Death in Indonesia: estimating all-cause mortality, cause-specific mortality, and fatal burden attributable to smoking and air pollution

Sarah Wulf Hanson

A dissertation

submitted in partial fulfillment of the

requirements for the degree of

Doctor of Philosophy

University of Washington

2019

Reading Committee:

Theo Vos, Chair

Laura Dwyer-Lindgren

Emmanuela Gakidou

Christopher J. L. Murray

Program Authorized to Offer Degree:

Global Health

University of Washington

Abstract

Death in Indonesia: estimating all-cause mortality, cause-specific mortality, and fatal burden attributable to smoking and air pollution

Sarah Wulf Hanson

Chair of Supervisory Committee:

Theo Vos

Department of Health Metrics Sciences

Indonesia is a populous, diverse country that has not been well studied in terms of health. This analysis addresses that knowledge gap in three aims by first, estimating the number deaths in Indonesia by province, year, age, and sex; second, developing a cause list based on cause of death assignment accuracy and estimating cause-specific mortality by province, age, and sex in 2014 according to that cause list; and third, comparing four methods to attribute deaths to risk factors that can be measured by fine particulate matter concentration.

The first aim adapted existing demographic techniques used by the Global Burden of Disease study to estimate subnational all-cause mortality over time by age and sex in Indonesia. Province-level life tables were generated from survey data about household deaths given the lack of a vital registration system in Indonesia and included in the analysis, along with all available data for population, education, income, child mortality, and adult mortality.

The second aim consists of two parts. First, an analytical cause list was generated based on the accuracy of cause of death assignments in physician-certified verbal autopsy data. Second, this cause list was

applied to all available physician-certified verbal autopsy data in Indonesia in order to estimate cause-specific mortality by province, age, and sex in 2014 using small area estimation methods.

The third aim compares four methods of calculating attributable burden to risk factors related to fine particulate matter—ambient air pollution, household air pollution, active smoking, and secondhand smoke—for fatal health outcomes that have an established relationship between fine particulate matter exposure level and risk ratio of the outcome.

Acknowledgments

This work would not have been feasible or finished without the support shown me by so many people along the way.

I would like to thank the Institute for Health Metrics and Evaluation and University of Washington for their financial and intellectual support: in particular, the IT and Data teams for their ever-ready help and humor, Admin and Ops for a corner office for our fledgling years when the PhD program began, and the UW libraries for faithfully fulfilling my many obscure article requests.

I also thank my collaborators at the National Institute for Health and Research Development in Indonesia led by Dr. Soewarta Kosen, who welcomed me into their office, shared tea and stories and data with me, and convinced me to taste durian. Terima kasih banyak Pak Alo, Endah, dan kolega-kolega saya yang luar biasa.

I give my deep gratitude to my committee for their loyal support and feedback throughout my dissertation process and for setting and maintaining a high bar. To Laura Dwyer-Lindgren, for her expertise in small area estimation and encouragement. To Chris Murray, for inspiring and facilitating my choice of studying burden of disease in Indonesia and for his keen methodological insights. To Emmanuela Gakidou, for creating this program and leading us students and candidates through the PhD process with grace and understanding. To Bob Mugerauer, for his kindness and invaluable perspective on facing difficulties during a PhD. And to Theo Vos, for his patient commitment, care, and guidance through the long years, interruptions, periods of slow progress, and many chapter drafts.

I am grateful to my colleagues both near and far. To my early officemates David Phillips, Grégoire Lurton, and Roy Burstein for inspiring laughter and curiosity at our intellectual hours, and to Joe Dieleman for inspiring the practice. To my close friends and all my fellow PhD students, for their support, optimism, and encouragement along the way. To the Facebook™ group "PhD and Early Career Researcher Parents" for the shared experiences, commiseration, encouragement, and companionship among researcher parents across the world. And to my church community, whose love and care exemplify that of Jesus.

