United States following Hurricane Katrina in 2005, supplemental research was prioritized over VR data.

In locations where we produced estimates at the subnational level for GBD 2015, deaths due to all fatal discontinuity causes were assigned to the relevant subnational location(s) when that information could be obtained either through the data sources mentioned above or through additional online research. If no subnational location could be found, the deaths were split proportionally by population across all subnational locations.

A systematic literature review was not used to identify input data for fatal discontinuities, though some literature sources were identified through online supplemental research.

Modeling strategy

All input data for fatal discontinuity causes were run through the Causes of Death data formatting and mapping process detailed in Part 2.

VR de-duplication

For country-years where deaths due to fatal discontinuity causes were recorded in both VR and other utilized data sources, the higher of the two estimates were taken in the case of deaths due to war and collective violence and exposure to forces of nature.

For the other injury causes that also have a Cause of Death Ensemble model (CODEm), a process was established to avoid duplication of fatal discontinuity deaths in the two models. First, location-years with greater than 50 deaths due to the cause were identified. If these location-cause-years had VR death

estimates that were greater than 40% higher than the immediately surrounding years, the identified years were reviewed. Those that represented a true diversion from the trend of VR and could be linked to a specific fatal discontinuity event were marked as outliers in the VR data and the difference between the outlier year and the average of the surrounding years was included in the relevant cause in the fatal discontinuities database. The deaths from the identified events were subtracted from the all-cause VR estimates used in the all-cause mortality estimation process.

Uncertainty analysis for input and draw-level input to age-sex splitting

Uncertainty intervals (UIs) for deaths due to collective violence and legal intervention were generated using UCDP high and low death estimates. In cases where low and high estimates are not provided by the original source, regional average relative UI by type of fatal discontinuity is applied to the mean input that is available.

We assumed a normal distribution using the mean deaths and standard deviation based on high and low estimates. The standard deviation was capped at the mean divided by 1.96 in order to ensure that 95% of the 1,000 draws generated were greater than 0. Any negative draws were dropped from final calculations of means and uncertainty intervals.

Age-sex splitting

All compiled data were run through the causes of death age-sex splitting process detailed in Part 2.

Changes from GBD 2013

Only collective violence and legal intervention and exposure to forces of nature were modeled as fatal discontinuities (previously called mortality shocks) in GBD 2013. GBD 2015 also includes fatal discontinuity models for protein energy malnutrition (previously included as "famine" in exposure to forces of nature) and additional injury models (motor vehicle road injuries; other transport injuries; exposure to fire, heat, and hot substances; poisoning; and other exposure to mechanical forces). These new causes (unlike collective violence and legal intervention and exposure to forces of nature) are also modeled in CODEm and thus have two models (a shock model and a CODEm model) that are combined to produce the final estimates of deaths for these causes.

The VR de-duplication process and the use of the causes of death age-sex splitting process for fatal discontinuity causes were added in GBD 2015.

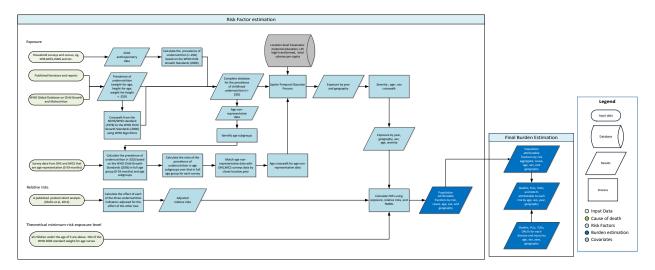
References

1. UCDP/PRIO Armed Conflict Dataset Codebook. Uppsala Conflict Data Program (UCDP); Centre for the Study of Civil Wars, International Peace Research Institute, Oslo (PRIO), 2013.

Childhood Undernutrition SDG Capstone Appendix

Flowchart

Childhood Undernutrition



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompasses indicators associated with child undernutrition: 2.2.1 and 2.2.2a.

Indicator 2.2.1

As a component of SDG Goal 2. End hunger, achieve food security, and improved nutrition, SDG Target 2.2, by 2030, end all forms of malnutrition, including achieving, by 2025, the internationally agreed targets on stunting and wasting in children under 5 years of age, and address the nutritional needs of adolescent girls, pregnant and lactating women and older persons, is measured using SDG Health Index Indicator 2.2.1, Prevalence of stunting among children under 5 (lower than two standard deviations from the median height for age of the reference population).

Indicator 2.2.2a

As a component of SDG Goal 2. End hunger, achieve food security, and improved nutrition, SDG Target 2.2, by 2030, end all forms of malnutrition, including achieving, by 2025, the internationally agreed targets on stunting and wasting in children under 5 years of age, and address the nutritional needs of adolescent girls, pregnant and lactating women and older persons, is measured using Indicator 2.2.2a, Prevalence of wasting among children under five (lower than two standard deviations from the median weight for height of the reference population).

Case definition

The exposure of childhood undernutrition was modeled by evaluating three anthropometric indicators which include underweight, wasting, and stunting. The definition of the three indicators are as follows:

Childhood underweight: Proportion of children aged 0 to 59 months in a given population who fall below 2 standard deviations (SD) of the WHO 2006 standard weight-for-age (wfa) curve. (1)

Childhood stunting: Proportion of children aged 0 to 59 months in a given population who fall below 2 standard deviations (SD) of the WHO 2006 height-for-age (hfa) curve.

Childhood wasting: Proportion of children aged 0 to 59 months in a given population who fall below 2 standard deviations (SD) of the WHO 2006 weight-for-height (wfh) curve.

Input data

There are two main inputs in the GBD 2015 undernutrition database—survey dataset and tabulated dataset. Survey dataset includes the standard multi-country or country-specific survey series such as: Reproductive and Health Surveys (RHS), Multiple Indicator Cluster Surveys (MICS), Demographic and Health Surveys (DHS), Living Standards Measurement Surveys (LSMS), China Health and Nutrition Survey (CHNS), and others. In the absence of survey data we used tabulated data from survey reports or published literature that have been extracted at IHME, downloaded from external databases or obtained from personal communication with external collaborators. The last update for tabulated dataset was conducted for GBD 2010. Tabulated data include survey reports or published literature from databases from UNICEF(2), the United Nations (UN) Statistics Division (3), and the WHO Global Database on Child Growth and Malnutrition(4).

Tabulated data based on the National Center for Health Statistics (NCHS)/WHO international growth reference (the NCHS reference) (5) were converted into data based on the World Health Organization (WHO) Child Growth Standards (the WHO 2006 standard) using WHO <u>algorithms</u> (6). Estimates that were not representative of all children under the age of 5 were adjusted based on age groups.

Modeling strategy

Exposure Estimate

To generate a complete time series of prevalence of childhood underweight, wasting, and stunting, we employed a three-step ST-GPR modeling strategy that uses linear regression, spatiotemporal regression and Gaussian Process Regression (GPR) which is specified in the main text of this manuscript. Identical strategies and covariates were used for each undernutrition indicator. A variety of combinations of socioeconomic and environmental covariates in different transformation format were tested by running mixed-effect models with exposure data to decide the inclusion and exclusion. The final list of covariates included in the childhood undernutrition models are mean years of education of women of reproductive age, log transformed lagged-distributed income and total caloric availability (kcal per capita), which remained the same as GBD 2013. Uncertainty in the estimates was based on the data variance, then calculated through ST-GPR.

The final step of exposure estimate is to calculate the distribution of undernutrition prevalence across different levels of severity and age- sex- groups. The levels of severity are defined as follows:

Severe: individuals less than 3SD below the median (<-3SD);

Moderate: individuals between 3SD and 2SD below the median (-3SD to -2SD);

Mild: individuals between 2SD and 1SD below the median (-2SD to -1SD).

In GBD 2013, prevalence of undernutrition in each of severity categories was predicted by applying a linear regression model of the prevalence of undernutrition in each of severity categories against the prevalence of undernutrition below -2SD of the reference median at global level using microdata from 179 DHS surveys. We assumed no difference in the prevalence of undernutrition at any severity level across age and sex among children under 5.

This strategy has experienced a major change in GBD 2015. We estimated the prevalence of undernutrition by GBD age-sex groups, assuming the distribution of undernutrition of different severity categories are difference across age and sex among children under 5. Using available microdata, we first created a pooled global database that consisted of binary indicators of undernutrition by GBD age-sex groups at individual level. Then we ran a logit regression model to predict the proportion of undernutrition outcome in most-detailed severity category (e.g. <-3SD) among the broader severity category (e.g. <-2SD) against the effects of age group and sex. We also took into account the covariance of the proportions among different age-sex groups by using variance-covariance matrix. Last, we applied the proportions by GBD age-sex group generated above onto our GPR estimates.

Theoretical minimum-risk exposure level

Theoretical minimum risk exposure levels (TMREL) for underweight, stunting, and wasting where all children under the age of 5 are above -1SD of the WHO 2006 standard weight-for-age, height-for-age, and weight-for-height curves respectively.

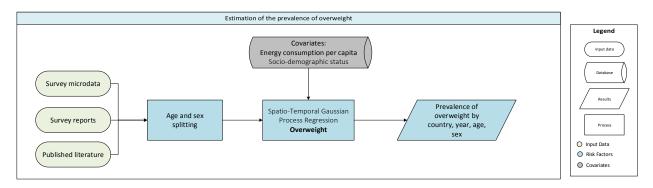
Relative risks

Relative risks (RRs) of risk-outcome pairs were extracted based on a study that conducted a pooled cohort analysis (7), which remained the same as GBD 2013. The final list of outcomes paired with childhood undernutrition risks included lower respiratory infections (LRIs), diarrhea, measles, and protein energy malnutrition (PEM). Originally in GBD 2013, upper respiratory infections (URIs) and otitis media were considered as analogies for LRI considering the similar pathological pathways they share. However, they were dropped from analysis in GBD 2015 due to the lack of evidence on the causal relationships with undernutrition risks. We also attributed 100% of PEM to childhood wasting and underweight but not stunting. A literature search was conducted for GBD 2015 searching for meta-analysis on the association of risk-outcome pairs published after January 1st, 2013, no updated results was found.

The RRs were adjusted using an optimization algorithm we developed at IHME for GBD 2013 that takes into account covariance between the three undernutrition indicators.

Body Mass Index SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompasses SDG Health Index Indicators associated with childhood overweight: 2.2.2b.

Indicator 2.2.2b

As a component of SDG Goal 2. End hunger, achieve food security, and improved nutrition, SDG Target 2.2, by 2030, end all forms of malnutrition, including achieving, by 2025, the internationally agreed targets on stunting and wasting in children under 5 years of age, and address the nutritional needs of adolescent girls, pregnant and lactating women and older persons, is measured using SDG Health Index Indicator 2.2.2b, Prevalence of children aged 2 to 4 years with a body-mass index (BMI) exceeding the overweight cut-offs established by the International Obesity Task Force (IOTF) for each sex and by month of age.

Case definition

Exposure to overweight is defined using metrics related to national and subnational estimates of BMI. If a person has a BMI of greater than IOTF cutoff for each sex and age (in month), they are considered overweight.

Input Data

We searched Global Health Data Exchange (GHDx) database for individual level data from multi-country survey programs, national surveys, and longitudinal studies providing measured data on height and weight. Additionally we searched GHDx for published literature providing data on measured height and weight. We included surveys, reports, or studies that provided nationally or subnationally representative estimates of overweight. Sources were excluded if using alternative standards for defining child obesity

which were not comparable with the IOTF standard; utilizing alternative measurement methods (e.g., hydrodensitometry, MRI, CT, skin-fold thickness, and waist-circumference) to estimate the prevalence of overweight; or reporting data on children under the age of 2. We also excluded studies that did not select a random sample of the population; studies conducted among a particular population group (e.g., specific employment status, economic status, pregnant women, patient groups); studies that did not provide adequate details of the sampling method or the sample composition; and studies with a sample size of less than 100. Data points from surveys with high level of missingness caused by incomplete entries on height and weight (>15%) were also excluded. Report and literature data with age groups wider than the standard age groups or with data on both sexes combined were split using the approach used by Ng et al. Briefly, age-sex patterns were identified using sources with data on multiple age-sex groups and these patterns were applied to split aggregated report data. Uncertainty in the age-sex split was propagated by multiplying the standard error of the data by the square root of the number of splits performed.

Modeling strategy

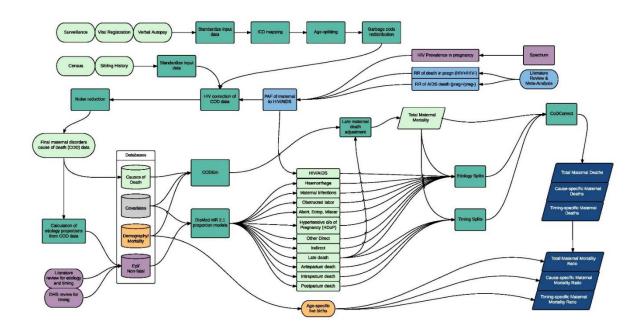
We used a spatiotemporal Gaussian process regression (ST-GPR) model to estimate the prevalence of overweight among children aged 2 to 4 years. To borrow strength across age groups, we included overweight data from adults in modeling of the prevalence of overweight among children. The mean functions used in ST-GPR were estimated using the following hierarchical mixed-effects linear regressions, run separately by sex:

$$logit(ow_{c,a,t}) = \beta_0 + \beta_1 energy_{c,t} + \beta_2 SDS_{c,t} + \sum_{k=3}^{19} \beta_k I_{A[a]} + \alpha_s + \alpha_r + \alpha_c + \epsilon_{c,a,t}$$

where energy_{c,t} is a 10-year moving average of energy intake per capita; SDS (socio-demographic status) is a composite measure of social status combining economic, social, and demographic factors; $I_{A[a]}$ is an indicator variable for specific age group A that the overweight prevalence point $ow_{c,a,t}$ is capturing, and $ow_{c,a,t}$

Maternal Mortality SDG Capstone Appendix

Flowchart



Input data & Methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with maternal mortality (3.1.1).

Indicator 3.1.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.1, by 2030, reduce the global maternal mortality ratio to less than 70 per 100,000 live births, is measured using SDG Indicator 3.1.1, maternal mortality ratio (maternal deaths per 100,000 live births).

For the present analysis, we report on MMR estimates generated for women aged 15-49 to align with the proposed indicator definitions by the IAEG-SDGs. For the Global Burden of Disease Study 2015 (GBD 2015), MMR estimates are estimated for a wider age range (10-54 years); MMR results for women aged 15-49 are derived from broader maternal mortality analysis and thus we provide the data and methodological details on the broader definition for MMR.

Input Data

Maternal mortality data identification and processing

Appendix Figure 1a and 1b show the high-level view of data inputs, analytical steps, and outputs of the cause of death analysis frame. The complexity of the overall process can be usefully divided into three broad phases: data inputs on the event of death going into the cause of death database that are analyzed using CODEm; data inputs on precursors to death that are modeled through a variety of strategies; and the integration of these streams of analysis into a single set of cause of death estimates by age, sex, year, and geography with uncertainty through the CoDCorrect algorithm. The process for cancer and for HIV/AIDS is somewhat different, and described in more detail below.

Cause of death data identification

1. Overview of data types

The cause of death database contains six types of data sources: vital registration, verbal autopsy, cancer registry, police records, sibling history, surveillance, and survey/census. The highest-quality data will have detailed characteristics of each demographic group and detailed causes of death across the time series. Countries with complete vital registration systems are considered to be high-quality. For counties with incomplete vital registration systems, vital statistics for causes of death can be supplemented with other data types to provide cause-specific estimates.

2. ICD detail

A majority of the cause of death data is vital registration data obtained from either the WHO Mortality Database, country-specific mortality databases operated by official offices, provided by trusted country collaborators. It is considered to be the highest-quality data, as it is the most comprehensive. Each cause is coded directly to the most detailed cause of death when possible, whereas cause codes in International Classification of Diseases (ICD) tabulated data are coded to aggregated cause groups. The cause of death database contains 8,044 location-years of detailed data from 1980 to 2014, which includes underlying causes of deaths coded with 3-5 digit se codes, by country, year, sex, and age groups. Detail causes are coded to one of the following ICD detail coding systems: ICD8, ICD9, or ICD10. Each coding system has a similar cause hierarchy and cause list that has continually developed over time. ICD10 is the current standard and most exhaustive cause list. Within the cause lists 5-digit codes are truncated to 4-digit codes to condense the cause lists. Updates to ICD detail occur biannually as WHO releases new versions or as country collaborators provide additional data. Updates to data from WHO increasingly include ICD10 cause of death data, as it is the most current classification of cause of death, while updates to ICD8 and ICD9 detailed lists are less common. In the case of overlapping data, preference is given to data from predetermined country collaborators, which is updated annually.

3. ICD tabulations lists

The ICD tabulation lists include the ICD8 List A (ICD8A), ICD9 Basic Tabulation List (BTL), ICD10 Mortality Tabulation, Russia Tabulation list, and India Medical Certification of Cause of Death (MCCD). These data sources make up 2,664 location-years from 1980 to 2014 in the cause of death database. All are condensed versions of the ICD9 and ICD10 detail lists, with some differences in the format of cause lists depending on the data source. ICD8A, ICD9 BTL, and ICD10 Mortality Tabulation cause of death are assigned to subtotal groups, referred to as chapters, and cause groups respective to ICD detail groups. Additionally ICD9 BTL includes ICD9 detail codes for some cancers and a custom tabulation scheme for

the former USSR countries. The Russia Tabulation lists and India Medical Certification of Causes of Death (MCCD) cause lists each have custom nomenclatures based on ICD detail cause codes.

Two of the drawbacks in data using tabulation lists are discrepancies in the accuracy of death counts and lack of detail to due to aggregated cause groups. There are instances where the sum of deaths in chapter subtotals are not equal to the sum of cause groups within the chapter. To account for any missing or duplicate deaths reported within the cause groupings, death counts are systematically adjusted, by calculating the differences between subtotals and sub-causes within the cause groups. Any differences are assigned to a remainder cause group. To account for the lack of cause code detail, select cause groups are disaggregated (Step 1.1) to create a complete cause list. Updates to ICD Tabulation lists obtained from WHO occur less frequently compared to ICD detailed lists, as more countries are reporting deaths in ICD detail. In instances of overlapping data, preference is given first to data from country collaborators' data from WHO, then preference to ICD detail data from WHO, before choosing to use ICD tabulation lists.

China DSP/ China CDC

The two primary sources of data for China are surveillance data from the China Disease Surveillance Points (DSP) system and vital registration collected by the Chinese Center for Disease Control and Prevention (CDC). In the China DSP data, deaths were reported across 145 disease surveillance points used from 1991 to 2003, and 161 points used from 2004 to 2007. While China DSP with ICD10 code is considered surveillance data, it provides national coverage and cause detail. Thus it receives similar processing and treatments to the China CDC vital registration from 2008 to 2012. From 2008 to 2012, all of the deaths and cause of death information from the Disease Surveillance Points system and other system points throughout China were collected and reported via the Mortality Registration and Reporting System, an online reporting system of the Chinese CDC. The deaths in these data are reported at the strata level, a metric that is specific to China. Counties are stratified by urban and rural classification, but definitions of urbanity vary across counties. In Step 7.1 we use a method developed to scale up deaths from strata level to the province level.

India CRS

The India Civil Registration System (CRS) is a continuous systematic recording of deaths and births. We consider these data to be similar to vital registration as the coverage is national. Usable cause-specific death data are in the form of a custom cause list and exist between 1983 and 1995, with 1983-1984 and 1986-1987 including deaths data specific to rural or urban locations. Due to the lack of complete India subnational locations we state-split the data after age-sex splitting to fill in data gaps (Step 1.2). Though this system is continuous we have not found any cause-specific data past 1995. The more recent years of the report only include data for all-cause mortality.

India MCCD

The India MCC) is the largest data source we have for India, with nationally representative data in a majority of the urban states and also union territories beginning in 1980. Deaths reported in this data source have been medically certified and are considered vital registration. The causes of death are reported in a tabulation list with a unique numbering scheme that conform to ICD9 and ICD10 detail codes, which must be disaggregated. Similar to the CRS, MCCD is state-split to fill in data gaps (Step 1.2 State Splitting); however, in these data state splitting occurs prior to age-sex splitting.

4. Verbal autopsy

VA coded to ICD 10 and VA coded to other lists

In countries without vital registration systems, verbal autopsy studies are a viable data source to inform cause of death. Data are obtained by trained interviewers who use a standardized questionnaire to ask relatives about the signs, symptoms, and demographic characteristics of recently deceased family members. Based on the answers to the questionnaires a cause of death is assigned.

Verbal autopsy (VA) data are highly heterogeneous: studies use different instruments, different cause lists from single causes to full ICD cause lists, different methods for assigning cause of death based on a completed verbal autopsy, different recall periods, and different age groups, quite apart from cultural differences in the interpretation of specific questions. The validity of cause of death must be considered when mapping to a GBD cause. Verbal autopsies are likely accurate in assigning cause of death to road injury or homicide, but less accurate for causes requiring medical certification, such as cardiovascular causes. Studies can also occur once in a particular country or as part of an extended network, such as INDEPTH. INDEPTH is a continuous surveillance source with several Demographic Surveillance Systems sites that collect data which is coded to ICD detail causes.

INTERVA modeled VA

In previous years, INTERVA modeled VA was excluded from our analysis. Verbal autopsies used in our analysis are non-INTERVA, as they use questionnaires and modules consistent with WHO standards. The Population Health Metrics Research Consortium (PHMRC) published a study that shows results of INTERVA-modeled VA are not compelling enough to be credible, thus we have decided to exclude data for all causes due to low validations with the exception of injuries in Sub-Saharan Africa. We lack data in sub-Saharan Africa and use INTERVA to fill in gaps and stabilize injuries patterns.

India SCD and SRS

Deaths reported in verbal autopsy studies in rural Indian states can be accounted for in the Survey of Causes of Death (SCD) from 1980 to 1994 and in the Sample Registration System (SRS) post-1998 in urban and rural states. Data in the SCD were collected through a verbal autopsy survey from a sample of villages. To expand our estimates to more states and causes we used methods of state splitting post-mapping to GBD causes (Step 1.2). Like the SCD, SRS also records deaths in a sample of villages but also includes urban blocks in its sample. This survey is collected by the Registrar-General of India, and since 1999 SRS has merged with SCD to provide ongoing coverage on both urban and rural areas.² The maternal data reported in SRS are given in the maternal mortality ratio (MMR), which we convert to deaths by state and urbanity by using our estimates for the SRS population and neonatal mortality rates.

5. Other data types

Maternal mortality data

In locations with low-quality vital registration or no vital registration, maternal mortality metrics can be found in surveillance, surveys, census, and sibling history data sources. The best data have death counts due to maternal causes and the total number of deaths for women within the reproductive ages of 10 to 54 (previously 15 to 49) by year. If a data source is missing these components, it is necessary to create a complete cause list using live births and all-cause mortality deaths (Step 1.4). Though death counts is the preferred metric, maternal mortality (MM) is often measured using MMR, which is easily converted to

deaths using live births. An additional adjustment that must be applied to the China Maternal and Child Surveillance data is scaling data from the strata to the province level (Step 7).

Surveys and censuses reporting fraction of deaths due to selected injuries

Surveys and censuses are often used in countries with less developed VR systems, or in countries with adequate VR these data source are supplementary. Much like the VAs, the validity of cause of death is a concern due to lack of medical certification at the time of death. For these data sources we keep only causes related to maternal mortality and injuries. The remaining causes are accounted for as a remainder of total deaths in the sample size.

Police records

In most countries, police and crime reports are an important source of information for some types of injury deaths, notably road injuries and interpersonal violence. Our police data comes from reports on road traffic and crime trends. The police reports used in this analysis were obtained from published studies, national agencies, and institutional surveys such as the UN Crime Trends survey and United Nations Office on Drugs and Crime Global study on Homicides. We can assess whether police reports were likely to be complete and cover the entire country if police trends are close to trends seen in vital registration. Data are excluded in instances where police data for road traffic injuries are significantly lower than our vital registration. The threshold for exclusion is less than 80% of the cause fraction of the road traffic injuries in VR. Police data that meet our inclusion criteria and provide complete coverage are uploaded to the database for injuries causes.

6. Population-based cancer registries

Cancer registries with incidence

Data on cancer incidence was sought from individual population based cancer registries as well as from databases that include multiple registries, for example "Cancer Incidence in Five Continents" (CI5) (NID 133224), NORDCAN (NID: 113386), or EUREG (114368). Cancer registries were identified through the membership list of the International Association of Cancer Registries (IACR), through the GBD collaborator network, or through the GHDx. Registries were excluded if they were not representative of the coverage population, if they did not contain incidence data tabulated by cancer site, if the data were limited to years prior to 1980, if the source did not provide details on the population covered, or if the list of cancer sites included was not comprehensive.

Cancer registries with incidence and high quality mortality

In addition to incidence, some high-quality cancer registries also report cancer mortality data. These data were also extracted and used as inputs into the mortality-to-incidence (MI) ratio model.

General Modeling Strategy

Step 1. Standardize input data

The input data to the Cause of Death (CoD) database are received in various formats and must be standardized to run through central CoD machinery to then upload to the database. Raw data inputs come from data sources such as mortality databases, literature reviews, or reports. Usable data sources

must have a clear sample size of the number of deaths in the population and exhaustive cause lists. The complexity of the cleaning process varies drastically across data sources. For vital registration micro-data with the location, age, sex, year, and ICD-coded cause of every death, very little effort is necessary to standardize it into a consistent structure. Other sources may require weeks of careful review to accurately extract scans of hardcover cause of death reports into spreadsheets that can be transformed and standardized.

At this point, data are assigned source identifiers so that they can be linked to the Global Health Data Exchange (GHDx) and cited appropriately. Any aggregate age and sex categories are flagged for age-sex splitting. The methods of cause-of-death assignment and data collection are reviewed to determine which source type to assign; for example, we distinguish sibling history data from surveys with a verbal autopsy module. Only data at the most detailed level of the Global Burden of Disease location hierarchy are used. Documentation from the source is reviewed to determine if the population is representative of the location or only a subset of the population in that location. Data sources representing a subset of the population are flagged as non-representative; this flag is used by CODEm to increase the variance associated with such data points.

Finally, diagnostics are reviewed at this stage to avoid sending cleaning errors downstream. We review cause-specific deaths for each demographic group to ensure the data are reasonable. For example, it is unlikely that male breast cancer deaths are higher than female breast cancer or deaths from neonatal causes occur in age groups over one year. All deaths totals are compared with the sum of cause-specific deaths to ensure the observed deaths are accounted for and sample size is complete.

1.1 Disaggregation

Causes of death in tabulated vital registration data are condensed into aggregated groups, some of which can be mapped directly to GBD causes while other aggregated cause groups are not informative and cannot be mapped to GBD causes. To correct for this, aggregated causes were mapped and split onto multiple ICD9 and ICD10 detail causes, or targets, based on the ICD groupings within the aggregated causes. Both ICD9 and ICD10 detail codes serve as targets because they are the highest quality vital registration data and enabled the calculation of proportions used to split the aggregated cause data into detailed causes. The proportions of deaths from nearby countries within the super region were used to fill in data gaps as they were likely to have similar cause of death trends.

We determined the targets based on detail causes missing from the tabulated cause list. For example, in ICD9 BTL, the tabulated cause list includes a viral diseases group. In the hierarchy of causes, this group consists of measles, yellow fever, encephalitis, hepatitis, rabies, other infectious diseases, garbage code, and remainder of viral diseases. We did not consider this list to be an exhaustive list of viral diseases based on the range of ICD detail codes given in the ICD9 BTL documentation. To make the cause list exhaustive and inclusive of other viral diseases, we split the remainder of viral diseases group into: other meningitis, other infectious diseases, herpes, dengue, other neglected tropical diseases, and garbage code. After a list of targets was determined, the aggregated deaths were disaggregated to the target causes using ICD9 and ICD10 detail proportions generated at the super region level for the corresponding sex and age groups across all years in the time series. For example, in ICD9 detail data, 54.8% of deaths in males in Latin American and Caribbean within the target group for BTL Viral Diseases were designated "other meningitis", so 54.8% of deaths in the tabulated group, "remainder of viral diseases", were

assigned to "other meningitis" for any country within that particular super region. For any cause and demographic group where we lacked ICD detail, global proportions were used.

1.2 State splitting

Two important sources for cause of death estimation in India are the MCCD report, which reports medically certified deaths from health facilities in mostly urban areas ³, and the SCD, which collects information via verbal autopsy on about one-half of 1% of all rural deaths in India, based on populations living in about 1,300 primary health care centers spread throughout the country. ⁴ For both of these reports, data missingness impedes estimation of trends at the state level. We used a first-order, log-linear model of the four-way contingency table of deaths by sex, age, state, and year to estimate the missing state-years. We fit the model to all available data for MCCD and SCD separately for each cause, including state-specific all-age measurements and age-specific national measurements. From this, we produced estimates for each combination of sex, age, state, and year. We then used these estimates wherever the raw data did not include sex-, age-, state-specific death counts.

For MCCD, the model was fit separately for ICD10- and ICD9-based reports using the tabulated cause list present in the data. In the SCD report, the model was fit for each GBD cause in the data. As data from the SCD reports were relatively sparse, the pooling of like causes together led to an improved model fit.

1.3 Region-sex-cause to state-urbanicity-age-sex-cause algorithm

We also made use of the Special Survey of Deaths in 2001–2003 and 2004–2005, a representative, national and subnational verbal autopsy study under the Sample Registration System in India. Data for the top 10 causes of death were available by region and sex, but we require data by urban and rural state in India. To achieve this, we first split the regions into states in proportion to the number of GBD-estimated deaths in each state. We then determined the urbanicity of the resulting state-cause-deaths by applying the SRS distributions of urban and rural deaths by cause in India. We then used a relative-rate splitting algorithm that accounts for the population structure of urban and rural areas in the state. Finally, we applied the SRS all-India age distribution to the all-age urban-rural-state-data with a similar approach, accounting for the age-specific population structure of urban and rural areas in each state.

1.4 Calculate non-maternal deaths

In cases when maternal mortality metrics do not include both deaths due to maternal causes and deaths due to non-maternal causes for women of reproductive ages, live births, and all-cause mortality estimates can be used to calculate deaths. Many studies report maternal deaths as the MMR. MMR is the number of maternal deaths per 100,000 live births and can be used to calculate deaths when it has been derived from primary data and not estimated. Maternal deaths were calculated using MMR and live births, if live births were missing we substituted live birth estimates and used the following equation:

Maternal deaths = (MMR/100,000) * Live births

If a study was non-representative we extracted sample size and live births from that study. After maternal deaths were calculated, we used the difference from all-cause mortality estimates to determine non-maternal deaths.

A more accurate and data inclusive method of calculating maternal and non-maternal deaths incorporates coverage and splits deaths for a range of years into individual years. If there were live births in the study we adjusted the coverage.

Coverage = live births / GBD estimated live births

After coverage was calculated, totals deaths were scaled to be more representative. This gives a more accurate death count since the envelope assumes representative coverage. Using all-cause mortality as an all-cause total, non-maternal deaths were subsequently calculated.

Maternal envelope with coverage = maternal envelope * coverage

An additional adjustment can be applied to maternal data spanning over a range of consecutive years, which allows for more data inclusion. The years within specified year ranges are separated into individual years and total deaths within the year range were split between each individual year using the fixed proportions of maternal deaths from vital registration in that particular country. We only used vital registration to inform the proportions because it was both high quality and representative.

Step 2. Map to GBD cause list

In GBD 2015 we developed 411 maps to translate causes found in the input data to the GBD 2015 cause list. This included 40 maps for vital registration data, 279 for verbal autopsy data sources, and 92 for other data types. The largest and most universal maps used were those for ICD9 and ICD10 detail vital registration data. The input data causes varied from 3-4 digit ICD codes to custom cause lists with cause names such as cholera or hepatitis. Our mapping process made it possible to compare these various data sources across demographic groups.

Appendix Table 1 shows the ICD10-detail and ICD9-detail codes included in the mapping of each GBD cause.

2.1 India urban/rural splitting

Another source of data for urban and rural state estimation of cause of death trends in India is the CRS, from which we retrieved usable data over the period 1983-1995. From 1983-1987, data were available for the urban and rural populations of each state in the system. However, after 1988, only state-level data were available.

We can only use data at the most detailed location level that we estimated for cause of death models. As a result, in order to use of all years of the CRS data split state-level mortality into urban and rural state-level mortality. To do this, we assumed the same relative rate of cause-specific urban and rural mortality in 1988-1995 as was present in the data in 1983-1987. We applied the same algorithm that is used for age-sex splitting, modified for this purpose:

$$D_{s,u,y,a,x,c} = R_{s,u,c}N_{s,u,y,a,x} \frac{D_{s,y,a,x,c}}{\sum_{u=urban}^{u=rural} (R_{s,u,c}N_{s,u,y,a,x})}$$

Where:

 $D_{s,u,y,a,x,c}$ = Number of observed deaths in state s, urbanicity u (either rural or urban), year y, age a, sex x, and cause c

 $R_{s,u,c}$ = Death rate in state s, urbanicity u, cause c from 1983-1987; data were not suitable for age or sex specific rates.

 $N_{s,u,y,a,x}$ = GBD-estimated population in state s, urbanicity u, year y, age a, sex x

 $D_{s,y,a,x,c}$ = Number of observed deaths in state s, year y, age a, sex x, and cause c (the deaths that are being split)

The result was a full time series of CRS data for both rural and urban populations of each state in the CRS system.

2.2 State splitting

This step is described in step 1.2, above.

Step 3. Age-sex splitting

3.1 Generate global age-sex weights by cause

Different sources, particularly verbal autopsy studies, report deaths for a wide range of age-groups with varying intervals. For the analysis of causes of death, we mapped these different age intervals to the GBD standard set of age-groups. The approach to undertake this mapping was the same as in the prior two GBD studies, GBD 2013 and GBD 2010.

In the process of assembling a consolidated demographic database, perhaps the most impairing source of inconsistency is the aggregation of age groups. It is conventional to report such data in broad age groupings such as "0-4, 5-14, 15-49," or to report data with both sexes together. The issue of comparability between age-sex groups arose when assembling the GBD cause of death database. The compiled database included 22 distinct tabulation formats for infants and 141 distinct tabulation formats for non-infants. We developed a tool, which we call age-sex splitting, that takes aggregated age groupings, and likewise the "both sexes combined" grouping, and divides them into what their constituent age groups would likely have been using respective cause-specific and country-specific age distributions. The analytical framework for the GBD includes three infant age categories: Early neonatal (0-6 days), late neonatal (7-27 days) and post neonatal (28 days to one year), and 17 non-infant age categories starting with age one to four years, then proceeding in five-year age groups until the terminal age group of 80+. We treat unknown ages and sexes in the same manner we treated the "all ages combined" age category and "both sexes combined" sex group. Through this process, we were able to directly compare all data sources on even terms.

The approach to age splitting is based on the following formula. The key assumption underlying this formula is that the relative risk of death by age group compared to a reference age group is invariant across populations. While this assumption is likely violated in specific cases, there is a strong biologically based pattern of the relative risk of death for a cause by age that is observed for most causes. The basic formula is as follows:

$$D_a = R_a N_a (\frac{D_a^{a+x}}{\sum_a^{a+x} (R_a N_a)})$$

Where:

 D_a = the number of deaths from a cause in age group a

 R_a = the relative risk of death in age group a compared to a reference group

 N_a = the country-year-sex-specific population in age group a

 D_a^{a+x} = the number of deaths in the age group a to a+x

With the assumption of invariant relative risks of death by age with respect to a reference age group, this equation can be used, along with population distribution by age, to split an aggregate number of deaths for the age groups a to a+x into specific deaths for each age group within the aggregate interval.

In some cases, deaths are reported for an aggregate age group for both sexes combined. The task in this case is more complicated, but the same principle can be applied. In this case we assumed that the relative risks of death by and sex are constant.

$$D_{as} = R_{as} N_{as} (\frac{D_{as}^{a+x,s}}{\sum_{a}^{a+x} (R_{as} N_{as})})$$

Where:

 D_{as} = the number of deaths from a cause in age group a, sex s

 R_{as} = the relative risk of death in age group a compared to a reference group for sex s

 N_{as} = the country-year-sex-specific population in age group a for sex s

 $D_a^{a+x,s}$ = the number of deaths in the age group a to a+x for sex s

This equation can be used to split data aggregated over age and sex. The assumption, however, of invariant relative risks across age and sex is a stronger assumption. Fortunately, data pooled across sexes are less common in the published or unpublished cause of death data.

The relative risk of death in a particular age group for a given sex is derived from the global distribution of cause-specific mortality rates found in available vital registration data. Location-years from the following code systems are used, provided they report the requisite age- and sex-detail: ICD7, ICD8, ICD9 BTL, ICD10 tabulated, ICD9, and ICD10. Upon compiling these data, we mapped them to GBD causes, and aggregated up to cause level 3. This is the level at which a particular cause is split – that is, any daughter cause of a level 3 parent is split using the age distribution of that parent (so, chronic kidney disease due to diabetes would be split using the age pattern of chronic kidney disease).

We next adjusted separately for estimated adult and child vital registration completeness. Location-year-age-sex-specific deaths and population were then aggregated across all location-years, in order to produce cause-specific mortality rates by age and sex. These were used to determine the risk of death at any age relative to any reference age group.

Step 4. Correct age-sex violations

Occasionally, data sources will include deaths by a cause for which there is medical consensus that death is impossible for the sex and age. For example, there may be some number of deaths due to cervical cancer in males, or deaths due to maternal causes in ages under 10. We have constructed a conservative list of age-sex restrictions. When deaths violate these restrictions, we redistribute them proportionally onto all causes.

Step 5. Redistribution

A crucial aspect of enhancing the comparability of data for cause of death is to deal with uninformative, so-called garbage codes. Garbage codes are codes to which deaths were assigned that cannot or should not be considered as the underlying cause of death, for example: heart failure, ill-defined cancer site, senility, ill-defined external causes of injuries, and septicaemia. The methods for redistributing these

garbage-coded deaths were outlined in detail in Naghavi et al,⁵ and the underlying algorithm for redistributing deaths assigned to these codes has not changed since GBD 2013.

5.1 Redistribute HIV-related garbage

The list of garbage codes known to be used to code deaths caused by HIV/AIDS can be found in Appendix Table 2.

Due to the disparate nature of HIV/AIDS mortality across space and time, dynamic redistribution of HIV/AIDS-related garbage codes was needed. To inform this redistribution, we generated target proportions for each garbage group by age band (Under 1 month, 1-59 months, 5-19 years, 20-49 years, 50-59 years, 60-69 years, 70-79 years, and 80+ years), 5-year time interval, and sex. The garbage groups will either target HIV or a remainder target. The allotment of deaths to either of these is based on the regional increase in the mortality rate of all codes in the group relative to the rates seen in 1980-1984 – an increase greater than 5% is assumed to be HIV/AIDS-related, and the proportion of those deaths exceeding 5% are redistributed to HIV/AIDS. Any increase \leq 5% is then assigned to the remainder target.

5.2 Regress garbage codes versus non-garbage

As in GBD 2013, the statistical analysis used to determine proportions for garbage code redistribution for ill-defined cancer sites, ill-defined external causes of injury, unspecified stroke, heart failure, hypertension, and atherosclerosis was based on the approach outlined by Ahern et al.⁶ For each redistribution package, we defined the "universe" of data as all deaths coded to either the package's garbage codes or the package's redistribution targets for each country, year, age, and sex. We then ran a regression based on the following equation, separately for each target group and sex:

$$TG_{crt} = \alpha + \beta_1 Gar_{crt} + \beta_2 Age_{crt} Gar_{crt} + \theta_r Gar_{crt} + \gamma_r + \varepsilon_{ct}$$

 TG_{crt} = percentage of deaths within the given garbage code's universe which were coded to a given target group, by country

 Gar_{crt} = percentage of deaths within the given garbage code's universe which were coded to a given set of garbage codes

 α = constant

 β_1 = slope coefficient describing the association between Gar_{crt} and G_{crt}

 β_2 = slope coefficient describing the association between the interaction $Age_{crt}Gar_{crt}$ and G_{crt}

 $\gamma_r =$ region specific random intercept (or super region if the random effect on region is not significant)

 $\theta_r=$ region specific random slope (or super region if the random effect on region is not significant)

 $\varepsilon_{ct}=$ standard error, normally distributed and calculated by bootstrapping

This regression was adjusted from GBD2013 to include fixed effects on the interaction of garbage and age to ensure smooth age patterns. We made this decision after investigating diagnostic visualizations that showed unlikely gaps between proportions assigned to different age groups.

Once proportions were produced for each country, sex, age, and target group, certain adjustments were made to conform our packages to the best medical evidence available. In some cases, we implemented restrictions on the proportions that the regressions could yield. For example, we did not allow any

redistribution onto Chagas disease outside of Latin America and the Caribbean, or suicide under the age of 15. In other cases, we capped the proportion for some targets to the level that would be produced from proportional redistribution; for example, hemoglobinopathies and hemolytic anemias were restricted to the level of proportional redistribution in the redistribution of left heart failure. Occasionally, further adjustments were made on a case-by-case basis per country, age, sex, and target group to suppress the impact of outliers based on existing epidemiological evidence and expert judgment.

5.3 VA anemia adjustment

To compensate for the over-representative cause fractions from anemia found in verbal autopsy studies, we redistributed these deaths based on the causal attribution of severe anemia from the GBD 2013 study. The proportions were country-year-age-sex specific.

Step 6. HIV/AIDS misclassification correction

In many location-years, certain causes of death known to be comorbid with HIV/AIDS (e.g., tuberculosis, other infectious diseases) are seen to have age-patterns that diverge from those observed in location-years without widespread HIV/AIDS epidemics, and are in fact more reflective of HIV/AIDS mortality trends. In order to identify these instances, a global relative age pattern is generated using all VR deaths in countries with observed HIV prevalence less than 1% using the following:

$$RR_{asc} = \frac{R_{asc}}{\bar{x}(R_{65sc}, R_{70sc}, R_{75sc})}$$

Where RR_{asc} is the relative death rate for age group a, sex s, and cause c; R_{asc} is the rate for that age group; and $\bar{x}(R_{65sc}, R_{70sc}, R_{75sc})$ is the mean of the rates in ages 65-69, 60-74, and 75-79 for that sex and cause. This is preferable to comparing mortality rates because we are able to isolate divergence in age pattern while accounting for varying levels of overall mortality by fixing death rates to age groups that are unlikely to be confounded by the presence of HIV. Expected deaths for an identified cause were then determined to be:

$$ED_{lyasc} = \bar{x}(R_{ly65sc}, R_{ly70sc}, R_{ly75sc}) * p_{lasc} * RR_{asc}$$

Where ED_{lasc} are deaths for location I, year y, age group a, sex s, and cause c; $\bar{x}(R_{l65sc}, R_{l70sc}, R_{l75sc})$ is the mean of the rates for ages 65-69, 60-74, and 75-79 for that location-year-sex-cause; p_{lasc} is the population for that location-year-age-sex-cause; and RR_{asc} is the global standard relative rate determined in the previous step for that age-sex-cause. The expected deaths remain attributed to that particular cause, while the difference between observed and expected are reallocated to HIV/AIDS.

Step 7. Scale strata to province

Over time, a higher proportion of deaths have been registered in China through the expansion of the DSP system and provincial and county efforts to increase cause of death registration. With the expansion of coverage, it is possible that province aggregates do not accurately represent the population distribution between urban and rural areas in each year. For this reason, we stratified the data preparation by urban and rural status for each county within each province. Stratification was based on the median level of urbanization across counties within each province as recorded in the 2010 China census. In the provinces of Tibet and Hainan, all counties were placed into one strata based on largely homogeneous urbanization levels within each province. This yielded a total of 62 analytical province-strata. Macao and Hong Kong were not included in this stratification system as the VR systems there are independent from that on the

mainland; no weighting scheme needs to be carried out in these complete VR systems with quality data on causes of death.

Within each province-strata, a larger proportion of deaths in-hospital might be reported than that of deaths outside of hospital because of the internet hospital reporting system. To avoid bias, we reweighted in-hospital and out-of-hospital deaths based on the age-sex-province-specific fraction of deaths in and out of hospital in the DSP system. DSP data have been used to establish these percentages because, in these communities, there is a concerted effort to identify all out-of-hospital deaths. Province-strata death rates are combined to produce overall province death rates by weighting each strata by population in each age-sex-year group. Province death rates are rescaled so that all-cause mortality equals the estimated death rate in each age-sex-year estimated in the life-table analysis. The Bayesian noise reduction algorithm was used to deal with zero counts and small number issues for rare causes.⁷

Step 8. Restrictions post-redistribution

Some causes of death can only be reliably assigned through an autopsy by a trained physician. For example, it is unlikely that a verbal autopsy would reliably distinguish between ischemic and hemorrhagic stroke.

In this step, it is ensured that the detail of the cause list at this point in the data prep process is reasonable given the detail of the original data source and the methods by which the cause of death was assigned. Two primary corrections are applied. First, any cause which is purely an artifact of the redistribution machinery targeting too detailed a cause is aggregated up to the parent cause. Second, a "bridge map" is applied over a certain set of sources to ensure that these sources do not contain causes which could not reliably be determined by the methodology. These two corrections are applied to ICD9-BTL, ICD10-tabulated, USSR tabulated ICD9, India MCCD reports, China-DSP-tabulated-ICD9, India SCD reports, and all verbal autopsy sources.

Step 9. Drop VR country years or mark as non-representative based on completeness

Lozano and colleagues⁸ describe the negative impact that low-completeness VR data can have on cause of death modeling for the GBD 2010. In particular, in settings where a data source does not capture all deaths in a population, the cause composition of deaths captured might be different from those that are not. However, a completeness sensitivity test found that low-completeness VR data had little impact on the cause-specific mortality trends at the global level.

For GBD 2015, we investigated the impact of these data at the country and subnational levels using the more thorough diagnostic visualizations available to us. It was determined that these data produced unlikely trends in the models affected. Despite the minimal impact on global trends, better models were produced by eliminating or marking as non-representative data with extremely low completeness. VR completeness was estimated using death distribution methods (DDM) described in the all-cause mortality section of the appendix.

For this round, vital registration location-years with completeness below 50% were dropped, while location-years with completeness between 50-69% were marked as non-representative.

The following country-years were dropped from the database:

Location Years Below 50% Completeness			
Location	Years Below 50% Completeness		
'Asir	1999 - 2012		
Bahah	1999 - 2012		
Eastern Province	1999 - 2003		
Ha'il	1999 - 2012		
Haiti	1981, 1999, 2002 - 2004		
Iran	1980 - 1985, 1987		
Jawf	1999 - 2012		
Jizan	1999 - 2012		
Makkah	1999 - 2005		
Maranhão	1985		
Najran	1999 - 2012		
Northern Borders	1999 - 2012		
Papua New Guinea	1980		
Qassim	1999 - 2003		
Riyadh	1999 - 2012		
Tabuk	1999 - 2002		
Turkey	1983, 1984, 1987 - 1995		

Step 10. Cause aggregation

The cause list is organized in a top down hierarchical format containing 4 levels. The first group, or level 1, sums all causes. Following all cause-mortality are level 2 causes, which include 3 broad groupings of causes of deaths: communicable, maternal, neonatal, and nutritional diseases; non-communicable diseases; and injuries. Within those level 2 groupings are finer levels used for modeling. Level 3, or parent causes, are aggregated, meaning the mortality estimate for a parent cause in the hierarchy represents the sum of the causes under that rubric. Sub-causes within level 3 causes – level 4 – are more detailed. For example, the parent cause "intestinal infectious diseases" contains the 3 sub-causes: typhoid fever, paratyphoid fever, and other intestinal infectious diseases. Included in the parent cause estimate are deaths mapped directly to the parent and any level 4 sub-causes. In data where there was not enough information to assign a level 4 cause, we aggregated to the level 3 parent cause. Exceptions to aggregating the level 4 sub-causes to the parent are instances when certain sub-causes are not present. The United Nations Crime Trends police data only identifies homicides, aggregating homicides to injuries would not accurately represent all injuries.

Step 11. Remove shocks and HIV/AIDS maternal adjustments

For GBD 2015, CODEm models use an HIV/AIDS- and shock-free envelope, as described in the all-cause mortality section of the appendix. In order to be comparable, cause fractions must also be HIV/AIDS- and shock-free. Cause fractions were uploaded to the CoD database as the number of deaths due to the cause over an adjusted sample in which the number of deaths due to HIV/AIDS, collective violence and legal intervention, and exposure to forces of nature were removed.

11.1 Remove HIV/AIDS, shocks from denominator where HIV/AIDS in cause list

The first step to generate HIV- and shock-free cause fractions was to remove any deaths from the sample which were directly coded to HIV/AIDS, collective violence and legal intervention, or exposure to forces of nature. The resulting equation for a cause fraction uploaded to the database is simple:

$$CF_{l,t,a,x,c} = \frac{D_{l,t,a,x,c}}{D_{l,t,a,x} - D_{l,t,a,x,hiv} - D_{l,t,a,x,war} - D_{l,t,a,x,disaster}}$$

In this equation, $CF_{l,t,a,x,c}$ is the cause fraction for a location (I), year (t), age (a), sex (x), and cause (c), $D_{l,t,a,x,c}$ is the number of deaths observed in the sample for the same, $D_{l,t,a,x}$ is the total number of deaths observed in the sample in the location, year, age and sex, and $D_{l,t,a,x,hiv}$, $D_{l,t,a,x,hiv}$, and $D_{l,t,a,x,disaster}$ are the number of deaths observed in the sample for HIV/AIDS, collective violence and legal intervention, and exposure to forces of nature, respectively.

Cause fractions for HIV/AIDS and shock causes were also uploaded to the database for use in separate estimation processes described in the all-cause mortality section of the appendix. In this case, cause fractions followed the standard equation, with variables following the same explanation as above:

$$CF_{l,t,a,x,c} = \frac{D_{l,t,a,x,c}}{D_{l,t,a,x}}$$

11.2 Remove HIV/AIDS deaths from maternal mortality sources

HIV-free cause fractions were also uploaded for sources on mortality due to maternal causes. In these cases, the sample of all deaths observed in the study is likely to contain some amount of deaths due to HIV/AIDS and shocks, but the sample only includes cause information on maternal deaths. To account for the presence of HIV/AIDS and shocks in the entire sample, we assumed the same proportion of total deaths due to HIV/AIDS by location, age, sex, and year as provided from the estimation of HIV/AIDS and all-cause mortality described in the all-cause mortality section of the appendix.

Maternal mortality studies were only corrected for HIV/AIDS if the sample of total deaths was provided in the data source. Where sources only provided the maternal mortality rate, we applied the rate to the HIV- and shock-free envelope produced by the analysis described in the all-cause mortality section of the appendix and thus did not need to adjust cause fractions (CFs) at this point in the process.

Where a correction was applied, we applied the following equation:

$$CF_{l,t,a,x,mat} = D_{l,t,a,x,mat} * \frac{E[D_{l,t,a,x,hiv_shock_free}]}{E[D_{l,t,a,x}]}$$

In this equation, X is the resulting cause fraction due to maternal causes for the location (I), year (t), age (a), and sex (x); $D_{l,t,a,x,mat}$ is the number of observed deaths in the sample due to maternal causes, $E[D_{l,t,a,x}]$ is the GBD estimate of all-cause mortality in the location, year, age, and sex, and $E[D_{l,t,a,x,hiv_shock_free}]$ is the GBD estimate of HIV- and shock- free mortality in the location, year, age, and sex.

11.3 HIV/AIDS correction of sibling history, census, and survey data

As described in our analysis from GBD 2013, many studies have failed to find increased mortality in HIV-positive pregnant mothers, but those who have advanced HIV are known to have increased baseline mortality. Prior to GBD 2013, we did not distinguish between deaths in HIV+ women that were caused by pregnancy and those for which the pregnancy was incidental to their death. In order to more explicitly

quantify the contribution of pregnancy to death in HIV+ women, and therefore more accurately estimate the maternal death count, we completed two additional analyses for GBD 2013. First, we determined the population attributable fraction (PAF) of HIV/AIDS to pregnancy-related death. Second, we determined the proportion of pregnancy-related deaths in HIV-positive persons that are aggravated by pregnancy and are therefore by definition maternal deaths.

$$PAF = \frac{p(RR - 1)}{1 + p(RR - 1)}$$

Where PAF is the population attributable fraction, p denotes the prevalence of HIV in pregnancy, and RR is relative risk of mortality in HIV+ vs HIV- pregnant females.

To recap our analysis for GBD 2013, we used the paper published by Calvert and Ronsmans to identify sources⁹ that could inform Step 1 of our HIV-correction analysis. We independently reviewed each of the component studies in Calvert and Ronsmans' review and extracted data directly, not from the systematic review paper. We only identified one additional study that was not used in Calvert and Ronsmans' analysis. We have, however, not used all the studies included in that review. Specific details are as follows: 1) Figueroa-Damian, et.al was excluded for not including any postpartum deaths at all. 2) In the case of Ryder, et al. and Zvandasara, et al. we excluded those deaths > 12 months after delivery. 3) We excluded the results from Chilongozi, et al. from the site that did not include any HIV-negative patients. 4) Leroy, et al. was not in the bibliography. We could not locate it for review so it was excluded. 5) Kourtis et al. was extracted with adjustment of the denominator based on the average number of hospitalizations per delivery in each group. 6) Ticconi, et al. was excluded for being both non-representative and including subgroup data from mothers with malaria infection. A total of 21 sources were included in our analysis of the increased mortality risk of HIV+ versus HIV- women in pregnancy. 9 We performed DerSimonian-Laird random effects meta-analysis to derive a pooled estimate of RR of death during pregnancy given HIV positivity. 10 The pooled effect size was 6.40 (95% uncertainty interval [UI] 3.98 - 10.29) which was then used to calculate an HIV PAF for each country, age group and year. In order to determine the proportion of those HIV-related deaths that were attributable to maternal causes, we performed a second systematic literature review. This time we sought evidence for the excess mortality risk of pregnancy in those women who are already HIV-positive. Most studies have failed to find such an effect, but most also did not stratify their study population by stage of HIV or antiretroviral therapy (ART) status. Only two studies did this stratification, with a pooled effect size of 1.13 (95% UI 0.73 - 1.77).

An updated literature review to inform the relative risk of mortality in pregnancy in HIV-positive versus HIV-negative women had 14 hits, but no usable sources. We completed this search on May 7, 2015, using the following two search strings:

```
(""HIV""[Mesh] OR ""Acquired Immunodeficiency Syndrome""[Mesh]) AND (""Pregnancy""[Mesh] OR
""Postpartum Period""[Mesh]) AND ""Mortality""[Mesh]
```

"HIV" (MeSH) AND ("pregnant" (Title/Abstract) OR "pregnancy" (Title/Abstract) OR "postpartum" (Title/Abstract) OR ""post partum" (Title/Abstract)) AND ("mortality" (Title/Abstract) OR "death" (Title/Abstract))"

Prevalence of HIV in pregnant women was calculated using UNAIDS' Spectrum model. Spectrum is a compartmental HIV progression model used to generate age-specific incidence, prevalence, and death rates from pre-calculated incidence curves and assumptions about intervention scale-up and local variation in epidemiology. For each location, we used UNAIDS' age-specific ratios of fertility in women

living with HIV to fertility in women not living with HIV. In most locations, this ratio is assumed to be greater than one in women aged 15-24 and less than one and decreasing as age increases beyond 24. Since Spectrum assumes fertile ages of 15-49, we used the ratio of HIV prevalence in pregnant women to HIV prevalence in the general population at either end of that range to extend estimates to age bands 10-14 and 50-54.

Unlike GBD 2013, when we applied the PAF correction to the envelope of maternal deaths predicted by CODEm, we instead applied country-year-age-group-specific PAF to maternal mortality input data prior to modeling in CODEm. This ensured that both the numerator and denominator of all cause fraction data were internally consistent in their exclusion of background HIV/AIDS mortality. The cause fractions for maternal deaths in sibling history, survey, and census data were therefore adjusted as follows:

$$CF_{l,t,a,x,mat_{adj}} = CF_{l,t,a,x,mat} * (1 - prop_{hiv_{l,t,a,x}})$$

$$prop_{hiv_{l,t,a,x}} = PAF_{l,t,a,x,hivpos} * (1 - \pi_{mat})$$

$$CF_{l,t,a,x,mat_{hiv}} = CF_{l,t,a,x,mat} * prop_{maternalhiv_{l,t,a,x}}$$

$$prop_{maternalhiv_{l,t,a,x}} = PAF_{l,t,a,x,hivpos} * \pi_{mat}$$

Where:

 $\pi_{mat} = .13/1.13$ = The proportion of HIV/AIDS deaths during pregnancy that were exacerbated by the pregnancy.

 $PAF_{l,t,a,x,hivpos}$ = The population-attributable fraction (PAF) that describes the percentage of all maternal deaths that were HIV-related for the location (I), year (t), age (a), and sex (x=Female)).

 $CF_{l,t,a,x,mat}$ = The proportion of deaths due to all maternal causes before HIV/AIDS correction for the location, year, age, and sex.

 $prop_{hiv_{l,t,a,x}}$ = The proportion of deaths in pregnancy for the location, year, age, and sex that are estimated to be incidental deaths due to HIV/AIDS, and therefore not a maternal cause of death.

 $prop_{maternalhiv_{l,t,a,x}}$ =The proportion of deaths in pregnancy for the location, year, age, and sex that are estimated to be HIV-positive and maternal deaths which are aggravated by HIV/AIDS.

 $CF_{l,t,a,x,mat_{adj}}$ = The proportion of deaths due to maternal causes after the adjustment for the location, year, age, and sex.

 $CF_{l,t,a,x,mat_{hiv}}$ = The proportion of deaths due to maternal deaths aggravated by HIV/AIDS after the adjustment for the location, year, age, and sex.

11.4 HIV/AIDS correction of other maternal mortality data

Although there are a specific subset of codes in ICD-10 that correspond to HIV/AIDS deaths aggravated by pregnancy, these codes are sparsely used and unreliable. We therefore adapted the method above to also correct VR and VA sources for the systematic exclusion of HIV-related maternal deaths. This correction was calculated in the same manner, using the same input data as above, with the only

difference that HIV correction of VR and VA sources resulted in a net increase in maternal CF. Maternal deaths aggravated by HIV/AIDS are calculated as the following:

$$CF_{l,t,a,x,mat_{hivvr}} = CF_{l,t,a,x,matvr} * prop_{maternalhiv_{l,t,a,x}}$$

$$prop_{maternalhiv_{l,t,a,x}} = \frac{PAF_{l,t,a,x,hivpos} * \pi_{mat}}{1 - PAF_{l,t,a,x,hivpos} * \pi_{mat}}$$

Where all symbols are the same as described above.

Step 12. Noise reduction

To deal with problems of zero counts in vital registration, verbal autopsy, cancer registries, or sibling histories for a given age group in a given year, we use a Bayesian noise reduction algorithm. For this algorithm, we assume a normal prior and a normal data likelihood. We estimate the normal prior for a given country series of data by estimating a negative binomial for the fraction of deaths in each age group due to each respective cause with dummy variables for age and year. With two notable exceptions (detailed below), these regressions are country-specific, so borrowing strength over age is only within a data type in a country. The variance of the prior, τ^2 , is estimated from the negative binomial regression, taking into account the variance-covariance matrix of the regression coefficients. For the data variance, we use the Wilson approximation which provides an estimate of σ^2 even in cases with a zero count of cause-specific deaths. The posterior estimate for each data point is:

$$Mean = \left(\frac{\tau^2}{\tau^2 + \sigma^2}X + \frac{\sigma^2}{\tau^2 + \sigma^2}\mu\right)$$

$$Variance = \left(\frac{\tau^2\sigma^2}{\tau^2 + \sigma^2}\right)$$

Where X is the mean of the data and μ is the mean of the prior. This approach to noise reduction avoids the problem that zero counts in an In rates model or a logit cause fraction model will be dropped from the regression and lead to upward bias in the estimates. This is particularly important in two settings: high-income countries with small numbers of cause-specific deaths, and in the analysis of sibling history data where for any given age group in any given year the number of deaths reported in the survey that are pregnancy-related or the number of deaths from all causes in that age group may be small.

Regarding the exceptions to the regression, the first is that country-years with populations under 1 million are pooled with the region data in order to prevent overdispersion and provide a stronger signal. Additionally, verbal autopsy data diverge from the above description in two ways. First, all data for a given super-region are pooled together and a study dummy variable is added, allowing for different studies and surveillance sites to borrow strength from one another within a super-region. Second, unless the data are part of a time series (e.g., Matlab), there is no year component to the regression.

Step 13. COD database and outlier identification

Death rates for different causes of death generally have a stable age pattern. In large populations, these patterns will not change very rapidly over time. We can assume a relatively stable pattern in death rates for all causes except for some epidemic diseases and specific types of injuries. Rare causes in large populations and prevalent causes in small populations usually have stochastic patterns. To correct for these stochastic patterns we implement a noise reduction process, explained in Step 12.

In vital registration data, we infrequently find one or more data points for specific geography/age/sex/years that lie very far from the stable pattern of death rates. In these situations, the model will usually ignore the data point(s). If the model fails to ignore these data, dramatic jumps or drops can occur in the death rates. When there is no logical explanation for variation in the death rates to this degree, we outlier the data point(s). The selection of data points to outlier occurs after data have been prepped for modeling, as well as during preliminary reviews of the models.

In non-vital-registration sources, data collection methods and data quality can vary widely from source to source. Where data points in each age-sex-geography-year are very sparse, extreme data points can have a bad effect on regional estimation. In these situations we investigate the study's methods and outlier lower-quality data points.

Identifying outliers in the cause of death data occurs prior to finalization of models for each cause. We do not automate the selection of outliers, but investigate the source of the offending data as well as reviewing other data sources for the same cause, geography, and year. Ultimately, outliers are identified based on the judgement of the modeler and senior faculty and are reversible to allow for decisions to be revisited in the future.

Modeling maternal mortality

2.1 Modeling Maternal disorders

CODEm models were informed by centrally prepped data stored in the CoD database. All data were corrected for incidental HIV deaths as described above. All data from all geographies were reviewed in CODEm models. Outliers were identified as those data where age patterns or temporal patterns were inconsistent with neighboring age groups or locations or where sparse data were predicting implausible overall temporal or age patterns for a given location. Overall maternal mortality was estimated with CODEm. Etiology-specific estimates were derived by multiplying the proportion outputs from DisMod-MR 2.1 by the total maternal deaths for that age-group, location, and year, which were scaled in relation to each other to equal one. HIV-related maternal deaths were estimated for all locations using the PAF approach described above for mortality data processing. Incidental HIV deaths during pregnancy were by definition excluded. DisMod-MR 2.1 is described in detail in Section 3.1.

2.2 CODFm

Overview of method

CODEm is a framework for modeling most cause-specific death rates in the GBD using five core principles: 1) Identify and use all the available data in the modeling process. Though data may vary in quality it all contains some signal of the true epidemiological process. 2) Develop a diverse set of plausible models to use for estimation. That is, build a number of models capturing well-documented associations to make estimates. 3) Assess the predictive validity of each plausible individual model and of an ensemble of models created from the pool of plausible models. 4) Choose the models and ensemble model with the best performance in the out-of-sample predictive validity tests.

For some causes, separate models were run for different age ranges when there was reason to believe that the relation between covariates and death rates might be different in different age ranges, for example, in children compared with adults. Separate models are developed for countries

with extensive, complete, and representative VR for every cause such that uncertainty can better reflect the more complete vital registration in these locations. A complete listing of countries with extensive, complete, and representative VR can be found in Appendix Table 3.

Model pool development

As many factors covary with a particular cause of death, a large range of plausible statistical models are developed for each cause. For the CODEm framework, four families of statistical models are developed using covariates (see 2x2 table in Foreman et al). These are mixed effects linear models of the natural log of the death rate, mixed effects linear models of the logit of the cause fraction, spatiotemporal Gaussian process regression (ST-GPR) models of the log of the death rate, and ST-GPR of the logit of the cause fraction. All plausible relationships between covariates and relevant cause are identified, and all possible permutations of selected covariates are tested in linear models where the logit cause fraction or log death rate is the response variable. Because we test all permutations of covariates, multicollinearity between covariates may produce implausible signs on coefficients or unstable coefficients. All models where the sign on the coefficient is in the direction expected based on the literature and where the coefficient is statistically significant at p <0.05 are retained. We run covariate selection for both cause fractions and death rates and then create both mixed effects only and ST models for each set of covariates. For a detailed explanation of the covariate selection algorithm see Foreman et al 2012.

Testing model pool on 15% sample

The performance of all component models and ensembles is evaluated using out-of-sample predictive validity tests. Thirty percent of the data are excluded from the initial model fits, and half of that (15% of total) is used to evaluate and rank component models and then build ensembles. Data are held out from the analysis using the pattern of missingness for each cause in the cause of death database. Out-of-sample predictive validity testing is repeated until stable model results have been obtained. The out-of-sample performance tests include the root mean squared error of the log of the cause-specific death rate, the direction of the trend in the prediction compared to the data, and the validity of the 95% UI. For every model, we show the in-sample root mean squared error (RMSE) of the log death rates and the out-of-sample performance in the 15% of data not used in the model building process.

Ensemble development

After component models are ranked on their out-of-sample predictive validity they are weighted based on their ranking and each component model contributes a portion to the final estimate. How much each submodel contributes is a function of its relative ranking as well as the value of psi chosen, which dictates that distribution of rankings (see Foreman et al 2012 for the details of psi distribution).¹

Testing ensembles

Using the second half of the holdout data (15% of total), the differently weighted ensembles and different values of psi are tested using the same predictive validity metrics as the component models. For every model, we show the in-sample RMSE of the log death rates and the out-of-sample performance in the 15% of data not used in the model building process. The ensemble with the best average trend and RMSE is chosen as the final ensemble weighting scheme.

Final estimation

After a model weighting scheme has been chosen, each model contributes a number of draws proportional to its weight such that 1,000 draws are created. The mean of the draws is used as the final estimate for the CODEm process and 95% UI are created from the 0.025 and 0.975 quantiles of the draws. The final assessment of ensemble model performance is the validity of the UIs; ideally, the 95% UI for a model would capture 95% of the data out-of-sample. Higher coverage suggests that UIs are too large and lower than 95% suggest UIs are too narrow.

3.1 Estimation of etiology and timing of maternal mortality DisMod-MR 2.1

Until GBD 2010, non-fatal estimates were based on a single data source on prevalence, incidence, remission, or a mortality risk selected by the researcher as most relevant to a particular geography and time. For GBD 2010, we set a more ambitious goal: to evaluate all available information on a disease that passes a minimum quality standard. That required a different analytical tool that would be able to pool disparate information presented in varying age groupings and from data sources using different methods. The DisMod-MR 1.0 tool used in GBD 2010 evaluated and pooled all available data, adjusted data for systematic bias associated with methods that varied from the reference and produced estimates by world regions with uncertainty intervals. For GBD 2013, the improved DisMod-MR 2.0 had increased computational speed allowing computations that were consistent between all disease parameters at the country rather than region level. The hundred-fold increase in speed of DisMod-MR 2.0 was partly due to a more efficient re-write of the code in C++ but also by changing to a model specification using log rates rather than a negative binomial model used in DisMod-MR 1.0. In cross-validation tests, the log rates specification worked as well or better than the negative binomial specification¹². For GBD 2015, the computational engine (DisMod-MR 2.1) remained substantively unchanged but we re-wrote the 'wrapper' code that organized the flow of data and settings at each level of the analytical cascade. The sequence of estimation occurred at five levels: global, super-region, region, country, and, where applicable, subnational geographical units (see flow diagram of DisMod-MR 2.1 cascade, below). The super-region priors were generated at the global level with mixed-effects, non-linear regression using all available data; the super-region fit, in turn, informed the region fit, and so on down the cascade. The wrapper gave analysts the choice to branch the cascade in terms of time and sex at different levels depending on data density. The default used in most models was to branch by sex after the global fit but to retain all years of data until the lowest level in the cascade. For GBD 2015, we generated fits for the years 1990, 1995, 2000, 2005, 2010, and 2015.

In updating the 'wrapper,' we consolidated the code base into a single language, Python, to make the code more transparent and efficient and to better deal with subnational estimation. The computational engine is limited to three levels of random effects; we differentiated estimates at the super-region, region, and country level. In GBD 2013, the subnational units of China, Mexico, and the United Kingdom (UK) were treated as 'countries' such that a random effect was estimated for every geography with contributing data. However, the lack of a hierarchy between country and subnational units meant that the fit to country data contributed as much to the estimation of a subnational unit as the fits for all other countries in the region. We found inconsistency between the country fit and the aggregation of subnational estimates when the country's epidemiology varied from the average of the region. Adding an additional level of random effects required a prohibitively comprehensive rewrite of the underlying DisMod-MR engine. Instead, we added a fifth layer to the cascade, with subnational estimation informed

by the country fit and country covariates, plus an adjustment based on the average of the residuals between the subnational unit's available data and its prior. This mimicked the impact of a random effect on estimates between subnationals.

For GBD 2015 we improved how country covariates differentiate non-fatal estimates for diseases with sparse data. The coefficients for country covariates were re-estimated at each level of the cascade. For a given geography, country coefficients were calculated using both data and prior information available for that geography. In the absence of data, the coefficient of its parent geography was used, in order to utilize the predictive power of our covariates in data sparse situations.

Modeling age-specific fertility and live births

Data

For locations where the United Nations Population Division provides age-specific fertility rate (ASFR) for age groups 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49 in their most recent update to the World Population Prospect (WPP), we start with their estimates for every five year time period (e.g. 1990-1995). We treat the given value as that of the midpoint year, so in the case of 1990-1995, we use the value for 1992. We then linearly interpolate in log space to generate values for the intervening years.

ASFR for locations not covered by the UNPOP

For locations not covered by UNPOP, including any subnational locations as well as countries such as Andorra, American Samoa, Bermuda, Northern Mariana Islands, and the United States (US) Virgin Islands, we took one of two approaches. If we could find relatively complete data for 1970's onwards, we would use those estimates. To address the small number of missing values in these datasets, we used a combination of linear mixed effects regression, simple linear interpolation, and 3-year rate of change extrapolation depending on the nature of values that were missing.

Linear mixed effects regression with age as categorical variable was applied to data when entire age groups were missing for a given location. Linear interpolation was applied to locations when missing ASFR values fell between years where ASFR was available. In locations where ASFR was missing for years where values did not fall between years where ASFR was available, but ASFR was present in years preceding or directly after the missing year, ASFR was calculated using annualized rate of change. Missing ASFR was interpolated based on the rate of change of ASFR of the 3 years preceding or following the missing year.

Secondly, in cases where there was little data or it did not cover most of the time period, we modeled ASFR using a database of fertility tables from the Human Fertility Database and from location-level surveys in the locations we were modeling. This process was as follows:

1. Calculating empirical weights: Using the database of tables, we created all possible pairs of tables. For each age category, we then calculated the difference between the two tables. These differences were then summed, producing a total difference for each pair of tables. We then created a series of indicator variables for each pair, indicating whether or not they were from the same country, region, or superregion, and how many years apart they were. We then average the difference for each category. So for example, we produced the mean difference for locations in the same super-region but not the same region or country that were 2 years apart in time. We then took the reciprocals of these differences to produce a weight, indicating how "close" a table is to another given their similarities in location and time.

2. Fit model relating difference in TFR to difference in ASFR: We again create all possible pairs of tables as in step 1. For each pair, we randomly select one table to be the predictor table. Then we fit the following model for each age group in 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, and 45-49:

$$ASFR_1 - ASFR_2 = \beta_1 (TFR_1 - TFR_2) + \beta_0$$

where table 1 is the table randomly designated as the predictor table.

3. Produce a standard tables and predict: Using the weights produced in the first step, we create a standard table from a selection of tables from the database. For each table, weights are calculated for all the other tables based on how far they are from the table in terms of year, and whether they are from the same country, region, or super-region. We then order by weight and take the first 300 tables. We then create an average table, weighted by weights calculated in step 1. This produces a standard table for each location-year. Due to the limited number of tables for many location, this can produce discontinuities from year to year. To prevent this, we applied a rolling mean over time to the standard, resulting in estimates that are relatively smooth over time. We then use this standard to predict the ASFR for each age group using the models produced in step 2 and the TFR for the country-year of interest:

$$ASFR_1 = \beta_1(TFR_1 - TFR_{standard}) + \beta_0 + ASFR_{standard}$$

where TFR₁ is the TFR in the location year where we are predicting ASFR.

Getting single year ASFR

Once we have five year ASFR values, we calculate single year ASFR using a spline and treating the ASFR values as midpoints for each age group. Though we do not use single-year ASFR for maternal calculations, they are used in other parts of the GBD, and so are incorporated in this process.

For high and low ages, we set fertility for 9 and below and 55 and above to 0, then used those in the interpolation. Because many sources do not have ages 10-14 and 50-54, which are necessary for our maternal estimates, we also extend our estimates to include these age groups. To do this, we created a linear interpolation between the value at age 15 and 0 at age 9 on the young side, and between the value at 50 and 0 at age 55. To these values, we then applied percentages of women who have gone through menarche^{29–31} or have not gone through menopause^{32–35}, respectively. These values are given in the following tables:

age	Pre-menopause (%)	age	Post-menarche (%)
50	36	10	4
51	28	11	14
52	20	12	40
53	14	13	77
54	9	14	98

Because of the steep climb in fertility in the teen years, we made sure that our estimates in 10-14 were in-line with what we would expect by scaling them to the 15-19 age category. Using the mean of the ratios between 10-14 and 15-19 from the Indian DHS, the US census, and the Democratic Republic of the

Congo DHS, we scaled the 10, 11, 12, 13, and 14 ages so that their mean has this ratio with the mean of 15-19.

Scaling to births

To get our final ASFR estimates, we scale ASFR so that the total implied births from our ASFR estimates and the GBD populations is the same as the GBD births. GBD births are generally derived from the WPP 2015 Revision, and WHO, though for some locations we use location-specific sources. This scaling ensures consistency between our fertility results and the populations that are used in other parts of the GBD process. The exception to this is South Africa. There we used subnational estimates from UNAIDS, calculated live births implied by these, then used their sum for the South Africa national ASFR estimates. These values were then substituted into the GBD births.

To re-calculate five year age groups, we calculate the number of births in each five year age group and divide by the population in that age group. These are the final ASFR estimates used in our maternal mortality calculations.

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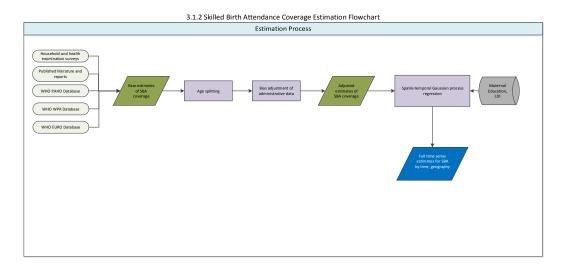
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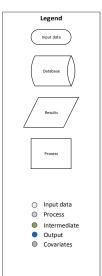
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Skilled Birth Attendance (SBA) Capstone Appendix

Flowchart





Input data & Methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with skilled birth attendance (3.1.2).

Indicator 3.1.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.1, by 2030, reduce the global maternal mortality ratio to less than 70 per 100,000 live births, is measured using SDG Indicator 3.1.2, the proportion of births attended by skilled health personnel (doctors, nurses, and midwives). This indicator is also a component of the UHC tracer indicator.

Input data

For the present analysis, we used individual-level microdata from population health surveys and tabulated survey report data on skilled birth attendance (SBA). As defined by the World Health Organization (WHO), SBA reflects the proportion of births in a given year where a doctor, nurse, or midwife was present.¹

Survey data which provided individual-level data, and specifically among female respondents, were identified and extracted. Major multi-country survey programs included in the analysis include the Demographic and Health Surveys (DHS),² Multiple Indicator Cluster Surveys (MICS),³ Reproductive Health Surveys (RHS),⁴ Living Standards Measurement Study (LSMS) surveys,⁵ and World Health Surveys (WHS).⁶ We also conducted a comprehensive search of the Global Health Data Exchange (GHDx),⁷as well as targeted internet searches and review of Ministry of Health websites, to identify national surveys and

other multi-country survey programs. In addition, we utilized tabulated report data from regional WHO databases, when available, including the PAHO, WHO WPR, and the WHO European Health for All databases.

We excluded all data sources that were not nationally representative or had high levels of missingness. We applied survey weights based on survey sampling frames whenever they were available to generate weighted national estimates of SBA coverage accompanied by estimates of standard error (SE). Estimates of SE, as well as sample sizes, were used to calculate uncertainty, as described below. Any point estimates with sample sizes less than 50 were reviewed to ensure that were not substantive outliers and would otherwise have an undue influence on our analysis.

Due to potential bias in recall, we limited our analysis to women who gave birth up to five years prior to the time of survey; due to data limitations, we used a limit of up to two years for some surveys. We also had to standardize the definition of "skilled health professional" across countries, which varied by differences in quality of training or health professional roles. For this analysis, doctors, nurses, and midwives were included as our foundational definition for SBA, and we extended this to include country-specific medical staff based on the number of years of training they received and/or their comparable ability to intervene in an emergency situation (eg, clinical officers). Care received during delivery by traditional health personnel was not considered a birth overseen by a skilled attendant.

Modeling strategy

Data processing

Age splitting

Most household surveys collection information on maternal and child health (MCH) indicators for children under 5 and/or mothers who gave birth within five years prior to the time of survey. To maximize data use for our model, we included SBA information for children aged 12 to 59 at the time of survey. Children younger than 12 months of age were excluded to minimize the influence of potentially censored observations. SBA coverage estimates were assigned to birth-cohort years based on a child's age prior to the time of survey: we used responses recorded for children aged 12 to 23 months for SBA coverage for one year prior to the time of survey, children aged 24 to 35 months for coverage two years prior to the time of survey, and so forth.

Age-specific estimates are easily computed from individual-level microdata, but many published reports and survey summaries present data in broader age aggregates (eg, SBA coverage for children aged 12 to 35 months). To standardize these age groups, we applied an age-splitting model used in the GBD study,⁹ as well as analyses that generated smoking and obesity prevalence by age group.^{10,11}

Using surveys with microdata as the reference, we used the following model to generate standardized age group-specific estimates for SBA:

$$\tilde{P}_{a,c,t,k} = P_{a,c,t,k}^{a+x} \frac{P_{a,c,t,j}}{P_{a,c,t,j}^{a+x}}$$

where $\tilde{P}_{a,c,k}$ is the adjusted estimate of coverage for target age group a in country c and year t of survey k; and $P_{a,c,k}^{a+x}$ is coverage reported from survey k, for country c in year t for the age group spanning age a to age (a+x). The ratio of coverage between the target age group and broader age group from a survey j with microdata from the same country-year was used to split data from survey k. Surveys to be split were ideally matched with DHS or MICS surveys. If microdata were not available for the same year, ratios within five years of the survey that required age-splitting were applied.

Bias adjustments

Intervention coverage estimates based on administrative sources can be biased, yet the direction and magnitude of such biases are not universal. Some studies show that coverage estimates from administrative data source are systematically higher than those of survey-based estimates, ¹² while other studies show that bias directionality is more heterogeneous. ¹³ Such biases may arise for a number of reasons, including discrepancies in the accurate reporting of services or interventions provided (eg, number of skilled attendants) and target population (eg, number of children born), as well as capturing these data in a timely manner from both public and private sector facilities and healthcare providers.

For SBA, we view individual-level data collected through population health surveys as the most accurate and least biased source of information, particularly for geographies with incomplete health information systems. We thus used SBA coverage estimates from household surveys to calculate country-specific adjustment factors:

$$logit\left(P_{s,c,t}\right) = \beta_0 + \beta_1 logit\left(\tilde{P}_{a,c,t}\right) + \sum_{k=2}^{2+B} \beta_k S_k + \varepsilon_{c,t}$$

where $P_{s,\,c,t}$ is the survey-based estimate for SBA coverage (s) in country c for year t; $\tilde{P}_{a,\,c,t}$ is the administrative estimate for coverage in country c in year t; S_k is a spline basis used to capture the secular trend in coverage; β_1 is the estimated adjustment factor used to correct for the administrative bias; and ε is the error term for country c in year t.

To quantify uncertainty for bias-adjusted estimates from the mixed-effects models described above, we calculated prediction error, \widehat{PE} , as follows:

$$\widehat{PE} = X^2 var(\hat{\beta})$$

where $var(\hat{\beta})$ is the variance for the estimated fixed-effects coefficient of the adjustment factor and X is the independent variable. Proper estimation of prediction errors is crucial as the data synthesis procedure, Gaussian process regression (GPR) (as described in the subsequent section), accounts for uncertainty from point estimates and bias adjustments when generating fitted values. More weight is given to data with less uncertainty. Prediction errors estimated from the bias adjustment were incorporated into the data variance and propagated through the GPR step to obtain estimates of SBA coverage and uncertainty intervals (UIs).

Trend estimation

We used a spatiotemporal Gaussian process regression (ST-GPR) to synthesize point estimates from multiple data sources and derive a complete time series for SBA coverage. This method has been used extensively in GBD and related studies, and accounts for uncertainty pertaining to each point estimate while borrowing strength across geographic space and time. ^{10, 11,15,16} Briefly, we assumed the Gaussian process was defined by a mean function $m(\bullet)$ and covariance function $Cov(\bullet)$.

We estimated the mean function using a two-step approach. Specifically, $m_c(t)$ can be expressed as:

$$m_c(t) = X\beta + h(r_{c,t})$$

where $X\beta$ is a linear model and $h(r_{c,t})$ is a smoothing function for the residuals; and $r_{c,t}$ is derived from the linear model. The following linear model was used for estimating SBA:

$$logit(P_{c,t}) = \beta_0 + \beta_1 medu_{c,t} + \beta_1 LDI_{c,t} + \alpha_c + \gamma_{R[c]} + \delta_c medu + \theta_{R[c]} medu + \varepsilon_{c,t}$$

where $P_{c,t}$ is SBA coverage for country c year t; $medu_{c,t}$ is the average years of education for women of reproductive age in country c and year t; $LDI_{c,t}$ is the lag-distributed income (LDI) in country c and year t; α_c and $\gamma_{R[c]}$ are country and region random intercepts, respectively. δ_c and $\theta_{R[c]}$ are country and region specific slope on education. These estimates were then run through ST-GPR, as documented elsewhere. ¹⁰

Random draws of 1,000 samples were obtained from the distributions above for every country for a given vaccine. Ninety-five percent uncertainty intervals were calculated by taking the ordinal 25 and 975th draws from the sample distribution.

To assess the accuracy of our estimates in each bias adjustment step and in the modeling process, we performed cross-validation analyses by randomly holding out 20% of the sample and, if available, the corresponding administrative estimates for the given indicator of the same country and year, 10 separate times. We computed the average root mean squared errors (RMSE) across each country. Error in the bias adjustments was calculated as the mean difference between the adjusted administrative estimate for a given country, year, and corresponding survey-level estimates (which were considered the "gold-standard"); error in the modeling process was calculated as the difference between the modeled estimates and the sample data.

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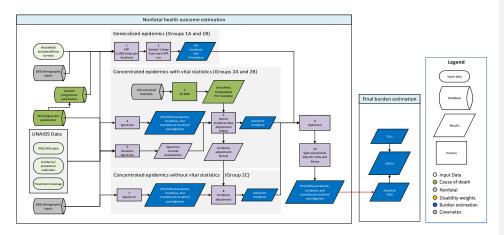
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HIV/AIDS SDG Capstone Appendix

Flowchart

HIV/AIDS



Input data & Methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with HIV incidence (3.3.1).

Indicator 3.3.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.3, by 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases, is measured using SDG Indicator 3.3.1, number of new HIV infections per 1,000.

Case definition

Infection with the human immunodeficiency virus (HIV) causes influenza-like symptoms during the acute period following infection and can lead to acquired immunodeficiency syndrome (AIDS) if untreated. HIV attacks the immune system of its host, leaving infected individuals more susceptible to opportunistic infections like tuberculosis. Although there are two different subtypes of HIV, HIV-1 and HIV-2, no distinction is made in our estimation process or presentation of results. For HIV, ICD 10 codes are B20-B24, C46-C469, D84.9; ICD 9 codes are 042-044, 112-118 (after 1980), 130 (after 1980), 136.3-136.8 (after 1980), 176.0-176.9 (after 1980), 279 (after 1980); and ICD9 BTL codes are B184-B185.

Input data

Model inputs

Household seroprevalence surveys

Geographically representative HIV seroprevalence survey results were used as inputs to the model for countries with generalized HIV epidemics where available.

GBD demographic inputs

Location-specific population, fertility, and HIV-free survival rates from GBD 2015 (see Part 1 for details on the generation of these data) and migration data from UNAIDS were used as inputs in modeling all locations.

UNAIDS data

Antenatal care, incidence, prevalence, and treatment coverage data from UNAIDS were used in modeling for all locations.

On-ART literature data

Data were identified by using search terms "HIV," "mortality," and "antiretroviral therapy" in PubMed searches across the literature. To be included, studies must include only HIV-positive people who receive antiretroviral therapy (ART) but who were ART-naïve prior to the study. In addition, studies must report either a duration-specific mortality proportion or a hazard ratio across age or sex, and must not include children.

For duration-specific survival data, studies must report uncertainty on mortality estimates or provide stratum-specific sample sizes and must include duration-specific data to allow for calculation of 0-6, 7-12, or 13-24 month conditional mortality. In addition, studies must either report separate mortality and loss-to-follow-up (LTFU) curves, be corrected for LTFU using vital registration (VR) data, or be conducted in a high-income setting. Finally, studies must report the percent of participants who are male, the median age of participants, and either specific data on the number of CD4 T lymphocytes (CD4 counts) or the median CD4 count used for the data.

Hazard ratio data for ages or sexes can only be used if the hazard ratios are controlled for other variables of interest (age, sex, and CD4 category).

Changes for GBD 2015

In GBD 2013, we identified 102 papers for extraction. For GBD 2015, we included 13 additional studies informing the duration-specific mortality estimation process and 26 studies informing the age and sex hazard ratio estimation process (some studies were used and counted in both). We also added one study to our LTFU analysis. In addition, we updated our data from the Antiretroviral Therapy Cohort Collaboration (ART-CC) with country-specific data pre- and post-2001 for enhanced use in estimating time trends for high-income countries. We excluded nine hazard ratio and four duration-specific mortality studies used in GBD 2013 which reported results on populations already present in other extracted studies. The inclusion of new ART-CC data necessitated the exclusion of four additional studies used in GBD 2013.

We also included on-ART cohort mortality data from 10 high-income nations with collaboration from ART-CC. These countries include Austria, Denmark, France, Germany, Italy, the Netherlands, Spain, Switzerland, the United Kingdom, and the United States. We excluded the US data because they were not fully representative of the complete with-HIV on-ART population at the time.

Off-ART literature data

In GBD 2013, to characterize uncertainty in the progression and death rates, we systematically reviewed the literature on mortality without ART. We searched terms related to pre-ART or ART-naive survival since seroconversion. After screening, we identified 13 cohort studies that included the cohorts used by UNAIDS from which we extracted survival at each one-year point after infection. Screening for additional, recently published studies for GBD 2015 identified no new cohort studies for inclusion in this analysis.

Severity splits & disability weights

The basis of the GBD disability weight survey assessments are lay descriptions of sequelae highlighting major functional consequences and symptoms. The lay descriptions and disability weights (DWs) for HIV/AIDS severity levels are shown below.

Severity level	Lay description	DW (95% CI)
Symptomatic HIV	has weight loss, fatigue, and	0.274
	frequent infections.	(0.184-0.377)
AIDS with antiretroviral	has occasional fevers and	0.078
treatment	infections. The person takes	(0.052-0.111)
	daily medication that sometimes	
	causes diarrhea.	
AIDS without antiretroviral	has severe weight loss,	0.582
treatment	weakness, fatigue, cough and	(0.406-0.743)
	fever, and frequent infections,	
	skin rashes, and diarrhea.	

The proportion of people living with HIV/AIDS who are being treated with ART is an output of Spectrum, the compartmental model used to make consistent incidence, prevalence, and mortality estimates described below.

Modeling strategy

In GBD 2015, our general modeling strategy for estimating HIV incidence, prevalence, and mortality is similar in many ways to the strategy used in GBD 2013. In GBD 2015, we continue to use the Spectrum program rewritten in Python for GBD 2013 to facilitate faster and more flexible execution necessary for our more intensive computational needs. We made several changes to Spectrum's assumptions comparing to the Spectrum software used by UNAIDS. A key change in GBD 2015 is the application of Estimation and Projection Package (EPP) using an open-source computer program in R written by Jeffrey Eaton. We ran EPP for all group 1 countries in order to produce incidence curves that were consistent with the demographic and epidemiological assumptions used in GBD 2015. This differed from GBD 2013, where we used the incidence curves provided by UNAIDS.

On-ART

First, we corrected reported probabilities of death for LTFU using an update of the approach developed by Verguet and colleagues. Serguet and colleagues used tracing and follow-up studies to empirically estimate the relationship between death in LTFU and the rate of LTFU.

After extracting the survival data into duration-specific conditional mortality, we used DisMod-MR 2.0 to synthesize the data into estimates of conditional probability of death over initial CD4 count. We modeled the data separately by duration and added a fixed effect on whether the study was conducted prior to 2002. Each analysis was conducted separately for high-income countries, GBD low-income countries outside of sub-Saharan Africa, and sub-Saharan Africa.

To create estimates of age-specific hazard ratios, we synthesized hazard ratio data in five broad age groups: 15-25, 25-35, 35-45, 45-55, and 55-100, and modeled the data using DisMod-MR 2.0.

To create estimates of sex-specific hazard ratios, we use the *metan* function in Stata to create estimates of relative risks separately by region, using female age groups as the reference group.

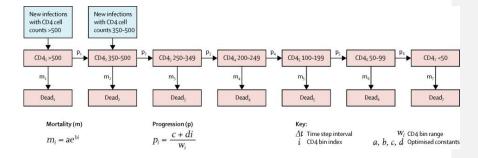
The age and sex hazard ratios were applied to the CD4-specific mortality rates, accounting for the distribution of ages and sexes in the mortality data. We then subtracted HIV-free mortality from the model life table process to calculate HIV-specific mortality, and used 1,000 draws from the posterior distribution for each age, sex, and CD4 category for conditional probabilities of death for 0-6 months, 7-12 months, and 13-24 months after initiation of ART as inputs into Spectrum.

Changes for GBD 2015

In GBD 2015, our primary methodological change was the analysis of on-ART mortality using a fixed effect on studies before/after 2002, only in the high-income region, to estimate conditional probability of death in DisMod-MR 2.0. By doing so, we incorporated changes over time in the quality of on-ART care, which may improve on-ART mortality. This change was also complemented by the inclusion of time-split data from ART-CC, which allowed us to incorporate this time trend across the large cohort. We then used the estimated post-2002 on-ART mortality through the rest of the on-ART estimation process.

Off-ART

Following UNAIDS assumptions, no-ART mortality is modeled as shown in the figure below.¹



The death and progression rates between CD4 categories vary by age according to four age groups: 15–24 years, 25–34 years, 35–44 years, and 45 years or older. We modeled the logit of the conditional probability of death between years in these studies using the following formula:

logit
$$(m_{ijk}) = \beta_0 + \sum_{i=1}^{4} \beta_{1i} \alpha_i + \sum_{j=1}^{12} \beta_{2j} t_j + u_k + \varepsilon_{ijk}$$

In the formula, m is conditional probability of death from year t_j to t_{j+1} , a_i is an indicator variable for age group at seroconversion (15–24 years, 25–34 years, 35–44 years, and 45 years or older), t_j is an indicator variable of year since seroconversion, and u_k is a study-level random effect.

By sampling the variance-covariance matrix of the regression coefficients and the study-level random effect, we generated 1,000 survival curves for each age group that capture the systematic variation in survival across the available studies. For each of the 1,000 survival curves, we used a framework modeled after the UNAIDS optimization framework in which we find a set of progression and death rates that minimizes the sum of the squared errors for the fit to the survival curve. ^{6,7}

Burden estimation overview

UNAIDS uses two key analytical components in their epidemiological estimation. EPP is used to estimate incidence trajectories that are consistent with prevalence surveys and other prevalence measurements such as antenatal clinic serosurveillance. Spectrum is a compartmental HIV progression model used to generate age-specific incidence, prevalence, and death rates from the EPP incidence curves and assumptions about intervention scale-up and local variation in epidemiology.

For GBD 2013, we created an exact replica of Spectrum in Python. This enabled us to run thousands of iterations of the model at once on our computing cluster and allowed for more flexible input data structures. Additionally, in order to generate estimates with more realistic ranges of uncertainty than those in UNAIDS 2012, we adjusted all input data by uniformly sampled factors between 0.9 and 1.1. These changes, along with our new estimation of with- and without-ART mortality and CD4 progression parameters, persist into GBD 2015.

We have made several substantial improvements elsewhere in the process for GBD 2015. Of particular note, we have integrated EPP into the modeling process when feasible, enabling more robust and internally consistent incorporation of parameter uncertainty in generalized epidemics, and we have vastly improved the accuracy of the incidence adjustment used to fit Spectrum to high-quality VR data. Details of the impacts are included in the descriptions of the appropriate country strategies.

Due to the substantial differences in the quality and types of data available across different countries, we used three different methodologies to produce year-, age-, and sex-specific estimates of HIV incidence, prevalence, and mortality.

Countries with seroprevalence surveys and antenatal clinic data (Groups 1A and 1B) We identified 43 countries – as well as 48 subnational locations from India, Kenya, Mozambique, and South Africa – with at least one geographically representative HIV seroprevalence survey. In order to ensure that our estimates of incidence and prevalence in these places were consistent with our estimates of HIV progression, we used a version of EPP written in R and C++ by Jeffrey Eaton to create new fits to the prevalence data in the UNAIDS files. By substituting in our own assumptions about HIV progression, we were able to ensure that the implied relationship between incidence and mortality/prevalence in EPP is similar to that in Spectrum.

In these locations, most of which experience generalized HIV epidemics, we expect estimates of HIV burden to exhibit substantial uncertainty. To reflect this, we induced a perfect correlation between the previously independent draws of HIV mortality with and without ART and CD4 progression. We paired the draws of the three parameter sets internally and with each other in the following way: we sorted without-ART mortality and CD4 progression internally by age (not CD4), meaning the highest draw of HIV mortality without ART for age a_i and CD4 category c_i will be paired with the highest draw of HIV mortality without ART for age a_k and CD4 category c_i . In the same way, we sorted with-ART mortality internally by age, sex, CD4 count at treatment initiation, and duration on treatment. After this sorting process, the lowest indexed draw of each parameter has the highest values and vice versa. This means that we will use the most extreme possible parameter sets in EPP and Spectrum and should see a commensurate expansion in the range of the uncertainty.

To ensure that this expanded uncertainty is replicated in EPP, we fit the model once for every set of paired draws of the progression parameters for every location. This means that the first iteration of EPP for Uganda sees the highest draws of all three sets of progression parameters. Such a procedure is necessary because EPP currently has no mechanism for incorporating uncertainty in any inputs except prevalence data. This process (Process 1 in the HIV/AIDS Estimation Flowchart), produced 1,000 sets of EPP output for each of the locations that make up the 47 countries in the group. Every set of EPP outputs contains 500 consistent draws of HIV incidence and prevalence in adults aged 15-49. In many cases, the algorithm used to fit EPP, incremental mixture importance sampling, failed, resulting in fewer than 1,000 sets of EPP results.

For every location in the group, we sampled one of the 500 incidence/prevalence draws from each of the sets of EPP results (Process 2 in the HIV/AIDS Estimation Flowchart). By sampling one draw from each set, we ensured that the distribution of progression parameters dictating the relationship between incidence and prevalence was exactly the same as the distribution of the sorted parameters generated in the previous step. In locations where not all 1,000 iterations of EPP fit successfully, we sampled one draw from every iteration that did succeed and then resampled with replacement from that set of draws. To maintain the link between the input progression draws and the resulting incidence and prevalence draws from EPP, we replaced any parameter draw associated with a failed run of EPP with the parameter draw that that failed draw was replaced with. At the end of this process, for every location in the set of 47 countries, we were left with 1,000 linked draws of adult incidence and prevalence and the exact progression parameters that generated those draws.

We then ran these results, along with the previously described demographic and HIV-specific inputs, through Spectrum to produce location-, year-, age-, and sex-specific estimates of HIV incidence, prevalence, and mortality (Process 9 in the HIV/AIDS Estimation Flowchart).

Countries with vital registration data (Group 2A and 2B)

VR is one of the highest-quality sources of data on HIV burden in many countries, so generating estimates that are consistent with these data, with necessary adjustment to account for any potential underreporting, is critical. We identified 116 countries – as well as 208 subnational locations from Brazil, China, Japan, Mexico, Saudi Arabia, Sweden, the United Kingdom, and the United States – with VR or sample registration systems (SRS) such as the Disease Surveillance Points (DSP) in China.

We imputed missing years of data to generate a complete time series for HIV from the estimated start year of the epidemic using spatiotemporal Gaussian process regression (ST-GPR). We analyzed mortality trends using ST-GPR starting in 1981, the year that HIV was first identified in the United States. For ST-GPR, we adjusted the lambda (time weight) and GPR scale according to the completeness of vital registration data, based on whether a country had 10 or more years of complete VR data as analyzed by the Death Distribution Methods (DDM) model described in Part 1. We produced separate splines by country/age group, up to the peak year of death rate. We then ran a linear regression with random effects on region, age, and sex. Following this, we ran space-time residual smoothing, in which time, age, and space weights are used to inform smoothing of the residuals between data points and the linear regression estimate. From this process, we generated space-time estimates with the applied weights, along with the median absolute deviation (MAD) of the space-time estimates from the data. The MAD was calculated at various levels of the geographic hierarchy (e.g., subnational and national), and was added into the data variance term. The data variance and space-time estimates were then analyzed using GPR to return a final estimate of mortality along with uncertainty.

Although Spectrum produces HIV mortality estimates that are within the realm of possibility in most countries using the incidence curves provided in the UNAIDS 2012/2015 country files, it is a deterministic model that has not yet been integrated into an optimizable framework. Therefore, in order to "fit" it to VR data, we need to adjust input incidence. For GBD 2013, we used a process that assumed several different durations between HIV infection and HIV death and adjusted incidence based on death some number of years in the future. Although that method worked relatively well and substantially reduced the disconnect between Spectrum and the VR data, it required very rigid and unrealistic assumptions about these survival durations. For GBD 2015, we have improved the performance of this method, allowing Spectrum to fit to the VR data more closely.

To improve the fit of this process, we restructured Spectrum to add compartments that identify groups of people living with HIV by year of infection (Process 5 in the HIV/AIDS Estimation Flowchart). With this version of Spectrum we can output, among many other metrics, HIV deaths by year, age, sex, and infection cohort. This enables us to adjust incidence to fit to death much more precisely and without making any rigid assumptions about the time from HIV infection to HIV death.

We have incorporated these improvements into a cohort incidence bias adjustment (CIBA) process. First, we ran Spectrum normally to produce 1,000 draws of incidence, prevalence and mortality (Process 4 in the HIV/AIDS Estimation Flowchart). Then, by year, age, and sex, we took the ratio of VR deaths to Spectrum deaths to quantify the amount of bias in Spectrum. Using the mean duration data from the new version of Spectrum, for every year-, age-, and sex-specific infection cohort, we calculated the share of all HIV deaths observed over the course of the projection period in that cohort that would occur in each year after the year of infection. For example, projecting from 1970 through 2015, we identified the cohort of men infected in 1992 at the age of 16, calculated the total number of HIV deaths in that cohort in all subsequent years through the end of 2015, and divided the annual number of deaths by that total. This showed us the distribution of deaths among that cohort over the projection period. In the most extreme case (infections in 2014), we could only produce one point of that distribution (2015), so that single value is exactly 1.0; 100% of the deaths observed in that cohort occurred in 2015.

We then used these distributions of death to weigh the ratio of VR deaths to Spectrum deaths, meaning that ratios in the years where we expect the largest share of deaths were weighed most heavily. We then

multiplied the initial size of that cohort from the normal run of Spectrum by the sum of the combined ratios to get a new estimate of new cases in that year/age/sex combination.

We can write this method mathematically in the following way:

$$\begin{split} r_t &= \frac{VR_t}{D_t} \\ \rho_t^{t-i} &= \frac{d_t^{t-i}}{\sum_{k=t-i+1}^n d_k^{t-i}} \\ \alpha^{t-i} &= \sum_{k=t-i+1}^n r_k * \rho_t^{t-i} \\ n_{\text{adjusted}}^{t-i} &= \alpha^{t-i} * n^{t-i} \end{split}$$

 VR_t is the number of HIV/AIDS deaths in year t from ST-GPR, and D_t is the number of HIV/AIDS deaths from the first run of Spectrum. In the second equation, d_t^{t-i} is the number of HIV/AIDS deaths among members of infection cohort t-i in year t, with $i \geq 1$, from the new, duration-tracking version of Spectrum, and n is final year of the projection. Therefore, ρ_t^{t-i} is the share of observed deaths in cohort t-i that we expect to occur in year t. It follows that α^{t-i} is the weighted adjustment ratio described above, which we multiply by the estimated initial size of infection cohort t-i as calculated in the first-stage Spectrum run to get the adjusted number of new cases, $n_{\mathrm{adjusted}}^{t-i}$. This process is run separately for every sex and single-age pair.

CIBA (Process 6 in the HIV/AIDS Estimation Flowchart) allows ratios in each year after a given infection year to influence the final adjustment to incidence. The size of that influence is determined by the relative importance of that year in the cohort-year's distribution of deaths over time. The result is a new set of 1,000 draws of incidence and a set of 1,000 ratios of post-adjustment incidence to pre-adjustment incidence. We perform this adjustment using mean durations from the new version of Spectrum in order to try to shift the mean of the regular distribution of deaths.

Finally, to produce location-, year-, age-, and sex-specific estimates of HIV incidence, prevalence, and mortality, we ran the new estimates of incidence and all previously input data through Spectrum (Process 9 in the HIV/AIDS Estimation Flowchart).

Countries without survey data and vital registration data (Group 2C)

The remaining 24 countries – as well as nine subnational locations from China and Saudi Arabia – had neither geographically representative seroprevalence surveys nor reliable VR systems. To produce estimates of HIV burden in these countries, we assumed that Spectrum is similarly biased as in other Group 2 countries. This involved running Spectrum (Process 7 in the HIV/AIDS Estimation Flowchart), adjusting incidence using 1,000 adjustment ratios randomly sampled from the entire set of CIBA results (Process 8), and rerunning Spectrum using the new draws of adjusted incidence (Process 9). As above, the estimates of incidence, prevalence, and mortality were incorporated into the rest of the machinery via the reckoning process.

Originally, Cambodia, which does have a prevalence survey, was included in this group because we have not yet coded the machinery necessary to reproduce the Asian Epidemic Model used by UNAIDS to model prevalence and incidence in Southeast Asian countries. The 2005 Demographic and Health (DHS) survey in Cambodia made clear that we were underestimating the burden due to HIV there by not using survey data during the modeling process. In order to more accurately represent the epidemic, we used the mortality profile from Thailand and scaled it by 80%, the ratio of estimated prevalence rate in Thailand in 2005 and the prevalence rate from the DHS survey in Cambodia. We then treated the scaled death series as VR data and added Cambodia to the 2B group that is run through CIBA.

Subnational splitting for India and Kenya

Spectrum results for India and Kenya subnational locations are modeled at higher levels of geography than our GBD locations. For example, Spectrum results for India are produced at the state level, while GBD 2015 estimates were produced at the state urban-rural level. Similarly, Spectrum is modeled at the province level, while we compute Kenyan subnational estimates for the 47 counties. To split the Spectrum results into more granular results for processing, we assign each GBD subnational unit to a Spectrum modeling unit. From this, we generate age/sex/year-specific proportions for population, HIV-specific death, and HIV-free mortality.

After this subnational splitting, results were incorporated into the all-cause mortality estimation machinery via the reckoning process described in Part 1.

HIV/AIDS resulting in other diseases

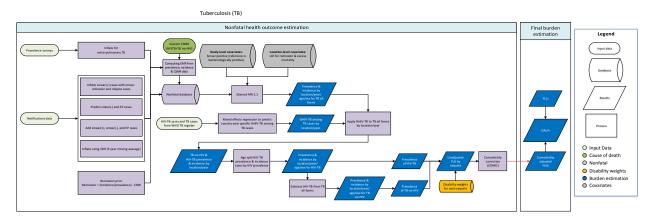
There are two Level 4 causes under the HIV/AIDS Level 3 cause in the GBD 2015 cause hierarchy. The modeling process for HIV/AIDS-tuberculosis is detailed in the capstone paper. We computed the number of people living with HIV resulting in other diseases by subtracting the number of people living with HIV/AIDS-tuberculosis from all people living with HIV/AIDS at the 1,000 draw evel.

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Tuberculosis (TB) Incidence SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with tuberculosis (TB) incidence (3.3.2).

Indicator 3.3.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.3, by 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases, is measured using SDG Indicator 3.3.2, number of new and relapsed TB cases per 1,000.

Case definition

TB is an infectious disease caused by *Mycobacterium tuberculosis*. The case definition includes all forms of TB including pulmonary TB and extrapulmonary TB which are bacteriologically confirmed or clinically diagnosed. For TB, the ICD 10 codes are A10-A14, A15-A19.9, B90-B90.9, K67.3, K93.0, M49.0, P37.0, Z03.0, Z11.1, Z20.1, Z23.2, and ICD 9 codes are 010-019.9, 137-137.9, 138.0, 138.9, 139.9, 320.4, 730.4-730.6, V01.1, V03.2, V12.01, V74.1. For HIV-TB, the ICD 10 code is B20.0.

Input data

For GBD 2015, input data included annual case notifications, data from prevalence surveys, and estimated cause-specific mortality (CSMR) of TB among HIV-positive and HIV-negative individuals. From these inputs, we calculated "priors" (expected values) on excess mortality and remission to give greater guidance to the model.

A systematic review done for GBD 2015 using the following PubMed search terms: (((tuberculosis[Title/Abstract]) OR TB[Title/Abstract]) OR Mycobacterium tuberculosis[Title/Abstract]) AND prevalence[Title/Abstract] Filters: Publication date from 2013/01/01 to 2015/12/31; Humans.

The exclusion criteria were:

- 1. Studies that were not population-based, e.g., hospital or clinic-based studies
- 2. Studies that did not provide primary data on epidemiological parameters, e.g., commentaries
- 3. Studies with a sample size of less than 150
- 4. Reviews

Modeling strategy

For GBD 2015, we used DisMod-MR 2.1, the GBD Bayesian meta-regression tool that adjusts for differences in methods between data sources and imposes consistency between data for different parameters.

Modeling TB incidence

We used the age- and sex-specific notifications in our analysis. There were age-specific missing data especially for younger age-groups in some countries. We imputed the missing age-groups for three forms of TB notifications (pulmonary smear-positive, pulmonary smear-negative, and extra-pulmonary). Smear-positive age-specific notifications were inflated with the proportion smear-unknown and relapsed cases only reported in the country-year data. Some countries reported only pulmonary smear-positive cases for selected years. Missing smear-negative and extrapulmonary cases were predicted from the adjusted smear-positive cases using a seemingly unrelated regression. All three types of notifications were added together and adjusted for undetected cases using WHO's estimates of country-year-specific case detection rates. We applied a 5-year moving average of case detection rates to avoid its fluctuation over time.

Modeling TB prevalence

Data from prevalence surveys reporting on pulmonary smear-positive TB and bacteriologically positive TB were included. We included a study covariate indicating whether it was bacteriologically positive TB (reference category) or smear-positive TB. We did not expect systematic bias between studies that used both symptoms and chest X-ray as screening methods and studies that used only one of the methods. We therefore did not adjust them for systematic bias but added more uncertainty to data points from studies that used only one of the screening methods. We also added more uncertainty to data points from subnational surveys. Because incidence data are for all forms of TB, we adjusted prevalence surveys to account for extrapulmonary cases. We predicted location-year-age-sex-specific proportions of extrapulmonary TB among all TB cases using data on the three forms of TB from the incidence data above and the lagged distributed income covariate from the IHME covariate database. We then computed the extrapulmonary inflation factor as 1+(proportion of extrapulmonary TB /(1- proportion of extrapulmonary TB)), and applied it to data from prevalence surveys.

Modeling remission and excess mortality

We matched each prevalence data point and TB CSMR (TB and HIV-TB combined) by location, year, age, and sex to calculate excess mortality rate (EMR) using the function in DisMod. We also matched each

incidence data point and TB CSMR by location, year, age, and sex to calculate EMR for data-rich countries. We calculated remission using data from countries where both incidence and prevalence data were available. We matched incidence and prevalence data by location, year, age, and sex, and calculated remission as *remission=(incidence/prevalence)-EMR*. For data-rich countries, we assumed a remission of 2.0 (1.8-2.2). We ran two DisMod models: one where we used the calculated remission for low- and middle-income countries, and another where we applied the remission assumption for data-rich countries.

HIV-TB incidence and prevalence

The output from the DisMod model described above is for all forms of TB in HIV-negative and HIV-positive individuals. To separate out HIV-TB from all forms of TB, we first estimated the proportions of HIV-TB cases among all TB cases for all locations and years, using the adult HIV death rate covariate in a mixed effects regression. The input data for this regression (i.e., proportions of HIV-TB cases among all TB cases) were based on the number of TB cases recorded as HIV-positive and the number of TB cases with an HIV test result recorded in the WHO TB register. We applied the predicted location-year-specific proportions to TB incident and prevalent cases from DisMod, respectively, to generate HIV-TB incident and prevalent cases by location and year, which were then age-sex split based on the age-sex pattern of estimated HIV prevalence to generate location-year-age-sex-specific HIV-TB incident and prevalent cases.

Betas and exponentiated values (which can be interpreted as an odds ratio) from the two DisMod models are shown in the tables below:

Betas and exponentiated values from the model using remission calculated based on incidence and prevalence data

Covariate	Parameter	Beta (95% CI)	Exponentiated beta (95% CI)
Smear positive TB	Prevalence	-0.75 (-0.76 — -0.75)	0.47 (0.47 — 0.47)
Sex (male)	Prevalence	0.79 (0.68 — 0.89)	2.19 (1.98 — 2.44)
Sex (male)	Incidence	0.61 (0.59 — 0.63)	1.85 (1.81 — 1.88)
LDI (log-transformed)	Remission	0.12 (0.070 — 0.22)	1.13 (1.07 — 1.24)
LDI (log-transformed)	Excess mortality	-0.10 (-0.10 — -0.10)	0.90 (0.90 — 0.90)

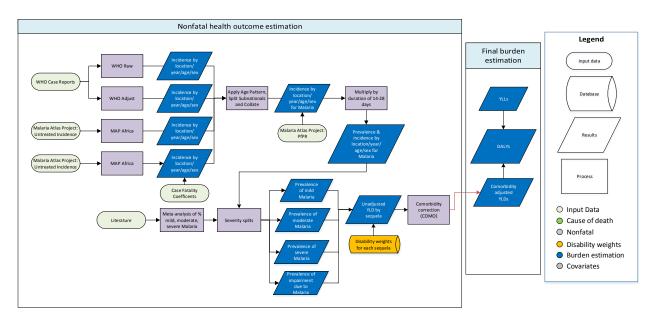
Betas and exponentiated values from the model applying the remission assumption for data-rich countries

Covariate	Parameter	Beta (95% CI)	Exponentiated beta (95%
			CI)
Smear positive TB	Prevalence	-0.75 (-0.76 — -0.75)	0.47 (0.47 - 0.47)
Sex (male)	Prevalence	0.77 (0.68 — 0.86)	2.16 (1.97 — 2.36)
Sex (male)	Incidence	0.60 (0.60 — 0.61)	1.83 (1.82 — 1.84)
LDI (log-transformed)	Remission	0.091 (0.045 — 0.18)	1.10 (1.05 — 1.19)
LDI (log-transformed)	Excess mortality	-0.10 (-0.10 — -0.10)	0.90 (0.90 — 0.90)

Malaria SDG Capstone Appendix

Flowchart

Malaria



Input data and methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with malaria incidence (3.3.3).

Indicator 3.3.3

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.3, by 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases, is measured using SDG Indicator 3.3.3, malaria cases per 1,000.

Case definition

Malaria is an acute parasitic mosquito-borne disease. An individual with uncomplicated malaria experiences one to two weeks of persistent fever, chills/shivering, sweating, joint pains and headache. The individual will likely be lethargic and feverish, causing loss of daily function during the attack. Individuals with an untreated *P. falciparum* infection may develop severe malaria, which includes the symptoms of uncomplicated malaria plus potentially swelling, difficulty breathing, unconsciousness, and death. Rapid diagnostic test or microscopy are considered the gold-standard diagnostic approaches for the purposes of the Global Burden of Disease Study (GBD). The relevant ICD-10 codes are B50-B54.

Input data

Model inputs

For GBD 2015 a systematic review of malaria was not conducted. Updates to systematic reviews are performed on an ongoing schedule across all GBD causes and an update for will be performed in the next one to two iterations. However, as described below, GBD 2015 does feature a substantial estimation change relative to GBD 2013 through the integration of work by the Malaria Atlas Project (MAP).

Data for the malaria modeling process come from three main sources. For endemic countries in continental Africa, we use estimates from MAP (1). Specifically, we use spatiotemporal (ST) cubes of incidence in three broad age-bins (0-5, 5-14 and 15+). It should be noted that these incidence estimates differ slightly from those published previously. First, the cube was re-estimated using a newer antimalarial coverage covariate that includes all types of antimalarial drugs rather than just artemisinin-based combination therapy (ACT). This change improves estimates occurring before ACT rollout in the early 2000s and otherwise allows for the expansion of the initial period of interest (from 2000-2015 to 1990-2015). Second, we combined the incidence estimates with a measure of drug efficacy to produce estimates of untreated incidence that then serve as our quantity of interest for burden assignment. Ultimately, we use MAP data for the following countries: Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Congo, Cote d'Ivoire, Democratic Republic of the Congo, Djibouti, Equatorial Guinea, Eritrea, Ethiopia, Gabon, Ghana, Guinea, Guinea-Bissau, Kenya (subnational), Liberia, Madagascar, Malawi, Mali, Mauritania, Mozambique, Namibia, Niger, Nigeria, Rwanda, Senegal, Sierra Leone, Somalia, South Sudan, Sudan, Swaziland, Tanzania, The Gambia, Togo, Uganda, Zambia, and Zimbabwe.

For most other countries with malaria transmission, we use confirmed cases reported in the 2013 World Malaria Report (WMR). These include: Afghanistan, Algeria, Argentina, Armenia, Azerbaijan, Bangladesh, Belize, Bhutan, Bolivia, Brazil (subnational), Cambodia, Cape Verde, China (subnational), Colombia, Comoros, Costa Rica, Dominican Republic, Ecuador, Egypt, El Salvador, Georgia, Guatemala, Guyana, Haiti, Honduras, Iran, Iraq, Kyrgyzstan, Laos, Malaysia, Mauritius, Mexico (subnational), Morocco, Nepal, Nicaragua, North Korea, Oman, Pakistan, Panama, Paraguay, Peru, Philippines, Sao Tome and Principe, Saudi Arabia (subnational), Solomon Islands, South Africa (subnational), South Korea, Sri Lanka, Suriname, Syria, Tajikistan, Thailand, Timor-Leste, Turkey, Turkmenistan, United Arab Emirates, Uzbekistan, Vanuatu, Venezuela, and Vietnam.

For the final data type, we used the GBD 2013 systematic review of studies with data on the clinical incidence of malaria. These studies were georeferenced so that the sites could be matched with spatially explicit covariates such as *Plasmodium falciparum* parasite rate (*Pf*PR). Data are standardized into four age categories: 0-4, 5-14, 15+ and all age. Countries whose results are estimated in this group include: India (subnational), Yemen, Indonesia, Myanmar and Papua New Guinea.

Severity splits

As in GBD 2013, we use a two-step process for determining malaria severity. For acute cases, severity splits for mild, moderate, and severe malaria were produced by analysis of Medical Expenditure Panel Survey data. These sequelae and their associated disability weights (DWs) are presented below.

Table 1. Severity level, lay description, and DW

Severity level	Lay description	DW (95% CI)
Mild	Has a low fever and mild discomfort but no	0.006
	difficulty with daily activities.	(0.002-0.012)
Moderate	Has a fever and aches and feels weak, which causes	0.051
	some difficulty with daily activities.	(0.032-0.074)
Severe	Has a high fever and pain and feels very weak,	0.133
	which causes great difficulty with daily activities.	(0.088-0.19)

To determine long-term neurological burden due to malaria, we use the work by Roca-Felter et al. (2008) that examined the number of uncomplicated cases that led to longer-term impairment. Analytically, this means multiplying incidence estimates (described in the section below) for persons under 20 by 0.00029 (0.000077-0.00057). This subset is then combined with excess mortality rates derived from all-cause mortality and standardized mortality ratios for neonatal encephalopathy (NE) in a DisMod model to produce prevalence estimates for all estimation years. Implicit in this process is an assumption that the disability and trend of impairment due to severe malaria follow NE. The subsequent severity splitting follows NF as well.

Modeling strategy

We stratify analysis of malaria morbidity into four different location sets: MAP Africa, WHO Raw, WHO Adjusted and Study Level.

MAP Africa:

For the subset of countries using data from MAP, we combine the broad age and sex untreated incidence estimates from the space-time cube with mortality estimates to generate incidence estimates by GBD standard age and sex groupings. As part of the mortality estimation process, we developed a regression model by age-bin where case fatality is a function of all-cause mortality and sex. More information can be found in the GBD 2015 mortality and causes of death paper. Using these models, we generated implied cases estimates where cases = death/case fatality for all age groups and both sexes. Subsequently, we scale the implied cases by age-bin so that they match the corresponding estimates from MAP. Although we carry forward the uncertainty from the MAP incidence estimates, we do not include the uncertainty of the age pattern.

WHO Raw:

Kyrgyzstan, Tajikistan, Belize, Panama, Iran, South Africa, China and Saudi Arabia, Tajikistan, Turkey, Azerbaijan, Uzbekistan, Georgia, South Korea, Argentina, Costa Rica, Armenia, Malaysia, Sri Lanka, Bhutan, Iraq, North Korea, Paraguay, Mexico, El Salvador, Ecuador, Cape Verde and Algeria all feature low case rates and are considered to have complete case reporting—particularly since the turn of the century. As such, our case rates are taken directly from those reported in the WMR. Because of systematic review cycles as well as the publication cycle of the WMR, we assume a flat trend of cases forward for the unmeasured years (e.g., 2014 and 2015). For some countries, data from the early 1990s were not reported. For these countries, cases were extrapolated via a mixed effects regression with reported cases as the outcome variable, year as the main predictor and random effects on location. The WMR does not provide information on the demographic characteristics of the malarial case, we apply the 5th percentile age pattern derived from the incidence estimates for MAP Africa to generate age- and sexspecific incidence estimates. In this case, the uncertainty in the broad age-bin pattern is taken into account.

In China, South Africa, and Saudi Arabia, we provide estimates at the subnational level, but the WMR only reports at the national level. Therefore, Chinese subnational estimates are derived from case reports from their malaria control program, while for South Africa and Saudi Arabia, we proportionally assign cases into the subnational geographies by MAP's 2010 world map of prevalence of *Pf*PR (2). This procedure is the assumption that the distribution of *Pf*PR is substantially similar to other malaria strains.

WHO Adjust:

For the remaining countries in the broader WHO grouping (see above) that feature either incomplete reporting systems or higher malaria burden, we adjust the WMR confirmed incidence rate upward via a proxy variable for health systems access. For Mexico, we replace the WHO estimates with subnational estimates from the national malaria control program.

First, we regress the confirmed incidence rate as a function of *Pf*PR and the interaction of health system access and malaria death rate with random effects on location. The regression is fit using all countries with complete or semi-complete case reports (e.g. not countries in MAP Africa or the study level approach) to derive stable relationships between *Pf*PR and the interaction term. Using the coefficients and associated uncertainty (fixed and random effects), we predict incidence rate with health system access set to the 95th percentile – thereby serving as a proxy for a more complete reporting system.

We apply the 5th percentile MAP Africa pattern to generate age- and sex-specific estimates. Although the uncertainty of the regression is captured, we do not capture uncertainty in the age and sex pattern.

Study Level:

For countries that feature a high malaria burden and poor case reporting infrastructure that are outside of Africa, we use an ordinary least squares regression with the natural log of incidence as the outcome. Predictors include the natural log of the malaria death rate, an indicator variable for Africa, the ratio between country and site level P PfPR , interaction terms between each age bin and the associated log malaria death rate and an indicator variable on whether the data point relies on passive case detection. We use the 5^{th} percentile age pattern derived from the MAP Africa estimates to distribute cases by age and sex.

Converting to prevalence

Once the separate incidence estimation procedures are complete, the results are combined and converted to prevalence by matching each draw with a draw of duration. Consistent with GBD 2013, we use a uniform distribution between 14 and 28 days for duration.

Comparison to GBD 2013

We have re-evaluated our location groupings for GBD 2013, moving several countries from the WHO adjustment category into the WHO Raw categories. Through the inclusion of the back-casting step, we account for the potential under-reporting that may have occurred pre-2000 in these reassigned countries.

GBD 2013 included an interpolation step to correct for jagged age-patterns as a correction for all-cause mortality during the age- and sex-splitting process. This step created overly smooth time and age trends and has since been removed through the inclusion of high- quality patterns from MAP. As such, we expect

more pronounced age patterns (higher in youth, less so in adults) and time trends (sharper declines since 2000) relative to GBD 2013.

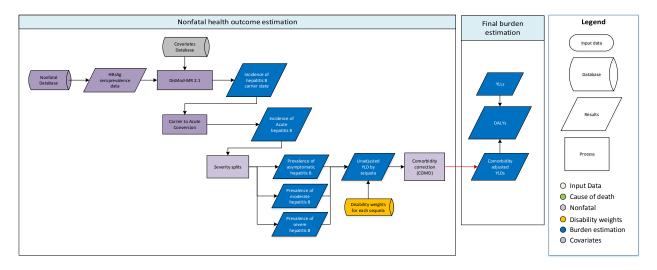
The inclusion of MAP incidence estimates in general marks a major step forward in better understanding malarial burden in Africa. In general, we expect a slightly higher amount of disability relative to GBD 2013. Future GBD iterations will expand the geostatistical model currently used in Africa to the rest of the world.

In conjunction with changes in the mortality estimation, GBD 2015 burden due to malaria now features a fully consistent set of results via the integration of MAP incidence data into the modeling framework.

Acute Hepatitis B SDG Capstone Appendix:

Flowchart

Acute hepatitis B



Input Data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with hepatitis B incidence (3.3.4).

Indicator 3.3.4

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.3, by 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases, is measured using SDG Indicator 3.3.4, hepatitis B incident cases per 100,000).

Case definition

We define acute hepatitis B as the period corresponding to initial infection with the hepatitis B virus, regardless of symptoms. It includes all ICD-10 codes under the heading B16 (Acute hepatitis B).

Input data

Model inputs

We use hepatitis B surface antigen (HBsAg) seroprevalence data from population-based studies and surveys for the incidence model.

Level	Prevalence
Data points	2,987
Studies	312
Locations	145
Regions	19

Updates to systematic reviews are performed on an ongoing schedule across all GBD causes.