I am indebted to the coffee shops within walking distance, where I have spent countless days writing code, debugging code, staring into space, creating too many figures and tables, sipping coffee, drafting and redrafting chapters, holding back tears, holding back jubilation, and reminding myself to stretch and drink some water occasionally.

Special gratitude is reserved for Bruce Johnson, whose willingness to put another's life before his own in the face of danger will always remind me of the goodness and grace of God, without whom I would likely not be here today.

I am so grateful to my family. To my brothers Ben and Adam, their wonderful families, my extended family, and my family-in-law for the love, interest, and care they have all shown as they cheered me on

to the final submission. To my mother for sacrificing many weeks to care for us during times of need, and to my father for his corresponding sacrifices at home; I would not have finished without you and am humbled by your gift.

I thank Graeme, who has only ever known me as a PhD student but who has always seen me as much more than that. Thank you for loving me apart from what I do and for also loving what I do. Your humility, selflessness, kindness, and humor inspire me daily, and I can't wait to watch Survivor with you tonight.

Luke and baby M, thank you for letting me be your mom. That is my greatest joy. Though you won't remember this time, know that you have inspired me to, as my dad says, "study hard, eat well, sleep lots, and play some." I pass this advice on to you, with plenty of play as your study.

Death in Indonesia: estimating all-cause mortality, cause-specific mortality, and fatal burden attributable to smoking and air pollution

Sarah Wulf Hanson, MPH
s.wulf.hanson@gmail.com

ADVISOR:

Theo Vos, MD, MSc, PhD
Professor, Health Metrics Sciences, University of Washington

READING COMMITTEE:

Laura Dwyer-Lindgren, PhD
Assistant Professor, Health Metrics Sciences, University of Washington

Emmanuela Gakidou, MSc, PhD
Professor, Health Metrics Sciences, University of Washington
Senior Director of Organizational Development and Training, Institute for Health Metrics and Evaluation

Christopher J.L. Murray, MD, DPhil
Chair and Professor, Health Metrics Sciences, University of Washington
Director, Institute for Health Metrics and Evaluation

GRADUATE STUDENT REPRESENTATIVE:

Robert Mugerauer, PhD
Professor, Urban Design and Planning, University of Washington
Professor, Architecture, University of Washington
Director, PhD in the Built Environment Program

Contents

Available as supplemental files through ProQuest Dissertations & Theses and at https://drive.google.com/open?id=1USmqxenZeI9BwNGr1M4vSilRNrdFqe6p:

Chapter 1 Supplementary Tables

Chapter 1 Supplementary Figures

Chapter 2 Supplementary Tables

Chapter 2 Supplementary Figures

Chapter 3 Supplementary Table

Chapter 3 Supplementary Figures

Introduction

The fourth most populous country in the world, Indonesia is home to a diverse 250 million people across thousands of islands (1). The health of this population has not been well characterized to date, in part due to the lack of a vital registration system. However, both the wealth of survey data and the recent scaling up of a sample registration system with verbal autopsies have enhanced the ability to estimate mortality levels and causes of death in Indonesia.

Estimates of many indicators are produced within Indonesia by the national statistics agency BPS (2), but with several limitations. First, the list of indicators is not comprehensive: there are estimates for maternal mortality, fertility, and neonatal mortality, but no all-cause child or adult mortality, and no cause-specific mortality even among maternal or neonatal deaths. Second, the health indicators are reported at the national level but rarely at the province level. Third, estimates are only produced for survey-years using a single source for each estimate rather than utilizing the strength of all available data to produce annual estimates of any indicator (3).

The Global Burden of Disease study (GBD) produces annual estimates of health outcomes in Indonesia, and the most recent publication in 2017 produced results at the province level (4–8). GBD utilizes the strength of most available data in Indonesia but does have some opportunities for improvement. For instance, the all-cause mortality process does not include any Indonesia-specific life tables, the spatial hierarchy of GBD treats all provinces equally rather than taking into account geographical proximity and the distinction between land and sea borders, and comparative risk analysis of GBD assumes the effects of risk factors defined by their level of fine particulate matter concentration are independent.