Modeling strategy

We model the incidence of chronic HBsAg carriage using a full DisMod model of HBsAg seroprevalence. We then convert incidence of chronic carriage to total incidence of hepatitis B infection by dividing agespecific estimates of the incidence of chronic carriage by age-specific estimates of the probability of infection resulting in carriage based on Edmunds et al. (1993).

$$P(carrier \mid age \le 6 \ months) = 0.885$$

$$P(carrier \mid 6 \ months \le age < 25 \ years) = e^{-0.645 \times age^{0.455}}$$

$$P(carrier \mid age \ge 25 \ years) = e^{-0.645 \times 25^{0.455}} = 0.061$$

We then split symptomatic cases into moderate (73%) and severe (27%) severities based on data from McMahon et al. (1985).

Sequela	Description	Disability weight
Moderate	Has a fever and aches, and feels weak, which causes	0.051
	some difficulty with daily activities.	(0.032-0.074)
Severe	Has a high fever and pain, and feels very weak, which	0.133
	causes great difficulty with daily activities.	(0.088-0.19)
Asymptomatic	Infection with no apparent illness.	NA

Changes from GBD 2013 to GBD 2015

We have updated the severity splits, but the modeling strategy remains otherwise unchanged from GBD 2013.

Neglected Tropical Diseases (NTDs) SDG Capstone Appendix

African trypanosomiasis, Chagas disease, cystic echinococcosis, cysticerosis, dengue, food-borne trematodiases, intestinal nematode infections, leishmaniosis, leprosy, lymphatic filariasis, onchocerciasis, rabies, schistosomiasis, and trachoma

Indicator definition

This modeling strategy encompassed the indicator associated with neglected tropical disease prevalence (3.3.5).

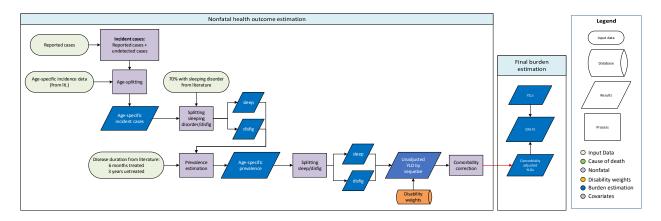
Indicator 3.3.5

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.3, by 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases, is measured using SDG Indicator 3.3.3, prevalence of neglected tropical diseases.

Human African Trypanosomiasis (HAT) SDG Capstone Appendix

Flowchart

African trypanosomiasis



Input Data & Methodological Summary

Case Definition

Human African Trypanosomiasis (HAT), also known as sleeping sickness, is a vector-borne disease which is transmitted by the bite of the tsetse fly. It is caused by the parasite *Trypanosoma brucei* with two subspecies, namely *T.b. rhodesience* (makes up less than 5% of total HAT cases) and *T.b. gambiense*. Cases are diagnosed through laboratory methods which rest on finding the parasite in body fluid or tissue by microscopy. In highly endemic or epidemic areas where the likelihood of false positives in serological tests is deemed lower, a seropositive individual is considered affected even in the absence of parasitological confirmation. The ICD-10 code for HAT are B56.0, B56.1 and B56.9.

Input data Model inputs

The input data for GBD 2015 included a) population at risk estimates from GBD 2010 ArcGIS analysis using geocoded case notifications for 2000 to 2009 [1] and population Count Grid estimates from Gridded Population of the World 3 [2, 3], b) population screened from 1997 to 2004 [4], c) historical data from GBD 2010 on total number of HAT cases reported [1, 4, 5], and d) cases reported annually to the WHO [6] – for Kenya, a study on cases reported subnationally [7] was used to split the national cases into five counties (HomaBay, Migori, Busia, Bungoma, Kakamega). A systematic review of literature was conducted in PubMed on 8/10/2016 using the following search string:

((African trypanosomiasis[Title/Abstract] AND incidence[Title/Abstract]) AND ("2009"[Date – Publication]): "2013"[Date – Publication])).

This yielded 72 studies of which only four met the inclusion criteria and were extracted. The inclusion criteria were:

- 1. Studies representative of the national population
- 2. Population-based studies
- 3. Studies with primary data on incidence
- 4. Studies of human African trypanosomiasis only (excluded studies on animal African trypanosimiasis)

The four studies extracted had national incidence data similar to the ones extracted from the WHO [6]. Therefore, only two studies with age-specific incidence data from active screening undertaken in the Democratic Republic of Congo [8] and Uganda [9] were used to inform age pattern for incidence and prevalence. Location-years with missing reported cases were excluded and five subnational locations for Kenya were added. The table below shows the number of studies included, and the number of countries or subnational units and GBD world regions represented.

	incidence
Studies	2
Countries/subnationals	34
GBD world regions	4

Severity splits/Sequelae

The basis of the GBD disability weight (DW) survey assessments are lay descriptions of sequelae highlighting major functional consequences and symptoms. The lay descriptions and disability weights for HAT sequelae due to HAT are shown below.

Sequela	Lay description	DW (95% CI)
Skin	has a visible physical deformity that causes others to	0.067 (0.044-0.096)
disfigurement,	stare and comment. As a result, the person is worried	
level 2	and has trouble sleeping and concentrating	
Motor plus	cannot move around without help, and cannot lift or	0.542 (0.37 – 0.702)
cognitive	hold objects, get dressed or sit upright. The person also	
impairments,	has very low intelligence, speaks few words, and needs	
severe	constant supervision and help with all daily activities	

Modeling strategy

The non-fatal model for HAT involved estimating prevalence from incidence. First, a multi-level mixed-effects linear regression of natural log-transformed incidence rate (ratio of HAT cases reported to population at risk) on natural log-transformed screening coverage (ratio of number screened for HAT to population at risk), with country random effects, was performed. Gaps were then filled using exponential interpolation between years and extrapolation from 2014 to 2015 for reported cases; for screening coverage only extrapolation from 2014 to 2015 was done. Then 1,000 draws of mortality among treated cases were generated, assuming that 0.7% - 6.0% of all treated (reported) cases die [10, 11, 12].

Using the mean and variance-covariance matrix from the regression as parameters, a multivariate normal distribution was used to generate 1,000 draws of case detection rate (CDR), given the expected screening coverage. Undetected deaths were then estimated as the difference between the ratio of reported cases to CDR and reported cases (reported cases/CDR – reported cases). Estimates of incidence were obtained by adding the reported cases to the undetected cases. Without information on sexspecific incidence, equal incidence rates between both sexes was assumed. Finally, an age-pattern was applied to the incidence estimates using the incidence studies from DRC and Uganda [8, 9]. Assuming the same proportion in treated and untreated cases, the incidence estimates were then split into the two sequelae, skin disfigurement and sleeping disorder. This was done by generating 1,000 draws of the splitting proportion for the sequelae (70%-74% with sleeping disorder) based on a study that reported presence of symptoms at admission of patients in treatment centers [13] – draws were generated from a beta distribution with alpha parameter = 1884 and beta parameter = 649.

To compute prevalence of HAT, 1,000 draws of total duration of symptoms in untreated cases was generated from a normal distribution with mean = $\{\ln(3) - 0.5 * \text{sigma^2}\}$, and standard deviation = sigma, where sigma = $\{\ln(4.39) - \ln(1.92)\}/(\text{invnormal}(0.975)*2)\}$ – these parameters were based on a study of *T.b. gambiense* [14] which estimated an average duration of three years to untreated cases. An estimated duration of six months was applied to cases that received treatment, based on findings from a paper about *T.b. rhodesiense* in Uganda [11]. Prevalence was then estimated from the incident cases before applying age pattern. Prevalence of treated and untreated cases were summed up, assuming that untreated cases have been prevalent up to their death for a certain duration. For untreated cases, it was assumed that half the duration is spent with sleeping disorder (severe motor and cognitive impairment) and disfigurement [14]. Treated (i.e., reported) cases are assumed to have been prevalent for 0.5 years, and for the fraction of treated cases that present with sleeping disorder, it was assumed that this is present for half the total duration and that the rest of the duration is spent suffering from disfiguring skin disease. Treated cases that don't present with sleeping disorder were assigned disfigurement for the entire duration. Lastly, an age-pattern was applied to the prevalence estimates using the incidence studies from DRC and Uganda [8, 9].

Results from the model were assessed by visualizing time trends of incident and prevalent cases across locations and age (similar trends were applied in both sexes). Maps of the global distribution of HAT and the two sequelae were also generated. In addition, the estimated incident cases were compared with the cases reported to the WHO across time – as expected, the estimates from GBD 2015 were higher than the WHO numbers because we accounted for undetected cases.

Changes from GBD 2013 included: a) inclusion of new data on reported cases from WHO [6] (years 2013 and 2014 for 23 locations), b) inclusion of the following country (years) based on available historical data post-1980: Botswana (1983), Ethiopia (1980-1983), Guinea-Bissau (1980-1983, 1985-1987), Rwanda (1980, 1982-1988), and Sierra Leone (1981-1982), c) adding five subnational locations (out of 49) for Kenya, thusd) correcting the age-split proportion such that a 0.32/0.68 proportion was used for adults/kids –in GBD 2013, this proportion was 0.25/0.75 for adults/kids.

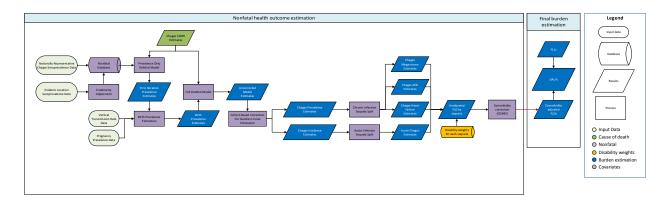
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Chagas Disease SDG Capstone Appendix

Flowchart

Chagas disease



Input Data & Methodological Summary

Case definition

Chagas disease is defined by infection with the protozoa *Trypanosoma cruzi*, which is transmitted by *Triatominae* insect vectors (most common), blood transfusion, organ transplant, and congenital transmission. It includes an acute phase corresponding with the time of infection, and is typically asymptomatic. Chronic infection may be latent (i.e., asymptomatic), or result in cardiovascular or digestive sequelae. It includes all ICD-10 codes under the heading B57 (Chagas disease), with codes B57.0-B75.1 corresponding to the acute phase, B57.2 corresponding to chronic cardiovascular sequelae, and B57.3 corresponding to chronic digestive sequelae.

Input data

Model inputs

For GBD 2015 estimation, we used seroprevalence data to model Chagas. The table below illustrates the geographic distribution of model input data for the estimation process.

Level	Prevalence
Data points	407
Studies	56
Locations	20
Regions	4

We also use CSMR estimates in the modeling process, which will be addressed in further detail below.

Modeling strategy

We modeled Chagas disease using a full DisMod-MR 2.1 Bayesian meta-regression model incorporating seroprevalence data, as above, and CSMR estimates. We assume no remission. We eliminate all new infections, except those via vertical transmission, in Chile and Uruguay for years after the interruption of vector-based transmission. For non-endemic countries, we estimate the prevalence of imported chronic infections based on migration. For each non-endemic country, we estimate the total number of people infected with Chagas as the sum of the number of immigrants from each endemic country multiplied by the corresponding prevalence of Chagas in that endemic country.

We estimate five sequelae: symptomatic acute infection from incidence; and megaviscera, heart failure, atrial fibrillation, and chronic asymptomatic infection from prevalence. We assume that 5% of acute infections will be symptomatic. The proportion of chronic infections resulting in a given sequela varies by sex and age: the prevalence of megaviscera among those infected with Chagas ranges from 0% in children to nearly 10% among older adults; the prevalence of atrial fibrillation attributable to Chagas ranges from 0% among children to approximately 10% in men over 80 years of age; and the prevalence of heart failure attributable to Chagas among those who are infected ranges from 0% among young children to a maximum of 23% among men over 80 years of age.

Severity splits and disability weights

Sequela	Description	Disability Weight
Atrial fibrillation and flutter due to Chagas disease	Has periods of rapid and irregular heartbeats and occasional fainting.	0.224 (0.151-0.312)
Mild heart failure due to Chagas disease	Is short of breath and easily tires with moderate physical activity, such as walking uphill or more than a quarter-mile on level ground. The person feels comfortable at rest or during activities requiring less effort.	0.041 (0.026-0.062)
Moderate heart failure due to Chagas disease	Is short of breath and easily tires with minimal physical activity, such as walking only a short distance. The person feels comfortable at rest but avoids moderate activity.	0.072 (0.047-0.103)
Severe heart failure due to Chagas disease	Is short of breath and feels tired when at rest. The person avoids any physical activity, for fear of worsening the breathing problems.	0.179 (0.122-0.251)
Mild chronic digestive disease due to Chagas disease	Has some pain in the belly that causes nausea but does not interfere with daily activities.	0.011 (0.005-0.021)
Moderate chronic digestive disease due to Chagas disease	Has pain in the belly and feels nauseous. The person has difficulties with daily activities.	0.114 (0.078-0.159)

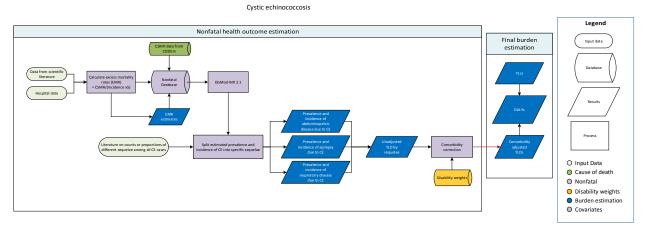
Acute Chagas disease	Has a fever and aches, and feels weak, which causes some difficulty with daily activities.	0.051 (0.032-0.074)
Asymptomatic Chagas disease	Latent Chagas infection (i.e., chronic infection with no apparent symptoms)	NA

Changes from GBD 2013 to GBD 2015

We have made no substantive changes in the modeling strategy for endemic countries from GBD 2013 for Chagas endemic countries. One notable improvement, however, is the estimation of Chagas disease among immigrants living in non-endemic countries which offers a more complete picture of Chagas' burden.

Cystic Echinococcosis SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Case definition

Ehinococcosis is a vector-borne disease caused by two species of tapeworm, *Echinococcus granulosis* (most common in sheep) which causes cystic echinococcosis (CE) and *E. multilocularis* (most common in foxes and wild dogs) which causes alveolar echinococcosis. Diagnosis is made by clinical findings, imaging, and serology. The ICD-10 codes for echinococcosis are B67-B67.9.

Input data

Model inputs

The nonfatal estimation for cystic echinococcosis (CE) focused on estimating incidence and prevalence of CE and its sequelae. A systematic review of literature was conducted in PubMed using the following search string:

("echinococcosis"[Title/Abstract] OR "hydatid disease"[Title/Abstract] OR "hydatidosis"[Title/Abstract] OR "echinococcul disease"[Title/Abstract] OR "Echinococcul granulosus infection"[Title/Abstract]) AND ("1990"[Date – Publication] : "2015"[Date – Publication]) AND (epidemiology OR incidence OR prevalence).

This yielded 1,619 studies of which 279 were included during the title/abstract screening. Following the full-text screening, 77 studies (32 incidence, 43 prevalence and 2 both) were included and extracted – studies were excluded because of one or more of the following reasons:

- 1. study not population-based
- 2. study does not have primary data on prevalence and/or incidence
- 3. study not in humans
- 4. study on sub-populations

5. review study

We combined the newly extracted studies with studies extracted during GBD 2013. The table below shows the number of studies finally included, and the number of countries or subnational units and GBD world regions represented.

	incidence	prevalence
Studies	47	58
Countries/subnationals	41	24
GBD world regions	12	8

Hospital data on incidence prepared by the GBD team was also used in the CE model. The table below shows the number of studies included in the hospital data, and the number of countries or subnational units and GBD world regions represented.

	incidence
Studies	38
Countries/subnationals	100
GBD world regions	8

Since we were interested in modeling symptomatic CE cases, we only used data on incidence of patients diagnosed by imaging techniques (mainly ultrasonography). Therefore we excluded prevalence data which were mostly from serological studies.

Two additional data sources that were used, including 1) data on echinococcosis endemicity (0=no cases/no data, 1=sporadic/mostly imported, 2=endemic/limited data, 3=highly endemic) provided by one of our echinococcosis collaborators, and 2) literature data on observed cases of abdominal, respiratory, and epileptic symptoms among echinococcosis cases [1].

Sequelae due to cystic echinoccocosis

The table below shows the sequelae due to echinococcosis and their associated disability weights.

Sequela	Lay description	DW (95% CI)
Chronic respiratory disease	has cough and shortness of breath after heavy physical activity, but is able to walk long	0.019 (0.011-0.033)
	distances and climb stairs.	
Abdominal problems	has pain in the belly and feels nauseous. The person has difficulties with daily activities	0.114 (0.078 – 0.159)
Epilepsy	(Combined DW)	NA

Modeling strategy

DisMod MR was used to model the nonfatal burden of symptomatic cystic echinococcosis (CE) using only incidence data. Mortality estimates from the custom mortality model were used to inform the excess mortality parameter (CODEm estimates used as cause-specific mortality rate data). Estimates of

excess mortality rate were obtained and used to estimate prevalence (CSMR/EMR). A remission of 0.15-0.25 per case per year (duration 2 – 6.7 years, average 5 years) was assumed. The following steps were followed to estimate excess mortality rate: 1) create custom age groups for CE deaths at the 1,000 draw level; 2) calculate CSMR as CSMR=deaths/population at the 1,000 draw level – calculate mean CSMR, uncertainty interval, and standard error; and 3) calculate EMR as EMR=CSMR/(prevalence), where prevalence = (incidence*5) – standard error of EMR was calculated taking into consideration the standard errors of both prevalence and CSMR.

After running DisMod, a thousand draws of proportions for abdominal, respiratory and epileptic symptoms among echinococcosis cases, that add up to 1, were generated. Uncertainty in the splitting proportions was captured by drawing them from a Dirichlet distribution, informed by published data on cysts localization [1]. On average, the proportions of abdominal, respiratory, and epileptic symptoms due to echinococcosis were 0.8, 0.19, and 0.01, respectively. These proportions were used to split the prevalence and incidence from DisMod into the three sequelae.

Model evaluation was done by separately assessing the fit of the DisMod MR model and checking the estimates produced after estimating incidence and prevalence of sequelae due to cystic echinococcosis. Plots of time trends of incidence and prevalence across locations and age were used to evaluate the results. In addition, maps of the global distribution of incidence and prevalence were assessed across time.

Changes from GBD 2013 included: a) estimation of excess mortality rate using literature/hospital incidence and CSMR data, b) inclusion of echinococcosis endemicity in as a country-level covariate in DisMod, and c) assuming that remission is 0.15-0.25 instead of 0.1 to 1.0 based on discussions with GBD 2015 CE model reviewers (an average remission of five years was used to calculate prevalence of CE).

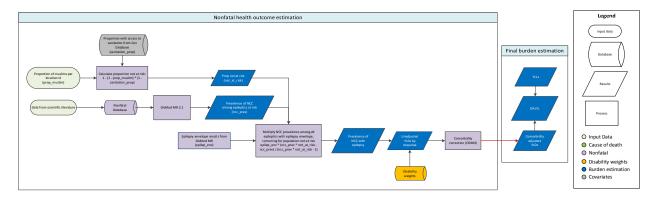
References

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Cysticercosis SDG Capstone Appendix

Flowchart

Cysticercosis



Input Data & Methodological Summary

Case Definition

Cysticercosis is a helminth disease caused by the pig tapeworm, *Taenia solium*, transmitted through the fecal oral route or by consumption of pork containing *T. solium* eggs. Diagnosis is made by Magnetic resonance imaging or CT brain scans for neurocysticercosis. The ICD-10 codes for cysticercosis are B69-B69.9.

Input data

Model inputs

The nonfatal estimation for cysticercosis focused on estimating prevalence of neurocysticercosis among epileptics at risk as well as the prevalence of neurocysticercosis with epilepsy. A systematic review of literature was conducted in PubMed using the following search string:

("cysticercosis"[Title/Abstract] OR "neurocysticercosis"[Title/Abstract] OR "cysticerciasis"[Title/Abstract] OR "Taenia solium"[Title/Abstract]) AND ("1990"[Date – Publication]: "2015"[Date – Publication]) AND (epidemiology OR prevalence)).

This yielded 1,038 studies of which 166 were included during the title/abstract screening. Following the full-text screening, 17 studies were included and extracted – studies were excluded because of one or more of the following reasons:

- 1. study not in epileptics
- 2. study not population-based
- 3. study does not have primary data on prevalence of neurocysticercosis among epileptics at risk
- 4. study not in humans (some studies were on cysticercosis in pigs)
- 5. study on comorbidities with neurocysticercosis (other than epilepsy)

- 6. study on sub-population, e.g., patients with neurological disorders
- 7. review study

We combined the newly extracted studies with studies extracted during GBD 2013. The table below shows the number of studies finally included, and the number of countries or subnational units and GBD world regions represented.

	prevalence
Studies	32
Countries/subnationals	23
GBD world regions	8

A study-level covariate was also created to indicate the type of diagnosis for each study, i.e., definitive or probable. Of the 77 rows of country-year-age-sex data, there were 15 rows with definitive diagnosis and 62 rows with probable diagnosis.

Three additional data sources that were used included 1) epilepsy envelope prevalence (from the epilepsy DisMod MR model), 2) proportion of the population with access to sanitation (from the GBD covariates database), and 3) proportion of the population that is Muslim (from the PEW Research Center [1].)(http://www.pewforum.org/2011/01/27/table-muslim-population-by-country/).

Modeling strategy

DisMod MR was used to model the prevalence (ONLY) of neurocysticercosis among epileptics at risk. In the model, pigs per capita and religion (binary, >50% Muslim) were used as country-level covariates. In addition, the prevalence of "definitive diagnosis" was crosswalked to that of "probable and definitive diagnosis" so as to not underestimate overall prevalence.

After running DisMod, we adjusted the fraction of people with epilepsy attributable to cysticercosis in endemic countries for the population at risk (based on the proportion of the population without access to sanitation and the proportion of the population that is Muslim). Predicted neurocysticercosis (NCC) prevalence among epileptics at risk such that Prevalence= $P\times(NM-N)/(NM-1)$, where P= prevalence of all-cause epilepsy in total population, N= proportion of NCC among epileptics at risk (non-Muslims without access to sanitation), and M= proportion of population not at risk of contracting NCC. It was assumed that the prevalence of epilepsy due to causes other than NCC is the same regardless of whether a population is at risk or not. It was also assumed that Muslims and non-Muslims have equal access to sanitation.

Model evaluation was done by separately assessing the fit of the DisMod MR model and checking the estimates produced after estimating prevalence of NCC with epilepsy. Plots of time trends of prevalence across locations and age were used to evaluate the results. In addition, maps of the global distribution of prevalence of NCC among epileptics at risk and prevalence of NCC with epilepsy were also assessed across time.

Other than using additional data extracted from literature, we updated the proportion of population with Muslim data by filling in subnational locations with national proportions – this was done due to lack of data on this covariate at the subnational level.

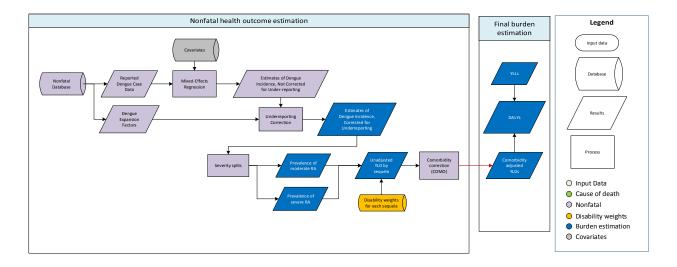
References:

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SDG Indicators Dengue Capstone Appendix

Flowchart

Dengue



Input Data & Methodological Summary

Case definition

Dengue is mosquito-borne viral infection that causes febrile illness and, in severe cases, jaundice, hemorrhage, and death. It includes all ICD-10 codes under the heading A90 (Dengue fever [classical dengue]) and A91 (Dengue hemorrhagic fever).

Input data

Model inputs

For GBD 2015, we modeled dengue incidence based on officially reported cases. The table below illustrates the geographic distribution of data points used in our analysis.

Level	Incidence	
Data points	2515	
Studies	70	
Locations	115	
Regions	14	

Updates to systematic reviews are performed on an ongoing schedule across all GBD causes, and an update for dengue fever will be performed in the next one to two iterations. While no systematic update was conducted, we did incorporate new expansion factor data that were provided by collaborators and have updated to the latest available case reports for GBD 2015.

Modeling strategy

We modeled dengue incidence using an improved variant of the methods used for GBD 2013, described by Stanaway et al. (2013). Briefly, we derive two dengue-specific covariates: first a variable to define the expected spatial distribution of the disease based on principal components analysis of dengue CSMR estimates and dengue transmission probability estimates (Bhatt et al. YEAR) and, second, a variable to define the country-specific trends, based on a mixed effects model of reported cases. We then estimate a mixed effects negative binomial model with number of reported cases as the dependent variable, fixed effects on the aforementioned spatial and temporal covariates, and random effects on location. These random effects are assumed to correspond to deviations in reporting completeness and, calibrating against published expansion factor data (i.e., estimates of the degree of underreporting), they are inflated to adjust for underreporting. The resulting incidence estimates are split into moderate (94.5%) and severe (5.5%) sequelae, based on the proportion of reported cases that were severe. We assume that 8.4% of symptomatic infections will produce post-acute chronic fatigue lasting an average of six months.

Severity splits and disability weights

Sequela	Description	Disability Weight
Moderate	Has a fever and aches, and feels weak, which causes some difficulty with daily activities.	0.051 (0.032-0.074)
Severe	Has a high fever and pain, and feels very weak, which causes great difficulty with daily activities.	0.133 (0.088-0.19)
Asymptomatic	Infection with no apparent illness.	NA

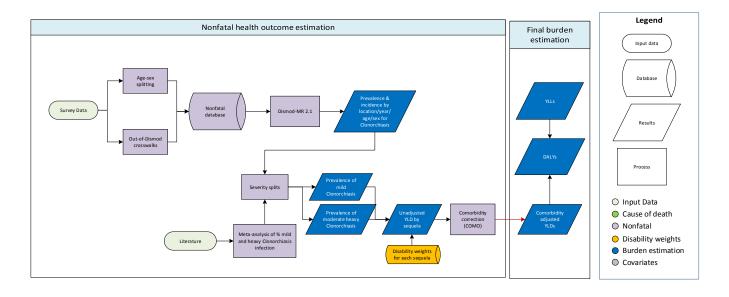
Changes from GBD 2013 to GBD 2015

The approach is largely the same as that used for GBD 2013. One notable change is the addition of the dengue trend covariate described above, which allows for dramatically improved trend estimates.

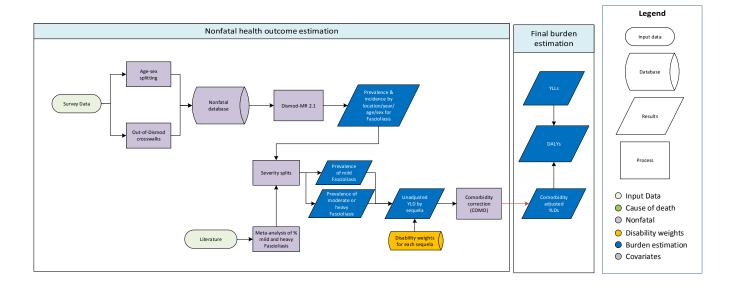
Foodborne Trematodiases SDG Capstone Appendix

Flowcharts

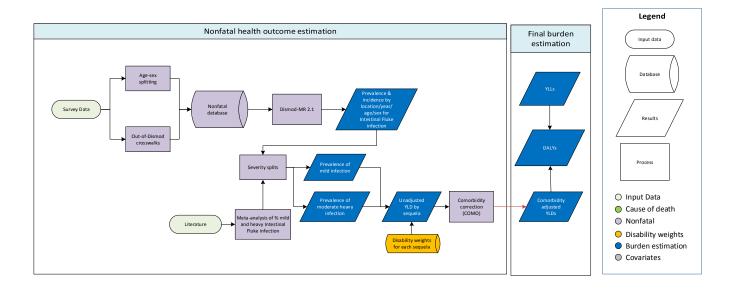
Clonorchiasis



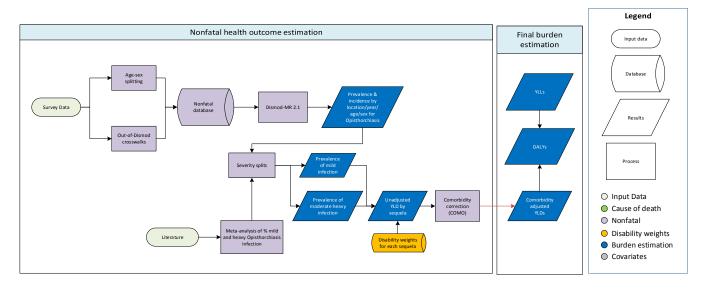
Fascioliasis



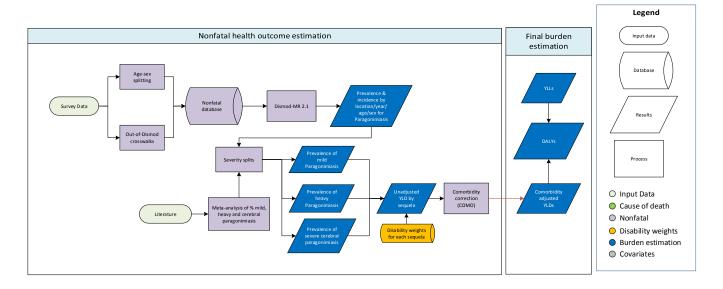
Intestinal fluke



Opisthorchiasis



Paragonimiasis



Input Data & Methodological Summary

Case definition

Human foodborne trematodiases (FBT) is defined as the infection with parasitic worms of the class trematoda, which are also known as flukes. Trematodes are transmitted via contaminated food and infection is highly related to food habits. Definitive hosts, including humans, become infected when ingesting viable metacercariae by consuming contaminated aquatic products (e.g. watercrest etc.). In the ICD-10, FBT are listed under code B66 [1].

FBT is subdivided into six types of FBT (see Table 1):

- Clonorchiasis
- Fascioliasis
- Intestinal fluke
- Opisthorchiasis
- Paragonimiasis (normal and cerebral infections)

Table 1. Subtypes of FBT

	Species of FBT	Also known as:	Carcinogen
1	Chlonorchiasis	(Chinese) Liver fluke	Associated with choliangiocarcinoma
2	Opisthorchiasis	Liver fluke	Associated with choliangiocarcinoma
	(O viverrini & O felineus)		(O viverrini)

3	Fascioliasis	Liver fluke	No available evidence
4	Intenstinal fluke	Liver fluke	No available evidence
5	Paragonimiasis	Lung fluke	

Thresholds for heavy infection and duration by species of FBT

The majority of people infected with FBTs are asymptomatic. When symptoms do occur they are often non-specific. Among the clinical symptomatic group, severity is associated with worm burden, typically measured by fecal egg counts, and the duration of infection. The thresholds for heavy infection and duration by species of FBT are shown in Table 2. The clinical presentation of FBT depends on the target organs (liver, lung, or intestines). Clonorchiasis and opisthorchiasis patients may suffer from loss of appetite, fullness, indigestion, diarrhoea, pain in the right upper quadrant, lassitude, weight loss, ascites, and oedema.[2, 3] Cholangitis, obstructive jaundice, intraabdominal mass, cholecystitis, and gallbladder or intrahepatic stones may occur as complications.[3, 4]

Table 2. Thresholds for heavy infection and duration by species of FBT

	Species of FBT	Case thresholds for heavy infection	Duration
1	Chlonorchiasis	10,000 eggs per g of feces	lifelong
2	Opisthorchiasis	10,000 eggs per g of feces	lifelong
3	Fascioliasis	1,000 eggs per g of faces	lifelong
4	Intenstinal fluke	1,000 eggs per g of faces	lifelong
5	Paragonimiasis	100 eggs per 5 ml sputum	lifelong
6	Cerebral paragonimiasis	Any infection of the brain with flukes and/or eggs of Paragonimus spp.	lifelong

Input data

Model inputs

For GBD 2010, the data came from the expert group and is the result of their analysis. The expert group analysis used the results of a systematic literature review performed by Furst et al. as a starting point for the analysis.[5] Furst et al. searched PubMed, WHOLIS, FAOBIB, Embase, CAB Abstracts, Literatura Latino Americana e do Caribe em Ciências de Saùde (LILACS), ISI Web of Science, BIOSIS preview, Science Direct, African Journals OnLine (AJOL), and the System for Information on Grey Literature in Europe (SIGLE), period Jan 1, 1980 to

Dec 31, 2008. The initial number of studies identified through the literature review was ~34,000 references. The literature review included extracted data from 181 studies. For GBD 2013 and GBD 2015 the search strategy was replicated to capture epidemiological studies published between 2008 and 2015.

Input data for the assessment of the total national number of infected people

Only studies that used countrywide surveys to estimate the national prevalence rates were included (or for China Province-wide surveys). Reason for choosing only national studies is that FBT shows a highly focal spatial distribution and local cross-sectional surveys would profoundly under- or overestimate true national prevalences. We decided not to model national and subnational together and get a coefficient on subnational, because there is not a one fits all relationship across the world. Infection is highly related to food habits and there are highly varying differences between national and sub-national prevalence rates. The final GBD 2015 dataset contained 29 prevalence studies from 17 countries. We used raw data from the selected studies as input for DisMod.

Prevalence intestinal fluke infection

Intestinal fluke is different from the other types of FBT, because there are several pathogens that fall under intestinal fluke infection. It can be caused by pathogens, such as Metagonimus spp., Echinostoma spp., Neodiplostomatidae.[6] When assessing the prevalence of intestinal fluke infection, we added the identified prevalence for each parasite species in order to obtain the overall prevalence of intestinal fluke infections. This approach may lead to a certain overestimation of the true prevalence, because people may be co-infected with more than one intestinal fluke species. There is no sufficient evidence about the proportion of co-infections, but the resulting overestimation of the true prevalence may be more than offset by the assumptions made in our previous modeling approach and the many challenges in generating the underlying epidemiological parameters (e.g., diagnostic inaccuracy in the detection of infections with the more than 50 intestinal fluke species). Also of note: the transmission source of intestinal fluke infections are species-specific and therefore vary. For instance, *Fasciolopsis buski* is usually transmitted by eating raw water plants with the infective parasite stage attached to the water plants, whereas Neodiplostomatidae are transmitted by eating undercooked and infested frogs, snakes, and tadpoles. Because of these different transmission pathways, the rate of co-infection might in fact be smaller than expected.

Input data to differentiate between asymptomatic and heavy infections

We estimated the proportion of heavily-infected among all infected in all available national and regional cross-sectional surveys. It is expected that heavy infection increases with age and there is data available on heavy infection by age group. We therefore decided to include age-dependent rates of heavy infection for clonorchiasis, opisthorchiasis, and intenstinal fluke infection. For (cerebral) paragonimiasis and fascioliasis there was not sufficient age-dependent data on high intensity FBT infection.

Modeling strategy

The GBD 2013 epidemiological modeling strategy for FBT made use DisMod-MR 2.0, a Bayesian meta-regression tool which built on GBD 2010's DisMod-MR. Updated characteristics of DisMod-MR 2.0 included the application of an offset lognormal rather than a negative binomial distribution. Dismod-MR 2.0 also executed its calculations in a cascade from global to country and (where applicable) from country to the subnational geographical unit, thereby

ensuring that estimates were consistent at all levels of the cascade. DisMod-MR 2.0 was used to estimate prevalence, by age, sex, year, and country for FBT.

We used a three-step process for the disease modeling of FBT. In the first step we used DisMod-MR to estimate assess the prevalence of FBT by age, sex, year, and country. In the second we differentiated between asymptomatic and heavy infections. MetaXL (a meta-analysis add in for Microsoft Excel) was used to estimate the proportion of heavy infected among all infected by age group for clonorchiasis, opisthorchiasis, and intenstinal fluke infection (see Table 3 and 4). These proportions were used to estimate the prevalence of heavy FBT infection. The third step consisted of deselecting countries that have no autochtonous case reports of FBT (input 34,000 references from literature review).

Table 3. Percentage of high intensity infection by age group and type of FBT (based on 8 FBT prevalence studies)

Age	Clonorchiasis		Op	Opisthorchiasis		Intestinal fluke infection			
category	Mean	Low	High	Mean	Low	High	Mean	Low	High
0-9	30%	17%	44%	10%	0%	29%	8%	3%	14%
10-19	15%	0%	43%	15%	0%	69%	11%	8%	14%
20-29	18%	10%	29%	16%	0%	52%	18%	15%	21%
30-39	17%	5%	34%	21%	0%	56%	22%	17%	28%
40-49	22%	13%	32%	28%	1%	68%	22%	13%	32%
50-59	18%	0%	49%	29%	0%	75%	17%	9%	28%
60+	32%	18%	47%	25%	0%	64%	15%	8%	23%

Table 4. Percentage of high intensity infection by type of FBT (based on 4 FBT prevalence studies)

_			
Type of FBT	Mean	Low	High
Paragonimiasis	23%	0%	59%
Fascioliasis	19%	3%	41%

Cerebral paragonimiasis

It was assumed that 0.8% of cerebral involvement in paragonimiasis. This proportion was used to estimate the prevalence of cerebral paragonimiasis. This proportion is based on one study. The study was performed in Paju, South Korea. This is an area with 6,738 inhabitants and according to the survey, it was estimated that 29.6% of all individuals would react to intradermal test (= an immunological reaction indicating previous or current contact to the parasite). 25% of all "positive reactors" may have eggs in their sputum (= active infection with the parasite currently present in the human host). If these rates are applied to the community as a whole, the number of patients with active paragonimiasis would be at least 498 (=6,738*0.296*0.250). Furthermore, four cases of cerebral paragonimiasis were found in this community. Therefore, four out of 498 individuals with active paragonimus infection suffered from cerebral infection (=0.80%; 95% confidence interval 0.019%-1.587%).

Severity splits and disability weights

For GBD 2015, FBT was not split into health states with different severities. The table below shows the GBD 2015 disability weights that were used to calculate the burden of FBT in YLDs.

Table 5. Disability weights that were used to calculate FBT YLDs

Sequelae	Severity description	Health state name	Disability weight
Asymptomatic clonorchiasis	Clonorchiasis, currently without symptoms	N/A	0.000 (0.000-0.000)
Hheavy clonorchiasis	Abdominal pain and nausea reported as moderate	Abdominopelvic problem, moderate	0.114 (0.078-0.159)
Asymptomatic opisthorchiasis	Opisthorchiasis, currently without symptoms	N/A	0.000 (0.000-0.000)
Heavy opisthorchiasis	Abdominal pain and nausea reported as moderate	Abdominopelvic problem, moderate	0.114 (0.078-0.159)
Asymptomatic fascioliasis	Fascioliasis, currently without symptoms	N/A	0.000 (0.000-0.000)
Heavy fascioliasis	Abdominal pain and nausea reported as moderate	Abdominopelvic problem, moderate	0.114 (0.078-0.159)
Asymptomatic intestinal fluke infection	Intestinal fluke infection, currently without symptoms	N/A	0.000 (0.000-0.000)
Heavy intestinal fluke infection	Abdominal pain and nausea reported as moderate	Abdominopelvic problem, moderate	0.114 (0.078-0.159)
Asymptomatic paragonimiasis	Paragonimiasis, currently without symptoms	N/A	0.000 (0.000-0.000)
Heavy paragonimiasis	Cough, fever and weight loss	Tuberculosis, not HIV infected	0.333 (0.224-0.454)
Cerebral paragonimiasis	Epilepsy due to cerebral paragonimiasis	Epilepsy, less severe (seizures < once per month)	0.263 (0.173-0.367)
		Epilepsy, severe (seizures >= once per month	0.552 (0.375-0.710)

Note. N/A: not applicable

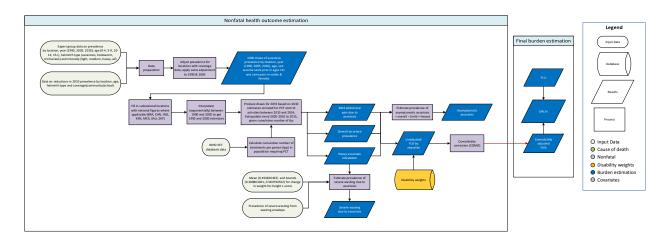
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Ascariasis SDG Capstone Appendix

Flowchart

Ascariasis



Case definition

Ascariasis is a helminth diseases caused by the parasitic roundworm, *Ascaris lumbricoides*. It is one of the three intestinal nematode infections (INI)/soil transmitted helminthiasis (STH) that we model in GBD. Diagnosis is made by microscopic exam of stool or by concentration procedures (recommended as eggs may be difficult to see). The ICD-10 codes for ascariasis are B77-B77.9.

Input data

Model inputs

Four different input data were used in the ascariasis nonfatal model. The first was prevalence data prepared by the expert group (EG) during GBD 2010 [1, 2]. They provided the data (mean, upper, lower) by location, year (1990, 2005, 2010), age (0-4, 5-9, 10-14, 15+ years), helminth type (ascariasis, hookworm disease, trichuriasis) and intensity of infection (light, medium, heavy, all). For the model, light infestation was not attributed any disability. The second data, also from the EG, was on reductions in prevalence in 2010, provided by location, age, helminth type, and coverage (community/school). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

	prevalence
Countries/subnationals	163
GBD world regions	16

The third input data was from the WHO PCT Databank [3]. This data was downloaded from the source website and represented 121 locations and six GBD world regions. The last input data was 1,000 draws

of wasting envelope prevalence among children under 5 years – the methods used to generate estimates of wasting prevalence are detailed elsewhere (part of risk factors documentation). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

	prevalence
Countries/subnationals	561
GBD world regions	21

Severity splits/Sequelae

The table below shows the list of sequelae due to ascariasis and the associated disability weights (DW). The sequelae were based on prevalence of medium and heavy infestation – medium infestation was assigned mild abdominopelvic problems; heavy infestation was assigned symptomatic worm infection; and light infestation was not attributed any disability.

Sequela	Lay description	DW
Mild abdominopelvic problems	has some pain in the belly that causes nausea but	0.011 (0.005-0.021)
	does not interfere with daily activities	
Heavy infestation	has cramping pain and a bloated feeling in the belly	0.027 (0.015-0.043)
Severe wasting	is extremely skinny and has no energy	0.128 (0.082-0.183)
Asymptomatic ascariasis	N/A	N/A

Modeling strategy

In the estimation of morbidity due to ascariasis, the EG data was first prepared by formatting the location names to be consistent with the GBD 2015 location names and applying the 2010 prevalence to 1990 and 2005 for sub-Saharan Africa countries — estimates for these two years were missing. This was followed by using the data on reductions in 2010 prevalence to adjust the prevalence for locations with coverage data. After this adjustment, only data for medium infection, heavy infection, and all infection was retained.

Using the mean prevalence and the upper and lower bounds of the mean provided by the EG, 1,000 draws of prevalence were generated. This was done by multiplying the mean estimates by the exponent of random draws from a normal distribution with mean = 0 and standard deviation = sd, where sd = abs(abs(ln(upper)-ln(lower))/(invnormal(0.975)*2). These draws were created for all GBD age-groups, assuming the same prevalence in ages 15+ and same prevalence in males and females. Since the draws were only at the national level, subnational locations were filled with national figures where applicable (Brazil, China, India, Kenya, Mexico, Saudi Arabia, and South Africa).

To get 1995 and 2000 estimates, exponential interpolation of estimates between 1990 and 2005 was performed. The draws for 2015 were produced based on 2010 estimates corrected for PCT control activities between 2010 and 2014 – this was done by extrapolating the 2005-2010 trend to 2015, given cumulative number of treatments per person calculated using data from the WHO PCT Databank [3]. The 2005-2010 trend was applied to all intensities of infection. Prevalence was assumed to be zero for

the countries with missing input data and also in children younger than 28 days. The resulting estimates were 1,000 draws of ascariasis prevalence by GBD location, year, age, sex, and intensity level (mild, heavy, overall infection). To estimate the prevalence of asymptomatic ascariasis, prevalence of mild and heavy infestation was subtracted from the overall ascariasis prevalence.

The final step in the modeling process was to estimate the prevalence of severe wasting due to ascariasis in age groups 28-364 days and 1-4 years. This was done separately using 1,000 draws of prevalence of heavy infestation due to ascariasis and the wasting envelope prevalence. The initial step in determining prevalence of severe wasting due to ascariasis was generating 1,000 draws of change in weight-for-height z-score per heavy prevalent case from a random normal distribution with mean = 0.493826493 and standard deviation = 0.04972834 (calculated from upper and lower bounds of the mean estimate). The mean, upper and lower bounds were provided by a GBD collaborator who calculated them based on a published article [4]. The prevalence of severe wasting due to ascariasis was then obtained as a function of change in weight-for-height z-score (z_change) such that prevalence = p_wasting_env = Phi(Phi_inv(p_wasting_env) - z_change*p), where p_wasting_env = wasting envelope prevalence, Phi_inv is the inverse standard normal cumulative distribution function (cdf), and p = prevalence of heavy ascariasis infestation.

Model evaluation was done by plotting prevalence of overall ascariasis and that of each sequelae against year for each location and age group. Maps of the global distribution of total ascariasis prevalence and prevalence of sequelae due to ascariasis were also assessed across time and age. Since we used the same data and model as that used in GBD 2013, we compared GBD 2015 estimates for each sequela with those from GBD 2013 for each country and age. As expected, our estimates were very similar to those from GBD 2013.

The only change made from GBD 2013 modeling strategy was the incorporation of updated data from the WHO PCT databank [3] in the correction of estimates for MDA activities.

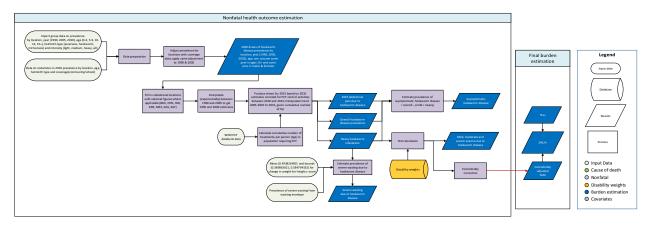
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Hookworm Disease SDG Capstone Appendix

Flowchart





Case definition

Hookworm disease is a helminth disease caused by the parasitic roundworms, *Ancylostoma duodenale* and *Necator americanus*. It is one of the three intestinal nematode infections (INI)/soil transmitted helminthiasis (STH) that we model in GBD. Diagnosis is made by a microscopic exam of stool or by concentration procedures (recommended as eggs may be difficult to see). The ICD-10 codes for hookworm disease are B76-B76.9.

Input data

Model inputs

Four different input data were used in the hookworm disease nonfatal model. The first was prevalence data prepared by the expert group (EG) during GBD 2010 [1, 2]. They provided the data (mean, upper, lower) by location, year (1990, 2005, 2010), age (0-4, 5-9, 10-14, 15+ years), helminth type (ascariasis, hookworm disease, trichuriasis) and intensity of infection (light, medium, heavy, all). For the model, light infestation was not attributed any disability. The second data, also from the EG, was on reductions in prevalence in 2010, provided by location, age, helminth type, and coverage (community/school). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

	prevalence
Countries/subnationals	163
GBD world regions	16

The third input data was from the WHO PCT Databank [3]. This data was downloaded from the source website and represented 121 locations and 6 GBD world regions. The last input data was 1,000 draws of wasting envelope prevalence among children under 5 years – the methods used to generate estimates of

wasting prevalence are detailed elsewhere (part of risk factors documentation). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

	prevalence
Countries/subnationals	561
GBD world regions	21

Severity splits/Sequelae

The table below shows the list of sequelae due to hookworm disease and the associated disability weights (DW). The sequelae were based on prevalence of medium and heavy infestation – medium infestation was assigned mild abdominopelvic problems; heavy infestation was assigned symptomatic worm infection; light infestation was not attributed any disability.

Sequela	Lay description	DW
Mild abdominopelvic problems	has some pain in the belly that causes nausea but	0.011 (0.005-0.021)
	does not interfere with daily activities	
Heavy infestation	has cramping pain and a bloated feeling in the belly	0.027 (0.015-0.044)
Severe wasting	is extremely skinny and has no energy	0.128 (0.082-0.183)
Asymptomatic hookworm	NA	NA
disease		
Mild anemia	feels slightly tired and weak at times, but this does	0.004 (0.001-0.008)
	not interfere with normal daily activities	
Moderate anemia	feels moderate fatigue, weakness, and shortness of	0.052 (0.034-0.076)
	breath after exercise, making daily activities more	
	difficult	
Severe anemia	feels very weak, tired and short of breath, and has	0.149 (0.101-0.210)
	problems with activities that require physical effort	
	or deep concentration	

Modeling strategy

In the estimation of morbidity due to hookworm disease, the EG data was first prepared by formatting the location names to be consistent with the GBD 2015 location names and applying the 2010 prevalence to 1990 and 2005 for sub-Saharan Africa countries — estimates for these two years were missing. This was followed by using the data on reductions in 2010 prevalence to adjust the prevalence for locations with coverage data. After this adjustment, only data for medium infection, heavy infection, and all infection was retained.

Using the mean prevalence and the upper and lower bounds of the mean provided by the EG, 1,000 draws of prevalence were generated. This was done by multiplying the mean estimates by the exponent of random draws from a normal distribution with mean = 0 and standard deviation = sd, where sd = abs(abs(ln(upper)-ln(lower))/(invnormal(0.975)*2). These draws were created for all GBD age-groups, assuming the same prevalence in ages 15+ and same prevalence in males and females. Since the draws

were only at the national level, subnational locations were filled with national figures where applicable (Brazil, China, India, Kenya, Mexico, Saudi Arabia, and South Africa).

To get 1995 and 2000 estimates, exponential interpolation of estimates between 1990 and 2005 was performed. The draws for 2015 were produced based on 2010 estimates corrected for PCT control activities between 2010 and 2014 – this was done by extrapolating the 2005-2010 trend to 2015, given cumulative number of treatments per person calculated using data from the WHO PCT Databank [3]. The 2005-2010 trend was applied to all intensities of infection. Prevalence was assumed to be zero for the countries with missing input data and also in children younger than 28 days. The resulting estimates were 1,000 draws of hookworm disease prevalence by GBD location, year, age, sex, and intensity level (mild, heavy, overall infection). To estimate the prevalence of asymptomatic hookworm disease, prevalence of mild and heavy infestation was subtracted from the overall hookworm disease prevalence.

The final step in the modeling process was to estimate the prevalence of severe wasting due to hookworm disease in age groups 28-364 days and 1-4years. This was done separately using 1,000 draws of prevalence of heavy infestation due to hookworm disease and the wasting envelope prevalence. The initial step in determining prevalence of severe wasting due to hookworm disease was generating 1,000 draws of change in weight-for-height z-score per heavy prevalent case from a random normal distribution with mean = 0.493826493 and standard deviation = 0.04972834 (calculated from upper and lower bounds of the mean estimate). The mean, upper and lower bounds were provided by a GBD collaborator who calculated them based on a published article [4]. The prevalence of severe wasting due to hookworm disease was then obtained as a function of change in weight-for-height z-score (z_change) such that prevalence = p_wasting_env - Phi(Phi_inv(p_wasting_env) - z_change*p), where p_wasting_env = wasting envelope prevalence, Phi_inv is the inverse standard normal cumulative distribution function (cdf) and p = prevalence of heavy hookworm infestation. The burden of anemia due to hookworm disease was estimated (see anemia documentation for details).

Model evaluation was done by plotting prevalence of overall hookworm disease and that of each sequelae against year for each location and age group. Maps of the global distribution of total hookworm disease prevalence and prevalence of sequelae due to hookworm disease were also assessed across time and age. Since we used the same data and model as that used in GBD 2013, we compared GBD 2015 estimates for each sequela with those from GBD 2013 for each country and age. As expected, our estimates were very similar to those from GBD 2013.

The only change made from GBD 2013 modeling strategy was the incorporation of updated data from the WHO PCT databank [3] in the correction of estimates for MDA activities.

References:

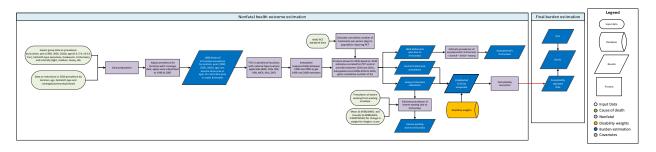
- 1. Brooker S, Pullan R, Smith J, and Hotez P. Chapter: Intestinal nematodes. Cluster D: Communicable Diseases, Neglected Tropical Diseases Group. Global Burden of Diseases, Injuries, and Risk Factors Study. 2011 (4 July). 1-24
- 2. Brooker S & Smith JL. Impact of hookworm infection and deworming on anaemia in non-pregnant populations: a systematic review. Tropical Medicine and International Health. 2010. 15,7,776-795
- 3. WHO PCT Databank. 2015; http://www.who.int/neglected_diseases/preventive_chemotherapy/sth/en/

4.	Hall A, Hewitt G, Tuffrey V, de Silva N. A review and meta-analysis of the impact of intestinal worms on child growth and nutrition. Maternal and Child Nutrition. 2008. 4. 118-236.		

Trichuriasis SDG Capstone Appendix

Flowchart

Trichuriasis



Case definitions

Trichuriasis is a helminth diseases caused by the parasitic roundworm *Trichuris trichiura*. It is one of the three intestinal nematode infections (INI)/soil transmitted helminthiasis (STH) that we model in GBD. Diagnosis is made by microscopic exam of stool or by concentration procedures (recommended as eggs may be difficult to see). The ICD-10 code for trichuriasis are B79.

Input data

Model inputs

Four different input data were used in the trichuriasis nonfatal model. The first was prevalence data prepared by the expert group (EG) during GBD 2010 [1, 2]. They provided the data (mean, upper, lower) by location, year (1990, 2005, 2010), age (0-4, 5-9, 10-14, 15+ years), helminth type (ascariasis, hookworm disease, trichuriasis) and intensity of infection (light, medium, heavy, all). For the model, light infestation was not attributed any disability. The second data, also from the EG, was on reductions in prevalence in 2010, provided by location, age, helminth type, and coverage (community/school). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

	prevalence
Countries/subnationals	163
GBD world regions	16

The third input data was from the WHO PCT Databank [3]. This data was downloaded from the source website and represented 121 locations and 6 GBD world regions. The last input data was 1,000 draws of wasting envelope prevalence among children under 5 years – the methods used to generate estimates of wasting prevalence are detailed elsewhere (part of risk factors documentation). The table below shows the number of countries or subnational units and GBD world regions represented in the data.

1
prevalence

Countries/subnationals	561
GBD world regions	21

Severity splits/Sequelae

The table below shows the list of sequelae due to trichuriasis and the associated disability weights (DW). The sequelae were based on prevalence of medium and heavy infestation – medium infestation was assigned mild abdominopelvic problems; heavy infestation was assigned symptomatic worm infection; light infestation was not attributed any disability.

Sequela	Lay description	DW (95% CI)
Mild abdominopelvic problems	has some pain in the belly that causes nausea but	0.011 (0.005-0.021)
	does not interfere with daily activities	
Heavy infestation	has cramping pain and a bloated feeling in the belly	0.027 (0.015-0.044)
Severe wasting	is extremely skinny and has no energy	0.128 (0.082-0.183)
Asymptomatic trichuriasis	N/A	N/A

Modeling strategy

In the estimation of morbidity due to trichuriasis, the EG data was first prepared by formatting the location names to be consistent with the GBD 2015 location names and applying the 2010 prevalence to 1990 and 2005 for sub-Saharan Africa countries — estimates for these two years were missing. This was followed by using the data on reductions in 2010 prevalence to adjust the prevalence for locations with coverage data. After this adjustment, only data for medium infection, heavy infection, and all infection was retained.

Using the mean prevalence and the upper and lower bounds of the mean provided by the EG, 1,000 draws of prevalence were generated. This was done by multiplying the mean estimates by the exponent of random draws from a normal distribution with mean = 0 and standard deviation = sd, where sd = abs(abs(ln(upper)-ln(lower))/(invnormal(0.975)*2). These draws were created for all GBD age-groups, assuming the same prevalence in ages 15+ and same prevalence in males and females. Since the draws were only at the national level, subnational locations were filled with national figures where applicable (Brazil, China, India, Kenya, Mexico, Saudi Arabia, and South Africa).

To get 1995 and 2000 estimates, exponential interpolation of estimates between 1990 and 2005 was performed. The draws for 2015 were produced based on 2010 estimates corrected for PCT control activities between 2010 and 2014 – this was done by extrapolating the 2005-2010 trend to 2015, given the cumulative number of treatments per person calculated using data from the WHO PCT Databank [3]. The 2005-2010 trend was applied to all intensities of infection. Prevalence was assumed to be zero for the countries with missing input data and also in children younger than 28 days. The resulting estimates were 1,000 draws of trichuriasis prevalence by GBD location, year, age, sex, and intensity level (mild, heavy, overall infection). To estimate the prevalence of asymptomatic trichuriasis, prevalence of mild and heavy infestation was subtracted from the overall trichuriasis prevalence.

The final step in the modeling process was to estimate the prevalence of severe wasting due to trichuriasis in age groups 28-364 days and 1-4 years. This was done separately using 1,000 draws of prevalence of heavy infestation due to trichuriasis and the wasting envelope prevalence. The initial step in determining prevalence of severe wasting due to trichuriasis was generating 1,000 draws of change in weight-for-height z-score per heavy prevalent case from a random normal distribution with mean = 0. 493826493 and standard deviation = 0.04972834 (calculated from upper and lower bounds of the mean estimate). The mean, upper and lower bounds were provided by a GBD collaborator who calculated them based on a published article [4]. The prevalence of severe wasting due to trichuriasis was then obtained as a function of change in weight-for-height z-score (z_change) such that prevalence = p_wasting_env - Phi(Phi_inv(p_wasting_env) - z_change*p), where p_wasting_env = wasting envelope prevalence, Phi_inv is the inverse standard normal cumulative distribution function (cdf), and p = prevalence of heavy trichuriasis infestation.

Model evaluation was done by plotting prevalence of overall trichuriasis and that of each sequelae against year for each location and age group. Maps of the global distribution of total trichuriasis prevalence and prevalence of sequelae due to trichuriasis were also assessed across time and age. Since we used the same data and model as that used in GBD 2013, we compared GBD 2015 estimates for each sequela with those from GBD 2013 for each country and age. As expected, our estimates were very similar to those from GBD 2013.

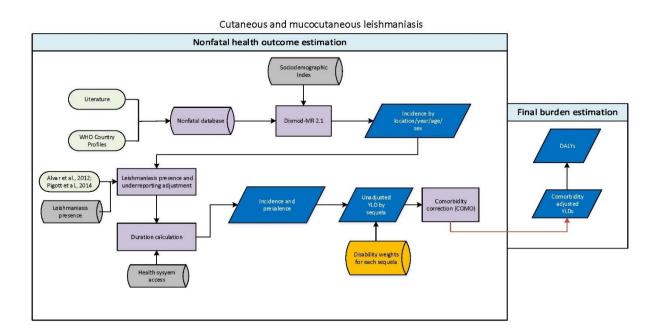
The only change made from GBD 2013 modeling strategy was the incorporation of updated data from the WHO PCT databank [3] in the correction of estimates for MDA activities.

References:

- 1. Brooker S, Pullan R, Smith J, and Hotez P. Chapter: Intestinal nematodes. Cluster D: Communicable Diseases, Neglected Tropical Diseases Group. Global Burden of Diseases, Injuries, and Risk Factors Study. 2011 (4 July). 1-24
- 2. Brooker S & Smith JL. Impact of hookworm infection and deworming on anaemia in non-pregnant populations: a systematic review. Tropical Medicine and International Health. 2010. 15,7,776-795
- 3. WHO PCT Databank. 2015; http://www.who.int/neglected_diseases/preventive_chemotherapy/sth/en/
- 4. Hall A, Hewitt G, Tuffrey V, de Silva N. A review and meta-analysis of the impact of intestinal worms on child growth and nutrition. Maternal and Child Nutrition. 2008. 4. 118-236.

Cutaneous and mucocutaneous leishmaniasis SDG Capstone Appendix

Flowchart



Input Data and Methodological Summary

Case Definition

Cutaneous leishmaniasis (CL) is the most common manifestation of disease caused by the *Leishmania* parasite, transmitted through the bite of phlebotomine sand flies. It causes the appearance of skin lesions, often beginning as papules or nodules and developing in/to ulcers, on parts of the body exposed to the bite of the sand fly. Mucocutaneous leishmaniasis (MCL) is a much more exceptional – and severe – presentation. Primarily isolated to Latin America, MCL infections can result in degradation of the mucous membranes, typically following an ulcerative sore from CL infection. Transmission varies by geographic region, as approximately 70 animal species have been identified as potential reservoir hosts of the parasite.

Input data

A systematic review of literature in the PubMed database was done on 17 July 2015 for prevalence and incidence data using the search term:

(leishmaniasis[Title/Abstract] OR "visceral leishmaniasis"[Title/Abstract] OR kalaazar[Title/Abstract] OR "black fever"[Title/Abstract] OR "dumdum fever"[Title/Abstract] OR "cutaneous leishmaniasis"[Title/Abstract] OR "mucosal leishmaniasis"[Title/Abstract] OR "mucocutanaeous leishmaniasis"[Title/Abstract] OR "oriental sore"[Title/Abstract] OR "tropical sore"[Title/Abstract] OR "chiclero ulcer"[Title/Abstract] OR "chiclero's ulcer"[Title/Abstract]) AND ("1990"[Date – Publication]: "2015"[Date – Publication]) AND (epidemiology OR prevalence OR incidence OR mortality OR fatality).

This search returned 3790 results, 258 of which passed title and abstract screening for CL and/or MCL. Upon full text review, 35 studies were selected – four reporting prevalence and 31 reporting incidence. For 66 countries, incidence from WHO country profiles were available.

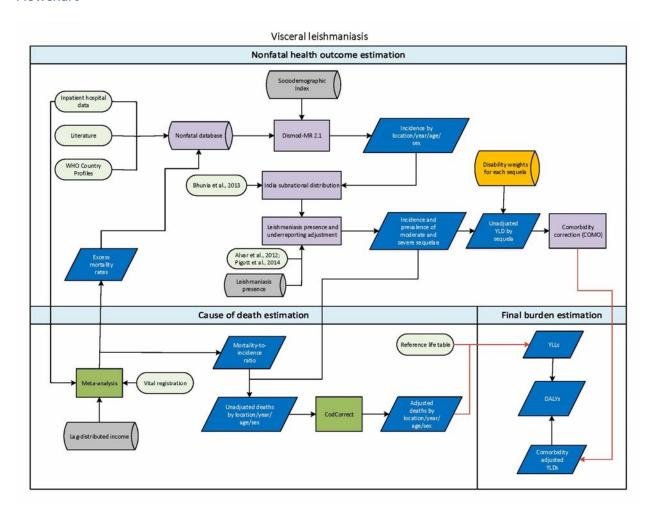
Modeling strategy

The minimal amount of prevalence data conflicted with incidence where available, and thus was excluded from the model. No study level covariates were used. The sociodemographic index (SDI) was used as a country level covariate on the incidence data, with a floor of exp(-1) – as to allow a degree of regional and subnational variation while constraining the predictive power such that predictions in hypo-endemic countries with low SDI values and no data would not be unduly high.

In order to control for DisMod fitting values to locations known to be devoid of CL, we replace estimates in these locations with zeros. Then for locations with confirmed CL presence, we apply an underreporting factor reported in Alvar et al. In order to distinguish prevalence of acute cases and those that endure lifelong disability, we used a normalized version of the health system access (HSA) covariate such that 47.6% of cases with poor access to healthcare – defined as (cases * (1 - norm(HSA)]) – would progress to the lifelong stage. All acute cases were assumed a six month duration.

Visceral Leishmaniasis SDG Capstone Appendix

Flowchart



Input Data and Methodological Summary

Case Definition

Visceral leishmaniasis (VL) is the most serious manifestation of disease caused by the *Leishmania* parasite, transmitted through the bite of phlebotomine sand flies. Those infected typically present with fever, weight loss, anemia, leukopenia, thrombocytopenia, and enlargement of the spleen and liver. If left untreated, it can be fatal. Transmission varies by geographic region, as approximately 70 animal species have been identified as potential reservoir hosts of the parasite. The ICD9 code related to visceral leishmaniasis is 085.0, and the ICD10 code is B55.0.

Input data

A systematic review of literature in the PubMed database was done on 17 July 2015 for prevalence and incidence data using the search term:

(leishmaniasis[Title/Abstract] OR "visceral leishmaniasis"[Title/Abstract] OR kala-azar[Title/Abstract] OR "black fever"[Title/Abstract] OR "dumdum fever"[Title/Abstract] OR "cutaneous leishmaniasis"[Title/Abstract] OR "mucosal leishmaniasis"[Title/Abstract] OR "mucocutanaeous leishmaniasis"[Title/Abstract] OR "oriental sore"[Title/Abstract] OR "tropical sore"[Title/Abstract] OR "chiclero ulcer"[Title/Abstract] OR "chiclero's ulcer"[Title/Abstract]) AND ("1990"[Date – Publication] : "2015"[Date – Publication]) AND (epidemiology OR prevalence OR incidence OR mortality OR fatality).

This search returned 3790 results, 274 of which passed title and abstract screening for VL. Upon full text review, 24 studies were selected – five reporting prevalence, 18 reporting exclusively incidence, and one reporting both.

Additionally, incidence data from WHO country profile reports were included for 50 countries, and inpatient hospital data from 75 unique locations was used in incidence estimation. In GBD 2013, we estimated country-year-specific MI ratios by running a linear regression of the logit of the MI ratio on the log of income per capita using vital registration and inpatient hospital data from Brazil and Spain, two countries in which we had both reliable mortality and incidence data at the national level. Those were used here. Assuming a duration of 3 months, excess mortality rates were calculated and used as well.

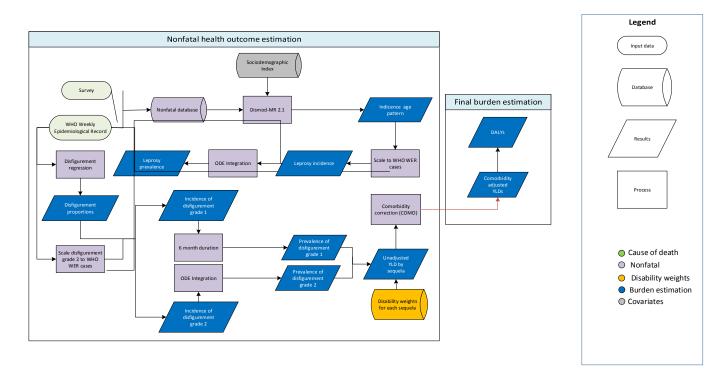
Modeling strategy

The minimal amount of prevalence data available conflicted with the relationship between incidence and excess mortality data, as well as the remission prior (set to 4 based on duration assumptions), and thus was excluded from the model. No study level covariates were used. The sociodemographic index (SDI) was used as a country level covariate on the incidence data, with a floor of exp(-1) – as to allow a degree of regional and subnational variation while constraining the predictive power such that predictions in hypo-endemic countries with low SDI values and no data would not be unduly high.

In order to best represent the documented distribution of VL in India, we used the national fit from the DisMod model and redistributed it amongst the Indian states based on data from Bhunia, et al. Further, in order to control for DisMod fitting values to locations known to be devoid of VL, we replace estimates in these locations with zeros. Then for locations with confirmed VL presence, we apply an underreporting factor reported in Alvar et al. Resultant incidence draws are then assumed to have a duration of three months, from which prevalence is calculated. Of those three months, three weeks are assumed to be spend with severe infection, and nine with moderate infection.

Leprosy Capstone Appendix

Flowchart



Input Data and Methodological Summary

Case Definition

Leprosy is a chronic bacterial infection caused by *Mycobacterium leprae*, primarily affecting the nervous system, skin, respiratory tract, and eyes. Transmission is facilitated through contact with fluid from the nose and mouth of an infected individual. Disability is associated with cases that develop to disfigurement, which is further subdivided into grade 1 and 2.

Sequela	Healthstate name	Healthstate description	Disability Weight (95% CI)	
Disfigurement level	Disfigurement,	has a slight, visible physical deformity that others	0.011 (0.005.0.031)	
1 due to leprosy	level 1	notice, which causes some worry and discomfort.	0.011 (0.005-0.021)	
		has a visible physical deformity that causes others		
Disfigurement level	Disfigurement,	to stare and comment. As a result, the person is	0.067 (0.044-0.096)	
2 due to leprosy	level 2	worried and has trouble sleeping and	0.007 (0.044-0.090)	
		concentrating.		

Input data

Due to the cyclical nature of systematic review for GBD causes, no data collection was scheduled for GBD 2015. As such, leprosy will be a priority for the next iteration of the study.

Modeling strategy

All available leprosy incidence data was modeled using Dismod-MR 2.1. Following this, the age-sex specific incidence rates are scaled to match the reported incidence in the WHO Weekly Epidemiological Record (WER). Where multiple years are available for a particular country, missing years are interpolated/extrapolated in order to maintain a complete time series. Countries for which only one year is present, other years are assumed to be zero. We them stream out prevalence of leprosy as a function of time by integrating the ordinary differential equation

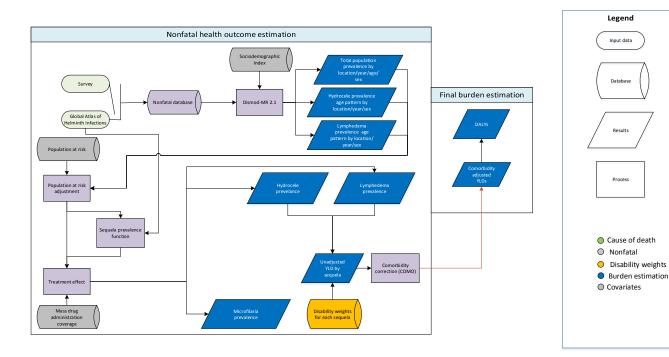
$$\frac{dp}{dt} = inc * (1 - prev) - prev * EMR$$

Where EMR = excess mortality rate, and is presumed to be zero for this condition.

In order to define the progression of leprosy incidence into disfigurement, we perform a generalized ordered logistic regression on WER data from Brazil, resulting in age-sex-specific probability of grade 2 disfigurement among incident cases of leprosy and grade 1 among incident cases of leprosy without grade 2 disfigurement. Then we use WER data to generate an envelope of grade 2, as was done for total leprosy. We are then able to take leprosy incidence, impose the age-sex-specific disfigurement proportions, and scale to the grade 2 envelope. From there, we apply the grade 1 proportions to the remaining leprosy cases that do not have grade 2 disability. For grade 1 prevalence, a duration of 6 months is applied. In deriving grade 2 prevalence, we use the same ODE integration function as described for total leprosy.

Lymphatic Filariasis Capstone Appendix

Flowchart



Input Data and Methodological Summary

Case Definition

Lymphatic filariasis (LF) is a neglected tropical disease spread in which threadlike nematodes invade the lymphatic system. The worms responsible – *Wuchereria bancrofti, Brugia malayi* and *Brugia timori* – are spread from human to human via mosquitoes. The most prominent clinical manifestations of LF are lymphedema (a swelling of the legs, also known in its more extreme manifestation as elephantiasis) and hydrocele (a collection of fluid in the sac around the testicles).

Input data

A systematic review of literature for GBD 2015 in the PubMed database was done on 10 August 2015 for prevalence and incidence data using the search ("lymphatic filariasis"[Title/Abstract] OR "filariasis"[Title/Abstract] OR "brugia"[Title/Abstract]) AND ("2013"[Date – Publication]: "2015"[Date – Publication]) AND (epidemiology OR incidence OR prevalence). This search returned 185 results, 121 of which passed title and abstract screening. Upon full text review, 37 studies were extracted for inclusion. Below is a summary of the geographic distribution of the data used.

Sequela	Data points	Regions	Countries	Subnational units
Prevalence of detectable				
microfilaria	1,552	10	40	28

Lymphedema due to lymphatic filariasis	511	10	25	15
Hydrocele due to lymphatic filariasis	265	8	22	12

Modeling strategy

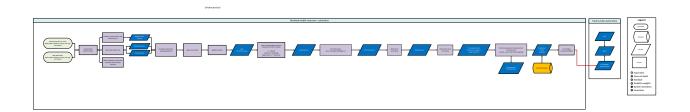
Data on prevalence of microfilaria is modeled using Dismod-MR 2.1. Due to the fact that data is collected in endemic locations, we then scaled according to at-risk population in order to attain nationally representative values. We then use non-linear regression to estimate the reduction of microfilaria as a function of treatments per person. Using mass drug administration (MDA) coverage, we are then able to reduce the total estimated prevalence by exposing modeled total prevalence according to treatment efficacy.

For lymphedema and hydrocele, we incorporate survey data from the Global LF Atlas in a non-linear error-in-variables regression that determines the prevalence of lymphedema and hydrocele as functions of microfilaria prevalence, which is then applied to the total microfilaria Dismod model in order to attain an envelope of cases by location-year. Separately, all available prevalence data for these conditions is modeled in Dismod in order to determine an age-sex pattern.

In the estimation of lymphedema and hydrocele prevalence, we perform the same population at-risk correction that is done on microfilaria prevalence. For hydrocele prevalence after treatment, we take the value before MDA rollout in 2000 and reduce that by the same treatment efficacy function described for microfilaria prevalence, using dosage-reduction data specific to hydrocele along with the location-year specific MDA coverage. For lymphedema, we assume no new cases appear among treated individuals. As such, we reduce lymphedema prevalence in post-treatment years in accordance with MDA coverage.

Onchocerciasis SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Case definition

Onchocerciasis, also known as river blindness, is a helminth disease caused by a parasitic worm, *Onchocerca volvulus*, transmitted by repeated bites by *Similium* blackflies. Diagnosis is made by different methods including skin snip biopsy to identify larvae, surgical removal of nodules and exam for adult worms, slit lamp exam of anterior part of the eye where larvae or lesions caused by them are visible, and antibody tests (mostly useful to visitors to areas with parasites). The ICD-10 code for onchocerciasis is B73.

Input data

Model inputs

To model nonfatal outcomes due to onchocerciasis, prevalence data prepared by the expert group (EG) during GBD 2013 was used. These included 1,000 draws of infection and morbidity (visual impairment, blindness, and skin conditions) cases with confidence intervals for years 1990, 1995, 2000, 2005, 2010, and 2013 categorized by country, age, and sex. Details of materials and methods used by the EG to generate draws can be found elsewhere [1-5]. The data only represented African countries included in the African Programme for Onchocerciasis Control (APOC) – Angola, Burundi, Cameroon, Central African Republic, Chad, Congo, Democratic Republic of Congo, Ethiopia, Equatorial Guinea, Liberia, Malawi, Nigeria, Sudan, Tanzania, and Uganda – and the Onchocerciasis Control Programme (OCP) – Benin, Burkina Faso, Côte d'Ivoire, Ghana, Guinea Bissau, Guinea, Mali, Niger, Senegal, Sierra Leone, and Togo. The table below shows (by program) the number of countries and GBD world regions represented.

	APOC	ОСР
Countries/subnationals	15	11
GBD world regions	3	1

We did not update the literature review for these data in GBD 2015. Updates to systematic reviews are performed on an ongoing schedule across all GBD causes, and an update for onchocerciasis will be performed in the next 1-2 iterations.

Severity splits/Sequelae

The table below shows the list of sequelae due to onchocerciasis, their lay descriptions, and the associated disability weights (DW).

Sequela	Lay description	DW
Moderate vision	has vision problems that make it difficult to	0.031 (0.019-0.049)
impairment	recognize faces or objects across a room	
Severe vision	has severe vision loss, which causes difficulty in	0.184 (0.125-0.258)
impairment	daily activities, some emotional impact (for	
	example worry), and some difficulty going	
	outside the home without assistance	
Blindness	is completely blind, which causes great difficulty	0.187 (0.124-0.260)
	in some daily activities, worry and anxiety, and	
	great difficulty going outside the home without	
	assistance	
Mild skin disease	has a slight, visible physical deformity that is	0.027 (0.015-0.042)
	sometimes sore or itchy. Others notice the	
	deformity, which causes some worry and	
	discomfort	
Mild skin disease	has a slight, visible physical deformity that others	0.011 (0.005-0.021)
without itch	notice, which causes some worry and discomfort	
Moderate skin	has a visible physical deformity that is sore and	0.188 (0.124-0.267)
disease	itchy. Other people stare and comment, which	
	causes the person to worry. The person has	
	trouble sleeping and concentrating	
Severe skin	has a visible physical deformity that is sore and	0.188 (0.124-0.267)
disease	itchy. Other people stare and comment, which	
	causes the person to worry. The person has	
	trouble sleeping and concentrating	
Severe skin	has an obvious physical deformity that makes	0.405 (0.275-0.546)
disease without	others uncomfortable, which causes the person	
itch	to avoid social contact, feel worried, sleep poorly,	
	and think about suicide	
Asymptomatic	NA	NA
onchocerciasis		

Modeling strategy

The nonfatal modeling for onchocerciasis included two major steps. In the first step, GBD 2013 prevalence was extrapolated to obtain GBD 2015 estimates. Acute skin disease level 2 and chronic skin disease level 2 were then summed up to create the "moderate skin disease" sequela. Within each of the OCP draws the number of cases with visual impairment and blindness was multiplied by a random value (the exponent of a normally distributed variable with mean zero and standard deviation 0.1) – this was

done to add some uncertainty to these estimates. Within each draw, the same randomly drawn value was applied to all country-year-age-sex. The other sequelae already had uncertainty quantified and were used as provided by the EG. Visual impairment was split into moderate and severe vision impairment by first multiplying the visual impairment estimates by a random value (from a normal distribution with mean 0.84 and standard deviation 0.0031) to generate moderate vision impairment, and then subtracting the resulting estimates from visual impairment to obtain estimates of severe vision impairment. Prevalence of sequelae was calculated by dividing the cases by the population.

The second step in modeling morbidity due to onchocerciasis was the adjustment of uncertainty in 1) conversion of nodule prevalence to microfilaria prevalence and 2) effects of mass treatment. To adjust for uncertainty in nodule to mf prevalence, the final draws from the first step were logit transformed, and then for OCP countries, we added uncertainty from a random value drawn from a normal distribution to the transformed estimates. The resulting estimates were then normalized and scaled using estimates published elsewhere [1]. To adjust for uncertainty due to MDA, the year when MDA with ivermectin started was set to 1990 and adjusted for some countries (1997 for Malawi; 1998 for Chad, Niger, and Tanzania; 1999 for Cameroon, Central African Republic, Equitorial Guinea, Liberia, Nigeria, and Uganda; 2001 for Congo, Ethiopia, and DRC; 2005 for Angola, Burundi, and South Sudan). Time trend uncertainty was then multiplied by the normalized prevalence estimates and then final prevalence was obtained by re-expanding the scaled normalized draws and adjusting the scale back from logit scale.

To estimate the prevalence of asymptomatic onchocerciasis, prevalence of morbidity (vision loss, blindness and skin conditions) was subtracted from the overall onchocerciasis prevalence – moderate vision impairment, severe vision impairment and blindness estimates were each multiplied by a factor of 8/33 before subtraction to account for cases that have concurring symptoms.

Model evaluation was done by separately assessing plots of time trends of prevalence across locations and age for each sequela. In addition, maps of the global distribution of total onchocerciasis prevalence and prevalence of sequelae due to onchocerciasis were also assessed across time. Since the modeling strategy was the same as that applied in GBD 2013, we compared our estimates with those from GBD 2013, and the correlation between the two was about 1.

We have made no substantive changes in the modeling strategy from GBD 2013.

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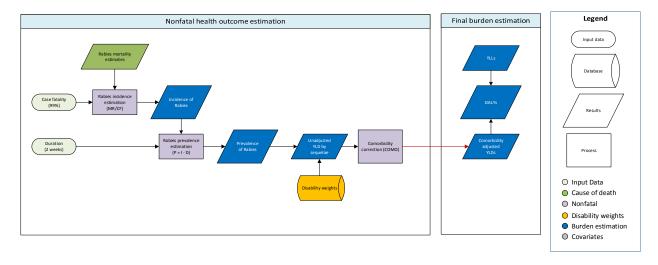
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Rabies SDG Capstone Appendix

Flowchart

Rabies



Input Data & Methodological Summary

Case definition

Rabies is a fatal viral infection, transmitted by animal bites. Without prophylactic vaccination the disease is almost universally fatal. The disease has a long incubation period (1-3 months), and early intervention with prophylactic vaccination is nearly 100% effective in preventing symptomatic disease. We model symptomatic infections, not including those infections in which intervention prevented the onset of symptomatic disease, corresponding to the ICD10 code A82.

Input data

Model inputs

As we derive our estimate of cases from our estimate of deaths, there are no incidence data used in the model. For GBD 2015, we modeled rabies mortality using all available data in the cause of death database. Data points were outliered if they reported an improbable number of rabies deaths (e.g., zero rabies deaths in a hyperendemic country) or if their inclusion in the model yielded distorted trends. In some cases multiple data sources for the same location differed dramatically both in their quality and reported rabies mortality (e.g., a verbal autopsy and vital registration source). In these cases the lower-quality data source was outliered.

Modeling strategy

We derive estimates of the number of symptomatic rabies infections (i.e., those not averted through prophylactic vaccination) based on rabies mortality estimates, assuming 99% case fatality. All cases are assumed to be severe.

We modeled rabies mortality using a two-model hybrid approach 1) a global CODEm model of all locations, using all data in the CoD database; and 2) a CODEm model restricted to data-rich countries. We have made two substantive changes in the modeling strategy from GBD 2013. First, we have changed from a single global model to the hybrid global/data rich model approach. Second, we conducted an exploratory analysis to determine the most predictive covariates for rabies and have updated the covariates used in the CODEm model accordingly.

Sequela description and DW

Sequela	Description	Disability Weight (95% CI)
Severe	Has a high fever and pain, and feels very weak,	0.133
	which causes great difficulty with daily activities.	(0.088-0.19)

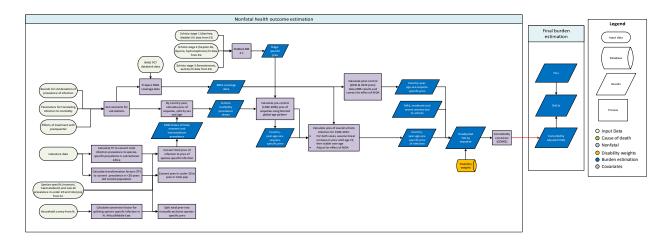
Changes from GBD 2013 to GBD 2015

We have made no substantive changes in the modeling strategy from GBD 2013.

Schistosomiasis SDG Capstone Appendix

Flowchart

Schistosomiasis



Input Data & Methodological Summary

Case definition

Schistosomiasis, also known as bilharzia or "snail fever," is a helminth disease caused by infection with five species of the parasite *Schistosoma* namely, *S. mansoni*, *S. japonicum*, *S. haematobium*, *S. mekongi*, and *S. intercalatuma*. The first three species cause the most infection and the last two rarely cause disease. Diagnosis is made by microscopic exam of stool or urine for parasite eggs. For less advanced infections, serologic techniques are used. The ICD-10 codes for schistosomiasis are B565-B65.9.

Input data

Model inputs

To model nonfatal outcomes due to schistosomiasis, data prepared by the expert group (EG) during GBD 2010 was used. To prepare the data, they compiled most recently published estimates of species-specific infection prevalence calculated using Bayesian geo-statistical models based on environmental predictors. Details of materials and methods used by the EG are referenced elsewhere [1-6]. Using citations compiled by the EG, a list of eight possible clinical sequelae and anemia sequelae were defined (mild infection, mild diarrhea, hematemesis (vomiting blood), hepatomegaly, ascites (build-up of fluid in the peritoneal cavity), dysuria (painful urination), bladder pathology, hydronephrosis (swelling of kidney due to build-up of urine in the kidney), mild anemia, moderate anemia, severe anemia).

Stage-specific prevalence data produced by the EG was also used as inputs for three separate single-parameter models in DisMod (stage 1: acute symptoms - diarrhea, active/mild schisto; stage 2: semi-acute/chronic symptoms - hepatomegaly, dysuria, and hydronephrosis; stage 3: long term chronic disease - haematemesis, ascites, and bladder pathology). The table below shows (by stage) the number

of literature studies included in GBD 2015, as well as the number of countries or subnational units and GBD world regions represented.

	Stage 1	Stage 2	Stage 3
Studies	76	40	10
Countries/subnationals	34	20	7
GBD world regions	8	8	5

Literature data on prevalence of infection by species (mansoni and haematobium) in under 20 and in the total population was also used [3-7] – for Brazil, this data was split into subnational using data from the Schostosomiasis Control Program (PCE) [8]. The table below shows the number of sources included, and the number of countries or subnational units and GBD world regions represented.

	prevalence
Sources	5
Countries/subnationals	94
GBD world regions	10

Additional literature sources were used to inform morbidity prevalence estimation. These are highlighted in modeling strategy section of this document.

We did not update the literature review for these data in GBD 2015. Updates to systematic reviews are performed on an ongoing schedule across all GBD causes, an update for schistosomiasis will be performed in the next 1-2 iterations.

Severity splits/Sequelae

The table below shows the list of clinical sequelae [8] (including mild, moderate, and severe anemia) due to schistosomiasis, their lay descriptions, and the associated disease stage and disability weights.

Clinical sequela	Lay description	Disease	Disability weights
		stage	
Mild infection	has a low fever and mild discomfort , but no	1	0.006 (0.002-0.012)
	difficulty with daily activities		
Mild diarrhea		1	0.056
Hepatomegaly	has some pain in the belly that causes nausea but	2	0.011 (0.005-0.021)
	does not interfere with daily activities		
Dysuria	has some pain in the belly that causes nausea but	2	0.011 (0.005-0.021)
	does not interfere with daily activities		
Hydronephrosis	has some pain in the belly that causes nausea but	2	0.011 (0.005-0.021)
	does not interfere with daily activities		
Haematemesis	vomits blood and feels nauseous	3	0.325 (0.209-0.463)
Ascites	has pain in the belly and feels nauseous. The	3	0.114 (0.078-0.159)
	person has difficulties with daily activities		

Bladder pathology	has some pain in the belly that causes nausea but	3	0.011 (0.005-0.021)
	does not interfere with daily activities		
Mild anemia	feels slightly tired and weak at times, but this	NA	0.004 (0.001-0.008)
	does not interfere with normal daily activities		
Moderate anemia	feels moderate fatigue, weakness, and shortness	NA	0.052 (0.034-0.076)
	of breath after exercise, making daily activities		
	more difficult		
Severe anemia	feels very weak, tired and short of breath, and	NA	0.149 (0.101-0.210)
	has problems with activities that require physical		
	effort or deep concentration		

Modeling strategy

The morbidity model for schistosomiasis involved six main steps. First, constants were set for calculations on 1) bounds for standard deviation of dispersion of logit prevalence of infection within countries (low=0.6, high=0.8), 2) parameters (a, b, c) for translating infection (x) to morbidity (y): $y = (a + bx^c)/(1 + bx^c) - a$ [9-11], and 3) effects (mean, low, high) of treatment with praziquantel (PZQ) [12, 13]. Next, the conversion factor for splitting species-specific (mansoni, haematobium) infection in North Africa and Middle East was calculated, informed by literature data [14]. In the third step, literature data [4, 5] was used to calculate transformation factors for converting 1) prevalence in individuals under 20 years of age to total population, and 2) total infection prevalence to species-specific prevalence in sub-Saharan Africa.

Next, infection data was prepared by 1) splitting total prevalence in North Africa/Middle East into mutually exclusive species-specific prevalence, 2) converting prevalence in under 20 to prevalence in the total population, and 3) converting total prevalence of infection in sub-Saharan Africa to prevalence of species-specific infection. To generate 1,000 draws of prevalence, the prevalence estimates were scaled using values drawn from a standard normal distribution. Resulting estimates were split by age and sex, based on general pattern (infection: linear increase until age 15, then stable) or DisMod global age pattern for the three different stages of disease, each with a different pattern. All stage-specific DisMod models were run as proportion models and included year as a country-level covariate.

The final step was the prediction of post-control prevalence of infection and morbidity, adjusted for the impact of treatment, given cumulative number of PCT treatments per person at risk (draws of expected reductions in overall infection prevalence were obtained using data from the WHO PCT Databank [15]) and estimated species-specific efficacy, as reported in literature. For reversible sequelae, it was assumed that treatment effects are the same as for infection. For irreversible symptoms (advanced hepatic disease, ascites, hematemesis), it was assumed that incidence among the treated fraction of the population is zero and 10% of individuals with these conditions die each year due their schisto-related sequelae. The burden of anemia due to schistosomiasis was estimated (see anemia documentation for details).

Model evaluation was done by separately assessing the fit of the three stage-specific DisMod models and checking the final estimates produced after age-sex splits. Plots of time trends of prevalence across locations, and age were used to evaluate the results. In addition, maps of the global distribution of total schistosomiasis prevalence and prevalence of sequelae due to schistosomiasis were also assessed across time.

The main change made from GBD 2013 was splitting Brazil national data into subnational using data from the Schistosomiasis Control program [8]. In addition, newly updated data from the WHO PCT databank was downloaded and used in the model.

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Non-communicable Diseases (NCDs) SDG Capstone Appendix

cardiovascular diseases, cancer except for liver cancer and non-melanom skin cancer, liver cancer, rheumatic heart disease, ischemic heart disease, cerebrovascular disease, ischemic stroke, hemorrhagic stroke, hypertensive heart disease, cardiomyopathy and myocarditis, other cardiovascular and circulatory diseases, chronic respiratory diseases, other chronic respiratory diseases, diabetes mellitus

Indicator definition

This modeling strategy encompasses the indicators associated with non-communicable disease mortality (3.3.5).

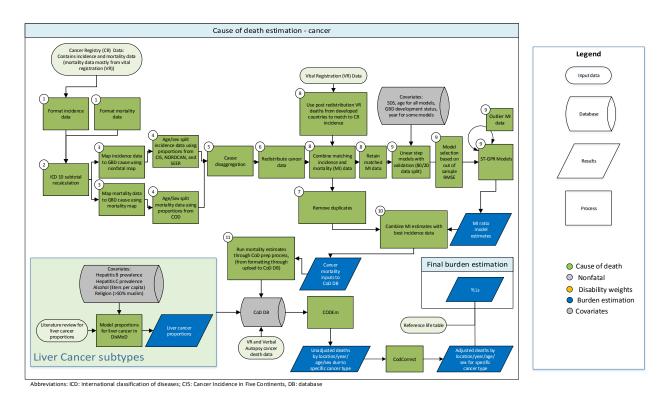
Indicator 3.4.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.4, by 2030, reduce by one third premature mortality from NCDs through prevention and treatment and promote mental health and well-being, is measured using SDG Indicator 3.4.1, deaths due to cardiovascular disease, cancer, diabetes, and chronic respiratory disease among populations aged 30 to 70 per 100,000.

Cancers SDG Capstone Appendix

Input data and methodological summary for all cancers except for liver cancer and non-melanoma skin cancer

Flowchart



Input Data & Methodological Summary

Input Data

The Cause of Death (COD) database contains multiple sources of cancer mortality data. These sources include vital registration, verbal autopsy, and cancer registry data. The cancer registry mortality estimates that are uploaded into the COD database stem from cancer registry incidence data that have been transformed to mortality estimates through the use of mortality-to-incidence (MI) ratios.

Data seeking processes

Cancer mortality data in the cause of death database other than cancer registry data Sources for cancer mortality data other than cancer registry data are described in the COD database description (Part 2).

Cancer registry data

All cancer registry data used for GBD 2010 were also included for GBD 2013, and the majority of these data were also used for GBD 2015 unless superseded by newer data (see step 7 in flow chart and

below). Most new data were added based on availability and collaborator recommendation. Some new data were acquired and approved for GBD 2013 but were received after the deadline for adding new data to GBD 2013. More than half (56%) of the final incidence data and 35% of the final MI model input data came from the Cancer Incidence in Five Continents series (CI5).^{1–10}

Cancer registry data were most often downloaded from a publicly available webpage or provided by collaborators. Most cancer registries only report cancer incidence. However, if a cancer registry also reported cancer mortality, mortality data were also extracted from the source to be used in the mortality-to-incidence estimation.

Inclusion and exclusion criteria

Only population-based cancer registries were included, and only those that included all cancers (no specialty registries), data for all age groups, and data for both sexes. Pathology-based cancer registries were included if they had a defined population. Hospital-based cancer registries were not included.

Cancer registry data were excluded from either the final incidence data input or the MI model input if a more detailed source (e.g., providing more detailed age or diagnostic groups) was available for the same population. Preference was given to registries with national coverage over those with only local coverage, except those from countries where the GBD study provides subnational estimates; thus some data were excluded because newly acquired national registry data could replace a regionally representative predecessor.

Data were excluded from the final incidence data input if the coverage population was unknown.

Bias of categories of input data

Cancer registry data can be biased in multiple ways. A high proportion of ill-defined cancer cases in the registry data requires redistribution of these cases to other cancers, which introduces a potential for bias. Changes between coding systems can lead to artificial differences in disease estimates; however, we adjust for this bias by mapping the different coding systems to the GBD causes. Underreporting of cancers that require advanced diagnostic techniques (e.g., leukemia and brain, pancreatic, and liver cancer) can be an issue in cancer registries from low-income countries. On the other hand, misclassification of metastatic sites as primary cancer can lead to overestimation of cancer sites that are common sites for metastases like brain or liver. Since many cancer registries are located in urban areas, the representativeness of the registry for the general population can also be problematic. The accuracy of mortality data reported in cancer registries usually depends on the quality of the vital registration system. If the vital registration system is incomplete or of poor quality, the mortality-to-incidence ratio can be biased to lower ratios.

Methods

Steps of analysis and data transformation processes

Cancer registry data went through multiple processing steps before integration with the COD database. First, the original data were transformed into standardized files, which included standardization of format, categorization, and registry names (#1 in flowchart).

Second, some cancer registries report individual codes as well as aggregated totals [e.g., C18, C19, and C20 are reported individually but the aggregated group of C18-C20 (colorectal cancer) is also reported in the registry data]. The data processing step "subtotal recalculation" (#2 in flowchart) verifies these totals and subtracts the values of any individual codes from the aggregates.

In the third step (#3 in the flowchart), cancer registry incidence data and cancer registry mortality data are mapped to GBD causes. A different map is used for incidence and for mortality data because of the assumption that there are no deaths for certain cancers. One example is basal cell carcinoma of the skin. In the cancer registry incidence data, basal cell carcinoma is mapped to non-melanoma skin cancer (basal cell carcinoma). However, if basal cell skin cancer is recorded in the cancer registry mortality data, the deaths are instead mapped to non-melanoma skin cancer (squamous cell carcinoma) under the assumption that they were indeed misclassified squamous cell skin cancers. Other examples are benign or in situ neoplasms. Benign or in situ neoplasms found in the cancer registry incidence dataset were simply dropped from that dataset. The same neoplasms reported in a cancer registry mortality dataset were mapped to the respective invasive cancer (e.g., melanoma in situ in the cancer registry mortality dataset was mapped to melanoma).

In the fourth data processing step (#4 in the flowchart) cancer registry data were standardized to the GBD age groups. Age-specific incidence rates were generated using CI5, SEER, and NORDCAN data, while age-specific mortality rates were generated from the CoD data through a method described in Part 2. Age-specific weights were then generated by applying the age-specific rates to a given registry population that required age-splitting to produce the expected number of cases/deaths for that registry by age. The expected number of cases/deaths for each sex, age, and cancer were then normalized to 1, creating final, age-specific proportions. These proportions were then applied to the total number of cases/deaths by sex and cancer to get the age-specific number of cases/deaths.

In the rare case that the cancer registry only contained data for both sexes combined, the now-age-specific cases/deaths were split and re-assigned to separate sexes using the same weights that are used for the age-splitting process. Starting from the expected number of deaths, proportions were generated by sex for each age (e.g., if for ages 15 to 19 years old there are six expected deaths for males and four expected deaths for females, then 60% of the combined-sex deaths for ages 15-19 years would be assigned to males and the remaining 40% would be assigned to females).

In the fifth step (#5 in the flowchart) data for cause entries that are aggregates of GBD causes were redistributed. Examples of these aggregated causes include some registries reporting ICD10 codes CO0-C14 together as, "lip, oral cavity, and pharyngeal cancer." These groups were broken down into subcauses that could be mapped to single GBD causes. In this example, those include lip and oral cavity cancer (C00-C08), nasopharyngeal cancer (C11), cancer of other parts of the pharynx (C09-C10, C12-C13), and "Malignant neoplasm of other and ill-defined sites in the lip, oral cavity, and pharynx" (C14). To redistribute the data, weights were created using the same "rate-applied-to-population" method employed in age-sex splitting (see step four above). For the undefined code (C14 in the example) an "average all cancer" weight was used, which was generated by adding all cases from SEER/NORDCAN/CI5 and dividing the total by the combined population. Then, proportions were

generated by sub-cause for each aggregate cause as in the sex-splitting example above (see step four). The total number of cases from the aggregated group (COO-C14) was then recalculated for each subgroup and the undefined code (C14). C14 was then redistributed as a "garbage code" in step six. Distinct proportions were used for C44 (non-melanoma skin cancer) and C46 (Kaposi's sarcoma). Population data were not used to redistribute data for these ICD codes. Non-melanoma skin cancer processing is described under section "Input data and methodological summary for non-melanoma skin cancer (squamous-cell carcinoma)." C46 entries were redistributed as "other cancer," HIV, and C80 (other and unknown cancers) using proportions described in Part 2.

In the sixth step (#6 in the flowchart) unspecified codes ("garbage codes") were redistributed. Redistribution of cancer registry incidence and mortality data mirrored the process of the redistribution used in the cause of death database (Part 2).

In the seventh step (#7 in the flowchart) duplicate or redundant sources were removed from the processed cancer registry dataset. Duplicate sources were present if, for example, the cancer registry was part of the CI5 dataset but we also had data from the registry directly. Redundancies occurred and were removed as described in "Inclusion and Exclusion Criteria," where more detailed data were available, or when national registry data could replace regionally representative data. From here, two parallel selection processes were run to generate input data for the MI models and to generate incidence for final mortality estimation. Higher priority was given to registry data from the most standardized source when creating the final incidence input (generally CI5 data), whereas preference was given to registry data from sources with matching mortality and incidence for the MI model input (in order to reduce confounding due to oppositional input biases when matching the two data types).

In the eighth step (#8 in the flowchart) the processed incidence and mortality data from cancer registries were matched by cancer, age, sex, year, and location to generate MI ratios. Because some cancer registries do not report mortality data – even though high-quality vital registration system data are available to the registry's coverage area – processed vital registration mortality data from the CoD database were matched to the registry's incidence data for some countries. This was the case for certain registries in the following countries: Australia, Austria, Belgium, Bulgaria, Denmark, Estonia, Finland, Hungary, Iceland, Ireland, New Zealand, Norway, South Korea, and Switzerland.

The ninth step involved creating and selecting the MI models. All models were run separately by cancer, and the best model was selected from the following list (see Table below).

```
1. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \epsilon_{c,a,s,t}

2. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \beta_4t + \theta_c + \epsilon_{c,a,s,t}

3. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \beta_4DS + \theta_c + \epsilon_{c,a,s,t}

4. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \beta_4DS + \beta_5t + \theta_c + \epsilon_{c,a,s,t}

5. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \lambda_{SR}(SDS_{c,t}) + \beta_4t + \epsilon_{c,a,s,t}

6. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \lambda_{SR}(SDS_{c,t}) + \epsilon_{c,a,s,t}

7. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \lambda_{SR}(SDS_{c,t}) + \beta_4DS + \epsilon_{c,a,s,t}

8. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \lambda_{SR}(SDS_{c,t}) + \beta_4DS + \epsilon_{c,a,s,t}

8. \log \operatorname{it}\left(MI\ ratio_{c,a,s,t}\right) = \alpha + \beta_1SDS_{c,t} + \sum_a^A\beta_2I_a + \beta_3I_s + \theta_c + \lambda_{SR}(SDS_{c,t}) + \beta_4DS + \epsilon_{c,a,s,t}
```

c: country; a: age group; t: time (years); s: sex

I: indicator variable

DS: binary variable for development status θ_c : random effect by country (intercept)

 $\lambda_{SR}(SDSc,t)$: random effect modifier between SDI and super-region (slope)

 $\epsilon_{c,a,s,t}$: error term

Table: MI models

All models were tested at multiple stages before creating the final model output. Models were initiated with an SDI covariate (Socio-Demographic Index) and first tested using the complete input dataset (Part 4). If after that initial test the SDI covariate's coefficient was negative (as expected), the next step was to outlier any data point for which the residual from the prediction was greater than three times the MAD from the mean residual. Next, data were marked as outliers due to a random effect criterion: if the country-level random effect for a lower-income country was lower than the random effect for the USA, all data points for that country were marked as outliers. This process was run iteratively until all lower-income countries had country-level random effects greater than that of the USA. All data points marked outliers were dropped from the final dataset, and that dataset was used to create the final model predictions.

If the SDI coefficient was found to be positive (unexpected) after the initial SDI test, it was assumed to indicate an excess of unrealistic data in the input dataset. To remove these unrealistic data, SDI was temporarily removed from the model formula. The model proceeded as above without SDI until all unrealistic data points were removed and the SDI coefficient was found to be negative. Unrealistic data were marked as outliers using the same residual MAD and random effect methodology described above. Once SDI was established as negative (expected) the model proceeded as usual.

To select the best model formula, the initial model results were tested by comparing mean MI predictions and the mean root-mean-squared error (RMSE) values of 10 random samples of 80%/20% splits from the input dataset. Mean MI predictions were compared between developing and developed countries. Models were eliminated if the mean MI for developing countries was lower than the mean MI ratio for developed countries. For RMSE testing, the dataset was split into an 80% dataset for model development and a 20% dataset for model testing. The process was repeated 10 times. The best model for each cancer was selected based on the lowest mean out-of-sample RMSE from those models remaining after checking the mean MI. The table below contains the final models selected for each cancer.

Cancer	Final model number (see numbering above)
Ovarian cancer	1
Uterine cancer	1
Gallbladder cancer	1
Kidney cancer	1

Larynx cancer	1
Acute lymphoid leukemia	1
Chronic myeloid leukemia	1
Lip and oral cavity cancer	1
Pancreatic cancer	1
Hodgkin lymphoma	2
Acute myeloid leukemia	2
Chronic lymphoid leukemia	2
Malignant skin melanoma	2
Bladder cancer	3
Brain and nervous system	3
cancer	
Esophageal cancer	3
Tracheal, bronchus, and	3
lung cancer	
Mesothelioma	3
Multiple myeloma	3
Other cancer	3
Prostate cancer	4
Testicular cancer	4
Breast cancer	4
Colorectal cancer	4
Leukemia	4
Liver cancer	4
Non-Hodgkin lymphoma	4
Non-melanoma skin cancer	4
(squamous cell carcinoma)	
Stomach cancer	4
Nasopharynx cancer	6
Cervical cancer	7
Other pharynx cancer	8
Thyroid cancer	8

Table: Final model selections

Once the best models were selected, data points were manually outliered based on the results of the first run of the model algorithm. Data points were outliered if they clearly influenced the model in an unrealistic way. For example, a data point was marked as an outlier if it created a single-year, single-agegroup spike in model predictions. This was mainly the case in countries with a small number of cases or deaths, or in age groups with small numbers of cases or deaths. Manual outliers were removed from the input dataset prior to initiating the second run of the model algorithm.

After best models were selected, all final outliers were dropped from the data input, and final linear predictions were created, the final linear predictions and residuals were used as input for space-time smoothing. Space-time smoothing is a spatiotemporal regression to smooth residuals over space, time, and age. ^{11–13} The weighted residuals were added to the linear model predictions and used as priors for

the third stage, a Gaussian process regression (GPR) implementing a Matern covariance function. ^{13–18} GPR is a nonparametric technique for interpolating non-linear trends that has been used extensively in the estimation of time series data. Final MI ratio predictions with 95% uncertainty intervals were obtained by back-transforming 1,000 draws from the posterior distribution.

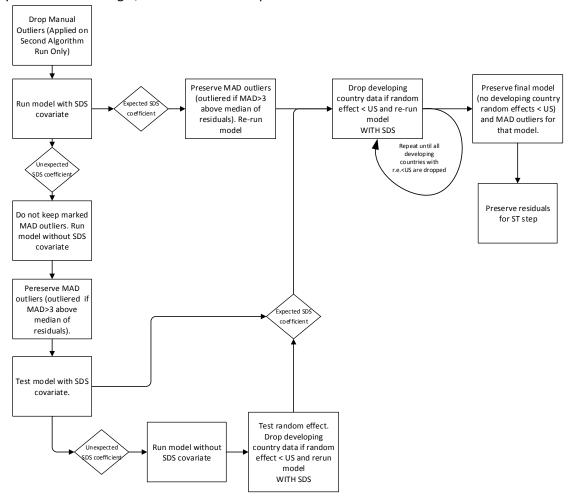


Figure 1: MI model estimation algorithm

Step 9 has undergone a revision compared to GBD 2010 and GBD 2013. In GBD 2010 and GBD 2013 only one model was used to predict all MI ratios, whereas for GBD 2015 we generated multiple models and chose a best model based on out-of-sample validation. Another major difference is that LDI (lagged distributed income) was used as a covariate in previous versions and was replaced by SDI for GBD 2015.

Final MI ratios were matched with the cancer registry incidence dataset in the ninth step (#10 in the flowchart) to generate mortality estimates (Incidence * Mortality/Incidence = Mortality). The final mortality estimates were then uploaded into the COD database (#11 in the flowchart).

After transforming cancer registry incidence data to mortality estimates, the modeling strategy followed the general CODEm process as described in Part 3.

Results

Interpretation of results

Cancer mortality estimates for GBD 2015 can differ from the GBD 2013 results for multiple reasons. First, compared to GBD 2013 more cancer mortality data were added to the cause of death database. Second, we added sources for cancer registry data, which were transformed into mortality estimates by using the MI ratio. Third, mapping of cancer ICD codes to the GBD cancer causes was updated slightly based on collaborator comments. One example is that mapping for the ICD10 code D46 (myelodysplastic syndrome) was changed from "other cancer" to "undefined cancer" for later redistribution to non-Hodgkin lymphoma and leukemia. The one major mapping change was the addition of subtypes for leukemia and non-melanoma skin cancer. Fourth, the method to redistribute undefined causes of death or undefined cancers changed compared to GBD 2013. Models for redistribution are now performed regionally rather than by super-region. Fifth, we updated and refined the mortality-to-incidence ratio estimation compared to GBD 2013. Whereas for GBD 2010 and GBD 2013 a single model was used to estimate the MI ratios for each location, by cancer, sex, and age, we developed multiple plausible models for GBD 2015 and chose the best model based on out-of-sample validation. Sixth, we reviewed the covariate inputs for the CODEm models and changed covariates when updated or improved covariates were available. Seventh, many covariates used in CODEm models were updated for GBD 2015 (Part 4).

The other group producing country-level cancer mortality estimates is the International Agency for Research on Cancer (IARC) with their GLOBOCAN database. Significantly different methods between the GBD study and GLOBOCAN can lead to differences in results. Whereas estimates in GLOBOCAN are based on the assumption that there are "In theory, [...] as many methods as countries," the cancer estimation process for the GBD study follows a coherent, well-documented method for all cancers, which allows cross-validation of models as well as determination of uncertainty. Another major difference is the ability in the GBD study to adjust single cause estimates to the all-cause mortality, which is being determined independently. This also allows us to adjust individual causes of death to the all-cause mortality envelope which permits us to correct for the underdiagnosis of cancer in countries with inadequate diagnostic resources. Redistribution of a fraction of undefined causes of death to certain cancers is another methodical advantage the GBD study has over GLOBOCAN, and estimates for cancer mortality can therefore differ substantially in countries with a large proportion of undefined causes of deaths in their vital registration data or a large proportion of undefined cancer cases in their cancer registry data.

Limitations

There are certain limitations to consider when interpreting the GBD mortality cancer estimates. First, even though every effort is made to include the most recently available data for each country, data-seeking resources are not limitless and new data cannot always be accessed as soon as they are made available. It is therefore possible that the GBD study does not include all available data sources for cancer incidence or cancer mortality. Second, different redistribution methods can potentially change the cancer estimates substantially if the data sources used for the estimated location contain a large number of undefined causes; however, neglecting to account for these undefined deaths would likely

introduce an even greater bias in the disease estimates. Third, using mortality-to-incidence ratios to transform cancer registry incidence data to mortality estimates requires accurate MI ratios. For GBD 2015 the methodology to estimate MI ratios was improved with development of multiple different models and implementation of model cross-validation, but the method is still sensitive to underdiagnosis of cancer cases or underascertainment of cancer deaths. However, given that the majority of data used for the cancer mortality estimation come from vital registration data and not cancer registry data this is not a major limitation.

Non-melanoma skin cancer (squamous-cell carcinoma)

Data

Data seeking processes

The input data were identified and processed using the same methods as all other cancers described above

Inclusion and exclusion criteria

Inclusion and exclusion criteria followed the same methods as described for other cancers (see above).

Bias of categories of input data

The potential biases of the input data are the same as for other cancers (see above).

Methods

Overall methodological process

The GBD produces estimates for non-melanoma skin cancer via two subgroups: non-melanoma skin cancer (basal cell carcinoma) and non-melanoma skin cancer (squamous cell carcinoma). While some cancer registries report non-melanoma skin cancer at the four- or five- digit level required to distinguish between the subtypes (e.g. "C44.01" vs. "C44.02", "173.01" vs. "173.02), most registries report these cancers at the three-digit level as "C44" or "173" ("Other and unspecified malignant neoplasm of skin"). Because of this, those incident cases that were reported at this three-digit level were split to "basal cell carcinoma" and "squamous cell carcinoma" based on proportions reported by Karagas et al during the cause disaggregation step (step #5 in the flowchart). Since mortality estimates are produced for squamous cell carcinoma under the assumption that basal cell carcinoma causes almost no deaths, all mortalities reported as "C44" or "173" were simply mapped to the "squamous cell carcinoma" GBD cause. Apart from this additional step for some incident cases, the remainder of the cancer registry processing was the same as for other cancers as described above.

Steps of analysis and data transformation processes

Non-melanoma skin cancer (squamous cell carcinoma) mortality estimation followed the same steps as the other cancers (see flowchart and description above) except for step #5 in the flowchart as described above.

Model selection

The modeling strategy for non-melanoma skin cancer (squamous cell carcinoma) followed the general CODEm process.

Model performance and sensitivity

The modeling performance and sensitivity for non-melanoma skin cancer (squamous cell carcinoma) mirrored that of the general CODEm process.

Uncertainty intervals

Uncertainty was determined using standard CODEm methodology.

Results

Interpretation of results

Non-melanoma skin cancer mortality estimates are not available from other sources. GLOBOCAN, for example, does not report deaths due to non-melanoma skin cancer. Even though the data availability for non-melanoma skin cancer is poor, the fact that it is the most common incident cancer with rates expected to rise makes it a necessity to include the disease in the GBD framework.

4–13 Limitations

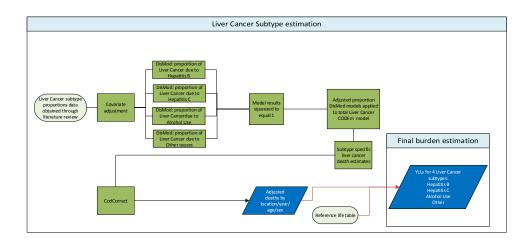
Cancer registry data for non-melanoma skin cancer incidence have to be interpreted with caution due to a substantial amount of underreporting or rules that only the first non-melanoma skin cancer has to be registered. Many cancer registries therefore do not include non-melanoma skin cancers at all. For vital registration data we make the assumption that there are no deaths due to non-melanoma skin cancer (basal cell carcinoma), therefore all deaths attributed to basal cell carcinoma were included instead as squamous cell carcinoma. Based on collaborator recommendations we will test this assumption formally for GBD 2016.

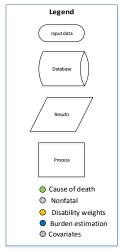
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Liver Cancer SDG Capstone Appendix





Input Data & Methodological Summary

Input data

Data seeking processes

The input data for the parent cause (liver cancer) were identified and processed using the same methods as all other cancers described above. To estimate the number of liver cancers for each of these sub-causes, DisMod-MR 2.1 was used to model the proportion of liver cancers due to the four subgroups. All publications used in GBD 2013 were included, and sources were supplemented with a systematic review of the published literature on the etiology of liver cancer. The literature search was performed in Pubmed on 8/13/15 with the following search string:

(("Carcinoma, Hepatocellular"[Mesh] AND "etiology"[Subheading] AND ("epidemiology"[Subheading] OR "epidemiology"[All Fields] OR "epidemiology"[MeSH Terms])) AND ("Hepatitis B"[Mesh] OR "Hepatitis C"[Mesh] OR ("ethanol"[MeSH Terms] OR "ethanol"[All Fields] OR "alcohols"[All Fields] OR "alcohols"[MeSH Terms] OR "alcohols"[All Fields]) OR autoimmune[All Fields])).

The duration was restricted to articles published between 2013 and 2015. 385 articles were found, of which eight studies were added for liver cancer due to alcohol use (49 studies included from GBD 2013), 11 studies were added for liver cancer due to hepatitis B and C (99 studies included for GBD 2013), and 7 studies were added for liver cancer due to other causes (25 studies included for GBD 2013).

Inclusion and exclusion criteria

Articles were included if it was possible to identify at least one of the causes (alcohol, hepatitis B, hepatitis C, or "other causes") as the only etiology.

Bias of categories of input data

The potential biases of the input data are the same as for other cancers (see above).

Modeling strategy

Overall methodological process

The modeling strategy for the parent cause "liver cancer" followed the general CODEm process. To estimate the fraction of liver cancer due to each etiology for each age-sex-geography-year DisMod-MR 2.1 was used.

Steps of analysis and data transformation processes

If the etiology was reported to be due to multiple causes, the cases due to multiple causes were split based on the proportion of cases in the individual etiologies reported in the publication. If cases were reported to be due to cryptogenic causes, these cases were removed from the denominator. A study covariate was used for publications that only assessed liver cancer in a cirrhotic population. The reference or "gold standard" that was used for crosswalking was the compilation of all studies that assessed the etiology of liver cancer in a general population. Smoothness (xi) was set at 0.1 to 0.3, heterogeneity (zeta) was set at 0.5 to 1. Time window for fit was 10 years, minimum coefficient of variation for global, super-region, region, and country was 0.4, 0.2, 0.1, and 0.1.

For liver cancer due to hepatitis C and hepatitis B, a prior value of 0 was set between age 0 and 0.01. For liver cancer due to alcohol a prior value of 0 was set for ages 0 to 5 years and a prior maximum value of 0.8 for ages older than 5.

For liver cancer due to hepatitis C, hepatitis C prevalence was used as a covariate with a predefined minimum of 0 and maximum of 10. Alcohol and hepatitis B prevalence were used as covariates with a pre-specified covariate of -1 to 0. A positive prior was set on the slope from age 0 to age 60.

For liver cancer due to hepatitis B, hepatitis B prevalence was used as a covariate with a predefined minimum of 0 and maximum of 10. Alcohol and hepatitis C prevalence were used as covariates with a pre-specified covariate of -1 to 0.

For liver cancer due to alcohol, alcohol (liters per capita) was used as a covariate with a predefined minimum of 0 and maximum of 10. Hepatitis B prevalence and hepatitis C prevalence were used as covariates with a pre-specified covariate of -1 to 0. A negative prior was set on the slope from age 0 to age 70.

For liver cancer due to other causes, hepatitis C prevalence, alcohol, and hepatitis B prevalence were used as covariates with a pre-specified covariate of -1 to 0.

To ensure coherent results between the cirrhosis and the liver cancer etiologies, the results from the liver cancer etiology models were transformed into covariates that were then used in the cirrhosis etiology models. The results from the cirrhosis etiology models were then used in the liver cancer proportion models.

The DisMod proportions for the underlying liver cancer etiologies were then squeezed to 100% and these final proportions were applied to the parent cause, "liver cancer," to derive the estimates for the

liver cancer etiologies.

Uncertainty intervals

Uncertainty was determined using standard DisMod methodology.

Results

Interpretation of results

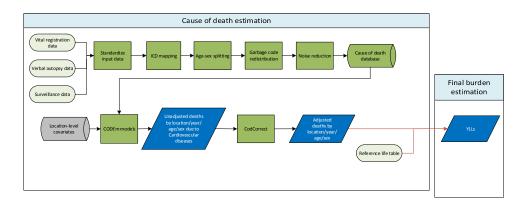
Results for the liver cancer subtype mortality can differ from GBD 2013 for multiple reasons. First, additional sources were added based on an updated literature review. Second, to ensure consistency between cirrhosis and liver cancer etiologies the results from each model for cirrhosis and for liver cancer subtypes were converted into covariates and used in the respective other model. Third, DisMod methods were updated for GBD 2015 (Section 3).

Limitations

The etiological proportion models for the liver cancer sub-causes depend on the availability of data sources that inform DisMod about the local patterns of liver cancer etiologies as well as age patterns. Unfortunately, not many data sources provide data on the etiology of liver cancer by sex or by different age groups. Age patterns were therefore determined based on the assumption that there are no cases of liver cancer due to hepatitis B, hepatitis C, or alcohol in young age groups.

Cardiovascular Diseases SDG Capstone Appendix

Flowchart





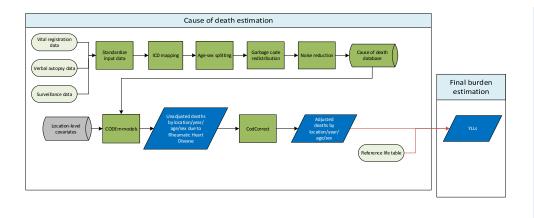
Input data

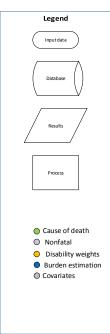
Vital registration, verbal autopsy, and surveillance data were used to model this cause. We outliered non-representative subnational verbal autopsies in a number of Indian states. We also outliered verbal autopsy data sources that were implausibly low in all age groups and ICD8 and ICD9 BTL data points that were inconsistent with the rest of the data and created implausible time trends.

Modeling strategy

We used a standard CODEm approach to model deaths from cardiovascular diseases. We have included two new variables, Socio-Demographic Index and the SEV scalar for rheumatic heart disease, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Rheumatic Heart Disease SDG Capstone Appendix





Input Data & Methodological Summary

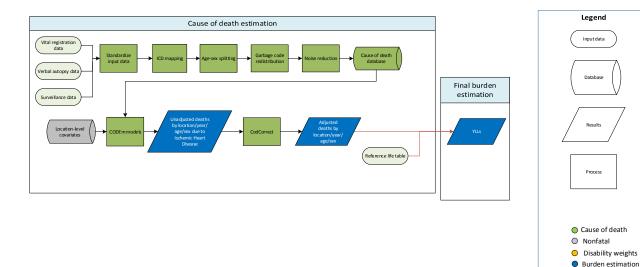
Input data

Vital registration, verbal autopsy, and surveillance data were used to model rheumatic heart disease. We outliered ICD8 and ICD9 BTL data points which were inconsistent with the rest of the data and created implausible time trends. We also outliered data points which were too high after the redistribution process in a number of age groups.

Modeling strategy

We used a standard CODEm approach to model deaths from rheumatic heart disease. We have included two new variables, Socio-Demographic Index and the SEV scalar for rheumatic heart disease, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Ischemic Heart Disease SDG Capstone Appendix



Input Data & Methodological Summary

Input data

Vital registration, verbal autopsy, and surveillance data were used to model ischemic heart disease. We outliered verbal autopsy data in countries and subnational locations where high-quality vital registration data were also available. We also outliered non-representative subnational verbal autopsy data points, ICD8 and ICD9 BTL data points which were inconsistent with the rest of the data and created implausible time trends, and data in a number of Indian states identified by experts as poor-quality.

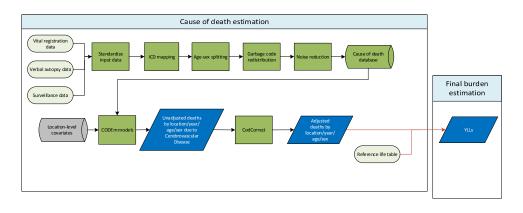
Modeling strategy

We used a standard CODEm approach to model deaths from ischemic heart disease. We have included two new variables, Socio-Demographic Index and the SEV scalar for ischemic heart disease, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Covariates

Cerebrovascular Disease SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

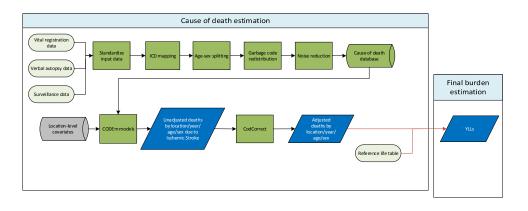
Verbal autopsy and vital registration data were used to model this cause. We outliered non-representative subnational verbal autopsy data points. We reassigned deaths from verbal autopsy reports for cerebrovascular disease to the parent cardiovascular disease for both sexes for those under 20 years of age. We also outliered ICD8, ICD9 BTL, and ICD10 Tabulated data points which were inconsistent with the rest of the data and created implausible time trends. Data points from sources which were implausibly low in all age groups and data points that were causing the regional estimates to be improbably high were outliered.

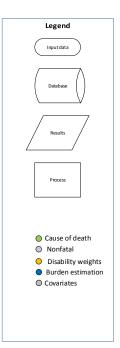
Modeling strategy

We used a standard CODEm approach to model deaths from cerebrovascular disease. We have included two new variables, Socio-Demographic Index and the SEV scalar for cerebrovascular disease, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Ischemic Stroke SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

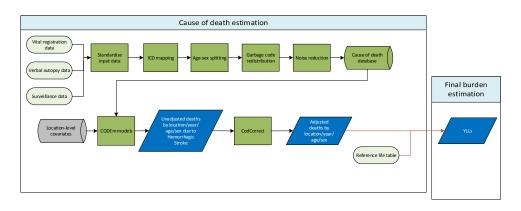
Vital registration, verbal autopsy, and surveillance data were used to model ischemic stroke. We reassigned deaths from verbal autopsy reports for ischemic stroke to the parent cardiovascular disease for both sexes for those under 20 years of age. We outliered ICD8 data points which were inconsistent with the rest of the data and created implausible time trends.

Modeling strategy

We used a standard CODEm approach to model deaths from ischemic stroke. In locations with limited data on ischemic stroke, the subtype-specific deaths were estimated by squeezing both ischemic and hemorrhagic stroke to the overall cerebrovascular envelope. We have included two new variables, Socio-Demographic Index and the SEV scalar for ischemic stroke, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Hemorrhagic Stroke SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

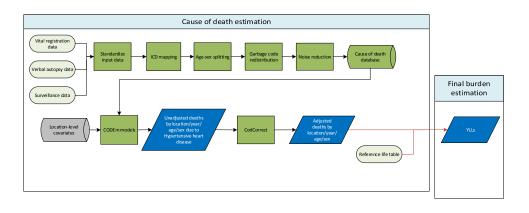
Vital registration, verbal autopsy, and surveillance data were used to model hemorrhagic stroke. We reassigned deaths from verbal autopsy reports for hemorrhagic stroke to the parent cardiovascular disease for both sexes for those under 20 years of age. We outliered ICD8 data points which were inconsistent with the rest of the data and created implausible time trends.

Modeling strategy

We used a standard CODEm approach to model deaths from hemorrhagic stroke. In locations with limited data on hemorrhagic stroke, the subtype-specific deaths were estimated by squeezing both ischemic and hemorrhagic stroke to the overall cerebrovascular envelope. We have included two new variables, Socio-Demographic Index and the SEV scalar for hemorrhagic stroke, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Hypertensive Heart Disease SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

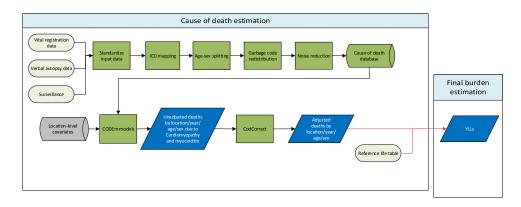
Vital registration, verbal autopsy, and surveillance data were used to model hypertensive heart disease. We outliered ICD9 BTL data points, which were inconsistent with the rest of the data and created implausible time trends.

Modeling strategy

We used a standard CODEm approach to model deaths from hypertensive heart disease. We have included two new variables, Socio-Demographic Index and the SEV scalar for hypertensive heart disease, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Cardiomyopathy and Myocarditis SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

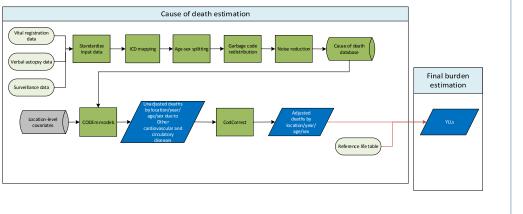
Vital registration, verbal autopsy, and surveillance data were used to model cardiomyopathy and myocarditis. We outliered data points in Central Asia and Central and Eastern Europe due to implausibly high values which we attributed to variation in local coding practices. We also outliered ICD8 data points in countries where they were discontinuous with other data in the time series.

Modeling strategy

We used a standard CODEm approach to model deaths from cardiomyopathy and myocarditis. We have included two new variables, Socio-Demographic Index and the SEV scalar for cardiomyopathy and myocarditis, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013. Finally, local differences in coding practices may explain some of the geographic variation that we see for deaths due to cardiomyopathy; we plan to explore this issue further in future iterations of GBD.

Other Cardiovascular and Circulatory Diseases SDG Capstone Appendix

Flowchart



Legend Inputdata Database Results Process Cause of death Nonfatal Disability weights Burden estimation Covariates

Input Data & Methodological Summary

Input data

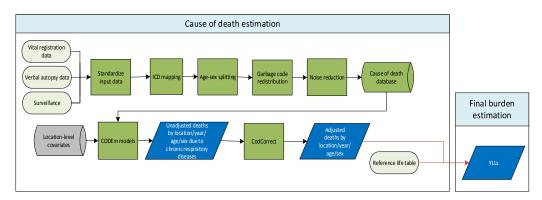
Vital registration, verbal autopsy, and surveillance data were used to model other cardiovascular and circulatory diseases. We outliered ICD8 and ICD9 BTL data points that were inconsistent with the rest of the data and created implausible time trends. We also outliered ICD8 data points which were not nationally representative.

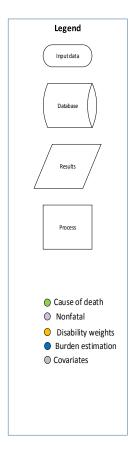
Modeling strategy

We used a standard CODEm approach to model deaths from other cardiovascular and circulatory diseases. We have included two new variables, Socio-Demographic Index and the SEV scalar for other cardiovascular and circulatory diseases, as possible covariates for selection in the ensemble modeling process. Otherwise, there have been no substantive changes from the approach used in GBD 2013.

Chronic Respiratory Diseases SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

Sources used to estimate chronic respiratory disease mortality included vital registration, verbal autopsy, and surveillance data from the cause of death (COD) database. Our outlier criteria excluded data points that (1) were implausibly high or low, (2) substantially conflicted with established age or temporal patterns, or (3) significantly conflicted with other data sources conducted from the same locations or locations with similar characteristics (i.e., Socio-Demographic Index).

For GBD 2015, there were two significant changes in the data preparation process that affect Chronic Respiratory Diseases and its children causes. First, the algorithm package that redistributes heart-failure-

related garbage codes has been updated to take into account the "side" of the heart failure – with right heart failure denoting an underlying respiratory disease. Second, verbal autopsy data are no longer used to inform children causes as they are thought to be unreliable below this cause level. Practically, this has a larger influence on the uncorrected children models than the parent Chronic Respiratory Diseases model discussed here.

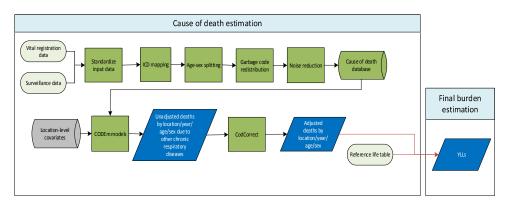
Modeling strategy

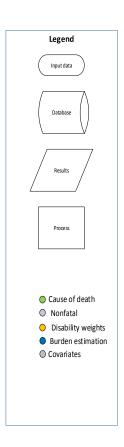
The standard CODEm modeling approach was applied to estimate deaths due to chronic respiratory diseases. Chronic respiratory diseases served as the parent cause to chronic obstructive pulmonary disease, pneumoconiosis (including silicosis, asbestosis, coal worker's pneumoconiosis, other pneumoconiosis), asthma, interstitial lung disease and pulmonary sarcoidosis, and other chronic respiratory diseases. Functionally, this means the death estimates for Chronic Respiratory Diseases serve as an envelope into which the children causes are squeezed by the CodCorrect algorithm. This approach allows us to use a broader range of data – specifically verbal autopsy data – which cannot be accurately mapped to a cause further down in the hierarchy.

Separate models were conducted for male and female mortality, and the age range for both models was 0 to 80+ years. The same covariates from GBD 2013 were used, with the addition of the Socio-Demographic Index (SDI) covariate. Although all covariates in this model received updates for GBD 2015, cumulative cigarettes, smoking prevalence, and health systems access received the larger overhauls. The updates to the smoking-based covariates were particularly helpful in developing these models. Beyond changes in the underlying covariates, there were no substantial deviations from the GBD 2013 approach.

Other Chronic Respiratory Diseases SDG Capstone Appendix

Flowchart





Input Data & Methodological Summary

Input data

Data used to estimate other chronic respiratory diseases included vital registration and surveillance data from the cause of death (COD) database. Our outlier criteria excluded data points that (1) were implausibly high or low, (2) substantially conflicted with established age or temporal patterns, or (3) significantly conflicted with other data sources conducted from the same locations or locations with similar characteristics (i.e., Socio-Demographic Index).

Modeling strategy

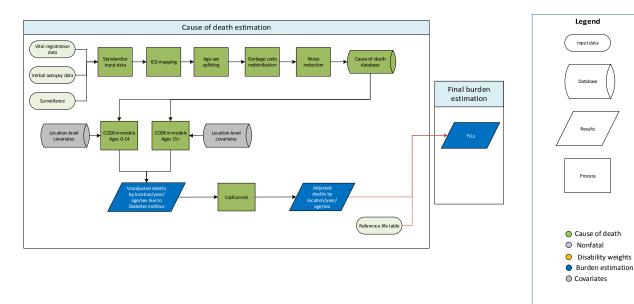
The standard CODEm modeling approach was applied to estimate deaths due to other chronic respiratory diseases. Separate models were conducted for male and female mortality, and the age range

for both models was 0 days to 80+ years. Like other respiratory causes, the mortality estimates from other chronic respiratory diseases were ultimately fit into the chronic respiratory envelope.

Besides general updates to the 2013 covariate set (specifically, the health systems and smoking-related ones), the modeling strategy remained unchanged from GBD 2013. For GBD 2015, we included two new covariates: the Socio-Demographic Index (SDI) covariate, and a standardized exposure variable (SEV) scalar (disease-specific values that reflect the combined effect of all GBD risks) for other respiratory diseases. However, as SEVs are essentially covariate/risk aggregate measures, no substantial changes were expected.

Diabetes Mellitus SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Input data

Verbal Autopsy Data

We outliered VA data points in urban Indian states where high-quality vital registration data were also available. We also outliered data points where the VA data were implausible in all age groups as we determined that these data sources were unreliable.

Vital Registration Data

We outliered all data in four urban Indian states where the source of the data was unreliable according to expert opinion. We also outliered ICD9BTL data points which were inconsistent with the rest of the data series and created unlikely time trends.

Modeling strategy

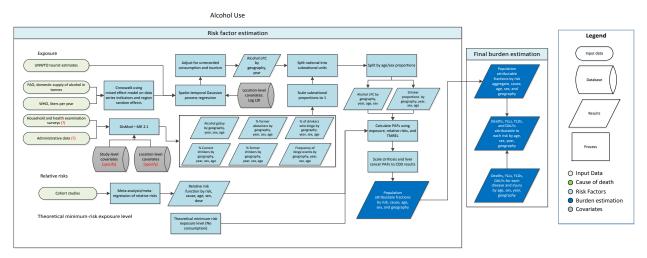
We used a slight variation on the standard CODEm approach to model deaths from diabetes mellitus. Since deaths in younger age groups are almost exclusively due to Type 1 diabetes while deaths in older ages are primarily due to Type 2, we used two models to estimate overall diabetes deaths. The first is for deaths in 0-14 year olds; the second is for deaths in 15-80+ year olds. In previous iterations of GBD, we used a similar approach, but the two models had age ranges of 0-24 and 25-80+. This change was made

due to the increasing prevalence of Type 2 diabetes at younger ages, and thus the increasing likelihood that diabetes-related deaths in the 15-24 year age groups are due to Type 2 diabetes.

We have included two new variables, Socio-Demographic Index and the SEV scalar for diabetes mellitus, as possible covariates for selection in the ensemble modeling process for the model in older ages.

Alcohol Use SDG Capstone Appendix

Flowchart



Input data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with alcohol use, as measured by the summary exposure value (SEV) (3.5.2).

Indicator 3.5.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.5, by 2030, strengthen the prevention and treatment of substance abuse, including narcotic drug abuse and harmful use of alcohol, is measured using SDG Indicator 3.5.2, Risk-weighted prevalence of alcohol use.

Case Definition

The impact of alcohol consumption on morbidity and mortality can be largely described by two separate but related dimensions. The 1st dimension is the individual level drinking and consists of four indicators:

- 1. Current drinkers, defined as the proportion of individuals who have consumed at least one alcoholic beverage (or some approximation) in the last 12 months.
- 2. Former drinkers, defined as the proportion of individuals who have ever consumed an alcoholic beverage, but not in the last 12 months.
- 3. Lifetime abstainers, defined as the proportion of individuals who have never consumed an alcoholic beverage.
- 4. Alcohol consumption (in grams per day), defined as grams of alcohol consumed by current drinkers, per day, over a 12 month period.

The 2nd dimension of alcohol consumption relates to the pattern of drinking and consists of two indicators;

- 5. Binge drinkers, defined as the proportion of drinkers who have had a binge event in the past 12 months. A binge event was defined as consuming 60 grams of alcohol (approximately five drinks or more) in a single occasion for males and 48 grams of alcohol in a single occasion for females.
- 6. Binge times, defined as the proportion of drinking events that are binge amongst binge drinkers i.e. the proportion of days that a binger has a binge event.

Input data

For GBD 2013, a systematic review of the literature was conducted to capture population survey data on all six alcohol use indicators. In summary, the search was conducted in three stages involving electronic searches of the peer-reviewed literature via PubMed, the grey literature and, expert consultation. Updates to systematic reviews via PubMed are performed on an ongoing schedule across all GBD causes and risk factors, an update for alcohol use will be performed in the next 1-2 iterations. For GBD 2015, stages two and three of the literature review were conducted, prioritizing countries for which subnational estimates were generated. The Global Health Exchange (GHDx), IHME's online database of health-related data, was searched for population survey data containing participant-level information from which we could formulate the required alcohol use indicators. Data-sources were included if they captured a sample representative of the geographic location under study and contained variables that could be used to formulate any of the six alcohol use indicators. Relevant survey variables from each data-source were documented in a Microsoft Excel codebook and extracted using STATA 13.1. A total of 629 potential data-sources were available in GHDx across countries with subnational locations, out of which 127 data-sources (66,108 data-points) were included across all six indicators.

To generate estimates of alcohol consumption in grams per day, data from population surveys were used in combination with estimates of per capita consumption from the Food and Agriculture Organization (FAO) and the Global Information System on Alcohol and Health (GISAH database). Per capita consumption is an aggregate measure of recorded, unrecorded, and tourist per capita consumption of alcohol (UNWTO database) derived from sales, production, and other economic statistics. While population-based surveys provide accurate estimates of the prevalence of lifetime abstainers, former drinkers and current drinkers, they typically underestimate real alcohol consumption levels. As a result, the all-age, both-sex per capita consumption figures from the FAO and GISAH are considered to be a better estimate of overall volume of consumption. Per capita consumption, however, does not provide age- and sex-specific consumption estimates needed to compute alcohol-attributable burden of disease. Therefore, we use the age-sex pattern of consumption among drinkers modeled from the population survey data and the overall volume of consumption from FAO and GISAH to determine the total amount of alcohol consumed by country.

Modeling strategy

DisMod-MR 2.1 was used to estimate country-, year-, age- and sex-specific proportions of current drinkers, former drinkers, lifetime abstainers, binge drinkers, and binge times; and alcohol consumption in grams per day. We have made no substantive changes in the modeling strategy from GBD 2013. We ran single-parameter models for each alcohol use indicator and included a combination of the location- and study-level covariates in each model. An alcohol liters per capita location-level covariate was used for

all six indicators to assist in the predictive power of the models. Additionally, study-level covariates were used to accommodate for known sources of variability in the raw data. In the current drinkers, former drinkers, binge drinkers and binge times models, we included two covariates which adjusted estimates derived in past week and past month towards those derived in the past year respectively. Estimates derived in the past year were considered to be the gold standard given the previously outlined definition for each indicator. In the alcohol consumption model, we included a covariate which flagged estimates from one specific data-source — WHO's World Health Surveys — which were systematically higher than estimates derived from other sources due to differences in how alcohol use was captured in these surveys. If other data-points causing higher or lower modelled output were identified during the modelling process for a given indicator, the plausibility of these data points was assessed and the study methodology reviewed. Data points with methodological limitations were excluded as outliers.

A spatiotemporal Gaussian process regression was used to model total alcohol liters per capita.

Theoretical minimum-risk exposure level

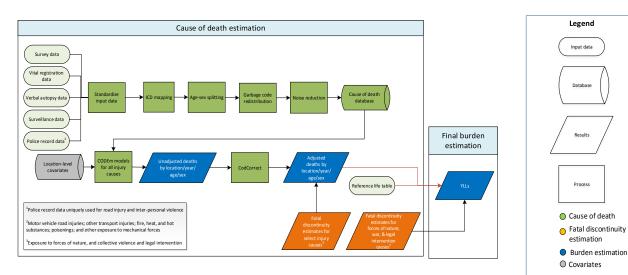
For alcohol use, the theoretical minimum-risk exposure level (TMREL) was assumed to be no alcohol use or, in other words, 0 g/day of alcohol consumption.

Relative risks

The relative risks have not changed significantly since GBD 2013.

SDG Capstone Appendix: Road traffic injuries, interpersonal violence, unintentional poisoning and self-harm mortality

Flowchart



Input data & Methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with mortality due to self-harm (3.4.2), road injury (3.6.1), unintentional poisonings (3.9.3) and interpersonal violence (16.1.1).

Indicator 3.4.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.4, reduce by one third premature mortality from NCDs through prevention and treatment and promote mental health and well-being, is measured using SDG Health Index Indicator 3.4.2, deaths due to selfharm per 100,000.

Indicator 3.6.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.6, by 2030, halve the number of global deaths and injuries from road traffic accidents, is measured using SDG Health Index Indicator 3.6.1, number of deaths due to road injuries per 100,000.

Indicator 3.9.3

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.9, by 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination, is measured using SDG Indicator 3.9.3, deaths due to unintentional poisoning per 100,000.

Legend

Input data

Indicator 16.1.1

As a component of SDG Goal 16. Promote peaceful and inclusive societies for sustainable development, provide access to justice for all and build effective, accountable and inclusive institutions at all levels, SDG Target 16.1, by 2030, significantly reduce all forms of violence and related death rates everywhere, is measured using SDG Health Index Indicator 16.1.1, number of deaths due to interpersonal violence per 100,000.

Input data

In GBD 2015, we estimated mortality due to road traffic injury, interpersonal violence, self-harm and unintentional poisoning from vital registration (VR), verbal autopsy (VA), mortality surveillance, censuses, surveys, and police record data. Police and crime reports were data sources uniquely used for the estimation of deaths from road injury and interpersonal violence. The police data were collected from published studies, national agencies, and institutional surveys such as the United Nations Crime Trends Survey and the WHO Global Status Report on Road Safety Survey. For countries with VR data we did not use police records, except if the recorded number of road injury and interpersonal violence deaths from police records exceeded that in the VR.

Infrequently, data points were marked as outliers. Outlier criteria excluded 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 conducted from the same locations or locations with similar characteristics (i.e., Socio-Demographic Index [SDI]).

Modeling strategy

Overview

In GBD 2015, the standard Cause of Death Ensemble model (CODEm) was applied to estimate deaths due to road injuries, unintentional poisonings, self-harm, and interpersonal violence.

GBD injury codes and categories

The International Classification of Diseases (ICD) was used to classify injuries because it is the standard diagnostic tool for epidemiology. In GBD, injury incidence and death are defined as ICD-9 codes E000-E999 and ICD-10 chapters V to Y.

There is one exception: deaths and cases of alcohol poisoning and drug overdoses are classified under drug and alcohol use disorders. We redistributed deaths coded as unspecified poisoning among the homicide, self-harm, unintentional poisoning, and drug use disorder causes. Upon close review of ICD-coded deaths for unspecified poisoning, we identified a specific age-pattern, in which the death rate spiked in ages 15–50 years. Noting this trend, we conducted further review of data and found that these deaths were likely due to drug use disorders. We then redistributed many unspecified poisoning deaths from unintentional poisoning to drug use disorders, and based on high-quality ICD-9 and ICD-10 data, reassigned these deaths to the drug use disorder sub-causes.

Preparation of data

The preparation of cause of death (CoD) data includes age splitting, age-sex splitting, smoothing, and outlier detection. These steps are described in detail by Naghavi et al and Lozano et al.^{1,2} The concept of "garbage codes" and redistribution of these codes was proposed in the GBD 1990.³ Garbage codes are causes of death that should not be identified as specific underlying causes of death but have been entered as the underlying cause of death on death certificates. A classic example of these types of codes in injuries chapters are "Exposure to unspecified factor" (X59 in ICD-10 and E887 in ICD-9) and all undetermined intent codes (Y10-Y34 in ICD-10 and E980-E988 in ICD-9). Other examples of garbage codes in injuries are the coding of an injury death to intermediate codes like septicemia or peritonitis or as an ill-defined and unknown cause of mortality (R99). Approximately 2% of total deaths in countries with VR data are assigned to these three injury garbage code categories.

Splitting into sublevel causes

In countries with non-detail ICD code data, cause-of-injury categories were proportionally split into sublevel cause-of-injury categories. The sublevel cause-of-injury causes were created in the CoDCorrect process. One of the countries with non-detail ICD code data is South Africa, and in GBD 2013 the proportions of sublevel cause-of-injury were based on VR data. For GBD 2015 the proportions were based on the paper by Matzopoulos et al. 2015.⁴

Limitations and model assumptions

We added police data for road injuries and interpersonal violence to help predict level and age patterns in countries with sparse or absent cause of death data even though we know from countries with near-complete vital registration data that police records tend to underestimate the true level of deaths. However, we applied police data estimates in instances where reported deaths were higher than VR numbers.

Table - Injury Cause List				
ID	Cause	Modeling Strategy	Covariate changes from GBD 2013	
1.1	Road injuries	CODEm	+Cause-specific risk scalar for road injuries, SDI	
1.1.a	Pedestrian road	CODEm	+ Cause-specific risk scalar for pedestrian road	
	injuries		injuries, SDS	
1.1.b	Cyclist road injuries	CODEm	+ Cause-specific risk scalar for cyclist road	
			injuries, SDI	
1.1.c	Motorcyclist road	CODEm	+ Cause-specific risk scalar for motorcyclist	
	injuries		injuries, SDI	
1.1.d	Motor vehicle road	CODEm and fatal discontinuity	+ Cause-specific risk scalar for motor vehicle	
	injuries	estimation	road injuries, SDI	
1.1.e	Other road injuries	CODEm	+ Cause-specific risk scalar for other road	
			injuries, SDI	
1.2	Other transport	CODEm and fatal discontinuity	+ Cause-specific risk scalar for other transport	
	injuries	estimation	injuries, SDI	
2.4	Poisonings	CODEm and fatal discontinuity	+ Cause-specific risk scalar for poisonings, SDI	
		estimation		
3.1	Self-harm	CODEm	+ Cause-specific risk scalar, SDI, major	
			depressive disorder prevalence	

3.2	Interpersonal violence	CODEm	+ Cause-specific risk scalar, SDI
3.2.a	Assault by firearm	CODEm	+ Cause-specific risk scalar for assault by
			firearm, SDI
3.2.b	Assault by sharp	CODEm	+ Cause-specific risk scalar for assault by sharp
	object		object, SDI
3.2.c	Assault by other	CODEm	+ Cause-specific risk scalar for assault by other
	means		means, SDI

References

- 1. Lozano R, Naghavi M, Foreman K, *et al.* Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet* 2012; **380**: 2095–128.
- 2. 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. *The Lancet* 2015; **385**: 117–71.
- 3. Murray CJL, Lopez AD, Harvard School of Public Health, World Health Organization, World Bank. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Cambridge, MA: Published by the Harvard School of Public Health on behalf of the World Health Organization and the World Bank: Distributed by Harvard University Press, 1996.
- 4. Matzopoulos R, Prinsloo M, Wyk VP, Gwebushe N, Mathews S, et al. Injury-related mortality in South Africa: a retrospective descriptive study of postmortem investigations. *Bull World Health Organ* 2015; **93**: 303–13.

Met Need for Family Planning with Modern Methods SDG Capstone Appendix

Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with the proportion of women aged 15 to 49 years with their family planning needs met with modern contraception methods (3.7.1). This indicator also is an individual component of Indicator 3.8.1, which is the composite indicator for universal health coverage (UHC) tracer interventions.

Indicator 3.7.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.7, by 2030, ensure universal access to sexual and reproductive health-care services, including for family planning, information and education, and the integration of reproductive health into national strategies and programmes, is measured using SDG Indicator 3.7.1 proportion of women of reproductive age (15 to 49 years) who are sexually active and have their need for family planning satisfied with modern methods (ie, female and male sterilization, oral hormonal pills, intra-uterine devices (IUD), male condoms, injectables, implants [including Norplant], vaginal barrier methods, female condoms, and emergency contraception)

Input data

We defined modern contraception methods as the current use of male or female sterilization, male or female condoms, spermicide foam/jelly, oral contraceptive, diaphragms, implants, injections, or use of an IUD. Traditional contraception methods were defined as the current use of methods including withdrawal, period abstinence, the rhythm method, and lactational amenorrhea method (LAM).

Women between the ages of 15 and 49 who were fecund, sexually active, and did not wish to become pregnant within the next two years or longer were defined as having need for family planning. Of women with need for family planning, we defined met need with modern methods as women who currently use a modern method of contraception.

The present study used two primary types of input data in order to ultimately generate a time series of met need for family planning with modern methods: (1) individual-level microdata from which met need for family with planning with modern methods could be directly estimated; and (2) tabulated data from which met need with modern methods could be indirectly calculated based on reported estimates of modern contraception coverage, any contraception coverage, and unmet need for family planning. In addition, we updated a systematic review, originally conducted for the 2010 iteration of the Global Burden of Disease Study (GBD 2010), and data extraction process for modern contraception coverage to use as a covariate in our eventual model for estimating met need with modern methods.

Our primary data sources for met need with modern methods included multi-country survey series, such as Demographic and Health Surveys (DHS), Multiple Indicator Cluster Surveys (MICS), and Centers for Disease Control and Prevention Reproductive Health Surveys (CDC RHS). In addition, we extracted data

from the Performance Monitoring and Accountability 2020 (PMA2020) surveys, to which we were granted access. We originally sought a wider universe of population surveys, but our search was somewhat restricted to the survey series for which information on contraception use by method and marital status was readily women for all women of reproductive age. Notably, relatively few microdata sources were available for higher-income countries; subsequently, we heavily relied on tabulated data for these geographies.

The below table shows the number of studies included in the 2015 SDG Capstone paper.

Surveys	Contraception Methods	Unmet Need
DHS	232	214
MICS	114	3
CDC RHS	25	0
PMA2020	12	12
Country-specific	497	43

Among the surveys for which we had access to microdata, we applied survey weights based on survey sampling frames to generate weighted national estimates of met need accompanied by estimates of standard error (SE). In the absence of microdata or survey sampling information, we used survey sample sizes as a mechanism for informing uncertainty estimation.

For a number of our data sources, we could not directly estimate met need with modern methods from microdata or survey reports did not include tabulated estimates of met need with modern methods; instead, the latter would include information on prevalence of modern contraception use, prevalence of any contraception use, and prevalence of unmet need for family planning among women of reproductive age. Following the recommended analytic approach from DHS and Inter-agency Expert Group on the SDG Indicators (IAEG-SDGs)^{1,2}, we estimated met need with modern methods based on this formula:

$$Prev_{MetMod} = \frac{Prev_{Mod}}{Prev_{Any} + Prev_{Unmet}}$$

where $Prev_{MetMod}$ is the prevalence of met need with modern methods among women aged 15 to 49 years; $Prev_{Mod}$ is the prevalence of current modern contraception use among women aged 15 to 49 years; $Prev_{Any}$ is the prevalence of any contraception use among women aged 15 to 49 years; and $Prev_{Unmet}$ is the prevalence of women who have need for family planning but are not currently using any method of contraception (capturing unmet need). In future iterations of this analysis, we will prioritize gaining access to microdata to these surveys, so that we can directly estimate met need with modern methods from individual-level data.

For a subset of surveys, contraception use and met need was only reported for women who were currently or had ever been married. To predict the prevalence of modern contraceptive use for all women, we ran a regression on observations where we had the prevalence of modern contraceptive use for all women as well as married women by age group and geographic region. Using this relationship we were able to cross-walk modern contraceptive use prevalence of all women for countries where only married women were surveyed. We repeated this prediction with met need for family planning, running a regression on observations where we had data on all women and married women by age group and

geographic region to predict the met need of women in countries for which only data on married women were available. Individual data points were reviewed by country and outliered accordingly.

Modelling strategy

For the present analysis, we implemented a two-part modelling approach: (1) generate a time series of modern contraception use for each country; and (2) generate a time series of met need with modern methods using modern contraception prevalence as a model covariate. Spatiotemporal Gaussian process regression (ST-GPR), a model used widely within the GBD study to synthesize coherent trends and uncertainty from multiple sources of data, was used for each of these steps.

Modern contraception prevalence

Based on cross-walked prevalence estimates of modern contraception use, we applied ST-GPR to generate a time series of modern contraception prevalence by geography and age group from 1990 to 2015. First, a mixed-effect linear model was fit based on a fixed effect on age and covariates on educational attainment among women of reproductive age and income per capita, and random effects for countries, GBD regions, and GBD super-regions. We used the predictions from that first-stage model to calculate residuals which were then smoothed over space and time. GPR was then used to compute prevalence of modern contraceptive use and corresponding uncertainty.

Met need with modern methods

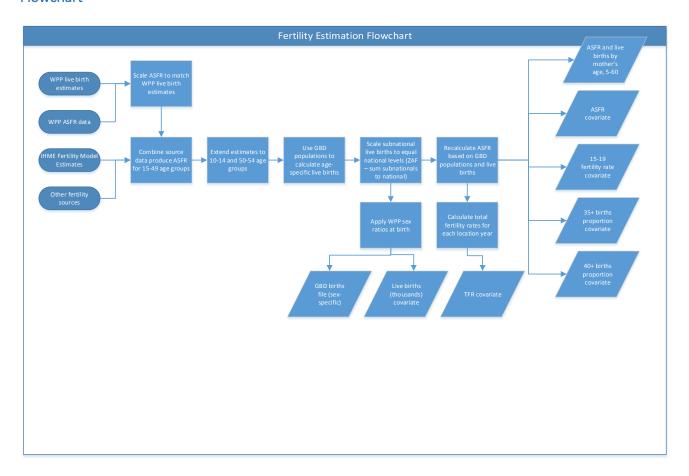
We leveraged the relationship between modern contraception prevalence and met need with modern contraption for modeling the latter as far more data sources had information on modern contraception prevalence than met need. In terms of ST-GPR for met need with modern contraception, the first stage included fitting a mixed-effect linear model with fixed effects on age, educational attainment, and income per capita; random effects for countries, GBD regions, and GBD super-regions; and modern contraception prevalence as a covariate. Smoothing over space time based on the residuals from the first-stage linear model then took place, followed by GPR to generate a cohesive time series of met need with modern contraception and uncertainty for all 188 countries and from 1990 to 2015.

References

- 1. Bradley, S. E. K., Croft, T. N. & Fishel, J. D. Revising Unmet Need for Family Planning: DHS Analytical Studies No. 25. 63 (2012).
- 2. United Nations Department of Economics and Social Affairs. *Goal 3: Ensure healthy lives and promote well-being for all at all ages.* (2015). at https://sustainabledevelopment.un.org/sdg3

Adolescent Birth Rates SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with adolescent birth rates (3.7.2).

Indicator 3.7.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.7, by 2030, ensure universal access to sexual and reproductive health-care services, including for family planning, information and education, and the integration of reproductive health into national strategies and programmes, is measured using SDG Indicator 3.7.2, birth rates (number of live births per 1,000 women) for women aged 10 to 14 years and women aged 15 to 19 years.

Input data

For locations where the United Nations Population (UNPOP) Division provides age-specific fertility rate (ASFR) for age groups 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49 in their most recent update to the

World Population Prospect (WPP), we start with their estimates for every five year time period (e.g. 1990-1995). We treat the given value as that of the midpoint year, so in the case of 1990-1995, we use the value for 1992. We then linearly interpolate in log space to generate values for the intervening years.

Modeling Strategy

ASFR for locations not covered by the UNPOP

For locations not covered by UNPOP, including any subnational locations as well as countries such as Andorra, American Samoa, Bermuda, Northern Mariana Islands, and the United States (US) Virgin Islands, we took one of two approaches. If we could find relatively complete data for 1970's onwards, we would use those estimates. To address the small number of missing values in these datasets, we used a combination of linear mixed effects regression, simple linear interpolation, and 3-year rate of change extrapolation depending on the nature of values that were missing.

Linear mixed effects regression with age as categorical variable was applied to data when entire age groups were missing for a given location. Linear interpolation was applied to locations when missing ASFR values fell between years where ASFR was available. In locations where ASFR was missing for years where values did not fall between years where ASFR was available, but ASFR was present in years preceding or directly after the missing year, ASFR was calculated using annualized rate of change. Missing ASFR was interpolated based on the rate of change of ASFR of the 3 years preceding or following the missing year.

Secondly, in cases where there was little data or it did not cover most of the time period, we modeled ASFR using a database of fertility tables from the Human Fertility Database and from location-level surveys in the locations we were modeling. This process was as follows:

- 1. Calculating empirical weights: Using the database of tables, we created all possible pairs of tables. For each age category, we then calculated the difference between the two tables. These differences were then summed, producing a total difference for each pair of tables. We then created a series of indicator variables for each pair, indicating whether or not they were from the same country, region, or super-region, and how many years apart they were. We then average the difference for each category. So for example, we produced the mean difference for locations in the same super-region but not the same region or country that were 2 years apart in time. We then took the reciprocals of these differences to produce a weight, indicating how "close" a table is to another given their similarities in location and time.
- 2. Fit model relating difference in total fertility rate (TFR) to difference in ASFR: We again create all possible pairs of tables as in step 1. For each pair, we randomly select one table to be the predictor table. Then we fit the following model for each age group in 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, and 45-49:

$$ASFR_1 - ASFR_2 = \beta_1 (TFR_1 - TFR_2) + \beta_0$$

where table 1 is the table randomly designated as the predictor table.

3. Produce a standard tables and predict: Using the weights produced in the first step, we create a standard table from a selection of tables from the database. For each table, weights are calculated for all the other tables based on how far they are from the table in terms of year, and whether they are from the same country, region, or super-region. We then order by weight and

take the first 300 tables. We then create an average table, weighted by weights calculated in step 1. This produces a standard table for each location-year. Due to the limited number of tables for many location, this can produce discontinuities from year to year. To prevent this, we applied a rolling mean over time to the standard, resulting in estimates that are relatively smooth over time. We then use this standard to predict the ASFR for each age group using the models produced in step 2 and the TFR for the country-year of interest:

$$ASFR_1 = \beta_1(TFR_1 - TFR_{standard}) + \beta_0 + ASFR_{standard}$$

where TFR₁ is the TFR in the location year where we are predicting ASFR.

Getting single year ASFR and extending age groups

Once we have five year ASFR values, we calculate single year ASFR using a spline and treating the ASFR values as midpoints for each age group. Though we do not use single-year ASFR for maternal calculations, they are used in other parts of the GBD, and so are incorporated in this process.

For high and low ages, we set fertility for 9 and below and 55 and above to 0, then used those in the interpolation. Because many sources do not have ages 10-14 and 50-54, which are necessary for our maternal estimates, we also extend our estimates to include these age groups. To do this, we created a linear interpolation between the value at age 15 and 0 at age 9 on the young side, and between the value at 50 and 0 at age 55. To these values, we then applied percentages of women who have gone through puberty or have not gone through menopause, respectively. These values are given in the following tables:

age	percent through
	puberty
10	4
11	14
12	40
13	77
14	98

age	percent fertile	
50	36	
51	28	
52	20	
53	14	
54	9	

Because of the steep climb in fertility in the teen years, we made sure that our estimates in 10-14 were in-line with what we would expect by scaling them to the 15-19 age category. Using the mean of the ratios between 10-14 and 15-19 from the Indian Demographic and Health Survey (DHS), the US census, and the Democratic Republic of the Congo DHS, we scaled the 10, 11, 12, 13, and 14 ages so that their mean has this ratio with the mean of 15-19.

Scaling to births

To get our final ASFR estimates, we scale ASFR so that the total implied births from our ASFR estimates and the GBD populations is the same as the GBD births. GBD births are generally derived from the WPP 2015 Revision, and WHO, though for some locations we use location-specific sources. This scaling ensures consistency between our fertility results and the populations that are used in other parts of the GBD process. The exception to this is South Africa. There we used subnational estimates from UNAIDS,

calculated live births implied by these, then used their sum for the South Africa national ASFR estimates. These values were then substituted into the GBD births.

To re-calculate five year age groups, we calculate the number of births in each five year age group and divide by the population in that age group. These are the final ASFR estimates used in our maternal mortality calculations.

Universal Health Coverage (UHC) Tracer Indicator SDG Capstone Appendix

Summary of methodological approach

Indicator definition

This modeling strategy involves the construction of a composite indicator of universal health coverage (UHC) tracer interventions (Indicator 3.8.1), which include vaccination coverage (coverage of three doses of diphtheria-pertussis-tetanus [DPT3], measles vaccine, and three doses of the oral polio vaccine or inactivated polio vaccine); met need for modern contraception; antenatal care (ANC) coverage (one ANC visit [ANC1] and four ANC visits [ANC4]); skilled birth attendance (SBA); in-facility delivery rates; coverage of antiretroviral therapy (ART) among people living with HIV; tuberculosis (TB) case detection rates; and malaria intervention coverage (household ownership of insecticide-treated nets [ITNs]) in malaria-endemic countries.

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, UHC composite indicator based on the geometric mean of coverage of individual tracer interventions for prevention and treatment services.

UHC tracer indicator input data

Individual UHC tracer interventions serve as the input data for the composite UHC tracer indicator, and their write-ups are included in this portion of the appendix.

UHC tracer intervention	Main text	Appendix content
Diphtheria-pertussis-tetanus vaccination, three		
doses (DPT3)		
Measles vaccination		
Polio vaccination, three doses		
Met need with modern contraception methods		
Antenatal care, 1 visit (ANC1)		
Antenatal care, 4 visit (ANC4)		
Skilled birth attendance (SBA)		
In-facility delivery rate (IFD)		
Tuberculosis (TB) case detection rate		
Antiretroviral therapy (ART) coverage among		
people living with HIV		
Insecticide-treated net (ITN) coverage in malaria		
endemic countries		

In sum, each tracer intervention is estimated within the broader GBD study, with many used as covariates to inform cause-specific models. Most of the individual tracer interventions use population health survey microdata, or tabulated report data when microdata are not publicly available, as their primary input data

sources. For a subset of tracer interventions, including vaccination, TB case detection, and ITN coverages, administrative data sources are also used to supplement survey-based estimates.

Composite UHC tracer indicator modeling strategy

To construct the composite UHC tracer indicator, we used draw-level coverage estimates as computed as part of GBD 2015 and took the geometric mean of the 1,000 draws for each intervention and every geography-year under analysis. Ninety-five percent uncertainty intervals (95% UIs) were calculated by taking the 25th and 975th draws. There were two exceptions: (1) TB case detection rates, which were based on case detection reports from the World Health Organization (WHO) and interpolated to construct a full time series; and (2) ITN coverage, which was estimated separately for 40 malaria-endemic countries in sub-Saharan Africa and endemic countries outside of sub-Saharan Africa. More detail can be found in the following appendix section.

Upon calculating the geometric mean for each intervention and geography-year, we took the geometric mean of across the individual interventions to compute the composite UHC indicator for each geography from 1990 to 2015.

We tested two different modeling strategies. The first was taking the geometric mean of each individual tracer intervention to directly compute the composite UHC tracer indicator. The second was grouping a subset of interventions – those related to maternal health (ANC1, ANC4, SBA, IFD, and met need with modern contraception) and vaccination (measles, DPT3, and polio) – and taking the geometric means of each subgroup. The UHC tracer indicator composite was then based on the geometric mean of five interventions: maternal health indicators, vaccine indicators, TB case detection, ART coverage, and ITN coverage. Our final strategy was first approach – taking the geometric mean of each tracer intervention – as the individual interventions within vaccination and particularly maternal health represent different modes of health service delivery and responsiveness to population health needs.

Models were evaluated by expert review and tracer interventions for which poor data quality or availability had large effects on the composite UHC tracer indicator were excluded; as more data become available or standardized data processing steps can be evaluated and implemented, we aim to include a broader range of UHC tracer interventions. Using the same data, geometric means generally result in lower point estimates than arithmetic means. For the present study, we preferred using the geometric mean for calculating the composite UHC tracer indicator to prevent the undue influence of any one particular intervention.

This is the first time that the composite UHC tracer intervention indicator has been calculated. As the annual GBD study expands its analyses to include more interventions and types of prevention or treatment services, we will also extend the number of UHC tracer interventions and services encompassed by the composite indicator.

UHC – ART SDG Capstone Appendix

Indicator definition

This modeling strategy encompassed the indicator associated with universal health coverage (3.8.1), specifically antiretroviral therapy (ART) coverage.

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, UHC composite indicator based on the geometric mean of coverage of tracer interventions for prevention and treatment services.

Input data

ART Coverage Data

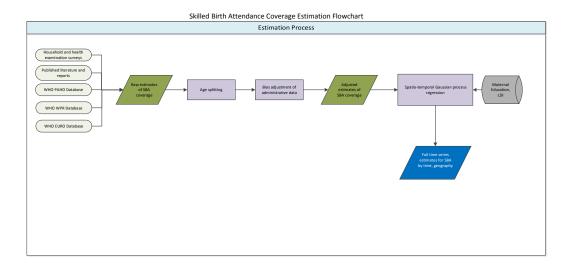
We define ART coverage as the percentage of individuals living with HIV who are receiving ART. This can be broken into two components: the numerator is the number of people receiving ART and the denominator is the number of people living with HIV. Location-, year-, and sex-specific data on the number of individuals receiving ART or the percentage of HIV positive individuals receiving ART were extracted from UNAIDS country files. Whether or not coverage is reported as a count or a percentage varies by year for a given location and sex. This data was estimated by UNAIDS using facility data reported to the WHO by ministries of health as well as data reported by non-profit organizations, private companies, and insurance companies (Cite: Estimation of antiretroviral therapy coverage: methodology and trends. Mary Mahy et al.).

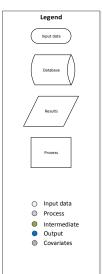
Modelling strategy

Full details of the modelling strategy can be found in the GBD 2015 HIV paper published in Lancet HIV. Spectrum, the compartmental model used for estimation of HIV burden, takes ART coverage as an input which informs the initiation of treatment by sub-group within Spectrum. We report the number of individuals found to be on treatment after running Spectrum so that our estimates of the number people receiving treatment are consistent with our estimates of the number of people living with HIV

UHC Tracer Indicator - Skilled Birth Attendance Capstone Appendix

Flowchart





Input data & Methodological summary

Indicator definition

This modeling strategy pertains to the composite universal health coverage (UHC) tracer indicator (Indicator 3.8.1) and specifically the estimation of skilled birth attendance (SBA).

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, as SBA. Note that SBA is also represented by Indicator 3.1.2.

Input data

For the present analysis, we used individual-level microdata from population health surveys and tabulated survey report data on skilled birth attendance (SBA). As defined by the World Health Organization (WHO), SBA reflects the proportion of births in a given year where a doctor, nurse, or midwife was present.¹

Survey data which provided individual-level data, and specifically among female respondents, were identified and extracted. Major multi-country survey programs included in the analysis include the Demographic and Health Surveys (DHS),² Multiple Indicator Cluster Surveys (MICS),³ Reproductive Health Surveys (RHS),⁴ Living Standards Measurement Study (LSMS) surveys,⁵ and World Health Surveys (WHS).⁶

We also conducted a comprehensive search of the Global Health Data Exchange (GHDx), ⁷as well as targeted internet searches and review of Ministry of Health websites, to identify national surveys and other multi-country survey programs. In addition, we utilized tabulated report data from regional WHO databases, when available, including the PAHO, WHO WPR, and the WHO European Health for All databases.

We excluded all data sources that were not nationally representative or had high levels of missingness. We applied survey weights based on survey sampling frames whenever they were available to generate weighted national estimates of SBA coverage accompanied by estimates of standard error (SE). Estimates of SE, as well as sample sizes, were used to calculate uncertainty, as described below. Any point estimates with sample sizes less than 50 were reviewed to ensure that were not substantive outliers and would otherwise have an undue influence on our analysis.

Due to potential bias in recall, we limited our analysis to women who gave birth up to five years prior to the time of survey; due to data limitations, we used a limit of up to two years for some surveys. We also had to standardize the definition of "skilled health professional" across countries, which varied by differences in quality of training or health professional roles. For this analysis, doctors, nurses, and midwives were included as our foundational definition for SBA, and we extended this to include country-specific medical staff based on the number of years of training they received and/or their comparable ability to intervene in an emergency situation (eg, clinical officers). Care received during delivery by traditional health personnel was not considered a birth overseen by a skilled attendant.

Modeling strategy

Data processing

Age splitting

Most household surveys collection information on maternal and child health (MCH) indicators for children under 5 and/or mothers who gave birth within five years prior to the time of survey. To maximize data use for our model, we included SBA information for children aged 12 to 59 at the time of survey. Children younger than 12 months of age were excluded to minimize the influence of potentially censored observations. SBA coverage estimates were assigned to birth-cohort years based on a child's age prior to the time of survey: we used responses recorded for children aged 12 to 23 months for SBA coverage for one year prior to the time of survey, children aged 24 to 35 months for coverage two years prior to the time of survey, and so forth.

Age-specific estimates are easily computed from individual-level microdata, but many published reports and survey summaries present data in broader age aggregates (eg, SBA coverage for children aged 12 to 35 months). To standardize these age groups, we applied an age-splitting model used in the GBD study, as well as analyses that generated smoking and obesity prevalence by age group. 10,11

Using surveys with microdata as the reference, we used the following model to generate standardized age group-specific estimates for SBA:

$$\tilde{P}_{a,c,t,k} = P_{a,c,t,k}^{a+x} \frac{P_{a,c,t,j}}{P_{a,c,t,j}^{a+x}}$$

where $\tilde{P}_{a,c,k}$ is the adjusted estimate of coverage for target age group a in country c and year t of survey k; and $P_{a,c,k}^{a+x}$ is coverage reported from survey k, for country c in year t for the age group spanning age a to age (a+x). The ratio of coverage between the target age group and broader age group from a survey j with microdata from the same country-year was used to split data from survey k. Surveys to be split were ideally matched with DHS or MICS surveys. If microdata were not available for the same year, ratios within five years of the survey that required age-splitting were applied.

Bias adjustments

Intervention coverage estimates based on administrative sources can be biased, yet the direction and magnitude of such biases are not universal. Some studies show that coverage estimates from administrative data source are systematically higher than those of survey-based estimates, ¹² while other studies show that bias directionality is more heterogeneous. ¹³ Such biases may arise for a number of reasons, including discrepancies in the accurate reporting of services or interventions provided (eg, number of skilled attendants) and target population (eg, number of children born), as well as capturing these data in a timely manner from both public and private sector facilities and healthcare providers.

For SBA, we view individual-level data collected through population health surveys as the most accurate and least biased source of information, particularly for geographies with incomplete health information systems. We thus used SBA coverage estimates from household surveys to calculate country-specific adjustment factors:

$$logit\left(P_{s,c,t}\right) = \beta_0 + \beta_1 logit\left(\tilde{P}_{a,c,t}\right) + \sum_{k=2}^{2+B} \beta_k S_k + \varepsilon_{c,t}$$

where $P_{s,\,c,t}$ is the survey-based estimate for SBA coverage (s) in country c for year t; $\tilde{P}_{a,\,c,t}$ is the administrative estimate for coverage in country c in year t; S_k is a spline basis used to capture the secular trend in coverage; β_1 is the estimated adjustment factor used to correct for the administrative bias; and ε is the error term for country c in year t.

To quantify uncertainty for bias-adjusted estimates from the mixed-effects models described above, we calculated prediction error, \widehat{PE} , as follows:

$$\widehat{PE} = X^2 var(\hat{\beta})$$

where $var(\hat{\beta})$ is the variance for the estimated fixed-effects coefficient of the adjustment factor and X is the independent variable. Proper estimation of prediction errors is crucial as the data synthesis procedure, Gaussian process regression (GPR) (as described in the subsequent section), accounts for uncertainty from point estimates and bias adjustments when generating fitted values. More weight is given to data with less uncertainty. Prediction errors estimated from the bias adjustment were incorporated into the data variance and propagated through the GPR step to obtain estimates of SBA coverage and uncertainty intervals (UIs).

Trend estimation

We used a spatiotemporal Gaussian process regression (ST-GPR) to synthesize point estimates from multiple data sources and derive a complete time series for SBA coverage. This method has been used extensively in GBD and related studies, and accounts for uncertainty pertaining to each point estimate while borrowing strength across geographic space and time. ^{10, 11,15,16} Briefly, we assumed the Gaussian process was defined by a mean function $m(\bullet)$ and covariance function $Cov(\bullet)$.

We estimated the mean function using a two-step approach. Specifically, $m_c(t)$ can be expressed as:

$$m_c(t) = X\beta + h(r_{c,t})$$

where $X\beta$ is a linear model and $h(r_{c,t})$ is a smoothing function for the residuals; and $r_{c,t}$ is derived from the linear model. The following linear model was used for estimating SBA:

$$logit(P_{c,t}) = \beta_0 + \beta_1 medu_{c,t} + \beta_1 LDI_{c,t} + \alpha_c + \gamma_{R[c]} + \delta_c medu + \theta_{R[c]} medu + \varepsilon_{c,t}$$

where $P_{c,t}$ is SBA coverage for country c year t; $medu_{c,t}$ is the average years of education for women of reproductive age in country c and year t; $LDI_{c,t}$ is the lag-distributed income in country c and year t; α_c and $\gamma_{R[c]}$ are country and region random intercepts, respectively. δ_c and $\theta_{R[c]}$ are country and region specific slope on education. These estimates were then run through ST-GPR, as documented elsewhere. ¹⁰

Random draws of 1,000 samples were obtained from the distributions above for every country for a given vaccine. Ninety-five percent uncertainty intervals were calculated by taking the ordinal 25 and 975th draws from the sample distribution.

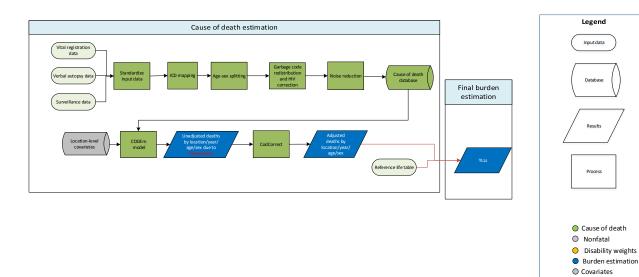
To assess the accuracy of our estimates in each bias adjustment step and in the modeling process, we performed cross-validation analyses by randomly holding out 20% of the sample and, if available, the corresponding administrative estimates for the given indicator of the same country and year, 10 separate times. We computed the average root mean squared errors (RMSE) across each country. Error in the bias adjustments was calculated as the mean difference between the adjusted administrative estimate for a given country, year, and corresponding survey-level estimates (which were considered the "gold-standard"); error in the modeling process was calculated as the difference between the modeled estimates and the sample data.

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Tuberculosis Case Detection SDG Capstone Appendix



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with universal health care – ITN ownership/TB case detection/immunization/ANC1 and ANC4/in-facility delivery rate/skilled birth attendant/ART coverage/modern contraceptive coverage (3.8.1).

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1.

Input data

Input data for modeling tuberculosis mortality among HIV-negative individuals include vital registration, verbal autopsy, and surveillance data from the World Health Organization (WHO). Vital registration data were adjusted for garbage coding (including ill-defined codes, and the use of intermediate causes) following GBD algorithms and misclassified HIV deaths (i.e., HIV deaths being assigned to other underlying causes of death such as tuberculosis or diarrhea because of stigma or misdiagnosis). This correction was done based on examining changes in the age pattern of diseases over time.

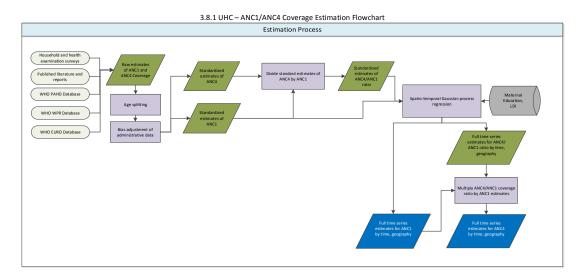
Verbal autopsy data in countries with age-standardized HIV prevalence greater than 5% were removed because of a high probability of misclassification, as verbal autopsy studies have poor validity in distinguishing HIV deaths from HIV-TB deaths. We also outliered data that were largely conflicting with the majority of data from other studies conducted either in the same or different countries (with similar sociodemographic characteristics) in the same region.

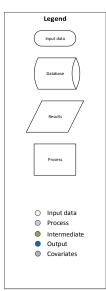
Modeling strategy

A general CODEm modeling strategy was used.

UHC – ANC 1/ANC4 Capstone Appendix

Flowchart





Input data & Methodological summary

Indicator definition

This modeling strategy encompassed the indicator associated with universal health care – ITN ownership/TB case detection/immunization/ANC1 and ANC4/in-facility delivery rate/skilled birth attendant/ART coverage/modern contraceptive coverage (3.8.1).

Indicator 3.8.1

As a component of SDG Goal 3, ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, coverage of tracer interventions for prevention and treatment services.

Input data

Our study included data from household level surveys as well as administrative reports of the proportion of skilled antenatal care attendance of at least one (ANC1) or at least four visits (ANC4) for a given birth, where skilled is defined by the WHO as visits where a doctor, nurse, or trained midwife was present. Survey data which provided person-level information on utilization of maternal health services were identified and extracted. Major multi-country survey programs included in the analysis include the Demographic and Health Surveys (DHS)², the Multiple Indicator Cluster Surveys (MICS)³, the Reproductive

Health Surveys (RHS)⁴, the Living Standards Measurement Studies⁵, and the World Health Surveys (WHS)⁶. In additional, a comprehensive search was performed on the Global Health Data Exchange (GHDx)⁷, as well as a targeted Google search and a search on the websites of national ministries of health, to identify national surveys and smaller multi-country surveys.

A comprehensive database of administrative estimates of maternal health service coverage were not available. Instead, we utilized administrative estimates from regional WHO databases, when available, including the PAHO, WHO WPR, and the WHO European Health for All databases.

Sources that were not nationally representative were excluded, as were data from sources with high levels of missingness for a given indicator. Survey weights and clustering methodology were applied, when available, to obtain weighted national estimates of coverage with accurate estimates of standard errors. In addition to prevalence estimates, we captured information on sample size and standard errors, which were utilized in subsequent analytical steps to capture uncertainty from the data. All data points with sample sizes less than 50 were carefully reviewed to ensure that small samples would not influence the accuracy of analysis. Such points with extreme coverage levels were excluded. We utilize a total of 8,406 data points in our estimation process.

Modeling strategy

Data processing

Age Splitting

Household-level surveys typically collect information about MCH indicators for children under 5 years of age or mothers who have given birth at most 5 years prior to the time of survey. For the sake of utilizing as much data as available, we incorporated estimates for births 0-59 months prior to the survey for analysis. For each indicator, estimates were assigned to a given birth cohort year based on the birth age prior to the time of interview—we used the responses recorded for children aged 12-23 months to estimate coverage 1 year prior to the survey, 24-35 months to estimate coverage 2 years prior to the survey, and so forth.

While information aggregated to these specific age ranges was easily extracted from surveys with person-level data, many published reports and summaries of surveys presented data in broader age groups. We disaggregated these data into the age grouping of interest in this study by applying a splitting model previously used in the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD)⁹, as well as in a studies estimating global smoking¹⁰ and obesity prevalence¹¹.

Using surveys that provided person-level data as references, the following model was applied on estimates with the broader age groups. Specifically, let $\tilde{P}_{a,c,k}$ be the adjusted estimate of coverage for a given indicator for the target age group a in country c and year t of survey k. To disaggregate data that were reported in a broader age group, the following formula was used:

$$\tilde{P}_{a,c,t,k} = P_{a,c,t,k}^{a+x} \frac{P_{a,c,t,j}}{P_{a,c,t,j}^{a+x}}$$

Where $P_{a,c,k}^{a+x}$ denotes the coverage reported from survey k, for country c in year t, but of the age group spanning age a to age (a+x). The ratio of coverage between the age group of interest and the broader age group from a survey j with person-level data from the same country and year was used to split data from survey k. Surveys to be split were ideally matched with DHS or MICS surveys. If person-level data was not available for the same year, data within 5 years to be split was used.

Bias adjustments

Administrative estimates of SBA are most typically produced using data gathered from supply-side registries. The quality and accuracy of the data therefore depends on the completeness of the nation's health information system.⁸ Previous studies have reported that administrative reports of MCH coverage indicators tend to be biased.^{8,12,13}

To reduce the impact of these biases on the final results, we performed adjustments on administrative data to account for overall systematic error. Using mixed effects models, we compared administrative data and survey data to derive appropriate adjustment ratios:

$$logit(P_{s,c,t}) = \beta_0 + \beta_1 logit(\tilde{P}_{a,c,t}) + \varepsilon_{c,t}$$

where $P_{s,\,c,t}$ is the survey-based coverage for a specific indicator for country c in year t, $\tilde{P}_{a,\,c,t}$ is the administrative coverage for country c in year t, β_1 is the estimated adjustment factor used to correct for the administrative bias.

Trend estimation

We used a spatiotemporal Gaussian process regression (ST-GPR) to synthesize information from the various data sources in order to derive a complete time series for each indicator for all countries. This method has been used extensively in other studies to combine information from different sources, taking into account uncertainty for each data point as well as to interpolate nonlinear trends by borrowing strength across geographic space and time. ⁹⁻¹¹ Briefly, we assumed the Gaussian process was defined by a mean function $m(\bullet)$ and covariance function $Cov(\bullet)$. The mean function was estimated using a mixed-effects linear regression as specified below:

$$logit(P_{c,t}) = \beta_0 + \beta_1 medu_{c,t} + \beta_2 gdp_{ct} + \tau_t + \alpha_c + \gamma_{R[c]} + \varepsilon_{c,t}$$
 (1)

where $P_{c,t}$ is the estimated coverage of SBA in country c and year t, $medu_{c,t}$ is the average years of education for women of reproductive age, α_c and $\gamma_{R[c]}$ are country and region random intercept respectively, and τ_t is a random intercept on year. The estimates were then run through ST-GPR, as documented in Ng et. al (2014). ¹⁰ Random draws of 1,000 samples were obtained from the distributions above for every country for a given indicator. The final estimated prevalence for each country was the mean of the draws. In addition, 95% uncertainty intervals were calculated by taking the 2.5 and 97.5 percentile of the sample distribution.

To assess the accuracy of our estimates in each bias adjustment step and in the modeling process, we performed cross-validation by randomly holding out 20% of the sample and, if available, the corresponding administrative estimates for the given indicator of the same country and year, 10 separate times. We computed the average root mean squared errors (RMSE) across each country by indicator. Error in the bias adjustments was calculated as the mean difference between the adjusted administrative estimate for a given country, year and corresponding survey-level estimates (which were considered the "gold-standard"); error in the modeling process was calculated as the difference between the modeled estimates and the sample data.

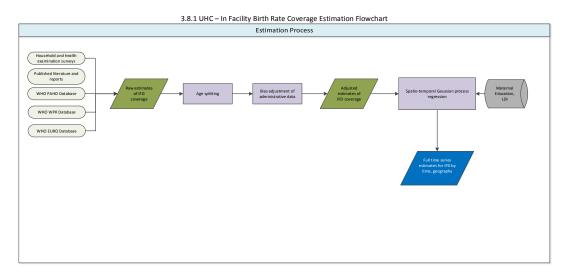
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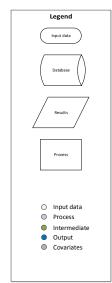
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UHC - In-facility Birth Rate (IFD) Capstone Appendix

Input data & methodological summary





Indicator definition

Indicator definition This modeling strategy encompassed the indicator associated with universal health care – ITN ownership/TB case detection/immunization/ANC1 and ANC4/in-fertility birth rate/ART coverage/modern contraceptive coverage (3.8.1).

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, in-facility birth rate (IFD).

Input data

Our study included data from household level surveys as well as administrative reports of in-facility delivery (IFD), defined by the WHO as the proportion of births in a given year delivered in a health facility. Survey data which provided person-level information were identified and extracted. Major multicountry survey programs included in the analysis include the Demographic and Health Surveys (DHS)², the Multiple Indicator Cluster Surveys (MICS)³, the Reproductive Health Surveys (RHS)⁴, the Living Standards Measurement Studies⁵, and the World Health Surveys (WHS)⁶. In additional, a comprehensive search was performed on the Global Health Data Exchange (GHDx)⁷, as well as a targeted Google search and a search on the websites of national ministries of health, to identify national surveys and smaller multi-country

surveys. In addition we utilized administrative estimates from regional WHO databases, when available, including the PAHO, WHO WPR, and the WHO European Health for All databases.

Sources that were not nationally representative were excluded, as were data from sources with high levels of missingness. Survey weights and clustering methodology were applied, when available, to obtain weighted national estimates of coverage with accurate estimates of standard errors. In addition to prevalence estimates, we captured information on sample size and standard errors, which were utilized in subsequent analytical steps to capture uncertainty from the data. All data points with sample sizes less than 50 were carefully reviewed to ensure that small samples would not influence the accuracy of analysis. Such points with extreme coverage levels were excluded. In total, we utilized 8,406 datapoints in our model.

Modeling strategy

Data processing

Age Splitting

Household-level surveys typically collect information about MCH indicators for children under 5 years of age or mothers who have given birth at most 5 years prior to the time of survey. For the sake of utilizing as much data as available, we incorporated estimates for births 0-59 months prior to the survey for analysis. For each indicator, estimates were assigned to a given birth cohort year based on the birth age prior to the time of interview—we used the responses recorded for children aged 12-23 months to estimate coverage 1 year prior to the survey, 24-35 months to estimate coverage 2 years prior to the survey, and so forth.

While information aggregated to these specific age ranges was easily extracted from surveys with person-level data, many published reports and summaries of surveys presented data in broader age groups. We disaggregated these data into the age grouping of interest in this study by applying a splitting model previously used in the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD)⁹, as well as in a studies estimating global smoking¹⁰ and obesity prevalence¹¹.

Using surveys that provided person-level data as references, the following model was applied on estimates with the broader age groups. Specifically, let $\tilde{P}_{a,c,k}$ be the adjusted estimate of coverage for a given indicator for the target age group a in country c and year t of survey k. To disaggregate data that were reported in a broader age group, the following formula was used:

$$\tilde{P}_{a,c,t,k} = P_{a,c,t,k}^{a+x} \frac{P_{a,c,t,j}}{P_{a,c,t,j}^{a+x}}$$

Where $P_{a,c,k}^{a+x}$ denotes the coverage reported from survey k, for country c in year t, but of the age group spanning age a to age (a+x). The ratio of coverage between the age group of interest and the broader age group from a survey j with person-level data from the same country and year was used to split data

from survey k. Surveys to be split were ideally matched with DHS or MICS surveys. If person-level data was not available for the same year, data within 5 years to be split was used.

Bias adjustments

Administrative estimates of IFD are most typically produced using data gathered from supply-side registries. The quality and accuracy of the data therefore depends on the completeness of the nation's health information system.⁸ Previous studies have reported that administrative reports of MCH coverage indicators tend to be biased.^{8,12,13}

To reduce the impact of these biases on the final results, we performed adjustments on administrative data to account for overall systematic error. Using mixed effects models, we compared administrative data and survey data to derive appropriate adjustment ratios:

$$logit(P_{s,c,t}) = \beta_0 + \beta_1 logit(\tilde{P}_{a,c,t}) + \varepsilon_{c,t}$$

where $P_{s,\,c,t}$ is the survey-based coverage for a specific indicator for country c in year t, $\tilde{P}_{a,\,c,t}$ is the administrative coverage for country c in year t, β_1 is the estimated adjustment factor used to correct for the administrative bias.

Trend estimation

We used a spatiotemporal Gaussian process regression (ST-GPR) to synthesize information from the various data sources in order to derive a complete time series for each indicator for all countries. This method has been used extensively in other studies to combine information from different sources, taking into account uncertainty for each data point as well as to interpolate nonlinear trends by borrowing strength across geographic space and time. $^{9-11}$ Briefly, we assumed the Gaussian process was defined by a mean function $m(\bullet)$ and covariance function $Cov(\bullet)$. The mean function was estimated using a mixed-effects linear regression as specified below:

$$logit(P_{c,t}) = \beta_0 + \beta_1 medu_{c,t} + \beta_2 gdp_{ct} + \tau_t + \alpha_c + \gamma_{R[c]} + \varepsilon_{c,t}$$
 (1)

where $P_{c,t}$ is the estimated coverage of IFD in country c and year t, $medu_{c,t}$ is the average years of education for women of reproductive age, α_c and $\gamma_{R[c]}$ are country and region random intercept respectively, and τ_t is a random intercept on year. The estimates were then run through ST-GPR, as documented in Ng et. al (2014). ¹⁰ Random draws of 1,000 samples were obtained from the distributions above for every country for a given indicator. The final estimated prevalence for each country was the

mean of the draws. In addition, 95% uncertainty intervals were calculated by taking the 2.5 and 97.5 percentile of the sample distribution.

To assess the accuracy of our estimates in each bias adjustment step and in the modeling process, we performed cross-validation by randomly holding out 20% of the sample and, if available, the corresponding administrative estimates for the given indicator of the same country and year, 10 separate times. We computed the average root mean squared errors (RMSE) across each country by indicator. Error in the bias adjustments was calculated as the mean difference between the adjusted administrative estimate for a given country, year and corresponding survey-level estimates (which were considered the "gold-standard"); error in the modeling process was calculated as the difference between the modeled estimates and the sample data.

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UHC Indicator: Modern Contraceptive Coverage

Input data

For the 2015 SDG Capstone paper, we built off a systematic review conducted for the 2010 GBD, focusing on data pertaining to modern contraceptive use prevalence and unmet need. In large, we limited our data search to survey series containing variables on contraceptive use by method, marital status and sexual activity for which microdata is readily accessible: the Demographic and Health Surveys (DHS), Multiple Indicator Cluster Surveys (MICS) and CDC Reproductive Health Surveys (CDC RHS). In addition we included the data from the Gates Foundation's Performance Monitoring and Accountability 2020 (PMA2020) surveys to which we had been granted access.

From the DHS and PMA2020 surveys we also extracted variables related to calculated unmet need.

The below table shows the number of studies included in the 2015 SDG Capstone paper.

	Contraception Methods	Unmet Need
DHS	232	175
MICS	114	0
CDC RHS	25	0
PMA2020	15	15
Country-specific	497	0

Modeling Strategy

For the purposes of our analysis modern contraceptive use was a binary variable defined as the current use of male/female sterilization, male/female condom, spermicide foam/jelly, oral contraceptive, diaphragms, implants, injections or use of an IUD. Unmet need was a binary variable defined as fecund, sexually active women ages 15–49 who are not using contraception and do not wish to become pregnant at all (unmet need for limiting) or within the next two years (unmet need for spacing).

Since the definition for unmet need has changed over time in DHS surveys, we re-computed the variable for DHS surveys prior to 2010 using a program provided by DHS for this exact purpose (http://dhsprogram.com/topics/upload/Stata-Revised-unmet-need-variable-general.zip).

Following data extraction we constructed prevalence rates for modern contraceptive use and unmet need by calculating survey-weighted means of the variables across countries and age groups. The age groups we defined were ages 15-19, 20-24, 25-29, 30-39, 40-49.

As some surveys asked contraception use questions only of women who were currently or had ever been married, we needed to cross-walk the prevalence of contraception use between women of different marital status. To accomplish this first used a spatio-temporal regression with the education and lagged-distributed income covariates to generate estimates of the proportion of women currently married and the proportion of women ever married across time and space. We then cross-walked modern

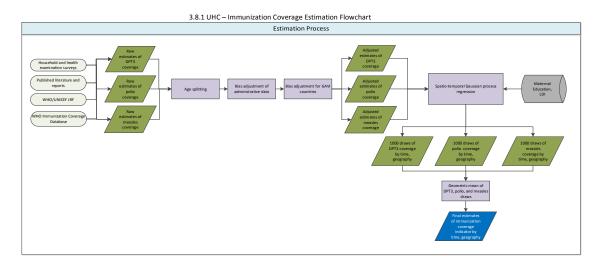
contraceptive prevalence for all women against the estimates of women ever married and currently married.

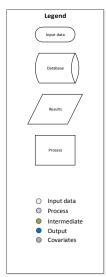
After cross-walking the data we graphed modern contraception use prevalence across time and space and identified outliers from the prevalence rate trends. These outliers were dropped. The final step to estimate prevalence of modern contraceptive use was to use the cross-walked prevalence data, sans outliers, as an input to space-time Guassian Process Regression (ST-GPR) to estimate prevalence for all relevant GBD years, age groups and countries. First, a mixed effect linear model was fit using a fixed effect on age, education and lagged-distributed income covariates, and random effects for countries, regions, and super-regions. The predictions from that model were used to calculate residuals that were smoothed over GBD space-time. Finally, GPR was used to generate estimates of modern contraceptive prevalence for all necessary units. These estimates were population-weighted to collapse to country years and used as an input for the UHC model.

For more information on modern contraceptive coverage, please refer to Indicator 3.7.1.

UHC Tracer Indicator – Immunization Capstone Appendix

Flowchart





Input data & Methodological summary

Indicator definition

This modeling strategy pertains to the composite universal health coverage (UHC) tracer indicator (Indicator 3.8.1) and specifically the estimation of immunization coverage for diphtheria-pertussis-tetanus (DPT3), measles vaccine, and three doses of the oral polio vaccine or inactivated polio vaccine (OPV3 or IPV3).

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, three measures of immunization coverage: DPT3, measles, and polio (OPV3 and/or IPV3) among children aged 12 to 23 months.

Input data

The present study used data from household level surveys as well as administrative reports of immunization coverage. Survey data which provided person-level information on immunization were identified and extracted. Major multi-country survey programs included in the analysis include the Demographic and Health Surveys (DHS),² Multiple Indicator Cluster Surveys (MICS),³ Reproductive Health Surveys (RHS),⁴ Living Standards Measurement Study (LSMS) surveys,⁵, and World Health Surveys (WHS).⁶ We also conducted a comprehensive search of the Global Health Data Exchange (GHDx),⁷as well as

targeted internet searches and review of Ministry of Health websites, to identify national surveys and other multi-country survey programs.

Administrative estimates of immunization coverage were obtained from the Joint Reporting Process (JRF),⁸ through which the World Health Organization (WHO) and UNICEF collates annual estimates of immunization coverage reported UN member states. These immunization coverage estimates are separate from those synthesized by WHO, and are calculated by dividing the number of doses of a given vaccine delivered to the target population (ie, children aged 12 to 23) by the number of individuals in that target population.

We excluded all data sources that were not nationally representative or had high levels of missingness. We applied survey weights based on survey sampling frames whenever they were available to generate weighted national estimates of vaccination coverage accompanied by estimates of standard error (SE). Estimates of SE, as well as sample sizes, were used to calculate uncertainty, as described below. Any point estimates with sample sizes less than 50 were reviewed to ensure that were not substantive outliers and would otherwise have an undue influence on our analysis.

Modeling strategy

Data processing

Age splitting

Most household surveys collection information on maternal and child health (MCH) indicators for children under 5 and/or mothers who gave birth within five years prior to the time of survey. To maximize data use for our model, we included immunization data for children aged 12 to 59 at the time of survey. Children younger than 12 months of age were excluded to minimize the influence of potentially censored observations. For each vaccine, coverage estimates were assigned to birth-cohort years based on a child's age prior to the time of survey: we used responses recorded for children aged 12 to 23 months for immunization coverage for one year prior to the time of survey, children aged 24 to 35 months for coverage two years prior to the time of survey, and so forth.

Age-specific estimates are easily computed from individual-level microdata, but many published reports and survey summaries present data in broader age aggregates (eg, DPT3 coverage for children aged 12 to 35 months). To standardize these age groups, we applied an age-splitting model used in the GBD study,⁹ as well as analyses that generated smoking and obesity prevalence by age group.^{10,11}

Using surveys with microdata as the reference, we used the following model to generate standardized age group-specific estimates of immunization coverage:

$$\tilde{P}_{a,c,t,k} = P_{a,c,t,k}^{a+x} \frac{P_{a,c,t,j}}{P_{a,c,t,j}^{a+x}}$$

where $\tilde{P}_{a,c,k}$ is the adjusted estimate of coverage for target age group a in country c and year t of survey k; and $P_{a,c,k}^{a+x}$ is coverage reported from survey k, for country c in year t for the age group spanning age a to age (a+x). The ratio of coverage between the target age group and broader age group from a survey j with microdata from the same country-year was used to split data from survey k. Surveys to be split

were ideally matched with DHS or MICS surveys. If microdata were not available for the same year, ratios within five years of the survey that required age-splitting were applied.

Bias adjustments

Intervention coverage estimates based on administrative sources can be biased, yet the direction and magnitude of such biases are not universal. Some studies show that immunization coverage estimates from administrative data source are systematically higher than those of survey-based estimates, ¹² while other studies show that bias directionality is more heterogeneous. ¹³ Such biases may arise for a number of reasons, including discrepancies in the accurate reporting of services or interventions provided (eg, number of vaccine doses administered) and target population (eg, number of children in need of vaccines), as well as capturing these data in a timely manner from both public and private sector facilities and healthcare providers.

For immunization coverage, we view individual-level data collected through population health surveys as the most accurate and least biased source of information of vaccination coverage, particularly for geographies with incomplete health information systems. We thus used vaccination coverage estimates from household surveys to calculate country-specific adjustment factors:

$$logit\left(P_{s,c,t}\right) = \beta_0 + \beta_1 logit\left(\tilde{P}_{a,c,t}\right) + \sum_{k=2}^{2+B} \beta_k S_k + \varepsilon_{c,t}$$

where $P_{s,\,c,t}$ is the survey-based estimate for immunization coverage (s) in country c for year t; $\tilde{P}_{a,\,c,t}$ is the administrative estimate for coverage in country c in year t; S_k is a spline basis used to capture the secular trend in coverage; β_1 is the estimated adjustment factor used to correct for the administrative bias; and ε is the error term for country c in year t.

Administrative estimates of immunization also may be subject to an additional bias from participation in performance-based health system support programs, such as the Global Alliance for Vaccines Initiative Immunization Services Support Program (Gavi ISS). It has previously been demonstrated that administrative estimates from participant countries are biased linearly with the number of year enrolled in the program. ¹⁴ To correct for this bias, we performed an additional bias adjustment on immunization coverage:

$$logit(P_{s,c,t}) = \beta_0 + \beta_1 logit(P_{a,c,t}) + \beta_2 T_{c,t}^g + \alpha_c + \varepsilon_{c,t}$$

where $P_{s,t}$ is the survey-based estimate for immunization coverage (s) for country c in year t; $P_{a,t}$ is the corresponding administrative coverage, T_t^g is the number of years of enrollment in the Gavi ISS program by year t; α_c is the country-specific random intercept to capture country-specific variation; β_2 is the estimated adjustment factor used to correct for the GAVI bias by the number of years of participation; and ε is the error term for country c in year t.

To quantify uncertainty for bias-adjusted estimates from the mixed-effects models described above, we calculated prediction error, \widehat{PE} , as follows:

$$\widehat{PE} = X^2 var(\hat{\beta})$$

where $var(\hat{\beta})$ is the variance for the estimated fixed-effects coefficient of the adjustment factor and X is the independent variable. Proper estimation of prediction errors is crucial as the data synthesis procedure, Gaussian process regression (GPR) (as described in the subsequent section), accounts for uncertainty from point estimates and bias adjustments when generating fitted values. More weight is given to data with less uncertainty. Prediction errors estimated from the bias adjustment were incorporated into the data variance and propagated through the GPR step to obtain estimates of coverage and uncertainty intervals (UIs).

Trend estimation

We used a spatiotemporal Gaussian process regression (ST-GPR) to synthesize point estimates from multiple data sources and derive a complete time series for each vaccine. This method has been used extensively GBD and related studies, and accounts for uncertainty pertaining to each point estimate while borrowing strength across geographic space and time. ^{10, 11,15,16} Briefly, we assumed the Gaussian process was defined by a mean function $m(\bullet)$ and covariance function $Cov(\bullet)$.

We estimated the mean function using a two-step approach. Specifically, $m_c(t)$ can be expressed as:

$$m_c(t) = X\beta + h(r_{c,t})$$

where $X\beta$ is a linear model and $h(r_{c,t})$ is a smoothing function for the residuals; and $r_{c,t}$ is derived from the linear model. The following linear model was used for the immunization indicators:

$$logit(P_{c,t}) = \beta_0 + \beta_1 medu_{c,t} + \beta_1 LDI_{c,t} + \alpha_c + \gamma_{R[c]} + \delta_c medu + \theta_{R[c]} medu + \varepsilon_{c,t}$$

where $P_{c,t}$ is vaccination coverage for country c year t; $medu_{c,t}$ is the average years of education for women of reproductive age in country c and year t; $LDI_{c,t}$ is the lag-distributed income in country c and year t; α_c and $\gamma_{R[c]}$ are country and region random intercepts, respectively. δ_c and $\theta_{R[c]}$ are country and region specific slope on education. These estimates were then run through ST-GPR, as documented elsewhere. δ_c

Random draws of 1,000 samples were obtained from the distributions above for every country for a given vaccine. Ninety-five percent uncertainty intervals were calculated by taking the 25 and 975th draws from the sample distribution.

To assess the accuracy of our estimates in each bias adjustment step and in the modeling process, we performed cross-validation analyses by randomly holding out 20% of the sample and, if available, the corresponding administrative estimates for the given indicator of the same country and year, 10 separate times. We computed the average root mean squared errors (RMSE) across each country by vaccine. Error in the bias adjustments was calculated as the mean difference between the adjusted administrative estimate for a given country, year, and corresponding survey-level estimates (which were considered the "gold-standard"); error in the modeling process was calculated as the difference between the modeled estimates and the sample data.

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UHC Tracer Indicator – Insecticide-Treated Net (ITN) Coverage Capstone Appendix

Input data & Methodological summary

Indicator definition

This modeling strategy pertains to the composite universal health coverage (UHC) tracer indicator (Indicator 3.8.1) and specifically the estimation of insecticide-treated net (ITN) coverage among malaria-endemic countries.

Indicator 3.8.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.8, achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all, is measured using SDG Indicator 3.8.1, which includes ITN coverage among malaria-endemic countries.

Countries which eliminated malaria prior to 2000 (the year at which ITNs became more widely available outside of ITN trials) or have been classified as at least pre-elimination according to the World Health Organization (WHO)¹ had 100% ITN coverage applied for all years of analysis; this applied to 115 of the 188 countries in the present study. This analytic decision was made because these countries have largely used other interventions to reduce their malaria burdens or fully eliminate the disease,² and in the cases where ITNs are distributed in these countries, their ownership and use is highly focal and seasonal.

Input data

ITNs were defined as either (1) a traditional ITN, which is treated with an insecticide designed to last up to one year and then needs retreatment at least every year thereafter to remain effective; or (2) a long-lasting insecticide-treated net (LLIN), which is impregnated with a type of insecticide meant to be effective for three to five years. However, since most household surveys combine LLINs received over three years ago as "more than 36 months [old]" rather than offering a more precise age measurement, LLINs obtained more than three years ago are no considered "active" ITNs.^{3–5}

Among the 73 malaria-endemic countries for which ITN coverage was estimated, three types of data sources were used to inform ITN models: (1) ITN delivery records provided by net manufacturers and reported to WHO; (2) ITN distribution records reported to WHO from national malaria control programs; and (3) ITN ownership and use accounts derived from household surveys. The latter were extracted from multi-country survey series, including the Demographic and Health Surveys (DHS), Multiple Indicator Cluster Surveys (MICS), and Malaria Indicator Surveys (MIS), as well as country-specific surveys. ITN coverage estimates were calculated from survey microdata whenever possible; in the absence of microdata, we used nationally representative survey reports of ITN coverage.

We excluded all data sources that were not nationally representative or only included coverage estimates of nets that were not treated with insecticide. We applied survey weights based on survey sampling

frames whenever they were available to generate weighted national estimates of ITN coverage accompanied by estimates of standard error (SE).

Estimates of populations at risk (PAR) for malaria were provided from the Malaria Atlas Project (MAP); these originated from the WHO and earlier iterations of the World Malaria Report. Among malaria-endemic countries and those not classified as at least pre-elimination, PAR ranged from 0.1 to 1.0.

Modeling strategy

Drawing from a Bayesian compartmental "stock-and-flow" model originally developed by Flaxman and colleagues, collaborators from MAP have honed these methods for estimating ITN coverage in sub-Saharan Africa over time. This modeling approach has been used to generate annual estimate of ITN coverage for the WHO's annual World Malaria Report since 2009. In brief, this model uses the relationships between net supply (from manufacturer deliveries), distribution (from national programs), and ownership by households (from population surveys), and tracks volumes of nets based on their "stock" or "flow" status throughout the delivery chain. Parameters on net discard rates at each stage were previously informed by continuously updated literature reviews; however, a substantive model improvement led by Bhatt and colleagues has been the development of a country-by-country loss function derived within the stock-and-flow model. In 16,12

Estimates of ITN coverage outside of sub-Saharan Africa (33 countries in the present analysis) using updated methods from MAP are currently in progress; thus we use previously generated results for ITN coverage for countries outside of sub-Saharan Africa for this study.

Sub-Saharan Africa

For 40 countries, we used national-level estimates of ITN coverage generated by Bhatt and colleagues from 2000 to 2015. ^{5,6} Five hundred draws of ITN coverage were provided for each geography-year, which resulted the duplication of these draws to create a total of 1,000 for uncertainty estimation. While this may have led to underestimated uncertainty for some geography-years, it is also possible that the duplicated draws represented a wider range of potential values (ie, an additional 500 draws all could have been within the minimum and maximum original draws) and thus uncertainty could have been overestimated. Future analyses will include the full 1,000 draws for these countries in sub-Saharan Africa.

Outside of sub-Saharan Africa

For 33 countries, we used national-level estimates of ITN coverage that were calculated for the GBD covariates database; the full 1,000 posterior draws also were available. These estimates spanned from 2000 to 2014, so we used 2014 estimates for 2015. This analytic necessity has the potential to underestimate ITN coverage (ie, if a country conducted a distribution campaign between 2014 and 2015) or overestimate coverage in the absence of continued or heightened net distribution in 2015.

Adjusting for minimum ITN coverage thresholds and population at risk

Prior to estimating ITN coverage for each geography-year, we applied two data adjustments. First, we replaced all draws of ITN coverage to 0.01 on a scale of 0 to 1 (1% on a scale of 0% to 100%) before 2000. This minimum was used to reflect the relatively minimal availability of ITNs before 2000, and to provide greater model stability (ie, analyses conducted in logit space require the replacement of zeros with a

small, non-zero value). In addition, we replaced all draws lower than 0.01 (1%) after 2000 to 0.01 (1%). Second, we divided all draws of ITN coverage by PAR, effectively weighting each draw for a geography's relative malaria risk (and thus need for ITNs). We replaced any draws that exceeded 1 (100%) with 1 (100%).

For each geography-year under analysis, ITN coverage estimates were generated by taking the mean of 1,000 draws of the posterior distribution, and the ordinal 25th and 975th draws of the distribution provided 95% uncertainty intervals (UIs).

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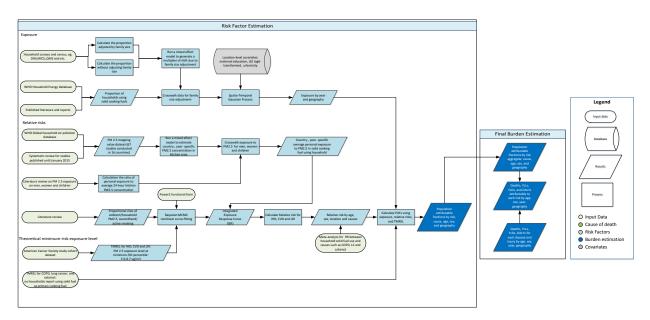
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SDG Indicator: Deaths Attributable to Household Air Pollution and Ambient Air Pollution

Household Air Pollution SDG Capstone Appendix

Flowchart

Household Air Pollution from Solid Fuels



Input data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with deaths attributable to household air pollution (3.9.1).

Indicator 3.9.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.9, by 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination, is measured using SDG Indicator 3.9.1, deaths attributable to household air pollution and ambient air pollution per 100,000 [PAF estimate].

Case Definition

Exposure to household air pollution from solid fuels (HAP) is defined as the proportion of households using solid cooking fuels. The definition of solid fuel in our analysis includes coal, wood, charcoal, dung, and agricultural residues.

Input data

Data were extracted from the standard multi-country survey series such as Demographic and Health Surveys (DHS), Living Standards Measurement Surveys (LSMS), Multiple Indicator Cluster Surveys (MICS), and World Health Surveys (WHS), as well as country-specific survey series such as Kenya Welfare Monitoring Survey and South Africa General Household Survey. To fill the gaps of data in surveys and censuses, we also downloaded and updated HAP estimates from WHO Energy Database and extracted from literature through systematic review done in IHME. Each nationally or sub-nationally representative data point provided an estimate for the percentage of households using solid cooking fuels. Estimates for the usage of solid fuels for non-cooking purpose were excluded, i.e. primary fuels for lighting. The database, with estimates from 1980 to 2015, contained 685 studies from 150 countries. Updates to systematic reviews are performed on an ongoing schedule across all GBD causes and risk factors, an update for household air pollution will be performed in the next 1-2 iterations.

Modeling strategy

Household air pollution was modeled at household level using a three-step modeling strategy ST-GPR that uses linear regression, spatiotemporal regression and Gaussian Process Regression (GPR). The first step is a mixed-effect linear regression of logit-transformed proportion of households using solid cooking fuels. The linear model contains maternal education and proportion of population living in urban areas as covariates and has nested random effect by country, GBD region, and GBD super region respectively. The full ST-GPR process is specified elsewhere in this appendix.

Compare with GBD 2013, we have made changes in terms of the covariates utilized in the linear model. In GBD 2013, year represented the only fixed effect in the robust linear model. While in GBD 2015, we switched to average years of maternal education and proportion of population living in urban areas as predictors for HAP.

Theoretical minimum-risk exposure level

For outcomes where we extracted RR based on direct epidemiological evidence i.e. COPD, lung cancer, and cataract, TMREL was defined such that no households would report using solid fuel as their primary cooking fuel. For outcomes that utilize evidence based on the Integrated Exposure Response (IER), the TMREL is defined as uniform distribution between 33.3 and 41.9 ug/m^3. TMREL for household air pollution did not change from GBD 2013.

Relative risks

The disease-outcomes paired with household air pollution has not changed since GBD 2013. The sources of relative risks (RRs) used depend on the disease outcomes. RRs for COPD, lung cancer and cataract paired with household air pollution rely on direct epidemiological evidence i.e. meta-analyses. The RRs from meta-analyses remain unchanged after a systematic review on meta-analysis for the risk-outcome pairs published after Jan 1st, 2014. RRs for IHD, stroke and LRI are generated from the IERcurves. The IER curves are updated to reflect the newly updated data and utilization of a new method that specified elsewhere.

PM2.5 mapping value

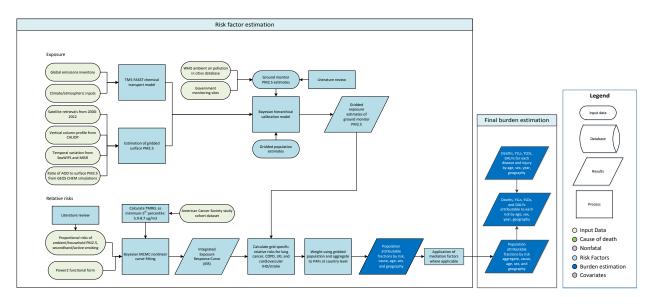
The relative risk estimates describing the association of HAP with outcomes including Ischemic Heart

Disease (IHD), cardiovascular disease (CVD), and lower respiratory infections (LRI) were derived from the IER curves. This is done by first estimating the crosswalk values that map household use of solid fuel to PM2.5 exposure because the IER curve measures exposure using PM2.5. This step of the analysis relied on 67 studies conducted in 16 countries to generate the PM2.5 mapping values, which remain the same sources as GBD 2013. The PM2.5 exposure was then cross-walked to men, women and children by generating the ratio of personal exposure to average 24-hour kitchen PM2.5 concentration based on a study after the literature review in GBD 2013.

Ambient Particulate Matter Pollution Capstone SDG Appendix

Flowchart

Ambient PM2.5



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with deaths attributable to ambient air pollution (3.9.1).

Indicator 3.9.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.9, by 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination, is measured using SDG Indicator 3.9.1, deaths attributable to household air pollution and ambient air pollution per 100,000 [PAF estimate].

Case definition

Exposure to ambient air pollution is defined as the population-weighted annual average mass concentration of particles with an aerodynamic diameter less than 2.5 micrometers ($PM_{2.5}$) in a cubic meter of air. This measurement is reported in $\mu g/m^3$.

Input Data

The data to estimate exposure to ambient air pollution is drawn from estimates of annual concentration of $PM_{2.5}$ – generated using satellite observations of aerosols in the atmosphere. To correct for bias in the satellite modeling approach, a spatially-varying flexible framework is used to combine modeled

concentrations with observations from ground-level monitoring of particles in more than 75 countries. All input data for GBD2015 was updated as follows:

Updated PM_{2.5} ground measurement database

For the GBD2015 update we updated the database of annual average PM measurements to include more recent data and to incorporate additional locations where measurement data have become available. To facilitate this we collaborated with WHO and contributed to their recently released WHO Air Pollution in Cities database. We then used disaggregated (monitor-specific values and not the city averages that are reported by WHO) measurements from this database with additional site-specific information (e.g. all monitors in a city, monitor geo coordinates, monitor site type) such as that included in the GBD2013 database. In total measurements of concentrations of PM₁₀ and PM_{2.5} were retrieved from 6,003 ground monitors with the majority contributing measurements from 2014 (as there is a lag in reporting measurements, little data from 2015 were available). Where data were not available for 2014 (2760 monitors), data was used from 2015 (18 monitors), 2013 (2155), 2012 (564), 2011 (60), 2010 (375), 2009 (49), 2008 (21) and 2006 (1). For locations with only PM₁₀ measurements, PM_{2.5} measurements were estimated from PM₁₀. This was done by a locally derived conversion factor (PM_{2.5}/PM₁₀ ratio) estimated as population-weighted averages of location-specific conversion factors for the country. Location-specific conversion factors were estimated as the mean ratio of PM_{2.5} to PM₁₀ of stations for the same year. If national conversion factors were not available, regional ones were used, which were obtained by averaging country-specific conversion factors.

Updated satellite-based estimates

The updated satellite-based estimates are described in detail in van Donkelaar et al. 2016^1 . These estimates (~11 x 11 km resolution at the equator) combine aerosol optical depth retrievals from multiple satellites with the GEOS Chem chemical transport model and land use information.

Updated population data

A comprehensive set of population data on a high-resolution grid was obtained from the Gridded Population of the World ($\underline{\text{GPW v4}}$) database. These data are provided on a 0.0417°×0.0417° resolution. To aggregate these estimates of population to each 0.1°×0.1° grid cell, the central 3 × 3 population cells were summed. As this accounted for a resolution higher than necessary, the same was done four other times, offset by one cell in a North, South, East and West direction. The average of five quantities was used as the aggregated population estimate for each cell. Estimates of population for 2000, 2005, 2010, 2015 and 2020 were extracted from GPW version 4 and estimates for 1990 and 1995 were extracted from GPW version 3 as described previously for GBD2013³.

Modeling Strategy

The methodology used to estimate the burden of ambient particulate matter pollution has seen significant changes since GBD2013.

The GBD2010 and GBD2013 estimates both used a single global function to calibrate the mean of the chemical transport model and satellite-based estimates to available ground measurements. In both instances the approach taken was recognized at the time to be a compromise between what could be easily implemented under tight timeframes and one that most efficiently utilized all of the data sources. In particular, the GBD2013 exposure estimates were known to underestimate ground measurements in specific locations (see discussion in Brauer et al., 2015²) such that it would be desirable to allow measurements to make a larger contribution to the final estimates where they were available. Therefore, for GBD2015 we implemented a Bayesian Hierarchical modelling approach using Integrated Nested Laplace Approximations (INLA) in which the satellite-based estimates, ground measurements and land use information are combined in a spatially varying flexible framework. Formal external evaluation using ground measurements was conducted and shown to lead to improved predictions of ground measurements in all super regions compared to GBD2013 estimates and an alternative geographically-weighted regression approach. Further, based on the external evaluation analyses, addition of the TM5 chemical transport model estimates of PM2.5 annual average did not improve the estimates and these were therefore not included.

Bayesian hierarchical models (BHM) provide an extremely useful and flexible framework in which to model complex relationships and dependencies in data. Uncertainty can also be propagated through the model allowing uncertainty arising from different components, both data sources and models, to be propagated through the models into estimates of uncertainty associated with the final estimates. In the hierarchical modeling approach coefficients associated with satellite-based estimates were estimated for each country. Where data were insufficient within a country, information can be 'borrowed' from a higher aggregation (region) and if enough information is still not available from an even higher level (super-region). Individual country level estimates were therefore based on a combination of information from the country, its region and super-region.

All modelling was performed on the log-scale with the unit of measurement being a grid cell. The model was constructed with the inclusion of all variables assessed statistically, based on model fit (DIC, a measure of the relative quality of a model and closely related to that of AIC but for Bayesian models) and predictive ability. The hierarchical structure was applied to the intercept and slope terms with all modelling on the log scale. The model was of the form:

$$log(PM2.5_i) = \beta_0 + \beta_1 log SAT_i + other variables + \varepsilon_i$$

where *i* denotes the grid cell. The following sets of variables were considering in developing the models:

Continuous explanatory variables:

- o (SAT) Estimate of PM_{2.5} (in μgm⁻³) for 2014 from satellite remote sensing on the log-scale.
- o (CTM) Estimate of PM $_{2.5}$ (in μgm^{-3}) for 2014 from chemical transport models on the log-scale.
- o Estimate of population for 2014 on the log-scale.

- o (SNAOC) Estimate of the sum of sulfate, nitrate, ammonium and organic carbon as estimated from GEOS Chem
- o (DST) Estimate of compositional concentrations for mineral dust from GEOS Chem
- (EDxDU) The log of the elevation difference between the elevation at the ground measurement location and the mean elevation within the GEOS Chem simulation grid cell multiplied by the inverse distance to the nearest urban land surface

Discrete explanatory variables:

- o Binary variable indicting whether exact location of ground measurement is known
- o Binary variable indicting whether exact type of ground monitor is known
- o Binary variable indicting whether ground measurement is PM_{2.5} or converted from PM₁₀

Random Effects:

- o Grid cell random effects on the intercept to allow for multiple ground monitors in a grid cell.
- o Country-region-super-region hierarchical random effects for the intercept
- o Country-region-super-region hierarchical random effects for the satellite remote sensing term.
- o Country-region-super-region hierarchical random effects for the coefficient associated with the difference between estimates from CTM and SAT.
- o Country-region-super-region hierarchical random effects for the coefficient log(POP)
- Country level random effects for intercept, satellite and difference between CTM and SAT are independent and identically distributed.
- o Country level random effects for population uses a neighbourhood structure allowing specific borrowing of information from neighbouring countries.
- o All region random effects are assumed to be independent and identically distributed.
- All super-region random effects are assumed to be independent and identically distributed.

Interactions:

o Interactions between the binary variables and the effects of log(SAT) and log(CTM/SAT)

Due to both the complexity of the models and the size of the data, notably the number of spatial predictions that are required in this setting, recently developed techniques that perform 'approximate' Bayesian inference based on integrated nested Laplace approximations (INLA) have been developed as a computationally attractive alternative to Markov Chain Monte Carlo methods. Computation was performed using the R interface to the INLA computational engine (R-INLA) with the size of the task of fitting the models and performing predictions for each of the ca. 1.4 million grid cells requiring the use of a high performance computing cluster (HPC) with high memory nodes. As in GBD2010 and GBD2013 the spatial model was built combining the different data sources for a single year (2014, corresponds to the most recent measurement data). The spatially-varying functions from this model were then applied to the satellite-based estimates from all other years - in other words assuming that the spatial relationship between the different data sources does not change over time. This is undoubtedly a simplification but to do otherwise would require assembling multi-year measurement databases which is not feasible given

current data availability and computational constraints. As the spatial model was built using the most recently available (2014) measurement and satellite-based estimates, 2015 estimates were based on extrapolation. Instead of extrapolating using an exponential model based on a 1-year trend as in GBD2013, splines based on a 5 year trend (2010-2014) were fit and applied to the 2014 grid-cell values to estimate levels for 2015. This reduced the likelihood of 2015 estimates being overly influenced by meteorological events in a specific year and to better represent the duration of exposure relevant to the epidemiologic studies included in the integrated exposure-response functions.

Model Evaluation

Model evaluation and comparison was performed by fitting models on a training set and predicting exposures at locations for which measurements were known (the validation set). The selection of the training (20%) and validation (80%) set consisted of taking a random sample of the total number of sites measuring PM2.5 (or having a value converted from PM10 measurements). Sampling was performed using sampling probabilities based on the cross-tabulation of PM2.5 categories (0-24.9, 25-49.9, 50-74.9, 75-99.9, 100+ μ g/m3) and super-regions. The resulting hold-out evaluation data set was a sample of 20% of the sites that have the same distribution over PM2.5 categories and super-regions as the entire set of sites.

This process was used to generate multiple training and validation set combinations, allowing for example cross-validation to be performed. In the evaluation, 25 sets of training/validation data were used. The following models were considered in the evaluation phase:

- (A) The GBD2013 model, using a simple linear regression with a fused estimate of SAT and CTM together with interactions with three binary variables representing whether the measurement was converted from PM10 and whether the exact site type and location is known.
- (B) A hierarchical model with SAT, the TM5 CTM estimates, population and the three binary variables described above
- (C) A hierarchical model with SAT, population, SNAOC, DST, EDxDU, population and the three binary variables
 - o Estimate of population for 2014 on the log-scale.
 - o Estimate of the sum of sulfate, nitrate, ammonium and organic carbon as estimated from GEOS Chem
 - o Estimate of compositional concentrations for mineral dust from GEOS Chem
 - o The log of the elevation difference between the elevation at the ground measurement location and the mean elevation within the GEOS Chem simulation grid cell multiplied by the inverse distance to the nearest urban land surface

For each training/evaluation set combination, model fit and prediction accuracy were evaluated for each of the 25 training/evaluation set combinations with the following metrics:

Model fit

- R²
- DIC

Predictive accuracy

- R² arising from a linear regression of predicted vs actual measurements at each location
- RMSE root mean squared error

- WRMSE weighted (by population) root mean squared error
- MSE mean square error
- MAE mean absolute error

This evaluation indicated the final model improved predictions of ground measurements in all super regions compared to GBD2013 estimates (median global R^2 [population-weighted RMSE] 0.82 (12.1 μ g/m³), 0.60 [13.5 μ g.m³] for GBD2015 and GBD2013, respectively).

Error! Reference source not found. shows the RMSE (median from the 25 runs) for each of the three models, by super-region. The accuracy of the prediction varies between super-regions, with lower errors being observed in areas where there are more monitoring sites. In each of the super-regions, the largest errors are seen for model A which are considerably higher than those for models B and C, with model C showing a small improvement over B (except in super-region 5, North Afirca/Middle East).

Figure 2 shows scatter plots of the observed and predicted measurements using the three models for each super-region. The predicted measurements are the median values over those obtained from the 25 training sets. Predictions from the two Bayesian hierarchical models (B&C) match the observed values more closely than the linear model (A) with much less spread around a straight line (with slope one and zero intercept, shown in red). In Central Europe and Sub-Saharan Africa it is noticeable that, in addition to reduced spread, models B&C are much better at predicting higher values. The same patterns of results in predictive ability were seen when looking at regions and individual countries. In all cases, model C performed better than model B with both being considerable better than model A.

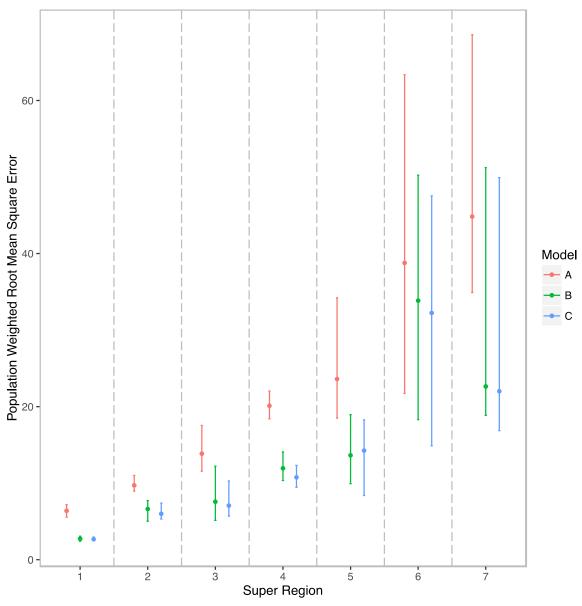


Figure 1: Comparison of RMSE from three models by super-region. Dots denote the median of the distribution from 25 training/evaluation sets and the vertical lines the range of values. Super-regions are 1: high income, 2: Central Europe, Eastern Europe, Central Asia, 3: Latin America and Caribbean, 4: Southeast Asia, East Asia and Oceania, 5: North Africa / Middle East, 6: Sub-Saharan Africa, 7: South Asia.

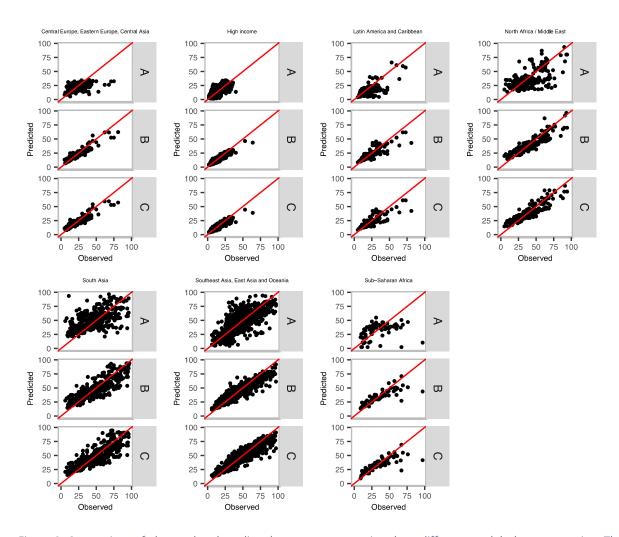


Figure 2: Comparison of observed and predicted measurements using three different models, by super-region. The red line has slope one and intercept zero.

Overall, the best model in terms of model fit and predictive ability was one with the following components:

- o Estimates of PM_{2.5} (in μ gm⁻³) from satellite remote sensing (SAT), population, and information on the GEOS Chem simulated chemical composition, elevation and distance to urban land use (SNAOC, DST and EDxDU).
- o Binary variables indicting whether exact location and type of ground measurement is known, and whether the measurement was $PM_{2.5}$ or converted from PM_{10} .
- o Interactions between the binary variables and the effects of estimates from satellite remote sensing.
- o Grid cell random effects on the intercept to allow for multiple ground monitors in a grid cell
- o Country-region-super-region hierarchical random effects for intercepts, satellite remote sensing and population terms.
- O Country level random effects for population using a neighbourhood structure allowing specific borrowing of information from neighbouring countries.

Relative Risk

Relative risks are generated using integrated exposure-response functions (IER) that are fit to available epidemiologic data using a Bayesian MCMC approach and a modified power function. The IER are estimated based on published relative risks for long-term exposure to ambient PM2.5, household air pollution, second-hand smoking, and active (cigarette) smoking. The concentration of particulate matter for each type of exposure is estimated based on literature values and used to map the relative risks to a curve generated for the entire range of exposure from these sources. The input data for this curve fitting process has been updated since GBD2013, adding new studies that estimate exposure at finer spatial scales, including studies of within-city exposure that focus on traffic-related air pollution. In addition, changes were made to the curve-fitting process. In order to account for differences in study design, temporal patterns of exposure and other differences among the studies of the different sources of PM2.5, a source-specific heterogeneity parameter was added to the IER. This resulted in much wider, and, in our view, more realistic, uncertainty intervals for the burden estimates, by propagating through the entire process the current uncertainty regarding the mechanisms and magnitude of health impacts of exposure to PM2.5 from diverse sources.

IER Functional Form

Data Likelihood

$$\log(RR_i) \sim \mathcal{N}ig(\mu_i, \sqrt{\sigma_i^2 + \delta_{source_i}}ig)$$

Model

$$\mu_i = \log \left(rac{1 + lpha imes \left(1 - e^{-eta imes (exposure_i - TMREL)^{\gamma}}
ight)}{1 + lpha imes \left(1 - e^{-eta imes (counter factual_i - TMREL)^{\gamma}}
ight)}
ight)$$

Data

 $RR_i: \mbox{measured relative risk for data point i} \\ \sigma_i: \mbox{variance of data point i} \mbox{based on study information} \\ source_i: \mbox{exposure source type (outdoor/household air pollution, secondhand/active smoking)} \\ TMREL: \mbox{theoretical minimum risk exposure level} \\ exposure_i: \mbox{measured exposure for data point i} \\ counterfactual_i: \mbox{counterfactual exposure for data point i} \\$

Priors

 $egin{aligned} lpha &\sim \Gammaig(1.0, 0.01ig) \ eta &\sim \Gammaig(1.0, 0.01ig) \ \gamma &\sim \Gammaig(1.0, 0.01ig) \ \delta &\sim \Gammaig(1.0, 0.01ig) \end{aligned}$

We also modified the way in which age-specific IER for IHD and stroke were estimated. In accordance with previously published work on other cardiovascular risk factors, the impact of air pollution on cardiovascular health is known to vary with age. To account for this phenomenon, age-specific RRs were based on a log-linear model of RR as a function of age, where the intercept (RR=1) is forced to age 110. In GBD2010 and GBD2013 the age for a relative risk estimate from a given study was estimated as the median age at death or disease incidence in that study. However, this may not accurately represent the age distribution of the entire study population so we re-estimated this variable as the mean age at enrollment + half of the average follow-up time to better represent the average age of the study population during the period of follow-up.. When compared to GBD2013, this change produced RRs that were generally lower for younger age groups, given that median age at event tends to produce a higher anchor age than average age during follow-up.

The relative risks are generated on the grid-level based on estimated exposure, and then applied to generate PAFs. These PAFs are aggregated using the grid-level population to create population-weighted national estimates of attributable burden, using the following formula:

PM2.5 Aggregation Formula

$$PAF_{A, C, L} = \frac{\sum ((RR_{A, C} - 1) * Pop_i)}{\sum (RR_{A, C} * Pop_i)}$$

A = age group

C = cause

L = location

i = grid

 $RR_{A-C}=$ grid-level RR based on $PM_{2.5}$ and given age/cause IER curve

TMREL

The TMREL for ambient PM is estimated using a uniform distribution between the minimum and 5th percentile of exposure observed in the studies used to generate the GBD estimates. This estimate was updated for GBD2015 as new studies were added to the analysis and studies used previously were updated through continued follow-up. The newer estimates included several large studies that included exposure at lower levels of PM2.5. As a result, the TMREL for GBD2015 was \sim U(2.4, 5.9), lower than GBD2013's distribution \sim U(5.9, 8.7), which had the effect, all things being equal, of increasing the estimated attributable burden relative to the GBD2013 estimate

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Deaths attributable to WaSH SDG Capstone Appendix

Input data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with deaths attributable to unsafe water, sanitation, and hygiene (WaSH) (3.9.2).

For GBD 2015, the WaSH category is an aggregate of the risk estimates for water (6.1.1), hygiene (6.2.1b) and sanitation (6.2.1a). These are modeled independently and then aggregated together to generate the overall risk estimates for deaths attributable to WaSH.

Indicator 3.9.2

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.9 by 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination, is measured using SDG Health Index Indicator 3.9.2, deaths attributable to unsafe WaSH per 100,000 [PAF estimate].

Input data

This indicator includes estimates made independently from unsafe hygiene, unsafe sanitation, and unsafe water risk factors. For both water and sanitation, nationally representative surveys and censuses are the primary source for input data. For hygiene, a combination of data from nationally representative surveys and epidemiological studies are extracted and used as input data.

Modeling strategy

Each of the three risks that make up WaSH are modelled using a three step process that includes linear regression, spatial-temporal smoothing, and Gaussian Process Regression (ST-GPR). In modeling water and sanitation exposure, socio-demographic index was used as a fixed effect, with random effects placed at geographic locations including GBD 2015-specific super regions and regions. The final step of modelling was GPR, which incorporated source-specific uncertainty estimates and produced full times series of each risk estimate for each location.

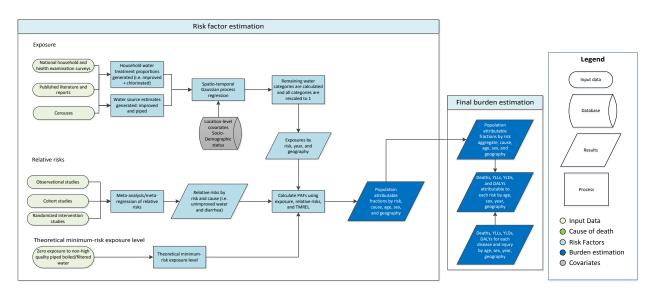
We estimate the PAF and burden of each of the 3 WaSH categories independently. This means that the calculation for PAF of all three is simply aggregating the PAF of each using this equation: 1 - ((1-PAF)(1-PAF)).

For additional information on the three risk models that are used to estimate Indicator 3.9.2, please refer to the write-ups for water, sanitation and hygiene in this appendix.

Unsafe Water SDG Capstone Appendix

Flowchart

Unsafe Drinking Water



Input data and Methodological Summary

Case Definition

For GBD 2015, exposure to unsafe water is defined based on reported primary water source used by the household and use of household water treatment (HWT) to improve the quality of drinking water before consumption. Water sources were defined as improved based on the JMP designation (The WHO), which includes piped water as improved water, and households with access to piped water connection to the house, yard, or plot were defined as having access to piped water supply. Solar treatment, chlorine treatment, boiling, or the use of filters were all assumed to be effective point-of-use household water treatments, and based on effect sizes published by Wolf et al. (2014) boiling or filtering was the most effective form of water treatment.

Input Data

The search for usable household surveys and censuses was conducted using the Global Health Data Exchange (GHDx) database. All surveys through December 2015 that provide household level micro-data on water source were added. Tabulated and report data was lower priority and was only updated when time permitted. HWT input data was limited to two large survey series (DHS and MICS) due to time constraints. An update to HWT input data is a top priority for estimating exposure to unsafe water in future iterations.

Modeling Strategy

Water source data is modeled in two distinct categories: household prevalence of improved water and household proportion of piped water within improved population in order to prevent the population with access to piped water from exceeding the population with access to improved water (which includes piped). HWT is modeled in 6 distinct categories based on the 3 water treatment categories (filtered/boiled, solar/chlorine, or untreated) and 2 water source categories (piped or improved). We have made no substantive changes in the modeling strategy from GBD 2013. By year and geography, each of the above categories are modeled using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR), which outputs full time series estimates for each GBD 2015 location. Socio-demographic status (SDS), an index metric that includes a measure of education and income level, was used as a fixed effect in the linear regression since it proved to have significant coefficients. Random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS proved to be the strongest predictor of unsafe water. Uncertainty in the estimates was initially formed based on standard deviation by survey, then propagated through ST-GPR modeling by means of confidence intervals around each data point that reflect the point-estimate specific variance.

Once models are fully vetted, full time series outputs from ST-GPR modeling are then converted from proportion to prevalence by year and geography and then rescaled to form 9 mutually exclusive categories that sum up to 1. The table below provides the final result of this rescaling.

Category	Definition
Unimproved, no HWT	Proportion of households that use unimproved source, and <i>do not</i> use any HWT to purify their drinking water.
Unimproved, chlorine/solar	Proportion of households that use unimproved source, and solar or chlorine treatment to purify their drinking water.
Unimproved, boil/filter	Proportion of households that use unimproved source, and boil or filter to purify their drinking water.
Improved water except piped, no HWT	Proportion of households that use improved sources other than piped water supply, and <i>do not</i> use any HWT to purify their drinking water.
Improved water except piped, chlorine/solar	Proportion of households that use improved sources other than piped water supply, and use solar or chlorine treatment to purify their drinking water.
Improved water except piped, boil/filter	Proportion of households that use improved sources other than piped water supply, and boil/filter their drinking water.
	Proportion of households that use piped water supply, and <i>do not</i> use any HWT to purify their drinking water
Piped water, no boil/filter	

Piped water, chlorine/solar	Proportion of households that use piped water supply, and <i>use</i> solar or chlorine water treatment to purify their drinking water.
Piped water, boil/filter	Proportion of households that use piped water supply, and boil or filter to purify their drinking water

Due to the nature of modeling piped water exposure as a proportion of total improved water access, we are limited in only using sources for piped water that also include total improved water values. It should be noted that high-income countries are assumed to have risk of unsafe water which could lead to an underestimate of unsafe water health burden in these countries. Another limitation in our analysis is the paucity of data on HWT. The inclusion of more location-specific data on water treatment utilization at the household level can greatly improve our estimates in future iterations. High-income countries were assumed to have 0 risk of unsafe water, and the TMREL was applied to these countries.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe water is defined as all households have access to high quality piped water that has been boiled or filtered before drinking. This exposure level is applied to all households in high-income countries, as well as households in countries in Southern Latin America region or Eastern Europe region that report piped water source and filtered or boiled water treatment.

Relative risks

GBD 2015 employ the same relative risks for unsafe water as was done for GBD 2013. There are 3 adverse health outcomes paired with unsafe water that comprise of diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Wolf et al. 2014 provides relative risk evidence for the relationship between unsafe water and diarrheal diseases. Wolf et al. 2014 publish relative risk values for water-source interventions and point-of-use treatment interventions separately so the combined effect of a source intervention and point-of-use intervention is assumed to be multiplicative in order to match GBD 2015 exposure definitions. In the absence of better data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease. Furthermore, it is assumed that there is a difference in piped water quality between Eastern Europe and Southern Latin America compared to rest of the developing world. As a result, we use effect sizes that are region-specific. The implication of this assumption is that no household in developing countries have access to high-quality piped water (TMREL). Please refer to appendix tables for more information on relative risk values and citations.

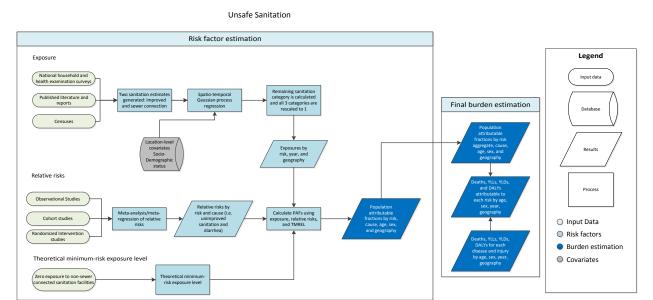
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Unsafe Sanitation SDG Capstone Appendix

Flowchart



Input data and Methodological Summary Case Definition

Exposure to unsafe sanitation were defined based on the primary toilet type used by households. Improved facilities are defined as such based on JMP designation (The WHO). Sewer connection toilets included flush toilets or any toilet with connection to the sewer or septic tank.

Input Data

The search for usable household surveys and censuses was conducted using the Global Health Data Exchange (GHDx) database. Searches were conducted from October 2015 to December 2015, with the final search household level micro-data on toilet type conducted on December 15, 2015. Due to the organized nature of the GHDx, the only search term used was "unsafe sanitation", which yielded just under 1400 results, of which 795 were extracted and used as inputs for modeling. Tabulated and report data was lower priority and was only updated when time permitted.

Modeling

There were no substantive changes in the modeling process from GBD 2015. Two distinct models are produced from sanitation data: prevalence of households with improved sanitation and the proportion of households with a sewer connection over the total improved sanitation population. Prevalence of households with a sewer connection is modeling with improved sanitation prevalence as the denominator in order to prevent the population with access to sewer connection from exceeding the population with access to improved sanitation. By each geography-year, both models are generated using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR), which outputs full time series estimates for each GBD 2015 location. Socio-

demographic status (SDS), an index metric that includes a measure of education and income level, was used as a fixed effect in the linear regression since it proved to have significant coefficients. Random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS proved to be the strongest predictor of unsafe sanitation. Uncertainty in the estimates was initially formed based on standard deviation by survey, then propagated through ST-GPR modeling by means of confidence intervals around each data point that reflect the point-estimate specific variance.

Once models are fully vetted, full time series outputs from ST-GPR modeling are then converted from proportion to prevalence by year and geography and then rescaled to form 3 mutually exclusive categories that sum up to 1. The table below provides the final result of this rescaling.

Category	Definition
	Proportion of households that use unimproved sanitation
Unimproved sanitation	facilities.
	Proportion of households that use improved sanitation
Improved sanitation, excluding sewer	facilities except those with sewer connection.
	Proportion of households that use toilet facilities with
Sanitation facilities with sewer connection	sewer connection.

Due to the nature of modeling sanitation with sewer connection as a proportion of total improved sanitation access, we are limited in only using sources for sewer connection that also include total improved sanitation values. It should be noted that high-income countries are assumed to have risk of unsafe sanitation which could lead to an underestimate of unsafe sanitation health burden in these countries. Another limitation that extends to the other two risk factors that comprise WaSH (unsafe water and unsafe hygiene) and can be improved upon in future iterations is taking into account covariance of access to water, sanitation and handwashing facilities. Currently, all three components of WaSH are modeled independently, which may lead to an overestimation of the burden of WaSH factors. High-income countries were assumed to have 0 risk of unsafe sanitation and the TMREL was applied to these countries.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe sanitation was defined as all households have access to a sanitation facility with sewer connection. Since it is assumed that all households in high-income countries have access to sewer-connected sanitation, this counterfactual exposure level is applied to all households in high-income countries.

Relative risks

GBD 2015 employ the same relative risks for unsafe water as was done for GBD 2013. Three adverse health outcomes are paired with unsafe sanitation, which comprise of diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Wolf et al. 2014 provides relative risk evidence for the relationship between unsafe sanitation and diarrheal diseases. In the absence of better data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease. Please refer to appendix tables for more information on relative risk values and citations.

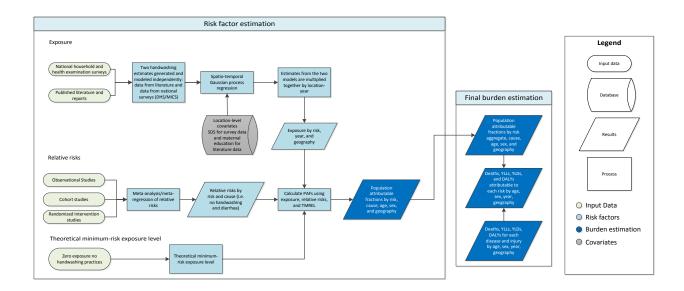
References

- 1. "Improved and Unimproved Water Sources and Sanitation Facilities." WHO / UNICEF Joint Monitoring Programme: Wat/san Categories. The WHO/UNICEF, n.d. Web. 08 June 2016
- 2. Wolf, Jennyfer, Annette Prüss-Ustün, Oliver Cumming, Jamie Bartram, Sophie Bonjour, Sandy Cairncross, Thomas Clasen, John M. Colford, Valerie Curtis, Jennifer De France, Lorna Fewtrell, Matthew C. Freeman, Bruce Gordon, Paul R. Hunter, Aurelie Jeandron, Richard B. Johnston, Daniel Mäusezahl, Colin Mathers, Maria Neira, and Julian P. T. Higgins. "Systematic Review: Assessing the Impact of Drinking Water and Sanitation on Diarrhoeal Disease in Low- and Middle-income Settings: Systematic Review and Meta-regression." Trop Med Int Health Tropical Medicine & International Health 19.8 (2014): 928-42. Web.

Unsafe Hygiene SDG Capstone Appendix

Flowchart

Unsafe Handwashing



Input data and Methodological Summary

Case Definition

Unsafe hygiene is composed of global handwashing practices. Handwashing is defined as the observed prevalence of handwashing with soap and water after using a toilet or after contact with excreta, including children's excreta. We estimate the burden of unsafe handwashing in both developed and developing settings.

Input Data

There were two main sources that were used in our estimation of handwashing practices, estimates from scientific literature and estimates from household survey series. Relevant literature on handwashing prevalence was gathered from a meta-analysis published recently by Freeman et al. (2014). Since water and soap availability data is very limited, only country-specific Demographic Health Surveys (DHS) and Malaria Indicator Survey Series (MICS) conducted after 2006 were able to be used as input data.

Modeling Strategy

Input data from scientific literature and input data from household survey series were modeled independently. Data from literature primarily measured a population's handwashing practices under ideal conditions, such as when water and soap was readily available. Additionally, these estimates from literature would likely be susceptible to acquiescence bias. Alternatively, data from DHS and MICS only provide insight into the availability of water, soap, and washing stations, which, alone, does not indicate how often a person may wash their hands after contact with excreta. Thus, after modeling data from

literature and data from surveys independently, these values were multiplied together by location-year in order to gain a more accurate representation of true handwashing prevalence.

Other than modeling literature data and survey data independently, we have made no substantive changes in the modeling strategy from GBD 2013. By year and location, estimates are generated using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR). When modeling survey input data, socio-demographic status (SDS), an index metric that includes a measure of education and income level, proved to have the most significant coefficient and was used as a fixed effect in the linear regression. To better inform our model that used scientific literature as input data, maternal education or average years of education for women ages 15-54, was implemented as a fixed effect in the linear regression. For both models, random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS and maternal education proved to be the strongest predictors of handwashing practices for their respective models. As mentioned above, once models were adequately vetted, full time series outputs from each of the models were multiplied together at each location-year.

A considerable limitation for when estimating handwashing practices for over 190 independent locations around the world is data sparseness. Even when data is published on handwashing prevalence, the definition is often altered from the GBD 2015 standard definition or it may only pertain to certain populations (such as hospital patients) and lacks representativeness at the geographic scale we require. The incorporation of questions about soap and water availability in DHS and MICS has added muchneeded information but there remains a large data gap that must be filled if we are to become more certain in handwashing estimates.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe hygiene is defined as all households engaging in handwashing with soap practices after any contact with excreta, including children's excreta.

Relative risks

GBD 2015 use the same relative risks for unsafe hygiene as was done for GBD 2013. There are 3 adverse health outcomes paired with unsafe hygiene that include diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Freeman et al. 2014 provides relative risk evidence for the relationship between unsafe hygiene and diarrheal diseases. In the absence of adequate data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease based on analogous transmission pathways (feco-oral pathway). Please refer to appendix tables for more information on relative risk values and citations.

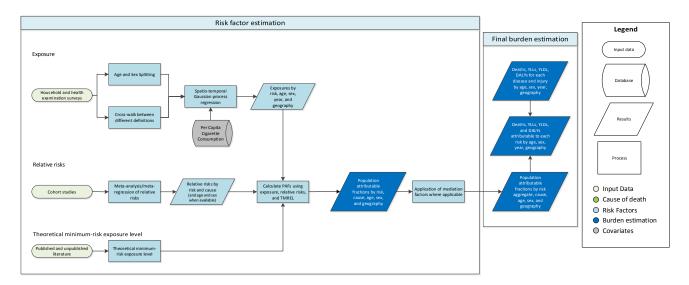
References

1. Freeman, M. C., Stocks, M. E., Cumming, O., Jeandron, A., Higgins, J. P., Wolf, J., Curtis, V. (2014). Systematic review: Hygiene and health: Systematic review of handwashing practices worldwide and update of health effects. *Trop Med Int Health Tropical Medicine & International Health, 19*(8), 906-916. doi:10.1111/tmi.12339

Smoking SDG Capstone Appendix

Flowchart

Tobacco Smoke



Input Data and Methodological Summary

Indicator definition

This modeling strategy encompassed the SDG health-related indicator associated with smoking prevalence (3.a.1).

Indicator 3.a.1

As a component of SDG Goal 3. Ensure healthy lives and promote well-being for all at all ages, SDG Target 3.a, strengthen the implementation of the World Health Organization Framework Convention on Tobacco Control in all countries, as appropriate, is measured using SDG Indicator 3.a.1, age-standardized prevalence of current tobacco use (smoking) among populations aged 18 and older.

Definition

We used the Smoking Impact Ratio (SIR) for modeling burden attributable to smoking for cancers, chronic obstructive pulmonary disease (COPD), interstitial lung disease, other chronic respiratory diseases, and pneumoconiosis. SIR is the population lung cancer mortality in excess of lung cancer mortality among never-smokers, relative to excess lung-cancer mortality observed in a known reference group of smokers. Currently, SIR is adjusted to account for differences in baseline never-smoker lung cancer mortality across geography, age, and sex, but not for differences across time.

We used 5-year lagged smoking prevalence, for modeling burden attributable to smoking for cardiovascular diseases, TB, diabetes, lower respiratory infections, asthma, cataracts, macular

degeneration, fractures, rheumatoid arthritis, and peptic ulcer disease. Smoking is a dichotomous exposure defined as current daily use of smoked tobacco.

A full list of outcomes included in GBD 2015 and their exposure definition is available in the table below.

Outcome	Exposure
Atrial fibrillation and flutter	5-year lagged smoking prevalence
Aortic aneurysm	5-year lagged smoking prevalence
Hypertensive heart disease	5-year lagged smoking prevalence
Ischemic heart disease	5-year lagged smoking prevalence
Other cardiovascular and circulatory diseases	5-year lagged smoking prevalence
Peripheral vascular disease	5-year lagged smoking prevalence
Hemorrhagic stroke	5-year lagged smoking prevalence
Ischemic stroke	5-year lagged smoking prevalence
Diabetes	5-year lagged smoking prevalence
Lower respiratory infections	5-year lagged smoking prevalence
Asthma	5-year lagged smoking prevalence
Tuberculosis	5-year lagged smoking prevalence
Peptic ulcer disease*	5-year lagged smoking prevalence
Rheumatoid arthritis*	5-year lagged smoking prevalence
Cataract*	5-year lagged smoking prevalence
Macular degeneration*	5-year lagged smoking prevalence
Hip fracture*	5-year lagged smoking prevalence
Non-hip fracture*	5-year lagged smoking prevalence
Bladder cancer	Smoking Impact Ratio (SIR)
Colon and rectum cancer	Smoking Impact Ratio (SIR)
Esophageal cancer	Smoking Impact Ratio (SIR)
Kidney cancer	Smoking Impact Ratio (SIR)
Leukemia	Smoking Impact Ratio (SIR)
Liver cancer	Smoking Impact Ratio (SIR)
Tracheal, bronchus, and lung cancer	Smoking Impact Ratio (SIR)
Lip and oral cavity cancer	Smoking Impact Ratio (SIR)
Nasopharynx cancer	Smoking Impact Ratio (SIR)
Pancreatic cancer	Smoking Impact Ratio (SIR)
Stomach cancer	Smoking Impact Ratio (SIR)
Larynx cancer*	Smoking Impact Ratio (SIR)
Chronic obstructive pulmonary disease	Smoking Impact Ratio (SIR)
Interstitial lung disease and pulmonary sarcoidosis	Smoking Impact Ratio (SIR)
Other chronic respiratory diseases	Smoking Impact Ratio (SIR)
Pneumoconiosis	Smoking Impact Ratio (SIR)

^{*} New outcome in GBD 2015

Input data

Consistent with GBD 2013, we used nationally representative survey data to estimate smoking prevalence. Survey and report data identified in the Global Health Data Exchange (GHDx), the WHO InfoBase, and the International Smoking Statistics (ISS) Database.

Inclusion Criteria

- Nationally representative
- Report current use of any of the following frequency-type combinations:
 - o Daily use of smoked tobacco
 - o Any use (both daily and occasional) of smoked tobacco
 - o Daily use of cigarettes
 - o Any use (both daily and occasional) of cigarettes
 - o Daily use of any tobacco (both smoked and smokeless)
 - Any use (both daily and occasional) of any tobacco (both smoked and smokeless)
 - o Daily use of any tobacco excluding cigarettes
- Report data within the time period of January 1, 1980 December 31, 2015 for any geography estimated in the GBD framework
- Smoking prevalence reported among individuals ages 10+

Global Health Data Exchange (GHDx)

Sources were identified through a systematic search of the GHDx.

• Search Terms (Keywords): Tobacco Use

• Time Period: January 1, 1980 – December 31, 2015

Data Type: Survey OR ReportSearch Date: February 16, 2016

Out of 3,912 sources identified in the GHDx, 2,664 sources were included.

WHO InfoBase and International Smoking Statistics (ISS) Database

An effort was made to replace database-derived estimates used in GBD 2013 with original extractions from primary data sources.

Outliers

Throughout the modeling process, data were assessed for bias and outliers were flagged. A data point was flagged as a candidate outlier if it was not consistent with the majority of other data points in a country with respect to level, age-pattern, sex-pattern, or temporal trend. In data-scarce countries, data points were also compared to data from other countries in a region. Candidate outliers were scrutinized for potential sources of bias and were ultimately excluded if the point or source was deemed to not be representative.

Modeling Strategy

Data Extraction

When possible, we extracted individual smoking status for all available frequency-type categories (listed above) from person-level microdata and collapsed these data to produce prevalence estimates in the standard GBD 5-year age-sex groups. If microdata were unavailable we extracted the most granular age-sex groups available from survey reports. Any available measures of uncertainty were extracted, including standard error, confidence or uncertainty intervals, and sample size.

Data Preparation: Crosswalking

Regressions to crosswalk other frequency-type categories to the gold-standard definition of daily use of smoked tobacco were estimated in the form:

$$p_{\text{daily-smoked,k}} = \beta_1 p_{i,k} + \epsilon_k$$

where $p_{daily\text{-}smoked,k}$ is the prevalence of daily smoking reported in survey k, and $p_{i,k}$ is the prevalence of an alternative frequency-type combination i also reported in survey k. Consistent with previous GBD smoking crosswalks, the intercept was omitted from the regression. The estimated regression coefficient β_1 was used to crosswalk alternative frequency-type categories to the gold-standard daily smoking definition in sources only providing the alternative category. Predication error at the data-point level was used to propagate uncertainty and was calculated using the following equation:

$$PE_k = \sigma_{\epsilon}^2 + X_k^2 var(\hat{\beta})$$

Compared to the separate frequency and type crosswalks used in GBD 2013, the combined frequency-type crosswalk used in GBD 2015 represents an improvement because patterns in frequency that may vary by type and patterns in type that may vary by frequency are captured.

Data Preparation: Age and Sex Splitting

Report data provided in age groups wider than the standard GBD 5-year age groups or as both sexes combined were split using the approach used in Ng et al. Briefly, age-sex patterns were identified using sources with data on multiple age-sex groups and these patterns were applied to split aggregated report data. Uncertainty in the age-sex split was propagated by multiplying the standard error of the data (including the predication error of the crosswalk) by the square root of the number of splits performed.

Modeling: Linear Model

After data preparation, the dataset consisted of prevalence estimates of daily smoked tobacco use in standard GBD country-year-age-sex groups. The mean function used in ST-GPR was estimated using the following hierarchical mixed-effects linear regression, run separately by sex:

$$logit(p_{c,a,t}) = \beta_0 + \beta_1 CPC_{c,t} + \sum_{k=2}^{16} \beta_k I_{A[a]} + \alpha_s + \alpha_r + \alpha_c + \epsilon_{c,a,t}$$

where $CPC_{c,t}$ is the annual tobacco consumption per capita covariate, $I_{A[a]}$ is a dummy variable indicating specific age group A that the prevalence point $p_{c,a,t}$ is capturing, and α_s , α_r , and α_c are super region, region, and country-specific random effects.

Modeling: Spatio-Temporal Gaussian Process Regression (ST-GPR)

The estimated mean function was then propagated through the ST-GPR framework to obtain 1,000 draws of smoking prevalence estimates for each location, year, age, and sex. Parameter selection for the ST-GPR hyper-parameters were selected through out-of-sample cross-validation using the strategy described elsewhere in this appendix.

Smoking Impact Ratio Estimation

We have made no substantive changes in the SIR estimation strategy from GBD 2013. The only change in input data for estimating never-smoker lung-cancer mortality was to update data from the China Kadoorie Biobank prospective cohort to include follow-up through 2014. Country-year-age-sex specific lung cancer mortality rates are derived from GBD 2015 Cause of Death estimation and detailed in that Capstone's appendix. The formula for calculating SIR is:

$$SIR = \frac{C_{LC} - N_{LC}}{S_{LC}^* - N_{LC}^*} \times \frac{N_{LC}^*}{N_{LC}}$$

C_{LC}: age-sex-specific lung cancer mortality rate in the population of interest

 N_{LC} : age-sex-specific lung cancer mortality rate of never-smokers in the population of interest S^*_{LC} : age-sex-specific lung cancer mortality rate for life-long smokers in a reference population N^*_{LC} : age-sex-specific lung cancer mortality rate for never smokers in the reference population

Theoretical minimum-risk exposure level

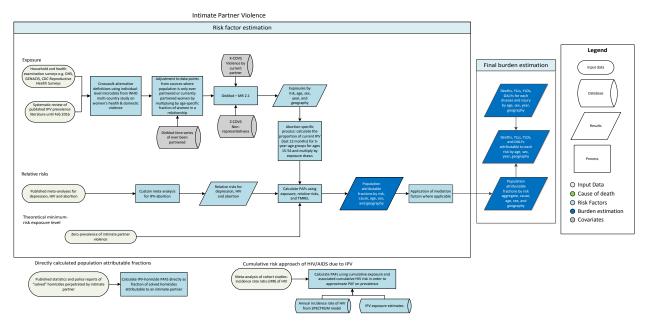
The theoretical minimum-risk exposure level is that no one in the population smokes tobacco; that is, the smoking impact ratio is zero and smoking prevalence is zero.

Relative risk

We have made no substantive updates to relative risks for outcomes included in GBD 2013. The following outcomes using 5-year lagged smoking prevalence as the exposure were added in GBD 2015: peptic ulcer disease, rheumatoid arthritis, cataracts, macular degeneration, hip fracture, and non-hip fracture. Larynx cancer was the only new outcome added using SIR as the exposure. Relative risks for rheumatoid arthritis, cataracts, and macular degeneration were derived from recent published meta-analyses. We performed out own meta-analyses of prospective cohort studies to derive relative risks for peptic ulcer disease, hip fracture, and non-hip fracture. We used Kontis et al.'s re-analysis of CPS-II smokers for the relative risk of larynx cancer.

Intimate Partner Violence SDG Capstone Appendix

Flowchart



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the SDG health-related indicator associated with prevalence of intimate partner violence (5.2.1).

Indicator 5.2.1

As a component of SDG Goal 5. Eliminate all forms of violence against all women and girls in the public and private spheres, including trafficking and sexual and other types of exploitation, SDG Target 5.2 eliminate all forms of violence against all women and girls in the public and private spheres, including trafficking and sexual and other types of exploitation, is measured using SDG Indicator 5.2.1, Prevalence of women aged 15 years and older who experienced intimate partner violence.

Case Definition

The case definition for intimate partner violence (IPV) is ever experienced one or more acts of physical and/or sexual violence by a current or former intimate partner since the age of 15 years. Estimated in females only because IPV is more common in females and there is more evidence quantifying the associated risk for health outcomes.

Physical violence is defined as: being slapped or having something thrown at you that could hurt
you, being pushed or shoved, being hit with a fist or something else that could hurt, being kicked,
dragged, or beaten up, being choked or burnt on purpose, and/or being threatened with or
actually having a gun, knife, or other weapon used on you.

- Sexual violence is defined as: being physically forced to have intercourse when you did not want
 to, having sexual intercourse because you were afraid of what your partner might do, and/or
 being forced to do something that you found humiliating or degrading (the definition of
 humiliating and degrading may vary across studies depending on the regional and cultural
 setting).
- Intimate partner is defined as: a partner to whom you are married or with whom you cohabit. In countries where people date, dating partners will also be considered (a partner with whom you have an intimate (sexual) relationship with but are not married to or cohabiting).

Input data

A systematic review of the intimate partner violence prevalence literature was conducted in Pubmed for anything published between November 2014 and February 2016. The following search terms were used to conduct the systematic review:

((("health surveys"[MeSH Terms] AND prevalence[Title/Abstract]) OR ("sentinel surveillance"[MeSH Terms] AND prevalence[Title/Abstract]) OR ("prevalence"[Title/Abstract] AND cross sectional studies[MeSH Terms])) AND (abuse, sexual[MeSH Terms] OR domestic violence[MeSH Terms] OR abuse, partner[MeSH Terms] OR abuse, spousal[MeSH Terms] OR rape[MeSH Terms]) NOT ("comment"[Publication Type] OR "letter"[Publication Type] OR "editorial"[Publication Type]))

This query produced 92 results, and of these, 33 data points were extracted for 13 different countries. In addition to literature, we supplemented this data with surveys tagged with "intimate partner violence" in the GHDx. Some of the big survey series that were updated or newly added include: all new Demographic and Health surveys, the National Youth Risk Behavior Survey, the Gender, Alcohol and Culture International Study (GENACIS), the CDC Reproductive Health Surveys, Mexican National Addiction Survey, USA Collaborative Psychiatric Epidemiology Surveys, and the Brazil National Alcohol and Drug Survey.

We get the proportion of solved homicides that were perpetrated by an intimate partner from crime statistics and police reports. For GBD 2013, the main source of these crime statistics and police reports came from an IPV-homicide systematic review in the Lancet in 2013.

In GBD 2015, an updated systematic review was done for IPV homicide sources in PubMed through April 2016. The query used for this Pubmed search was:

((IPV[All Fields] OR ("intimate partner violence"[MeSH Terms] OR ("intimate"[All Fields] AND "partner"[All Fields] AND "violence"[All Fields]) OR "intimate partner violence"[All Fields])) AND (("homicide"[MeSH Terms] OR "homicide"[All Fields])) OR femicide[All Fields])) AND ("2013/01/01"[PDAT] : "3000/12/31"[PDAT])

These literature sources were supplemented with sources from the GHDx that were tagged with Intimate partner violence AND Homicide.

Modeling strategy

For GBD 2015, we use three distinct approaches to estimate burden attributable to IPV, including 1) the traditional exposure and relative risk to PAF method for depression, suicide and abortion; 2) the direct

PAF approach for estimating the proportion of homicides that are perpetrated by an intimate partner; and 3) a cumulative risk approach for estimating the burden of HIV/AIDS attributable to IPV.

Estimating attributable burden to IPV for depression, suicide and abortion

Before upload to DisMod, we first adjust data with variable recall periods (previous 12 months versus lifetime), type of violence (sexual, physical, or both) and severity (severe only versus all levels). To convert data to our gold standard definition of ever having experienced any IPV, we use data from the WHO multi-country violence against women surveys to construct crosswalk regressions. The dependent variable in each of these regression was ever any IPV (gold standard), while the key independent variable was one of the 11 alternative metrics of IPV that were represented in our dataset:

- 1. Physical IPV in the past 12 months
- 2. Sexual IPV in the past 12 months
- 3. Severe IPV in the past 12 months
- 4. Severe physical IPV in the past 12 months
- 5. Severe sexual IPV in the past 12 months
- 6. Any IPV (physical and/or sexual) in the past 12 months
- 7. Ever any physical IPV
- 8. Ever any sexual IPV
- 9. Ever any severe IPV
- 10. Ever severe physical IPV
- 11. Ever severe sexual IPV

For alternate metrics 1-6 there is likely to be a relationship between current exposure and age. For these metrics we included a series of age dummies:

$$logit(GSait) = \beta + \beta 1 logit(ALTait) + \beta 2 Ia + \varepsilon$$

For alternate metrics 7-11, we ran the following regression:

$$logit(GSit) = \beta 0 + \beta 1 logit(ALTit) + \varepsilon$$

where GS refers to the gold standard metric of IPV prevalence, ALT is the alternate metric of IPV prevalence, *Ia* refers to the complete set of age-group indicators, *a* refers to an age-group, *i* refers to a country, and *t* refers to year. We included age-group indicators in the first six regressions because we expected the prevalence of recent IPV to vary by age. Using the intercepts, coefficients, and variance-covariance matrix from each of these eleven regressions, we were able to convert all of the alternate metrics of IPV prevalence in our dataset to estimates of "ever any IPV". We eliminated observations based on alternate metrics of IPV which came from studies that also provided estimates of IPV based on the gold standard definition (i.e. duplicates).

After applying crosswalks to the alternate metrics of IPV in the manner described above, we made an additional adjustment to the subset of our data that was based on only ever-partnered, currently partnered women currently married women or ever married women. To adjust these values so that they reflected IPV prevalence in the entire female population, regardless of partnered status, we multiplied estimates from these studies by the age-specific fraction of women who had ever been partnered.

An updated time series was generated in GBD 2015 using MICS and DHS data in a single parameter DisMod model to reflect the most recent data on proportion of women that have ever been partnered. This revised time series was used to adjust values for surveys with restricted partner status to reflect the prevalence among all women in the population.

After these pre-DisMod crosswalks and adjustments, a single-parameter prevalence model was run in DisMod with age mesh points at 0 14 15 20 30 40 50 60 80 & 100. A study-level covariate fixed effect (x-cov) was used to adjust data points where the survey question used to calculate prevalence only asked about violence perpetrated by the woman's spouse. A study-level fixed effect on integrand variance (z-cov) to indicate whether a study was nationally representative or not was used to account for the heterogeneity introduced by studies that are not generalizable to the entire population.

We tried using alcohol liters per capita, prevalence of binge drinking, and prevalence of male binge drinking in the GBD 2015 model as national-level fixed effects, but they were not significant so they were ultimately dropped.

Direct PAF for female homicides

The burden of homicides attributable to intimate partner violence is modeled as a direct PAF.

Input data all fed into a single-parameter proportion DisMod model, which has age mesh points at 0 10 20 45 & 100. The model has a study-level covariate fixed effect on integrand value (x-cov) for sources just including police reported homicides. We also included a study-level fixed effect on integrant variance (z-cov) to indicate whether a study was nationally representative or not.

In GBD 2015, we added prevalence of binge drinking to the model as a country-level covariate.

Cumulative risk approach for PAF of HIV/AIDS due to IPV

The third and final modelling approach that we used to assess burden attributable to intimate partner violence was a cumulative risk approach to measure the burden of HIV/AIDS attributable to IPV.

The approach itself remained the same in GBD 2015, but included updated intimate partner violence exposure numbers from the DisMod model described above, as well as revised HIV incidence numbers.

From the literature we have information on the incidence rate ratio (IRR) of HIV incidence from two cohort studies (Jewkes et al, Lancet 2010 & Kouyoumdijian, et al AIDS 2013). As we measure burden based on deaths and prevalence, we need to be able to quantify attributable fractions on prevalence and death rather than incidence. To get a PAF on prevalence we need to consider the history of exposure to IPV and the accumulated associated risk of incident HIV due to IPV, relative to the overall risk of HIV at the population level. The ratio of cumulative IPV-attributable HIV incidence to total HIV incidence is an approximation of the relevant PAF on HIV prevalence and we will assume this PAF can also applied to mortality.

$$\frac{\textit{Cumulative HIV incidence due to IPV}}{\textit{Cumulative HIV incidence overall}} = \frac{1 - \prod_{a=o}^{a=n} \left(1 - \textit{PAF}_{ay} * \textit{I}_{ay}\right)}{1 - \prod_{a=o}^{a=n} \left(1 - \textit{I}_{ay}\right)}$$

Where:

I = annual incidence rate of HIV

$$a = age (15-84)$$

 $y = year (1980-2013)$
 $PAF_{HIV\ incidence} = \frac{[Prevalence\ of\ IPV]_{ay^*}(IRR-1)}{[Prevalence\ of\ IPV]_{ay^*}(IRR-1)+1}$

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level is zero exposure to intimate partner violence, as defined above.

Relative risks

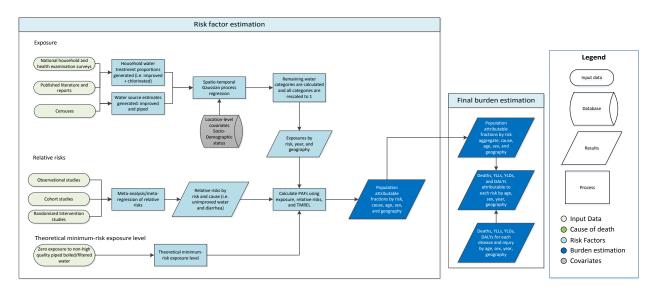
We estimate burden attributable to IPV for abortion, depression, suicide, interpersonal violence (i.e. homicide) and HIV incidence. We have added HIV as an outcome for GBD 2013 in response to bolstered causal evidence from a second prospective study published in 2013 (Kouyoumdjian, 2013). We use a pooled incidence rate ratio (IRR) of 1.59 (95% CI 1.3-1.94) from a meta-analysis of the two available prospective studies as of date.

The relative risks for depression and suicide come from a systematic review of longitudinal studies assessing intimate partner violence and incident depressive symptoms and suicide attempts. For the relative risk for IPV-abortion, we ran a custom meta-analysis in GBD 2013 that we continued to use in GBD 2015. An important methodological note with IPV-abortion is that we must apply the pooled relative risk for abortion to the current prevalence of IPV (in the previous 12 months), rather than lifetime prevalence. This is because the relevant exposure for abortion would be recent IPV, and because the case definition for all but one of the RR component studies was physical or sexual IPV in the past year.

Unsafe Water SDG Capstone Appendix

Flowchart

Unsafe Drinking Water



Input data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with water (SEV) (6.1.1).

Indicator 6.1.1

As a component of SDG Goal 6. Ensure availability and sustainable management of water and sanitation for all, SDG Target 6.1, by 2030, achieve universal and equitable access to safe and affordable drinking water for all, is measured using SDG Indicator 6.1.1, risk-weighted prevalence of population using unsafe/unimproved water sources.

Case Definition

For GBD 2015, exposure to unsafe water is defined based on reported primary water source used by the household and use of household water treatment (HWT) to improve the quality of drinking water before consumption. Water sources were defined as improved based on the JMP designation (The WHO), which includes piped water as improved water, and households with access to piped water connection to the house, yard, or plot were defined as having access to piped water supply. Solar treatment, chlorine treatment, boiling, or the use of filters were all assumed to be effective point-of-use household water treatments, and based on effect sizes published by Wolf et al. (2014) boiling or filtering was the most effective form of water treatment.

Input Data

The search for usable household surveys and censuses was conducted using the Global Health Data Exchange (GHDx) database. All surveys through December 2015 that provide household level micro-data on water source were added. Tabulated and report data was lower priority and was only updated when

time permitted. HWT input data was limited to two large survey series (DHS and MICS) due to time constraints. An update to HWT input data is a top priority for estimating exposure to unsafe water in future iterations.

Modeling Strategy

Water source data is modeled in two distinct categories: household prevalence of improved water and household proportion of piped water within improved population in order to prevent the population with access to piped water from exceeding the population with access to improved water (which includes piped). HWT is modeled in 6 distinct categories based on the 3 water treatment categories (filtered/boiled, solar/chlorine, or untreated) and 2 water source categories (piped or improved). We have made no substantive changes in the modeling strategy from GBD 2013. By year and geography, each of the above categories are modeled using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR), which outputs full time series estimates for each GBD 2015 location. Socio-demographic status (SDS), an index metric that includes a measure of education and income level, was used as a fixed effect in the linear regression since it proved to have significant coefficients. Random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS proved to be the strongest predictor of unsafe water. Uncertainty in the estimates was initially formed based on standard deviation by survey, then propagated through ST-GPR modeling by means of confidence intervals around each data point that reflect the point-estimate specific variance.

Once models are fully vetted, full time series outputs from ST-GPR modeling are then converted from proportion to prevalence by year and geography and then rescaled to form 9 mutually exclusive categories that sum up to 1. The table below provides the final result of this rescaling.

Category	Definition
Unimproved, no HWT	Proportion of households that use unimproved source, and <i>do not</i> use any HWT to purify their drinking water.
Unimproved, chlorine/solar	Proportion of households that use unimproved source, and solar or chlorine treatment to purify their drinking water.
Unimproved, boil/filter	Proportion of households that use unimproved source, and boil or filter to purify their drinking water.
Improved water except piped, no HWT	Proportion of households that use improved sources other than piped water supply, and <i>do not</i> use any HWT to purify their drinking water.

Improved water except piped, chlorine/solar	Proportion of households that use improved sources other than piped water supply, and use solar or chlorine treatment to purify their drinking water.
Improved water except piped, boil/filter	Proportion of households that use improved sources other than piped water supply, and boil/filter their drinking water.
Piped water, no boil/filter	Proportion of households that use piped water supply, and <i>do not</i> use any HWT to purify their drinking water
Piped Water, no bon/inter	
Piped water, chlorine/solar	Proportion of households that use piped water supply, and <i>use</i> solar or chlorine water treatment to purify their drinking water.
Piped water, boil/filter	Proportion of households that use piped water supply, and boil or filter to purify their drinking water

Due to the nature of modeling piped water exposure as a proportion of total improved water access, we are limited in only using sources for piped water that also include total improved water values. It should be noted that high-income countries are assumed to have risk of unsafe water which could lead to an underestimate of unsafe water health burden in these countries. Another limitation in our analysis is the paucity of data on HWT. The inclusion of more location-specific data on water treatment utilization at the household level can greatly improve our estimates in future iterations. High-income countries were assumed to have 0 risk of unsafe water, and the TMREL was applied to these countries.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe water is defined as all households have access to high quality piped water that has been boiled or filtered before drinking. This exposure level is applied to all households in high-income countries, as well as households in countries in Southern Latin America region or Eastern Europe region that report piped water source and filtered or boiled water treatment.

Relative risks

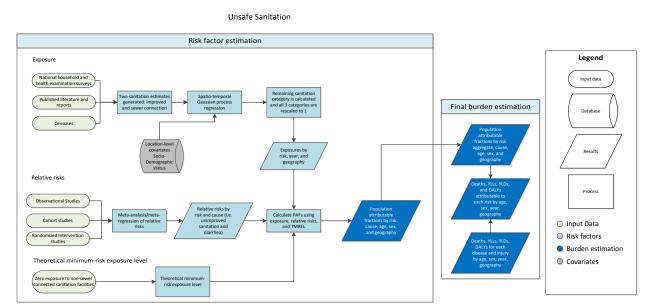
GBD 2015 employ the same relative risks for unsafe water as was done for GBD 2013. There are 3 adverse health outcomes paired with unsafe water that comprise of diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Wolf et al. 2014 provides relative risk evidence for the relationship between unsafe water and diarrheal diseases. Wolf et al. 2014 publish relative risk values for water-source interventions and point-of-use treatment interventions separately so the combined effect of a source intervention and point-of-use intervention is assumed to be multiplicative in order to match GBD 2015 exposure definitions. In the absence of better data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease. Furthermore, it is assumed that there is a difference in piped water quality between Eastern Europe and Southern Latin America compared to rest of the developing world. As a result, we use effect sizes that are region-specific. The implication of this assumption is that no household in developing countries have access to high-quality piped water (TMREL). Please refer to appendix tables for more information on relative risk values and citations.

References

- 1. "Improved and Unimproved Water Sources and Sanitation Facilities." WHO / UNICEF Joint Monitoring Programme: Wat/san Categories. The WHO/UNICEF, n.d. Web. 08 June 2016
- 2. Wolf, Jennyfer, Annette Prüss-Ustün, Oliver Cumming, Jamie Bartram, Sophie Bonjour, Sandy Cairncross, Thomas Clasen, John M. Colford, Valerie Curtis, Jennifer De France, Lorna Fewtrell, Matthew C. Freeman, Bruce Gordon, Paul R. Hunter, Aurelie Jeandron, Richard B. Johnston, Daniel Mäusezahl, Colin Mathers, Maria Neira, and Julian P. T. Higgins. "Systematic Review: Assessing the Impact of Drinking Water and Sanitation on Diarrhoeal Disease in Low- and Middle-income Settings: Systematic Review and Meta-regression." Trop Med Int Health Tropical Medicine & International Health 19.8 (2014): 928-42. Web.

Unsafe Sanitation SDG Capstone Appendix

Flowchart



Input data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with sanitation (SEV) (6.2.1a).

Indicator 6.2.1a

As a component of SDG Goal 6. Ensure availability and sustainable management of water and sanitation for all, SDG Target 6.2, by 2030, achieve access to adequate and equitable sanitation and hygiene for all and end open defecation, paying special attention to the needs of women and girls and those in vulnerable situations, is measured using SDG Indicator 6.2.1a, risk-weighted prevalence of population using unsafe sanitation practices.

Case Definition

Exposure to unsafe sanitation were defined based on the primary toilet type used by households. Improved facilities are defined as such based on JMP designation (The WHO). Sewer connection toilets included flush toilets or any toilet with connection to the sewer or septic tank.

Input Data

The search for usable household surveys and censuses was conducted using the Global Health Data Exchange (GHDx) database. Searches were conducted from October 2015 to December 2015, with the final search household level micro-data on toilet type conducted on December 15, 2015. Due to the organized nature of the GHDx, the only search term used was "unsafe sanitation", which yielded just

under 1400 results, of which 795 were extracted and used as inputs for modeling. Tabulated and report data was lower priority and was only updated when time permitted.

Modeling

There were no substantive changes in the modeling process from GBD 2015. Two distinct models are produced from sanitation data: prevalence of households with improved sanitation and the proportion of households with a sewer connection over the total improved sanitation population. Prevalence of households with a sewer connection is modeling with improved sanitation prevalence as the denominator in order to prevent the population with access to sewer connection from exceeding the population with access to improved sanitation. By each geography-year, both models are generated using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR), which outputs full time series estimates for each GBD 2015 location. Sociodemographic status (SDS), an index metric that includes a measure of education and income level, was used as a fixed effect in the linear regression since it proved to have significant coefficients. Random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS proved to be the strongest predictor of unsafe sanitation. Uncertainty in the estimates was initially formed based on standard deviation by survey, then propagated through ST-GPR modeling by means of confidence intervals around each data point that reflect the point-estimate specific variance.

Once models are fully vetted, full time series outputs from ST-GPR modeling are then converted from proportion to prevalence by year and geography and then rescaled to form 3 mutually exclusive categories that sum up to 1. The table below provides the final result of this rescaling.

Category	Definition
Unimproved sanitation	Proportion of households that use unimproved sanitation facilities.
	Proportion of households that use improved sanitation
Improved sanitation, excluding sewer	facilities except those with sewer connection. Proportion of households that use toilet facilities with
Sanitation facilities with sewer connection	sewer connection.

Due to the nature of modeling sanitation with sewer connection as a proportion of total improved sanitation access, we are limited in only using sources for sewer connection that also include total improved sanitation values. It should be noted that high-income countries are assumed to have risk of unsafe sanitation which could lead to an underestimate of unsafe sanitation health burden in these countries. Another limitation that extends to the other two risk factors that comprise WaSH (unsafe water and unsafe hygiene) and can be improved upon in future iterations is taking into account

covariance of access to water, sanitation and handwashing facilities. Currently, all three components of WaSH are modeled independently, which may lead to an overestimation of the burden of WaSH factors. High-income countries were assumed to have 0 risk of unsafe sanitation and the TMREL was applied to these countries.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe sanitation was defined as all households have access to a sanitation facility with sewer connection. Since it is assumed that all households in high-income countries have access to sewer-connected sanitation, this counterfactual exposure level is applied to all households in high-income countries.

Relative risks

GBD 2015 employ the same relative risks for unsafe water as was done for GBD 2013. Three adverse health outcomes are paired with unsafe sanitation, which comprise of diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Wolf et al. 2014 provides relative risk evidence for the relationship between unsafe sanitation and diarrheal diseases. In the absence of better data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease. Please refer to appendix tables for more information on relative risk values and citations.

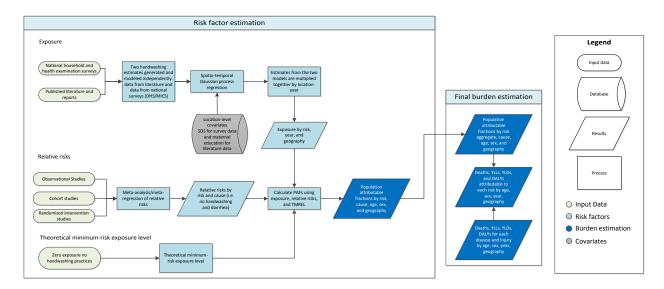
References

- 1. "Improved and Unimproved Water Sources and Sanitation Facilities." WHO / UNICEF Joint Monitoring Programme: Wat/san Categories. The WHO/UNICEF, n.d. Web. 08 June 2016
- 2. Wolf, Jennyfer, Annette Prüss-Ustün, Oliver Cumming, Jamie Bartram, Sophie Bonjour, Sandy Cairncross, Thomas Clasen, John M. Colford, Valerie Curtis, Jennifer De France, Lorna Fewtrell, Matthew C. Freeman, Bruce Gordon, Paul R. Hunter, Aurelie Jeandron, Richard B. Johnston, Daniel Mäusezahl, Colin Mathers, Maria Neira, and Julian P. T. Higgins. "Systematic Review: Assessing the Impact of Drinking Water and Sanitation on Diarrhoeal Disease in Low- and Middle-income Settings: Systematic Review and Meta-regression." Trop Med Int Health Tropical Medicine & International Health 19.8 (2014): 928-42. Web.

Unsafe Hygiene Capstone Appendix

Flowchart

Unsafe Handwashing



Input data and Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with hygiene [handwashing] (SEV) (6.2.1b).

Indicator 6.2.1b

As a component of SDG Goal 6. Ensure availability and sustainable management of water and sanitation for all, SDG Target 6.2, by 2030, achieve access to adequate and equitable sanitation and hygiene for all and end open defecation, paying special attention to the needs of women and girls and those in vulnerable situations, is measured using SDG Indicator 6.2.1b, risk-weighted prevalence of population with unsafe hygiene (no handwashing with soap).

Case Definition

Unsafe hygiene is composed of global handwashing practices. Handwashing is defined as the observed prevalence of handwashing with soap and water after using a toilet or after contact with excreta, including children's excreta. We estimate the burden of unsafe handwashing in both developed and developing settings.

Input Data

There were two main sources that were used in our estimation of handwashing practices, estimates from scientific literature and estimates from household survey series. Relevant literature on handwashing prevalence was gathered from a meta-analysis published recently by Freeman et al. (2014). Since water

and soap availability data is very limited, only country-specific Demographic Health Surveys (DHS) and Malaria Indicator Survey Series (MICS) conducted after 2006 were able to be used as input data.

Modeling Strategy

Input data from scientific literature and input data from household survey series were modeled independently. Data from literature primarily measured a population's handwashing practices under ideal conditions, such as when water and soap was readily available. Additionally, these estimates from literature would likely be susceptible to acquiescence bias. Alternatively, data from DHS and MICS only provide insight into the availability of water, soap, and washing stations, which, alone, does not indicate how often a person may wash their hands after contact with excreta. Thus, after modeling data from literature and data from surveys independently, these values were multiplied together by location-year in order to gain a more accurate representation of true handwashing prevalence.

Other than modeling literature data and survey data independently, we have made no substantive changes in the modeling strategy from GBD 2013. By year and location, estimates are generated using a 3-step modeling scheme of mixed effect linear regression followed by spatio-temporal Gaussian process regression (ST-GPR). When modeling survey input data, socio-demographic status (SDS), an index metric that includes a measure of education and income level, proved to have the most significant coefficient and was used as a fixed effect in the linear regression. To better inform our model that used scientific literature as input data, maternal education or average years of education for women ages 15-54, was implemented as a fixed effect in the linear regression. For both models, random effects were placed at GBD 2015 region and super-region levels.

The process of vetting and validating models was accomplished primarily through an examination of ST-GPR scatter plots by GBD 2015 location from 1990-2015. Any unfitting data points were re-inspected for error at the level of extraction and survey implementation, and subsequently excluded from analysis if deemed appropriate. In addition to SDS, a number of different potential fixed effects were considered, including lag-distributed income and urbanicity, but SDS and maternal education proved to be the strongest predictors of handwashing practices for their respective models. As mentioned above, once models were adequately vetted, full time series outputs from each of the models were multiplied together at each location-year.

A considerable limitation for when estimating handwashing practices for over 190 independent locations around the world is data sparseness. Even when data is published on handwashing prevalence, the definition is often altered from the GBD 2015 standard definition or it may only pertain to certain populations (such as hospital patients) and lacks representativeness at the geographic scale we require. The incorporation of questions about soap and water availability in DHS and MICS has added much-needed information but there remains a large data gap that must be filled if we are to become more certain in handwashing estimates.

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level for unsafe hygiene is defined as all households engaging in handwashing with soap practices after any contact with excreta, including children's excreta.

Relative risks

GBD 2015 use the same relative risks for unsafe hygiene as was done for GBD 2013. There are 3 adverse health outcomes paired with unsafe hygiene that include diarrheal diseases, typhoid fever, and paratyphoid fever. A meta-analysis by Freeman et al. 2014 provides relative risk evidence for the relationship between unsafe hygiene and diarrheal diseases. In the absence of adequate data, the relative risk for typhoid and paratyphoid fevers were assumed to be the same as the relative risk for diarrheal disease based on analogous transmission pathways (feco-oral pathway). Please refer to appendix tables for more information on relative risk values and citations.

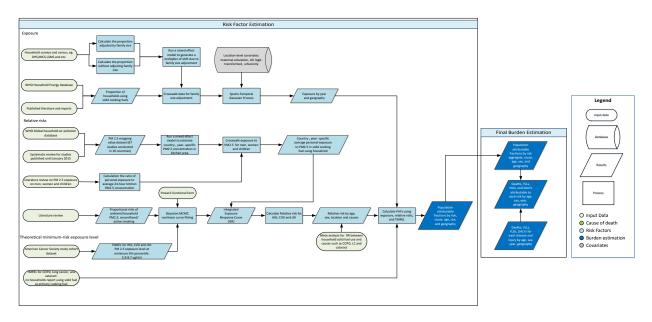
References

1. Freeman, M. C., Stocks, M. E., Cumming, O., Jeandron, A., Higgins, J. P., Wolf, J., Curtis, V. (2014). Systematic review: Hygiene and health: Systematic review of handwashing practices worldwide and update of health effects. *Trop Med Int Health Tropical Medicine & International Health*, 19(8), 906-916. doi:10.1111/tmi.12339

Household Air Pollution SDG Capstone Appendix

Flowchart

Household Air Pollution from Solid Fuels



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with risk weighted prevalence of population using unsafe cooking fuel (7.1.2).

Indicator 3.9.1

As a component of SDG Goal 7. Ensure access to affordable, reliable, sustainable, and modern energy for all, SDG Target 7.1, by 2030, ensure universal access to affordable, reliable and modern energy services, is measured using SDG Health Index Indicator 7.1.2, risk weighted prevalence of population using unsafe cooking fuel, which comes from household air pollution (HAP).

Case definition

Exposure to household air pollution from solid fuels (HAP) is defined as the proportion of households using solid cooking fuels. The definition of solid fuel in our analysis includes coal, wood, charcoal, dung, and agricultural residues.

Input data

Data were extracted from the standard multi-country survey series such as Demographic and Health Surveys (DHS), Living Standards Measurement Surveys (LSMS), Multiple Indicator Cluster Surveys (MICS), and World Health Surveys (WHS), as well as country-specific survey series such as Kenya Welfare Monitoring Survey and South Africa General Household Survey. To fill the gaps of data in surveys and censuses, we also downloaded and updated HAP estimates from WHO Energy Database and extracted

from literature through systematic review done in IHME. Each nationally or sub-nationally representative data point provided an estimate for the percentage of households using solid cooking fuels. Estimates for the usage of solid fuels for non-cooking purpose were excluded, i.e. primary fuels for lighting. The database, with estimates from 1980 to 2015, contained 685 studies from 150 countries. Updates to systematic reviews are performed on an ongoing schedule across all GBD causes and risk factors, an update for household air pollution will be performed in the next 1-2 iterations.

Modeling strategy

Household air pollution was modeled at household level using a three-step modeling strategy ST-GPR that uses linear regression, spatiotemporal regression and Gaussian Process Regression (GPR). The first step is a mixed-effect linear regression of logit-transformed proportion of households using solid cooking fuels. The linear model contains maternal education and proportion of population living in urban areas as covariates and has nested random effect by country, GBD region, and GBD super region respectively. The full ST-GPR process is specified elsewhere in this appendix.

Compared with GBD 2013, we have made changes in terms of the covariates utilized in the linear model. A variety of combinations of socioeconomic and environmental covariates in different transformation format were tested by running mixed-effect models with exposure data. The final list of covariates included in the exposure model are maternal education and the proportion of population living in urban area.

Theoretical minimum-risk exposure level

For outcomes where we extracted RR based on direct epidemiological evidence i.e. COPD, lung cancer, and cataract, TMREL was defined such that no households would report using solid fuel as their primary cooking fuel. For outcomes that utilize evidence based on the Integrated Exposure Response (IER), the TMREL is defined as uniform distribution between 33.3 and 41.9 ug/m^3. TMREL for household air pollution did not change from GBD 2013.

Relative risks

The disease-outcomes paired with household air pollution has not changed since GBD 2013. The list of outcomes paired with household air pollution has not changed since GBD 2013, which included lower respiratory infections (LRI), stroke, Ischemic Heart Disease (IHD), chronic obstructive pulmonary disease (COPD), lung cancer and cataract. The relative risks of all outcomes but not cataract were generated by using the integrated exposure-response functions (IER). The relative risks for cataract were extracted from a meta-analysis paper (1). The IER curves are updated to reflect the newly updated data and utilization of a new method that specified elsewhere.

PM2.5 mapping value

The relative risk estimates describing the association of HAP with outcomes including Ischemic Heart Disease (IHD), cardiovascular disease (CVD), and lower respiratory infections (LRI) were derived from the IER curves. This is done by first estimating the crosswalk values that map household use of solid fuel to PM2.5 exposure because the IER curve measures exposure using PM2.5. This step of the analysis relied on 67 studies conducted in 16 countries to generate the PM2.5 mapping values, which remain the same sources as GBD 2013. The PM2.5 exposure was then cross-walked to men, women and children by

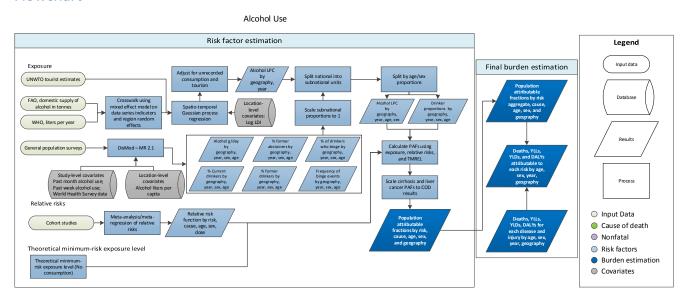
generating the ratio of personal exposure to average 24-hour kitchen PM2.5 concentration based on a study after the literature review in GBD 2013.

References

1. Smith KR, Bruce N, Balakrishnan K, Adair-Rohani H, Balmes J, Chafe Z, et al. Millions Dead: How Do We Know and What Does It Mean? Methods Used in the Comparative Risk Assessment of Household Air Pollution. Annu Rev Public Health. 2014;35(1):185–206.

Alcohol Capstone Appendix

Flowchart



Input Data and Methodological Summary

Exposure

Case definition

The impact of alcohol consumption on morbidity and mortality can be largely described by two separate but related dimensions. The 1st dimension is the individual level drinking and consists of four indicators;

- 1. Current drinkers, defined as the proportion of individuals who have consumed at least one alcoholic beverage (or some approximation) in the last 12 months.
- 2. Former drinkers, defined as the proportion of individuals who have ever consumed an alcoholic beverage, but not in the last 12 months.
- 3. Lifetime abstainers, defined as the proportion of individuals who have never consumed an alcoholic beverage.
- 4. Alcohol consumption (in grams per day), defined as grams of alcohol consumed by current drinkers, per day, over a 12 month period.

The 2nd dimension of alcohol consumption relates to the pattern of drinking and consists of two indicators:

- 5. Binge drinkers, defined as the proportion of drinkers who have had a binge event in the past 12 months. A binge event was defined as consuming 60 grams of alcohol (approximately five drinks or more) in a single occasion for males and 48 grams of alcohol in a single occasion for females.
- 6. Binge times, defined as the proportion of drinking events that are binge amongst binge drinkers i.e. the proportion of days that a binger has a binge event.

Input data

For GBD 2013, a systematic review of the literature was conducted to capture population survey data on all six alcohol use indicators. In summary, the search was conducted in three stages involving electronic searches of the peer-reviewed literature via PubMed, the grey literature and, expert consultation. Updates to systematic reviews via PubMed are performed on an ongoing schedule across all GBD causes and risk factors, an update for alcohol use will be performed in the next 1-2 iterations. For GBD 2015, stages two and three of the literature review were conducted, prioritizing countries for which subnational estimates were generated. The Global Health Exchange (GHDx), IHME's online database of health-related data, was searched for population survey data containing participant-level information from which we could formulate the required alcohol use indicators. Data-sources were included if they captured a sample representative of the geographic location under study and contained variables that could be used to formulate any of the six alcohol use indicators. Relevant survey variables from each data-source were documented in a Microsoft Excel codebook and extracted using STATA 13.1. A total of 629 potential data-sources were available in GhDx across countries with subnational locations, out of which 127 data-sources (66,108 data-points) were included across all six indicators.

To generate estimates of alcohol consumption in grams per day, data from population surveys were used in combination with estimates of per capita consumption from the Food and Agriculture Organization (FAO) [1] and the Global Information System on Alcohol and Health (GISAH database [2]) Per capita consumption is an aggregate measure of recorded, unrecorded, and tourist per capita consumption of alcohol (UNWTO database [3]) derived from sales, production, and other economic statistics. While population-based surveys provide accurate estimates of the prevalence of lifetime abstainers, former drinkers and current drinkers, they typically underestimate real alcohol consumption levels. As a result, the all-age, both-sex per capita consumption figures from the FAO and GISAH are considered to be a better estimate of overall volume of consumption. Per capita consumption, however, does not provide age- and sex-specific consumption estimates needed to compute alcohol-attributable burden of disease. Therefore, we use the age-sex pattern of consumption among drinkers modeled from the population survey data and the overall volume of consumption from FAO and GISAH to determine the total amount of alcohol consumed by country.

To generate estimates of alcohol consumption in liter per capita, raw inputs were obtained from FAOSTAT [1] and WHO GISAH database [2]. To provide more stable time trends in the model, FAO sales data was transformed to a lagged 5-year average. FAO data was used when WHO data wasn't available. Otherwise, FAO and WHO data was adjusted (crosswalked) by running a mixed effect model on the log average of the data with indicators for the FAO and WHO data series, as well as random effects on super region, region, country, and time. Each data point was adjusted by the predicted betas on super-region and region.

 $Log\ Average\ Data = D + (Super\ Region\ |\ D, Region\ |\ D, Country\ |\ D, Year\ |\ D)$

Transformed data = data * $e^{\widehat{\beta_1} + \widehat{\beta_3}}$

Where D = Indicator variable for data source

To generate uncertainty, a Lowess model was run on the adjusted data and the standard deviation between the difference of the Lowess smoothed model and the adjusted data points was used for data points missing uncertainty.

Unrecorded consumption was incorporated into the alcohol LPC data using estimates provided by the WHO [4]. WHO estimates were only reported for the years 1990, 2005, and 2010 so for missing years, estimates were interpolated. For years outside this range, unrecorded estimates were carried forward or backwards from the closest year. Unrecorded consumption estimates were reported in liters per capita so estimates were added to adjusted data points to account for unrecorded consumption.

Tourism data was obtained through the UNWTO [4]. A crosswalk was applied across different tourist categories, similar to the one used for FAO and WHO data, to estimate tourist proportions for a given country. Tourism consumption was incorporated after modeling unadjusted alcohol LPC as outlined below.

Data Preparation & Modeling strategy

DisMod-MR 2.1 was used to estimate country-, year-, age- and sex-specific proportions of current drinkers, former drinkers, lifetime abstainers, binge drinkers, and binge times; and alcohol consumption as a continuous variable in grams per day. We have made no substantive changes in the modeling strategy from GBD 2013. We ran single-parameter models for each alcohol use indicator and included a combination of location- and study-level covariates in each model. An alcohol liters per capita location-level covariate was used for all six indicators to assist in the predictive power of the models. Additionally, study-level covariates were used to accommodate for known sources of variability in the raw data. In the current drinkers, former drinkers, binge drinkers and binge times models, we included two covariates which adjusted estimates derived in the past week and past month towards those derived in the past year respectively. Estimates derived in the past year were considered to be the gold standard given the previously outlined definition for each indicator.

In the alcohol consumption model, we included a separate study-level covariate flagging data points derived from The World Health Organization's World Health Surveys (WHS) conducted across multiple countries. There was considerable variability in estimates derived from the WHS which may have been influenced by methodological differences in how alcohol use was captured. This study-level covariate looked for unsystematic bias between data-points and added more uncertainty onto those from the WHS. If other data-points causing higher or lower modelled output were identified during the modelling process for a given indicator, the plausibility of these data points was assessed and the study methodology reviewed. Data points with methodological limitations, for instance those derived from survey items not entirely representatively of the alcohol use indicators required, with small sample sizes, or derived from samples not entirely representative of the general population were excluded.

A spatial-temporal Gaussian process regression was used to model total alcohol in liters per capita (see appendix, section 2). Parameters and a random effect model for the prior were chosen using out-of-sample cross validation. This produced estimates of alcohol LPC for a complete time series for the years 1980-2015 by country.

Alcohol LPC was adjusted for each country hosting tourists using the following equations:

$$Alcohol\ LPC_{H} = Unadjusted\ Alcohol\ LPC_{H} + Alcohol\ LPC_{Consumption\ abroad} \\ - Alcohol\ LPC_{Tourist\ consumption}$$

Alcohol LPC Consumption abroad =

$$\frac{\sum_{V} Proportion\ of\ tourists\ _{H,V}*Unadjusted\ Alcohol\ _{LPC}}{Population\ _{H}}*\frac{^{Average\ length\ of\ stay\ _{H,V}}}{_{365}}*Tourist\ _{Population\ _{H}}$$

 $Alcohol\ LPC_{Tourist\ consumption} =$

$$\frac{\sum_{V} Proportion \ of \ tourists \ _{V}*Unadjusted \ Alcohol \ _{LPC_{V}}*\frac{Average \ length \ of \ stay \ _{V}}{365}*Tourist \ Population \ _{H}}{Population \ _{H}}$$

Where H = Host country, V = Visiting country

Or, in other words, alcohol LPC was adjusted by adding in the per capita rate of consumption abroad and subtracting the per capita rate of tourist consumption domestically.

After adjusting alcohol LPC by tourist consumption and unrecorded consumption for all location/years reported, sex-specific and age-specific estimates were generated by incorporating estimates modeled in Dismod for percentage of current drinkers within a location/year/sex/age, as well as consumption trends modeled in Dismod g/day by location/year/sex/age, using the following equations.

```
\begin{split} &Proportion \ of \ total \ consumption \ _{l,y,s,a} = \\ & \frac{Alcohol \ g/day \ _{l,y,s,a} * Population \ _{l,y,s,a} * \% \ Current \ drinkers \ _{l,y,s,a}}{\sum_{s,a} Alcohol \ g/day \ _{l,y,s,a} * Population \ _{l,y,s,a} * \% \ Current \ drinkers \ _{l,y,s,a}} \\ &Alcohol \ LPC \ _{l,y,s,a} = \frac{Alcohol \ _{LPC \ _{l,y}} * Population \ _{l,y} * Proportion \ of \ total \ consumption \ _{l,y,s,a}}{Population \ _{l,y,s,a}} \end{split}
```

Where L = location, Y = Year, S = Sex, A = Age

A similar scalar was applied so that total subnational consumption equaled national consumption.

Theoretical minimum-risk exposure level

For alcohol use, the theoretical minimum-risk exposure level (TMREL) was assumed to be no alcohol use, i.e. 0 g/day of alcohol consumption. This diverges from the definition of other theoretical minimum-risk exposure level of risks because, for some alcohol-use relative risks, there's a preventative effect for low levels of consumption. However, due to the modeling of alcohol relative risks outlined below, it was found that 0 g/day provided the most consistency between the definition of alcohol-use TMREL and other GBD risk's TMREL. This is an area of improvement for future GBD iterations. Current research suggests that the preventative effect noted in studies may be due to issues in estimating abstainer populations. [5-7] If this is the case, a TMREL of 0 would still be valid.

Relative risk

Relative risks were derived for each GBD cause by mapping functions to the dose-response relationships found in meta-analysis. [11-22] Due to data availability, for high levels of consumption, uncertainty in

the relative risk functions increases greatly. To minimize the uncertainty of these measures, relative risks were estimated up to the 90th percentile of exposures in men (85 g/day) and the 95th percentile of exposures in women (60 g/day). For exposures beyond this, the associated relative risk was carried forward from these chosen percentile exposure levels. Though a dose-response relationship is evident at higher levels of exposure, the shape of the relative risk function is highly uncertain for higher levels of exposure both due to a lack of observations at these exposure levels, as well as confounding variables affecting estimation of the relative risk of these populations. Thusly, our relative risk estimates are likely an underestimate for the top 10% of male exposures and 5% of female exposures. For exact relative risks used, see appendix section 4.

Population Attributable Fraction

For chronic conditions, PAF was defined as

$$PAF(x) = \frac{P_A + P_{F^*}RR_F + \int_0^{150} P(x) * RR_C(x) dx - 1}{P_A + P_{F^*}RR_F + \int_0^{150} P(x) * RR_C(x) dx} \qquad P(x) = P_C * \frac{\Gamma(k, \theta)}{\int_{0.1}^{150} \Gamma(k, \theta)}$$

where:

x = alcohol consumption in g/day P_A = Prevalence of lifetime abstainers

 P_F = Prevalence of former drinkers

P(x) = Prevalence of alcohol consumption

RR_F = Relative risk of former drinkers

 $RR_C(x)$ = Relative risk function for drinkers

$$k = \frac{\overline{x}^2}{\sigma(\overline{x})^2}$$

$$\theta = \frac{\sigma(\overline{x})^2}{\overline{x}^2}$$

A thousand draws were taken of PAFs to generate uncertainty. The gamma distribution was used to estimate individual level variation within drinking populations [8-9]. Binge drinkers were not taken into account for chronic causes since the pattern of drinking has not been found to be an indicator of most outcomes [10].

For non-chronic conditions, such as injuries, binge drinking was accounted for in the model since patterns of drinking is significant.

$$PAF(x) = \frac{P_A + P_F + P_C + P_{C+B} * RR_{C+B}(x) - 1}{P_A + P_F + P_C + P_{C+B} * RR_{C+B}(x)}$$

$$RR_{C+B}(x) = P_D * P_{D+B} * (RR_{crude}(x) - 1) + 1$$

where:

 P_{C+B} = Prevalence of current drinkers who binge RR_{C+B} = Relative risk of current drinkers who binge RR_{crude} = Relative risk for a given mean level of consumption

 P_D = Proportion of a day that is a binge event P_{D+B} = Proportion of all days where a binge event occurs

The estimated PAF draws were then used to estimate YLL, YLDs, and DALYs, as per the other risk factors (see appendix section 2).

References

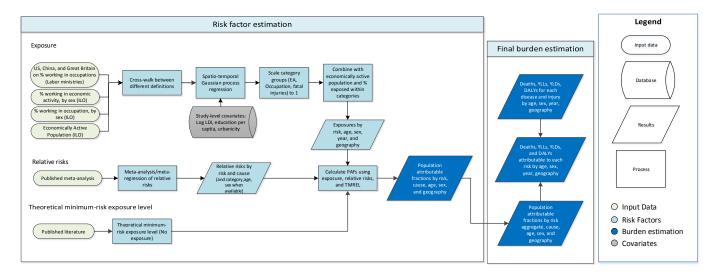
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Occupational Exposures & Risks SDG Capstone Appendix

Flowchart

Occupational Risk Factors (except abestos and injuries)



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with DALY rates attributable to occupational risks (8.8.1).

Indicator 8.8.1

As a component of SDG Goal 8. Promote sustained, inclusive and sustainable economic growth, full and productive employment and decent work for all, SDG Target 8.8, protect labour rights and promote safe and secure working environments for all workers, including migrant workers, in particular women migrants, and those in precarious employment, is measured using SDG Indicator 8.8.1, age-standardised all-cause DALY rates (per 100,000) attributable to occupational risks.

Occupational Asbestos SDG Capstone Appendix

Input Data & Methodological Summary

Case definition

We define exposure to occupational asbestos as the cumulative exposure to asbestos using mesothelioma death rate in a smoking impact ratio analogue. This definition assumes that all exposure to mesothelioma in the population is occupational. We estimate the burden of occupational asbestos for both sexes for ages 15 and above in both developed and developing countries.

Input data

The following were the data inputs required for the estimation of cumulative exposure to occupational asbestos in GBD 2013.

1. Mortality rate due to mesothelioma in 1990, 2005, 2010, and 2013

Cause-specific mortality rates for mesothelioma, C_{LC} by country, age, and sex were generated by causes of death models for GBD 2013¹.

2. Mortality rate due to mesothelioma in a population not exposed to asbestos

We calculated the background mortality of mesothelioma, N_{LC} , from the model used by Lin et al². Using the uncertainty around the coefficients, we created 1,000 draws of the mortality due to mesothelioma if there was no asbestos consumption in a country. The mean value for background mortality is 0.73 and 0.47 deaths per million males and females, respectively.

3. Mortality rate due to mesothelioma in a population highly exposed to asbestos

We found the mortality rate for highly exposed individuals from asbestos workers, C^*_{LC} from Goodman et al³. We used all studies that reported both the number of person-years followed and the number of cases of mesothelioma and found the death rate of all individuals included in the studies. The death rate for highly exposed individuals is 226 per million people.

Modeling strategy

The approach used to estimate the burden of cancer caused by occupational exposure to asbestos was different than the approach used for all other occupational carcinogens. This approach, analogous to the

¹ Naghavi, M & GBD Mortality and Causes of Death Collaborators. (2014). Global, regional, and national levels of 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 2014*. In press.

² Lin R-T, Takahashi K, Karjalainen A, *et al.* Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis. *Lancet* 2007; **369**: 844–9.

³ Goodman M, Morgan RW, Ray R, Malloy CD, Zhao K. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. *Cancer Causes Control* 1999; **10**: 453–65.

Peto-Lopez Smoking Impact Ratio (SIR)⁴, uses mesothelioma deaths as a marker for exposure to asbestos in order to take into account issues of latency and risk accumulation.

For asbestos, the excess lung cancer deaths analogous measurement for a population is the excess deaths due to mesothelioma observed in that population divided by the excess deaths in a population that is heavily exposed to asbestos. The asbestos impact ratio (AIR) gives a measurement for the exposure level of a population to asbestos. We then use the AIR and relative risks to calculate the Population Attributable Fraction (PAF) for each cause related to asbestos. Formally, the AIR is defined as:

$$AIR = \frac{C_{LC} - N_{LC}}{C_{LC}^* - N_{LC}}$$

Where:

 C_{LC} = (country-sex-specific) mesothelioma mortality rate in the study population N_{LC} = mesothelioma mortality rate in a population not exposed to asbestos C_{LC}^* = mesothelioma mortality rate in a population highly exposed to asbestos

Relative Risks

Relative risks for occupational asbestos were searched for and provided by the expert groups (same as GBD 2010). Based on expert group recommendation, we updated the relative risks for lung cancer with a recent meta-analysis which was unavailable during GBD 2010. *Table 1* outlines the relative risk estimates along with the outcomes used to estimate the burden attributable to occupational exposure of asbestos. These numbers have remained unchanged since GBD 2010.

Outcome	Sex	Age	High Exposure RR (95% CI)	Low Exposure RR (95% CI)	RR application	Source
Larynx cancer	Both	15-79	1.38 (1.17- 1.60)	1	Mortality and Morbidity	IOM, 2006 ⁵
Lung cancer (males)	Both	15-79	2.27 (1.67-2.85)	1.65 (1.50-1.79)	Mortality and Morbidity	Lenters et al, 2011
Lung cancer (females)	Both	15-79	1.86 (1.56-2.15)	1.52 (1.46-1.58)	Mortality and Morbidity	Lenters et al, 2011
Mesothelioma	Both	15-79	38.4 (13.5- 110)	20.7 (6.5-65.5)	Mortality and Morbidity	Rake et al 2009 ⁶
Ovarian cancer	Female	15-79	1.77 (1.37- 2.28)	1	Mortality and Morbidity	Camargo et al, 2011 ⁷

Table 1: Relative risks for occupational asbestos used in GBD 2013.

⁴ Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet* 1992; **339**: 1268–78.

⁵ Institute of Medicine of the National Academies (IOM), Asbestos: selected cancers. 2006, The National Academies Press: Washington, DC.

⁶ Rake C, Gilham C, Hatch J, Darnton A, Hodgson J, Peto J. Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study. *Br. J. Cancer* 2009; **100**(**7**): 1175–1183.

⁷ Camargo MC, Stayner LT, Straif K, Reina M, Al-Alem U, Demers PA, et al. Occupational exposure to asbestos and ovarian cancer: a meta-analysis. *Environ. Health Perspect.* 2011; **119(9)**: 1211–1217.

Theoretical Minimum-Risk Exposure Level

TMREL draws for occupational asbestos were prepped to feed into the central PAF calculate using the following code:

"J:\WORK\2013\05_risk\01_database\02_data\occ\03_tmred\code\fix_occ_asbestos_tmred.do"

Attributable Burden Estimation

Population attributable fractions (PAFs) for occupational asbestos were calculated centrally using the following formula similar to all occupational carcinogens. The following equation which takes into account exposure distribution (P_i); relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i); and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i (RR_i - 1)}{\sum_{i=1}^{n} P_i (RR_i - 1) + 1}$$

GBD 2010 vs. GBD 2013

- Mesothelioma was not part of cause list in GBD 2010. As a result, cause specific mortality rate for mesothelioma for all country years had to be modeled in GBD 2010 separately. In GDB 2013, mesothelioma was added to the cause list. As a result, causes of death output of cause-specific mortality rates of mesothelioma for all country years were used in GBD 2013.
- Updated relative risks for lung cancer associated with occupational asbestos based on a newly published meta-analysis.

Occupational Asthmagens SDG Capstone Appendix

Input data & Methodological summary

Case definition

Exposure to occupational asthmagens is defined as the proportion of the population exposed based on distribution of the population in eight occupational groups. The burden of occupational asthmagens is only estimated for ages 15 and older. We model the burden of occupational asthmagens in both developed and developing countries.

Input Data

Description of data identification and prep

The primary data type used to estimate exposure to occupational asthmagens is data on economic activity by country obtained from the ILO database. Data in the ILO database are primarily from censuses and national labor force surveys. The database was supplemented with subnational data for UK and China. Data processing involved sex-splitting estimate of industry groups to generate sex-specific estimates and crosswalking between different ISIC versions (classification systems used by ILO.)

Modeling strategy

- 1. Calculate a time series of the percentage of the workforce working in 8 occupational groups. This step is modeled using ST-GPR. This first step of generating the distribution of the labor force across occupational groups is analogous to all other occupational risks (except injuries and asbestos). The ST-GPR model is run separately for each economic activity by sex for ages 15 and above.
- 2. Run an additional spacetime model to further subdivide the 7th occupational group by economic activities. This was done to match the exposure categories in the reported relative risks.
- 3. Rescale GPR output such that the proportion of individuals employed in all economic activity groups add up to 1 i.e 100% of the labor force.
- 4. Calculate the population level exposure to all occupational asthmagens using proportion of the population that is economically active population, proportion that is employed in each economic activity/occupation group, and the prevalence of asthmagen exposure in each economic activity based on data provided the expert group.

Exposure group j = % of pop that is economically active * % of economically active in economic activity i * % of occupation/economic activity i that is exposed to level j

5. Prep and save draws of occupational asthmagens to feed into the PAF calculator.

Relative Risks

The relative risks and the outcomes associated with occupational asthmagens was systematically searched for and generated by the occupational risk expert group in GBD 2010. For GBD 2013, there were

no major updates made to relative risks used in GBD 2010. The table below outlines the relative risk values used for occupational asthmagens in GBD 2013 provided by the expert group.

		Males			Females	
	RR	95% LL	95% UL	RR	95% LL	95% UL
Background	1			1		
Administrative	1			1		
Technical	1.05	0.98	1.12	1.06	1.03	1.1
Sales	1.14	1.05	1.23	1.13	1.08	1.18
Agriculture	1.5	1.11	2.03	1.5	1.11	2.03
Mining	1.95	1.58	2.40	1.95	1.58	2.40
Transport	1.31	1.22	1.40	1.22	1.13	1.31
Manufacturing	1.56	1.47	1.65	1.33	1.27	1.39
Services	1.53	1.42	1.66	1.41	1.35	1.46

All RRs except for that for agricultural occupations came from a study by Karjalainen and co-workers. This is the only comprehensive national population study of incident asthma. Relative risks for agricultural occupations were based on a study by Kogevinas and co-workers, with an inverse variance-weighted estimate obtained using the separate estimates for "farmers" and "agricultural" workers provided in the paper. There were limited updates made to the relative risk draw files used for occupational asthmagens in GBD 2013 compared to 2010. Relative risk draws for occupational asthmagens were centrally migrated from GBD 2010 infrastructure to GBD 2013. As a result, code files that prep RR draws from scratch do not exist for all relative risks in the table above.

Theoretical Minimum-Risk Exposure Level

The theoretical minimum risk exposure level is defined as no occupational exposure to asthmagens. TMREL for occupational asthmagens remained unchanged since GBD 2010. Code files that prep TMREL draws for occupational asthmagens from scratch do not exist because they were centrally migrated from GBD 2010 to GBD 2013 infrastructure.

Attributable Burden Estimation

Population Attributable Fractions (PAFs) were calculated centrally using input draws of exposure, relative risks, and theoretical minimum level prepared by the modeler (Astha). The following equation which takes into account exposure distribution (P_i); relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i); and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i (RR_i - 1)}{\sum_{i=1}^{n} P_i (RR_i - 1) + 1}$$

GBD 2010 vs. GBD 2013

- Data updates included using economic activity data from newly downloaded ILO database and additional of subnational data for China and the UK.
- Methodology from GBD 2010 has remained unchanged

Occupational Carcinogens SDG Capstone Appendix

Input data & Methodological summary

Case definition

Exposure to occupational carcinogens is defined as the proportion of people ever exposed (taking into account worker turnover) to each carcinogen (listed below) based on the distribution of the population in nine economic activity groups. The burden of occupational carcinogens is only estimated for ages 15 and older. We model the burden of occupational carcinogens in both developed and developing countries.

Input data

Description of data identification and prep

The primary data type used to estimate exposure to occupational carcinogens is data on economic activity by country obtained from the ILO database. Data in the ILO database are primarily from censuses and national labor force surveys. The database was supplemented with subnational data for UK and China. Data processing involved sex-splitting estimate of industry groups to generate sex-specific estimates and crosswalking between different ISIC versions (classification systems used by ILO.)

Data on prevalence of carcinogens by industry was estimated using data from the CAREX database. This was provided by the expert group for GBD 2010. The proportion of workers within an industry who were exposed to each carcinogen was distributed between "high" and "low" exposure. This was done to account for varying levels of exposure to occupational carcinogens within each industry. The carcinogen prevalence were distributed 10:90 (high:low) for developed regions and 50:50 (high:low) for developing countries. This assumption was made based on the methodology devised by the expert group in GBD 2010 and also for GBD 2013. Occupational turnover factors used in the analysis were provided by the expert group for use in GBD 2010 and were used as is for GBD 2013. See notes on future iterations for more information on turnover factors.

Modeling strategy

- 6. Calculate a time series of the percentage of the workforce working in 9 economic activity groups. This step is modeled using ST-GPR. This first step of generating the distribution of the labor force across 9 economic activities is used for occupational carcinogens as well as particulates, noise, ergonomic factors and asthmagens. The ST-GPR model is run separately for each economic activity by sex for ages 15 and above.
- 7. Rescale GPR output such that the proportion of individuals employed in all economic activity groups add up to 1 i.e 100% of the labor force.
- 8. Calculate the population level exposure to all occupational carcinogens (except asbestos) using proportion of the population that is economically active population, proportion that is employed in each economic activity, turnover rates for each economic activity, prevalence of carcinogen exposure in each economic activity based on CAREX, and exposure partition factor (EPF).

Industry	Covariates
Agriculture,	1) Proportion of the population living in levels of population density (150-300,
Hunting, Forestry	300-500, 500-1,000 and over 1,000 people per square km);
and Fishing	2) Proportion of population living in bands of latitude (15-30, 30-45 and over
O	45 degrees of latitude);
	3) Proportion of the population living in levels of altitude (100-500, 500-1500
	and over 1500 meters of elevation);
	4) mean years of education of the population by sex;
	5) Lagged-distributed income in 2005 international dollars (a smoothed
	measure of gross domestic product).
Mining and	1) Proportion of the population living in levels of population density (150-300,
Quarrying	300-500, 500-1,000 and over 1,000 people per square km);
	2) Proportion of the population living in levels of altitude (100-500, 500-1500
Wholesale and	and over 1500 meters of elevation);
Retail Trade	3) number of 4 wheel vehicles per capita;
and Restaurants	4) mean years of education of the population by sex;
and Hotels	5) Lagged-distributed income in 2005 international dollars (a smoothed
	measure of gross domestic product).
Manufacturing	1) Proportion of the population living in levels of population density (150-300,
	300-500, 500-1,000 and over 1,000 people per square km);
Electricity, Gas and	2) Proportion of population living in bands of latitude (15-30, 30-45 and over
Water	45 degrees of latitude);
	3) Proportion of the population living in levels of altitude (100-500, 500-1500
Transport, Storage	and over 1500 meters of elevation);
and	4) number of 4 wheel vehicles per capita;
Communication	5) mean years of education of the population by sex;
	6) Lagged-distributed income in 2005 international dollars (a smoothed
Construction	measure of gross domestic product). 1) Proportion of the population living in levels of population density (150-300,
Construction	300-500, 500-1,000 and over 1,000 people per square km);
	2) Proportion of the population living in levels of altitude (100-500, 500-1500
	and over 1500 meters of elevation);
	3) mean years of education of the population by sex;
	4) Lagged-distributed income in 2005 international dollars (a smoothed
	measure of gross domestic product).
Financing,	1) Proportion of the population living in levels of population density (150-300,
Insurance, Real	300-500, 500-1,000 and over 1,000 people per square km);
Estate and	2) mean years of education of the population by sex;
Business Services	3) Lagged-distributed income in 2005 international dollars (a smoothed
	measure of gross domestic product).
Community, Social	
and	
Personal Services	

9. Prep and save draws of occupational carcinogens (except asbestos) into the PAF calculator.

Table below outlines the country-level covariates used to model the 9 economic activities.

Relative Risks

The relative risks and the outcomes associated with each carcinogen was systematically searched for and generated by the occupational risk expert group in GBD 2010. For GBD 2013, there were n omajor updates made to relative risks used in GBD 2010. We added RR for kidney cancer associated with occupational trichloroethylene to the list of relative risks used in GBD 2013 based on IARC's reclassification of tricholorethylene as a Group 1 carcinogen. Relative risks outline for each carcinogen-cancer pair outlined in the table below were extracted from a single key study or meta-analysis.

Carcinogen	Outcome	RR (high)	LL (high)	UL (high)	RR (low)	LL (low)	UL (low)	Source
Arsenic	Trachea, bronchus, and lung cancers	2.05	1.43	2.85	1	1	1	Lee-Feldstein 1986
Benzene	Leukemia	2.62	1.57	4.39	1.64	1.10	2.39	Khalade et al, 2010
Beryllium	Trachea, bronchus, and lung cancers	1.17	1.08	1.28	1	1	1	Schubauer-Berigan et al, 2011
Cadmium	Trachea, bronchus, and lung cancers	1.19	1.09	1.29	1	1	1	Verougstraete et al, 2003
Chromium VI	Trachea, bronchus, and lung cancers	1.18	1.12	1.25	1	1	1	Cole & Rodu, 2005
Diesel engine exhaust	Trachea, bronchus, and lung cancers	1.47	1.29	1.67	1	1	1	Lipsett & Campelman, 1999
Second hand smoke	Trachea, bronchus, and lung cancers	1.24	1.18	1.29	1	1	1	Stayner et al, 2007
Formaldehyde	Leukemia	1.47	1.19	1.83	1	1	1	Collins and Lineker, 2004
Formaldehyde	Nasopharynx cancer	2.1	1.05	4.21	1	1	1	Hauptmann et al, 2004
Nickel	Trachea, bronchus, and lung cancers	2.1	1.3	3.2	1	1	1	Grimsrud et al 2005
PAHs	Trachea, bronchus, and lung cancers	1.31	1.16	1.48	1	1	1	Armstrong et al, 2004
Silica	Trachea, bronchus, and lung cancers	1.32	1.24	1.41	1	1	1	Kurihara & Wada, 2004
Sulpuric acid	Larynx cancer	4.28	2.13	8.58	1.91	0.97	3.78	Soskolne et al, 1992
Trichloroethylene	Kidney cancer	1.24	1.06	1.45	1	1	1	Kelsh et al, 2010

There were limited updates made to the relative risk draw files used for occupational carcinogens in GBD 2013 compared to 2010. Relative risk draws were centrally migrated from GBD 2010 infrastructure to GBD 2013. As a result, code files that prep RR draws from scratch do not exist for all relative risks in the table above.

Theoretical Minimum-Risk Exposure Level

The theoretical minimum risk exposure level is defined as no occupational exposure to carcinogens. TMREL draws were prepped by the modeler (Astha). TMREL for all carcinogens remained largely unchanged since GBD 2010. Code files that prep TMREL draws for occupational carcinogens from scratch do not exist because they were centrally migrated from GBD 2010 to GBD 2013 infrastructure.

Attributable Burden Estimation

Population Attributable Fractions (PAFs) were calculated centrally by Stan Biryukov (Data Specialist) using input draws of exposure, relative risks, and theoretical minimum level prepared by the modeler (Astha). The following equation which takes into account exposure distribution (P_i); relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i); and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i(RR_i - 1)}{\sum_{i=1}^{n} P_i(RR_i - 1) + 1}$$

GBD 2010 vs. GBD 2013

- Both groups 1 and 2A carcinogens, as classified by IARC, were analyzed in GBD 2010 and only group 1 was reported. In GBD 2013, we generated estimates for only those carcinogens that we reported in GBD 2010 which includes group 1 carcinogens.
- Based on the reclassification of tricholorethylene from group 2A carcinogen to group 1 we included it to the list of occupational carcinogens that were analyzed in GBD 2013.
- Data updates included using economic activity data from newly downloaded ILO database and additional of subnational data for China and the UK.

Occupational Injuries SDG Capstone Appendix

Input data & Methodological summary

Case definition

Proportion of injuries (fatal or non-fatal) attributed to occupational exposure estimated based on rates of fatal injuries by country-year as reported to the ILO database. The burden of occupational injuries is only estimated for ages 15 and older. We model the burden of occupational injuries in both developed and developing countries.

Input Data

Description of data identification and prep

The primary data type used to estimate the burden of occupational injuries is data on rates of fatal injury reported by economic activity and country year from the ILO database. This was updated in GBD 2013 by using a newly downloaded ILO database.

Modeling strategy

- 1. Data on fatal injury rates reported by economic activity and country-year from the ILO database is prepped for modeling.
- 2. Based on a linear regression using fatal injury data from the ILO database generate a time series of fatal injury rates for all country years for all 9 economic activities (EA). Also, generate a full time series using a similar model for the total rate of fatal injuries for all country years.
- 3. Rescale EA-specific fatal injury rate such that it adds up to the total fatal injury rate modeled for that country year. This is done to ensure that the EA-specific rates are add up to the reported total rate of fatal injuries.
- 4. Generate injury counts using modeled rates of fatal injuries for all economic activity groups for all country years. The following equation was used to generate injury counts.

of injuries by industry for each age = % of workforce in industry * economically active population by age * rate of injuries / 100,000 people

5. Calculate injury PAFs using the following equation. The denominator in the PAF equation, total unintentional injuries was estimated by summing across injury deaths estimated by GBD causes of death models:

PAF = (total occupational injuries - theoretical minimum) / total unintentional injuries

Where, total occupational injuries = injury count calculated from step 4, theoretical minimum is assumed to be zero i.e. all occupational injuries can be avoided, total unintentional injuries = sum of injury deaths in each cause that is selected by economic activity. NOTE: Although, the PAF estimated

above is for fatal injuries given that we don't have a good way of estimating non-fatal occupational injury PAFs we assume that YLL PAFs = YLD PAFs.

6. Finally, calculate and save draws of occupational injury PAFs to feed into the GBD infrastructure.

Relative Risks

There are no relative risk estimates for occupational injuries because PAFs are estimated directly.

Theoretical Minimum-Risk Exposure Level

The theoretical minimum risk exposure level is defined as 0 injury deaths per 1,000,000 person-years.

Attributable Burden Estimation

Population Attributable Fractions (PAFs) for occupational injuries are estimated directly by the modeler (Astha). PAF is calculated using the equation and code files outlined in step 5 of the modeling strategy section. The PAF estimation uses GBD outputs from causes of death. Therefore, occupational injury PAFs should be calculated using the finalized cause of death numbers.

GBD 2010 vs. GBD 2013

- Data updates included using fatal injury data from newly downloaded ILO database.
- For GBD 2010 rates of fatal injuries were modeled using ST-GPR in 3 broad groups combining economic activities (agriculture, industry, and service). In GBD 2013, we changed this to model rates of fatal injury rates by detailed groups i.e. 9 economic activities using a simple linear regression.
- Use of occupation group and injury type matrix to exclude implausible injury type and occupation pairing e.g. snake bite deaths among clerical workers etc. In GBD 2010, we assumed that all unintentional injuries were included in the denominator of the PAF calculation. However, in GBD 2013, we calculated PAFs for each economic activity separately. Only the injury types marked by a "x" in the matrix outlined in the above table contributed to the denominator of the PAF equation for the respective economic activity
- For GBD 2013, we estimated the non-fatal burden of occupational injuries which wasn't done in GBD 2010. We don't have data sources estimating non-fatal injuries as a result we assumed that the burden of non-fatal would be equal to the fatal burden of occupational injuries i.e. YLL PAFs = YLD PAFs for occupational injuries.
- TMREL changed from 5 injury deaths per 1,000,000 person-years, based on rates of clerical workers in the US to 0 injury deaths per 1,000,000 person-years. This was done to be consistent with the new definition of TMREL for GBD based on the risk consultative meeting in winter 2015.

Occupational Ergonomic Factors SDG Capstone Appendix

Input data & Methodological Summary

Case definition

Exposure to occupational ergonomic factors is defined as the proportion of the population exposed based on distribution of the population in occupation groups. Occupation groups of the population was used as a proxy because there aren't direct measures of exposure to occupational ergonomic factors associated with low back pain. The burden of occupational ergonomic factors is only estimated for ages 15 and older. We model the burden of occupational ergonomic factors in both developed and developing countries.

Data inputs

Description of data identification and prep

The primary data type used to estimate exposure to occupational ergonomic factors is data on occupational groups by country obtained from the ILO database. Data in the ILO database are primarily from censuses and national labor force surveys. The database was supplemented with subnational data for UK and China. Data processing involved sex-splitting estimate of industry groups to generate sex-specific estimates and crosswalking between different ISIC versions (classification systems used by ILO.)

Modeling strategy

The modeling strategy for occupational exposure to ergonomic factors associated with low back pain has remained unchanged since GBD 2010⁸. The strategy used to estimate exposure has been summarized below.

There are few direct measurements of exposure to ergonomic factors in the workplace. We estimated risk of exposure to occupational ergonomic factors by using the composition of a country's workforce in seven different occupational groups. This work has been published since GBD 2010⁹. The number of workers in a particular occupation group was determined by multiplying the percentage of the workforce in the occupation group by the percentage of economically active people. The steps involved in estimating exposure are outlined below.

7. Calculate a time series of the percentage of the workforce working in 8 occupational groups. This step is modeled using ST-GPR. This first step of generating the distribution of the labor force across occupational groups is analogous to all other occupational risks (except injuries and asbestos). The ST-GPR model is run separately for each economic activity by sex for ages 15 and above.

⁸ Lim SS, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet 2012*; **380**: 2224-60.
⁹ Driscoll T, Jacklyn G, Orchard J, Passmore E, Vos T, Freedman G, Lim S, Punnett L. The global burden of occupationally related

low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014 Jun;73(6):975-81. doi: 10.1136/annrheumdis-2013-204631. Epub 2014 Mar 24.

- 8. Rescale GPR output such that the proportion of individuals employed in all economic activity groups add up to 1 i.e. 100% of the labor force.
- 9. Calculate the population level exposure to all occupational ergonomic factors using proportion of the population that is economically active, and proportion that is employed in each occupation group.

$$P_{c,y,a,s,i} = EAP_{c,y,a,s,i} X EAC_{c,y,a,s}$$

Where:

 $P_{c,y,a,s,i}$ = Prevalence of occupational exposure to ergonomic factors associated with low back pain; $EAP_{c,y,a,s,i}$ = Proportion of the population that is economically active, by industry, country, year, age, and sex; $EAC_{c,y,a,s}$ = Proportion of the population that is economically active, by country, year, age, and sex;

```
c = Country;

y = Year;

a = GBD age group;

s = Sex;

i = classification of occupation (7 types).
```

10. Prep and save draws of exposure to feed into the PAF calculator.

Relative Risks

The relative risks and the outcomes associated with occupational ergonomic factors was systematically searched for and generated by the occupational risk expert group in GBD 2010. For GBD 2013, there were no major updates made to relative risks used in GBD 2010. The table below outlines the relative risk values used for occupational ergonomic factors in GBD 2013 provided by the expert group which has been published since GBD 2010².

DD (OEO/ CI)

	KR (95% CI)	
Background	1	
Professional, technical, and related workers	1.17 (1.06-1.28)	
Administrative and managerial workers	1.20 (0.96-1.50)	
Clerical and related workers	1	
Sales workers	1.21 (1.01-1.44)	
Service workers	1.47 (1.38-1.57)	
Agriculture, animal husbandry and forestry workers,	3.73 (2.61-5.33)	
fishermen, and hunters		
Production and related workers	1.54 (1.41-1.68)	
Transport equipment operators and laborers	1.54 (1.41-1.68)	

Relative risk draws for occupational ergonomic factors were centrally migrated from GBD 2010 infrastructure to GBD 2013. As a result, code files that prep RR draws from scratch do not exist for all relative risks in the table above.

Theoretical Minimum-Risk Exposure Level

The theoretical minimum risk exposure level is defined as no occupational exposure to asthmagens. TMREL for occupational asthmagens remained unchanged since GBD 2010. Code files that prep TMREL

draws for occupational carcinogens from scratch do not exist because they were centrally migrated from GBD 2010 to GBD 2013 infrastructure.

Attributable Burden Estimation

Population Attributable Fractions (PAFs) were calculated centrally by *Stan Biryukov (Data Specialist)* using input draws of exposure, relative risks, and theoretical minimum level prepared by the modeler (Astha). The following equation which takes into account exposure distribution (P_i) ; relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i) ; and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i(RR_i - 1)}{\sum_{i=1}^{n} P_i(RR_i - 1) + 1}$$

GBD 2010 vs. GBD 2013

- Data updates included using data on occupational groups from newly downloaded ILO database and additional of subnational data for China and the UK.
- Methodology remained unchanged since GBD 2010

Occupational Noise SDG Capstone Appendix

Input data & Methodological summary

Case definition

Definition of occupational noise as a risk factor is determined by noise levels in decibels (dB) which is associated with varying levels of severity in hearing loss (see Table 1.)

Occupational exposure and outcome pairs

Exposure (dB)	Outcome
Low (85-90dB)	Mild hearing loss
High (90dB +)	Moderate, moderately severe, severe,
	profound, and complete hearing loss

In the absence of direct exposure data however, exposure to occupational noise is defined based on the proportion of the population exposed based on the distribution of the population in nine economic activities. The burden of occupational noise is only estimated for ages 15 and older. We model the burden of occupational ergonomic factors in both developed and developing countries.

Input Data

Economically Active Population by Industry

One of the primary inputs in our exposure model is the composition of a country's workforce. We generated a complete time series of estimates of the proportion of economically active population $(EAP_{c,y,a,s})$ by industry, country, year, age, and sex from 1980-2013 using spacetime GPR. Data on economic activity by country and industry over time was obtained from the International Labor Organization¹⁰. ILO data are primarily from censuses and national labor force surveys; these were also supplemented with data from the National Bureau of Statistics of China and the 1991 and 2001 India Censuses. For countries which did not provide sex specific estimates of industry groups to the ILO but did provide total estimates, the regional average of male to female participation in the industry was applied in order to determine sex specific estimates.

Economically Active Population

The proportion of the population that is economically active was based on estimates produced by the ILO³. ILO estimates are produced separately by age for males and females for all but 10 countries between 1990 and 2013. We used the regional average as an estimate for the EAP in these countries. Because there were no estimates for 70-80 year olds, the 65-70 estimates were carried forward. The data

¹⁰ International Labor Organization (ILO). ILOSTAT Database. http://www.ilo.org/ilostat/. Accessed: Sept 16, 2013

for the population aged younger than 15 was too sparse and this age range was excluded. This data was provided by the expert group.

Prevalence of Noise Exposure by Industry

The exposure levels by industry were assumed to vary by industry and the country's development status. Australian national data on noise exposure in various industries (sampled across a range of tasks) provided the basis for the mean and standard deviation of noise exposure in each industry⁴. This information was in turn used to estimate the proportion of workers exposed at low levels of noise (85-90db) and high levels of noise (90+db). No account was made of the use of hearing protection, except for the mining industry, where mean exposure levels were decreased by 3dB to take this into account. These proportions were used for developed countries for 1990, 2005, 2010, and 2013. This data was provided by the expert group. The prevalence of noise exposure by industry were modified for developing countries, to take into account the likely higher exposure levels in developing countries due to the less extensive use of noise controls. For developing countries, the mean exposure was estimated to be three dB higher (double the noise level compared to developing countries) ¹. Table 2 shows the exposure levels for both high and low exposure by country's development status. This data was provided by the expert group.

Prevalence of exposure by industry (per 100,000 workers)

Industry	Developed, 90+ dB	Developed, 85-90 dB	Developing, 90+ dB	Developing, 85-90 dB
Agriculture, Hunting, Forestry and Fishing	0.180	0.144	0.261	0.167
Mining and Quarrying	0.393	0.294	0.572	0.254
Manufacturing	0.106	0.245	0.233	0.322
Electricity, Gas and Water	0.204	0.123	0.274	0.138
Construction	0.251	0.194	0.362	0.210
Wholesale and Retail Trade and Restaurants and Hotels	0	0.018	0.001	0.231
Transport, Storage and Communication	0.079	0.202	0.180	0.287
Financing, Insurance, Real Estate and Business Services	0	0.031	0.004	0.230
Community, Social and Personal Services	0.09	0.131	0.159	0.176

Modeling strategy

The modeling strategy for exposure to occupational noise has remained unchanged since GBD 2010. The strategy used to generate exposure estimates for occupational noise has been summarized below.

- 1. Calculate a time series of the percentage of the workforce working in 9 economic activities/industries. This step is modeled using ST-GPR. This first step of generating the distribution of the labor force across economic activities is the same for a number of other occupational risks. The ST-GPR model is run separately for each economic activity by sex for ages 15 and above.
- 2. Rescale GPR output such that the proportion of individuals employed in all economic activity groups add up to 1 i.e. 100% of the labor force.

3. Calculate the population level exposure to occupational noise by industry using proportion of the population that is economically active, proportion that is employed in each economic activity group, and prevalence of noise exposure associated with each economic activity/industry. Population exposure is then determined as the sum of each industry's exposure for that country, age, and sex group. This equates to:

$$P_{c,a,s,j,t} = \sum_{i=1}^{9} (EAP_{i,s,j,t} \ X \ CP_{i,c} \ X \ EAC_{i,s,j,t})$$

 $P_{c,a,s,j,t}$ = Prevalence of exposure to occupational noise, by country, year, age, sex, and level of exposure; $EAP_{i,s,j,t}$ = Proportion of the population that is economically active, by industry, country, year, age, and sex; $CP_{i,c}$ = Prevalence of noise exposure by industry, and exposure level; $EAC_{c,y,a,s}$ = Proportion of the population that is economically active, by country, year, age, and sex; c = exposure level (high or low); a = 5-year age group; s = sex; s = country; s = country; s = repar; s = Industry.

4. Prep and save draws of exposure to feed into the PAF calculator.

Relative Risks

Relative risks for occupational noise were estimated by occupational risk expert group for GBD 2010. See expert group documentation from GBD 2010 on details of how these were estimated. The tables below list RRs by age and noise exposure level.

Relative risk by age and noise exposure level (41db or more)

High exposure (>90dB)			Low exposure (85-90dB)			
Age	RR	LL	UL	RR	LL	UL
15-19	7.96	4.74	13.37	2.92	1.74	4.91
20-24	7.96	4.74	13.37	2.92	1.74	4.91
25-34	6.63	4.74	9.28	3.42	2.45	4.79
35-44	5.93	4.24	8.29	3.79	2.71	5.30
45-54	5.55	3.94	7.81	3.88	2.75	5.46
55-64	3.56	2.53	5.02	2.66	1.89	3.75
65-74	2.14	1.62	2.84	1.80	1.35	2.39
75-84	1.29	1.07	1.56	1.22	1.01	1.47
85+	1.00	1.00	1.22	1.00	1.00	1.22

Relative risk by age and noise exposure level (25db or more)

High exposure (>90dB)			Low exposure (85-90dB)			
Age	RR	LL	UL	RR	LL	UL
15-19	7.29	4.91	10.82	2.74	1.85	4.07
20-24	7.29	4.91	10.82	2.74	1.85	4.07
25-34	5.44	4.32	6.86	2.91	2.31	3.66
35-44	3.07	2.71	3.48	2.17	1.91	2.46
45-54	2.55	2.36	2.77	1.99	1.83	2.15

55-64	1.85	1.71	2.00	1.55	1.43	1.68
65-74	1.45	1.37	1.52	1.30	1.23	1.37
75-84	1.13	1.05	1.21	1.09	1.02	1.17
85+	1.00	1.00	1.07	1.00	1.00	1.07

The underlying sources for these RR estimates are:

Agrawal Y, Platz E, Niparko J. Prevalence of hearing loss and differences by demographic characteristics among US adults. Data from the National Health and Nutrition Examination Survey, 1999-2004. Archives of Internal Medicine 2008;168(14):1522-1530.

Davis A. The prevalence of hearing impairment and reported hearing disability among adults in Great Britain. International Journal of Epidemiology 1989,18: 911-917.

Wilson D, Walsh P, Sanchez L, Davis A, Taylor A, Tucker G, Meagher I. The epidemiology of hearing impairment in an Australian adult population. International Journal of Epidemiology 1999;28:247-252.

The relative risks used to assess the attributable burden of occupational exposure to noise levels that are associated with hearing loss has remained unchanged since GBD 2010. The relative risk draws were migrated centrally from GBD 2010 to GBD 2013 infrastructure. As a result, code that preps RR draws from scratch do not exist for occupational noise.

Theoretical Minimum-Risk Exposure Level

The TMREL for occupational noise is defined as all individuals are exposed to background noise levels. This definition of TMREL has remained unchanged since GBD 2010. The TMREL draws were migrated centrally from GBD 2010 to GBD 2013 infrastructure. As a result, prep code for this step does not exist.

Attributable Burden Estimation

Population Attributable Fractions (PAFs) were calculated centrally by *Stan Biryukov (Data Specialist)* using input draws of exposure, relative risks, and theoretical minimum level prepared by the modeler (Astha). The following equation which takes into account exposure distribution (P_i); relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i); and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i (RR_i - 1)}{\sum_{i=1}^{n} P_i (RR_i - 1) + 1}$$

Occupational Particulates SDG Capstone Appendix

Input data & Methodological Summary

Case definition

The exposure definition for occupational particulates is a proxy measure based on industry data which were grouped to be consistent with exposure data in studies that provide relative risk information. COPD is the only outcome associated with occupational particulates exposure. Burden of occupational particulates is estimated for age groups 15 and beyond in both developing and developed countries.

Input Data

Economically Active Population by Industry ($EAP_{c,v,a,s}$)

Data on economic activity by country and industry over time was obtained from the International Labor Organization¹¹. ILO data are primarily from censuses and national labor force surveys; these were also supplemented with data from the National Bureau of Statistics of China and the 1991 and 2001 India Censuses. For countries which did not provide sex specific estimates of industry groups to the ILO but did provide total estimates, the regional average of male to female participation in the industry was applied in order to determine sex specific estimates. These numbers were downloaded directly from the ILO database.

Economically Active Population ($EAC_{c,y,a,s}$)

The proportion of the population that is economically active was based on estimates produced by the ILO². ILO estimates are produced separately by age for males and females for all but 10 countries between 1990 and 2013. We used the regional average as an estimate for the EAP in these countries. Because there were no estimates for 70-80 year olds, the 65-70 estimates were carried forward. The data for the population aged younger than 15 was too sparse and this age range was excluded. These numbers were provided to us by the expert group.

Exposure Partition Factor (EPF_{level})

The exposure levels vary by industry. We assumed that the exposure levels by industry also varied by a country's development status⁴. Table 2 shows the exposure levels for both high and low exposure by country's development status. These numbers were provided by the expert group.

Economic Activity	Developed, high	Developed,	Developing,	Developing,
		low	high	low
Agriculture, Hunting, Forestry and Fishing	0.1	0.5	0.1	0.7
Mining and Quarrying	0.1	0.7	0.4	0.4
Manufacturing	0.1	0.7	0.4	0.4
Electricity, Gas and Water	0.1	0.5	0.1	0.7
Construction	0.1	0.7	0.4	0.4

¹¹ International Labor Organization (ILO). ILOSTAT Database. http://www.ilo.org/ilostat/. Accessed: Sept 16, 2013

Wholesale and Retail Trade and	0	0.05	0	0.1
Restaurants and Hotels				
Transport, Storage and Communication	0.1	0.5	0.1	0.7
Financing, Insurance, Real Estate and	0	0.05	0	0.1
Business Services				
Community, Social and Personal Services	0	0.05	0	0.1

Modeling strategy

The modeling strategy for occupational exposure to occupational particulates associated with COPD has remained unchanged since GBD 2010¹². The modeling strategy has been summarized below.

There are few direct measurements of exposure to particulates, gasses and fumes in the workplace. We estimated exposure to occupational particulates using the composition of a country's workforce in nine different industries¹³. Exposure to particulates in a particular industry was determined by multiplying the percentage of the workforce in the industry by the percentage of economically active people and by the prevalence of exposure to particulates in the industry. Population exposure is then determined as the sum of each industry's exposure for that country, age and sex group.

Formally, this is given by:

$$P_{c,y,a,s,level} = \sum_{i=1}^{9} EAP_{c,y,a,s,i} \ X \ EAC_{c,y,a,s} \ X \ EPF_{l}$$

 $P_{c,y,a,s,level}$ = Prevalence of exposure to occupational particulates, by country, year, age, sex, and level of exposure; $EAP_{c,y,a,s,i}$ = Proportion of the population that is economically active, by industry, country, year, age, and sex; $EAC_{c,y,a,s}$ = Proportion of the population that is economically active, by country, year, age, and sex;

 EPF_{level} = Exposure partition factor, by level of exposure; c = Country;

y = Year;
 a = 5-year age group;
 s = Sex;
 I = Level of exposure (high or low)
 i = Industry (9 types).

Relative Risks

Relative risks for occupational particulates were estimated by the occupational risk expert group by conducting a systematic review of international literature and meta-analysis of relevant studies. The table below outlines the effect sizes associated with occupational particulates and COPD used in GBD 2013.

Relative risk by age and particulates exposure level

¹² Lim SS, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet 2012*; **380**: 2224-60. ¹³ Driscoll T, et al. The global burden of non-malignant respiratory disease due to occupational airborne exposures. *American Journal of Industrial Medicine 2005*; **48(6)**: 432-445.

		H	ligh exposure		Lo	Low exposure		
Outcome	Age	RR	LL	UL	RR	LL	UL	
COPD	15-80	2.31	1.45	3.73	1.44	1.07	1.95	

The relative risks used for occupational particulates remained unchanged since GBD 2010. Draws of relative risks for occupational particulates were migrated centrally from GBD 2010 to GBD 2013 infrastructure. As a result, code that preps relative risks from scratch do not exist.

Theoretical Minimum-Risk Exposure Level

The TMREL for occupational particulates is defined as no occupational exposure to particulates, gases, and fumes.

Attributable Burden Estimation

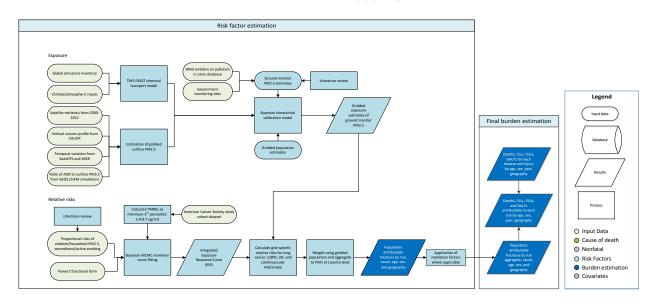
Population Attributable Fractions (PAFs) were calculated centrally using input draws of exposure, relative risks, and theoretical minimum level prepared by the modeler (Astha). The following equation which takes into account exposure distribution (P_i); relative risks i.e. measure of effect on outcome associated with each level of exposure (RR_i); and the counterfactual level of risk exposure (TMREL) was used to calculate PAFs centrally.

$$PAF = \frac{\sum_{i=1}^{n} P_i(RR_i - 1)}{\sum_{i=1}^{n} P_i(RR_i - 1) + 1}$$

Ambient Particulate Matter Pollution SDG Capstone Appendix

Flowchart

Ambient PM2 5



Input Data & Methodological Summary

Indicator definition

This modeling strategy encompassed the indicator associated with population-weighted PM2.5 (fine particulate matter) (11.6.2)

Indicator 11.6.2

As a component of SDG Goal 11. Make cities and human settlements inclusive, safe, resilient and sustainable, SDG Target 11.6, by 2030, reduce the adverse per capita environmental impact of cities, including by paying special attention to air quality and municipal and other waste management, is measured using SDG Indicator 11.6.2, Population-weighted mean levels of fine particulate matter (PM2.5).

Case definition

Exposure to ambient air pollution is defined as the population-weighted annual average mass concentration of particles with an aerodynamic diameter less than 2.5 micrometers ($PM_{2.5}$) in a cubic meter of air. This measurement is reported in $\mu g/m^3$.

Input data

The data to estimate exposure to ambient air pollution is drawn from estimates of annual concentration of $PM_{2.5}$ – generated using satellite observations of aerosols in the atmosphere. To correct for bias in the satellite modeling approach, a spatially-varying flexible framework is used to combine modeled concentrations with observations from ground-level monitoring of particles in more than 75 countries. All input data for GBD2015 was updated as follows:

Updated PM_{2.5} ground measurement database

For the GBD2015 update we updated the database of annual average PM measurements to include more recent data and to incorporate additional locations where measurement data have become available. To facilitate this we collaborated with WHO and contributed to their recently released WHO Air Pollution in Cities database. We then used disaggregated (monitor-specific values and not the city averages that are reported by WHO) measurements from this database with additional site-specific information (e.g. all monitors in a city, monitor geo coordinates, monitor site type) such as that included in the GBD2013 database. In total measurements of concentrations of PM₁₀ and PM_{2.5} were retrieved from 6,003 ground monitors with the majority contributing measurements from 2014 (as there is a lag in reporting measurements, little data from 2015 were available). Where data were not available for 2014 (2760 monitors), data was used from 2015 (18 monitors), 2013 (2155), 2012 (564), 2011 (60), 2010 (375), 2009 (49), 2008 (21) and 2006 (1). For locations with only PM₁₀ measurements, PM_{2.5} measurements were estimated from PM₁₀. This was done by a locally derived conversion factor (PM_{2.5}/PM₁₀ ratio) estimated as population-weighted averages of location-specific conversion factors for the country. Location-specific conversion factors were estimated as the mean ratio of PM_{2.5} to PM₁₀ of stations for the same year. If national conversion factors were not available, regional ones were used, which were obtained by averaging country-specific conversion factors.

Updated satellite-based estimates

The updated satellite-based estimates are described in detail in van Donkelaar et al. 2016^1 . These estimates (~11 x 11 km resolution at the equator) combine aerosol optical depth retrievals from multiple satellites with the GEOS Chem chemical transport model and land use information.

Updated population data

A comprehensive set of population data on a high-resolution grid was obtained from the Gridded Population of the World ($\underline{\mathsf{GPW}}\ \mathsf{v4}$) database. These data are provided on a 0.0417°×0.0417° resolution. To aggregate these estimates of population to each 0.1°×0.1° grid cell, the central 3 × 3 population cells were summed. As this accounted for a resolution higher than necessary, the same was done four other times, offset by one cell in a North, South, East and West direction. The average of five quantities was used as the aggregated population estimate for each cell. Estimates of population for 2000, 2005, 2010, 2015 and 2020 were extracted from GPW version 4 and estimates for 1990 and 1995 were extracted from GPW version 3 as described previously for GBD2013³.

Modeling strategy

The methodology used to estimate the burden of ambient particulate matter pollution has seen significant changes since GBD2013.

The GBD2010 and GBD2013 estimates both used a single global function to calibrate the mean of the chemical transport model and satellite-based estimates to available ground measurements. In both instances the approach taken was recognized at the time to be a compromise between what could be easily implemented under tight timeframes and one that most efficiently utilized all of the data sources. In particular, the GBD2013 exposure estimates were known to underestimate ground measurements in specific locations (see discussion in Brauer et al., 2015²) such that it would be desirable to allow measurements to make a larger contribution to the final estimates where they were available. Therefore, for GBD2015 we implemented a Bayesian Hierarchical modelling approach using Integrated Nested Laplace Approximations (INLA) in which the satellite-based estimates, ground measurements and land use information are combined in a spatially varying flexible framework. Formal external evaluation using ground measurements was conducted and shown to lead to improved predictions of ground measurements in all super regions compared to GBD2013 estimates and an alternative geographically-weighted regression approach. Further, based on the external evaluation analyses, addition of the TM5 chemical transport model estimates of PM2.5 annual average did not improve the estimates and these were therefore not included.

Bayesian hierarchical models (BHM) provide an extremely useful and flexible framework in which to model complex relationships and dependencies in data. Uncertainty can also be propagated through the model allowing uncertainty arising from different components, both data sources and models, to be propagated through the models into estimates of uncertainty associated with the final estimates. In the hierarchical modeling approach coefficients associated with satellite-based estimates were estimated for each country. Where data were insufficient within a country, information can be 'borrowed' from a higher aggregation (region) and if enough information is still not available from an even higher level (super-region). Individual country level estimates were therefore based on a combination of information from the country, its region and super-region.

All modelling was performed on the log-scale with the unit of measurement being a grid cell. The model was constructed with the inclusion of all variables assessed statistically, based on model fit (DIC, a measure of the relative quality of a model and closely related to that of AIC but for Bayesian models) and predictive ability. The hierarchical structure was applied to the intercept and slope terms with all modelling on the log scale. The model was of the form

$$log(PM2.5_i) = \beta_0 + \beta_1 log SAT_i + other variables + \varepsilon_i$$

where *i* denotes the grid cell. The following sets of variables were considering in developing the models:

Continuous explanatory variables:

- o (SAT) Estimate of PM_{2.5} (in μ gm⁻³) for 2014 from satellite remote sensing on the log-scale.
- o (CTM) Estimate of PM $_{2.5}$ (in μgm^{-3}) for 2014 from chemical transport models on the log-scale.

- o Estimate of population for 2014 on the log-scale.
- o (SNAOC) Estimate of the sum of sulfate, nitrate, ammonium and organic carbon as estimated from GEOS Chem
- o (DST) Estimate of compositional concentrations for mineral dust from GEOS Chem
- (EDxDU) The log of the elevation difference between the elevation at the ground measurement location and the mean elevation within the GEOS Chem simulation grid cell multiplied by the inverse distance to the nearest urban land surface

Discrete explanatory variables:

- o Binary variable indicting whether exact location of ground measurement is known
- o Binary variable indicting whether exact type of ground monitor is known
- \circ Binary variable indicting whether ground measurement is PM_{2.5} or converted from PM₁₀

Random Effects:

- o Grid cell random effects on the intercept to allow for multiple ground monitors in a grid cell.
- o Country-region-super-region hierarchical random effects for the intercept
- o Country-region-super-region hierarchical random effects for the satellite remote sensing term.
- o Country-region-super-region hierarchical random effects for the coefficient associated with the difference between estimates from CTM and SAT.
- o Country-region-super-region hierarchical random effects for the coefficient log(POP)
- o Country level random effects for intercept, satellite and difference between CTM and SAT are independent and identically distributed.
- Country level random effects for population uses a neighbourhood structure allowing specific borrowing of information from neighbouring countries.
- o All region random effects are assumed to be independent and identically distributed.
- All super-region random effects are assumed to be independent and identically distributed.

Interactions:

o Interactions between the binary variables and the effects of log(SAT) and log(CTM/SAT)

Due to both the complexity of the models and the size of the data, notably the number of spatial predictions that are required in this setting, recently developed techniques that perform 'approximate' Bayesian inference based on integrated nested Laplace approximations (INLA) have been developed as a computationally attractive alternative to Markov Chain Monte Carlo methods. Computation was performed using the R interface to the INLA computational engine (R-INLA) with the size of the task of fitting the models and performing predictions for each of the ca. 1.4 million grid cells requiring the use of a high performance computing cluster (HPC) with high memory nodes. As in GBD2010 and GBD2013 the spatial model was built combining the different data sources for a single year (2014, corresponds to the most recent measurement data). The spatially-varying functions from this model were then applied to the satellite-based estimates from all other years - in other words assuming that the spatial relationship between the different data sources does not change over time. This is undoubtedly a simplification but to

do otherwise would require assembling multi-year measurement databases which is not feasible given current data availability and computational constraints. As the spatial model was built using the most recently available (2014) measurement and satellite-based estimates, 2015 estimates were based on extrapolation. Instead of extrapolating using an exponential model based on a 1-year trend as in GBD2013, splines based on a 5 year trend (2010-2014) were fit and applied to the 2014 grid-cell values to estimate levels for 2015. This reduced the likelihood of 2015 estimates being overly influenced by meteorological events in a specific year and to better represent the duration of exposure relevant to the epidemiologic studies included in the integrated exposure-response functions.

Model Evaluation

Model evaluation and comparison was performed by fitting models on a training set and predicting exposures at locations for which measurements were known (the validation set). The selection of the training (20%) and validation (80%) set consisted of taking a random sample of the total number of sites measuring PM2.5 (or having a value converted from PM10 measurements). Sampling was performed using sampling probabilities based on the cross-tabulation of PM2.5 categories (0-24.9, 25-49.9, 50-74.9, 75-99.9, 100+ μ g/m3) and super-regions. The resulting hold-out evaluation data set was a sample of 20% of the sites that have the same distribution over PM2.5 categories and super-regions as the entire set of sites.

This process was used to generate multiple training and validation set combinations, allowing for example cross-validation to be performed. In the evaluation, 25 sets of training/validation data were used. The following models were considered in the evaluation phase:

- (A) The GBD2013 model, using a simple linear regression with a fused estimate of SAT and CTM together with interactions with three binary variables representing whether the measurement was converted from PM10 and whether the exact site type and location is known.
- (B) A hierarchical model with SAT, the TM5 CTM estimates, population and the three binary variables described above
- (C) A hierarchical model with SAT, population, SNAOC, DST, EDxDU, population and the three binary variables
 - o Estimate of population for 2014 on the log-scale.
 - o Estimate of the sum of sulfate, nitrate, ammonium and organic carbon as estimated from GEOS Chem
 - Estimate of compositional concentrations for mineral dust from GEOS Chem
 - o The log of the elevation difference between the elevation at the ground measurement location and the mean elevation within the GEOS Chem simulation grid cell multiplied by the inverse distance to the nearest urban land surface

For each training/evaluation set combination, model fit and prediction accuracy were evaluated for each of the 25 training/evaluation set combinations with the following metrics:

Model fit

- R²
- DIC

Predictive accuracy

• R² arising from a linear regression of predicted vs actual measurements at each location

- RMSE root mean squared error
- WRMSE weighted (by population) root mean squared error
- MSE mean square error
- MAE mean absolute error

This evaluation indicated the final model improved predictions of ground measurements in all super regions compared to GBD2013 estimates (median global R^2 [population-weighted RMSE] 0.82 (12.1 $\mu g/m^3$), 0.60 [13.5 $\mu g.m^3$] for GBD2015 and GBD2013, respectively).

Error! Reference source not found. shows the RMSE (median from the 25 runs) for each of the three models, by super-region. The accuracy of the prediction varies between super-regions, with lower errors being observed in areas where there are more monitoring sites. In each of the super-regions, the largest errors are seen for model A which are considerably higher than those for models B and C, with model C showing a small improvement over B (except in super-region 5, North Afirca/Middle East).

Figure 2 shows scatter plots of the observed and predicted measurements using the three models for each super-region. The predicted measurements are the median values over those obtained from the 25 training sets. Predictions from the two Bayesian hierarchical models (B&C) match the observed values more closely than the linear model (A) with much less spread around a straight line (with slope one and zero intercept, shown in red). In Central Europe and Sub-Saharan Africa it is noticeable that, in addition to reduced spread, models B&C are much better at predicting higher values. The same patterns of results in predictive ability were seen when looking at regions and individual countries. In all cases, model C performed better than model B with both being considerable better than model A.

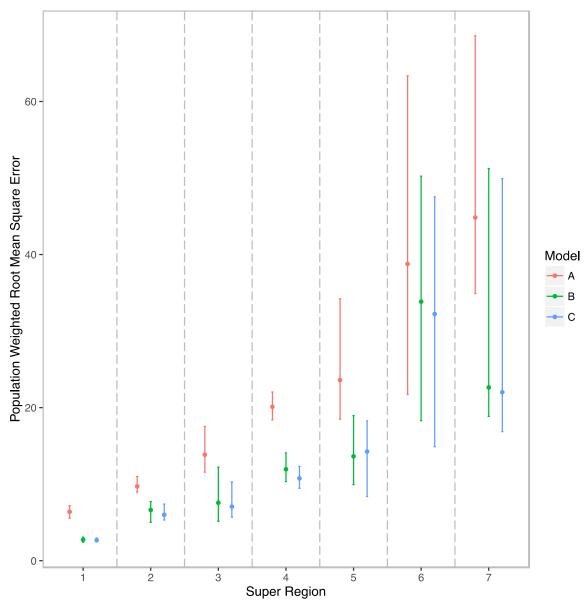


Figure 1: Comparison of RMSE from three models by super-region. Dots denote the median of the distribution from 25 training/evaluation sets and the vertical lines the range of values. Super-regions are 1: high income, 2: Central Europe, Eastern Europe, Central Asia, 3: Latin America and Caribbean, 4: Southeast Asia, East Asia and Oceania, 5: North Africa / Middle East, 6: Sub-Saharan Africa, 7: South Asia.

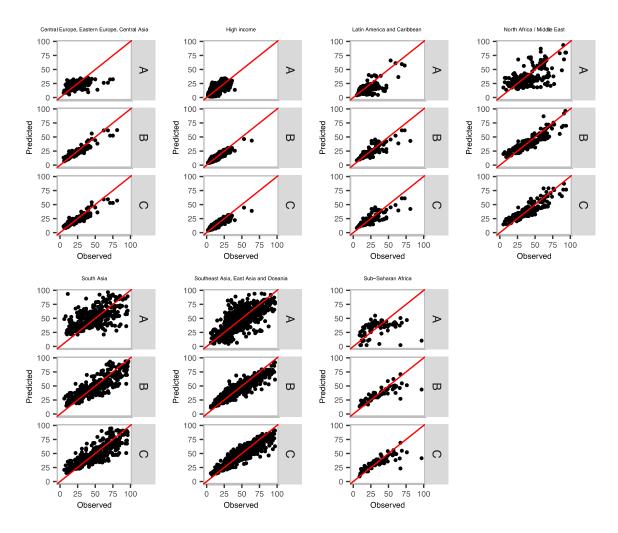


Figure 2: Comparison of observed and predicted measurements using three different models, by superregion. The red line has slope one and intercept zero.

Overall, the best model in terms of model fit and predictive ability was one with the following components:

- O Estimates of PM_{2.5} (in μgm⁻³) from satellite remote sensing (SAT), population, and information on the GEOS Chem simulated chemical composition, elevation and distance to urban land use (SNAOC, DST and EDxDU).
- o Binary variables indicting whether exact location and type of ground measurement is known, and whether the measurement was $PM_{2.5}$ or converted from PM_{10} .
- o Interactions between the binary variables and the effects of estimates from satellite remote sensing.
- o Grid cell random effects on the intercept to allow for multiple ground monitors in a grid cell.
- o Country-region-super-region hierarchical random effects for intercepts, satellite remote sensing and population terms.
- o Country level random effects for population using a neighbourhood structure allowing specific borrowing of information from neighbouring countries.

Relative Risk

Relative risks are generated using integrated exposure-response functions (IER) that are fit to available epidemiologic data using a Bayesian MCMC approach and a modified power function. The IER are estimated based on published relative risks for long-term exposure to ambient PM2.5, household air pollution, second-hand smoking, and active (cigarette) smoking. The concentration of particulate matter for each type of exposure is estimated based on literature values and used to map the relative risks to a curve generated for the entire range of exposure from these sources. The input data for this curve fitting process has been updated since GBD2013, adding new studies that estimate exposure at finer spatial scales, including studies of within-city exposure that focus on traffic-related air pollution. In addition, changes were made to the curve-fitting process. In order to account for differences in study design, temporal patterns of exposure and other differences among the studies of the different sources of PM2.5, a source-specific heterogeneity parameter was added to the IER. This resulted in much wider, and, in our view, more realistic, uncertainty intervals for the burden estimates, by propagating through the entire process the current uncertainty regarding the mechanisms and magnitude of health impacts of exposure to PM2.5 from diverse sources.

IER Functional Form

Data Likelihood

$$\log(RR_i) \sim \mathcal{N}ig(\mu_i, \sqrt{\sigma_i^2 + \delta_{source_i}}ig)$$

Model

$$\mu_i = \log \left(rac{1 + lpha imes \left(1 - e^{-eta imes (exposure_i - TMREL)^{\gamma}}
ight)}{1 + lpha imes \left(1 - e^{-eta imes (counterfactual_i - TMREL)^{\gamma}}
ight)}
ight)$$

Data

 RR_i : measured relative risk for data point i σ_i : variance of data point i based on study information $source_i$: exposure source type (outdoor/household air pollution, secondhand/active smoking) TMREL: theoretical minimum risk exposure level $exposure_i: \text{measured exposure for data point } i$ $counter factual_i: \text{counterfactual exposure for data point } i$

Priors

We also modified the way in which age-specific IER for IHD and stroke were estimated. In accordance with previously published work on other cardiovascular risk factors, the impact of air pollution on cardiovascular health is known to vary with age. To account for this phenomenon, age-specific RRs were based on a log-linear model of RR as a function of age, where the intercept (RR=1) is forced to age 110. In

GBD2010 and GBD2013 the age for a relative risk estimate from a given study was estimated as the median age at death or disease incidence in that study. However, this may not accurately represent the age distribution of the entire study population so we re-estimated this variable as the mean age at enrollment + half of the average follow-up time to better represent the average age of the study population during the period of follow-up. When compared to GBD2013, this change produced RRs that were generally lower for younger age groups, given that median age at event tends to produce a higher anchor age than average age during follow-up.

The relative risks are generated on the grid-level based on estimated exposure, and then applied to generate PAFs. These PAFs are aggregated using the grid-level population to create population-weighted national estimates of attributable burden, using the following formula:

PM2.5 Aggregation Formula

$$PAF_{A, C, L} = \frac{\sum ((RR_{A, C} - 1) * Pop_i)}{\sum (RR_{A, C} * Pop_i)}$$

A = age group

C = cause

L = location

i = grid

 $RR_{A,\ C}=$ grid-level RR based on $PM_{2.5}$ and given age/cause IER curve

TMREL

The TMREL for ambient PM is estimated using a uniform distribution between the minimum and 5th percentile of exposure observed in the studies used to generate the GBD estimates. This estimate was updated for GBD2015 as new studies were added to the analysis and studies used previously were updated through continued follow-up. The newer estimates included several large studies that included exposure at lower levels of PM2.5. As a result, the TMREL for GBD2015 was \sim U(2.4, 5.9), lower than GBD2013's distribution \sim U(5.9, 8.7), which had the effect, all things being equal, of increasing the estimated attributable burden relative to the GBD 2013 estimates.

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Part 2. SDG index sensitivity analyses

In this analysis we have constructed indices that represent overall performance on: the health-related SDG indicators (referred to as the SDG index); the indicators that were previous MDG indicators (MDG index); and indicators that are newly added compared to the MDGs (non-MDG index). These indices were constructed by first rescaling each health-related SDG indicator on a 0 to 100 scale with 0 being the lowest (worst) value observed and 100 being the highest (best) value observed over the time period 1990 to 2015. The health-related SDG index was then computed by first determining the geometric mean of each rescaled health-related SDG indicator for a given target and then taking the geometric mean of the resulting values across the targets. This approach weights each of the health-related SDG targets equally and assumes partial substitutability with high values on one target partly compensating for low values on another target.

In addition to this approach to constructing the index, we used three alternative construction methods, namely: (i) principal component analysis (PCA); (ii) arithmetic mean across the health-related targets; and (iii) minimum value across the health-related targets. For each of the alternative methods we first rescaled the individual health-related indicators as described above. The PCA identified five principal components with eigenvalues greater than one. The first principal component, however, included both positively, eg maternal mortality ratio, and negatively correlated indicators, eg. alcohol use. Having a negative correlation on some components is not in line with the goals of the SDG targets and we do not compare the results of the PCA further.

For the second approach, we first take the arithmetic mean of indicator values for a given target and then the arithmetic mean of the resulting values across the targets. In contrast to the approach using the geometric mean, this approach assumes complete substitutability whereby poor performance on a target is linearly compensated for by better performance on another target. The resulting index using this approach was highly correlated with the approach using the geometric mean both in terms of 2015 values of the health-related SDG index (Appendix Figure 7a; Pearson correlation coefficient = 0.99, p<0.0001) as well as the corresponding country ranks (Appendix Figure 7b; Spearman's rank correlation coefficient=0.99, p<0.0001).

For the third approach, we first take the geometric mean of indicator values for a given target then determine the minimum of the resulting values across the health-related targets. This approach adopts what is called zero substitutability, ie better performance on one target in no way compensates for poor performance on another target. The resulting index using this approach was also well correlated with the approach using the geometric mean both in terms of 2015 values of the health-related SDG index (Appendix Figure 8a; Pearson correlation coefficient = 0.88, p<0.0001) and country rank (Appendix Figure 8b; Spearman's rank correlation coefficient=0.91, p<0.0001), although not as highly as the approach taking the arithmetic mean.

Part 3. Comprehensive citation list

Methods Appendix Table 2. Comprehensive Citation List

Citation

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Max Planck Institute for Demographic Research, University of California, Berkeley. Slovenia Human Mortality Database Deaths Period Data.

Citation

Max Planck Institute for Demographic Research, University of California, Berkeley. Spain Human Mortality Database Deaths Period Data.

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Max Planck Institute for Demographic Research, University of California, Berkeley. Switzerland Human Mortality Database Deaths Period Data. Max Planck Institute for Demographic Research, University of California, Berkeley. Taiwan Human Mortality Database Deaths Period Data. Max Planck Institute for Demographic Research, University of California, Berkeley. Ukraine Human Mortality Database Deaths Period Data. Maxwell CA, Curtis CF, Haji H, Kisumku S, Thalib AI, Yahya SA. Control of Bancroftian filariasis by integrating therapy with vector control using polystyrene beads in wet pit latrines. Trans R Soc Trop Med Hyg. 1990; 84(5): 709-14. As it appears in London School of Hygiene and Tropical Medicine. Global Atlas of Helminth Infections - Lymphatic Filiariasis.

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Part 4. Online results and abbreviations

Part 1. Online Results

Further results are presented as dynamic visualizations at http://www.healthdata.org/results/data-visualizations.

Part 2. List of abbreviations

20q50: probability of death from age 50 years to 70 years

35q15: probability of death from age 15 years to 50 years

45q15: probability of death from age 15 years to 60 years

5q0: probability of death from birth to age 5 years

ANC: antenatal care

ART: antiretroviral therapy

ASFR: age-specific fertility rate

BMD: bone mineral density

BMI: body-mass index

BTL: basic tabulation list

CBH: complete birth histories

CDC: Center for Disease Control and Prevention

CDR: crude death rates

CF = cause fraction

CHERG: Child Health Epidemiology Research Group

CHNS: China Health and Nutrition Survey

CI5: Cancer Incidence in Five Continents

CKD: chronic kidney disease

CKD-DM: chronic kidney disease deaths attributable to diabetes

CoD: Cause of Death

CODEm: Cause of Death Ensemble model

COPD: chronic obstructive pulmonary disease

CR: cancer registry

CRA: comparative risk assessment

CRS: civil registration system

CSA: childhood sexual abuse

CVD: cardiovascular disease

DALY: disability-adjusted life-year

DDM: deaths distributions method

DHS: Demographic and Health Survey

DRI: data representativeness index

DSP: Disease Surveillance Points

ELISA: enzyme-linked immunosorbent assay

EM-DAT: Epidemiology of Disasters' International Disaster Database

EPEC: enteropathogenic E. coli

EPP: estimation and projection package

ETEC: enterotoxigenic E. coli

FAO: Food and Agriculture Organization

FBS: fasting blood sugar

FFQ: food frequency questionnaire

GATHER: Guidelines for Accurate and Transparent Health Estimates Reporting

GBD: Global Burden of Diseases, Injuries, and Risk Factors Study

GEMS: Global Enteric Multicenter Study

GHDx: Global Health Data Exchange

HDI: Human Development Index

Hib: haemophilus influenzae type B

HIV CDR: crude death rate due to HIV/AIDS

IAEG-SDGs: Inter-Agency and Expert Group on Sustainable Development Goal Indicators

IARC: International Agency for Research on Cancer

ICD: International Classification of Disease

IER: integrated exposure response

IHD: ischemic heart disease

IISS: International Institute for Strategic Studies

ILO: International Labour Organization

IOTF: International Obesity Task Force

IPV: intimate partner violence ITNs: insecticide-treated nets

LDI: lag distributed income per capita

LRI: lower respiratory infection

LSMS: Living Standards Measurement Survey

MAP: Malaria Atlas Project

MCCD: Medical Certification of Causes of Death

MCEE: Maternal and Child Epidemiology Estimation group

MDG: Millennium Development Goal

MI: mortality-to-incidence ratio

MICS: Multiple Indicator Cluster Survey

MM: maternal mortality

MMR: maternal mortality ratio

MMS: maternal mortality surveillance

NCDs: non-communicable diseases

NCHS: National Center for Health Statistics

NTDs: neglected tropical diseases

PAF: population attributable fraction

PCA: principal component analysis

PEM: protein-energy malnutrition

PHMRC: Population Health Metrics Research Consortium

PM2.5: particulate matter <2.5μm in diameter

PMTCT: prevention of mother-to-child transmission

PUFA: polyunsaturated fatty acids

RCT: randomised controlled trial

RHS: Reproductive Health Survey

RMSE: root mean square error

RSV: respiratory syncytial virus

SBA: skilled birth attendance

SBH: summary birth history

SBP: systolic blood pressure

SCD(R): survey of cause of death (rural)

SCD: survey of cause of death

SD: standard deviation

SDG: Sustainable Development Goals

SDI: Socio-Demographic Index

SDSN: Sustainable Development Solutions Network

SEER: Surveillance, Epidemiology, and End Results Program

SEV: summary exposure value

SIR: smoking impact ratio

SRS: sample registration system

SSB: sugar-sweetened beverages

ST-GPR: Spatiotemporal Gaussian process regression

TAB: Tabulation List

TAC: TaqMan Array Card

TB: tuberculosis

TFR: total fertility rate

TMREL: theoretical minimum risk exposure level

UCDP: Uppsala Conflict Data Program

UHC: universal health coverage

UI: uncertainty interval

UK: United Kingdom

UN: United Nations

URIs: upper respiratory infections

US: United States

VA: verbal autopsy

VR: vital registration

WaSH: water, sanitation, and hygiene

WHO: World Health Organization

WPP: World Population Prospect

YLD: years lived with disability

YLL: years of life lost