In this dissertation, I have developed and completed three research aims that contributed to GBD 2017 estimates for Indonesia (4–8), and that make further contributions to subnational cause of death analysis and to burden attribution to fine particulate matter. Briefly, I estimated the number deaths in Indonesia by province, assessed what diseases and injuries cause these deaths and how this fatal disease profile differs by province, and compared four methods to attribute deaths to risk factors that can be measured by fine particulate matter concentration: ambient air pollution, household air pollution, active smoking, and secondhand smoke.

Aim 1: To estimate all-cause mortality by age and sex for Indonesian provinces from 1990 to 2014

> All-cause mortality is the foundation for many important population-level health indicators, but it can be difficult to quantify accurately. This analysis utilized all available data on child and adult mortality levels to generate age- and sex-specific mortality rates by province in Indonesia with uncertainty. These results are useful not only for assessing differences in health in terms of premature death, but also as a crucial primary input into answering the necessary subsequent step of identifying the causes of these differences as well as the opportunities for prevention and intervention. I utilized GBD methods for most parts of this analysis, along with additional data extractions and analyses to make subnational estimates possible.

Several parts of this analysis directly contributed to the GBD 2017 province-level estimates for Indonesia: mapping province boundaries over time for consistency of annual estimates; annual estimates of population numbers by province, age, and sex; and a province-level time series of lag-distributed income (LDI) per capita. In addition, from this analysis I generated province-level empirical life tables in Indonesia to inform the all-cause mortality age patterns, which can contribute to future GBD iterations.

Aim 2: To estimate cause-specific mortality rates and years of life lost by age and sex in Indonesian provinces for 2014 utilizing physician-certified verbal autopsy data

Until 2014, knowledge about causes of death in Indonesia depended heavily on sparse and inconsistently-collected hospital data, self-report questions in surveys about a household or community, and expert knowledge from local leadership. The collection of a nationally-representative sample of verbal autopsy (VA) deaths in 2014 makes a more systematic and comparable set of cause of death estimates possible, and this analysis aims to fill that knowledge gap and contribute to the better understanding of health and death by generating province-level estimates of causes of death in Indonesia.

I generated an analytical cause list appropriate for physician-certified verbal autopsy data based on validation datasets created by the Population Health Metrics Research Consortium (PHMRC) (9); this has not been done before to my knowledge (after assessing all publications that cite the PHMRC work) and is a crucial first step in using VA data for cause of death estimates. Also, I created subnational estimates of cause-specific mortality utilizing small area estimation that incorporates province spatial information rather than treating all provinces as equal in one hierarchy level within the analysis regressions as in GBD.

Aim 3: To compare four methods of estimating attributable burden to all fine particulate matter related to smoking and air pollution in Indonesia

Accurately estimating risk factor exposures is a critical component of a burden of disease analysis to understand the health profile of a population and possible opportunities for intervention. This analysis compares four methods for calculating population-attributable fractions for four causes of death—ischemic heart disease, stroke, lung cancer, and lower respiratory infections—attributable to ambient air pollution, household air pollution, active smoking, and secondhand smoke. As inputs for all four methods, I utilized risk factor exposures and integrated exposure-risk curves from GBD 2017, and I applied cause-specific mortality results from Aim 2 above to obtain mortality estimates attributable to each risk factor and combination of risk factors.

As a result of this comparison, I recommend updated methods to account for the additive nature of fine particulate matter exposures rather than assuming independence among any of these risk factors.

References

1. Population [Internet]. BPS Statistics Indonesia; 2015. Available from: http://www.bps.go.id/Subjek/view/id/12#subjekViewTab3|accordion-daftar-subjek1

2. Statistics Indonesia [Internet]. [cited 2019 Jan 28]. Available from: https://www.bps.go.id/

3. Statistics Indonesia [Internet]. [cited 2018 Jul 26]. Available from: https://www.bps.go.id/subject/30/kesehatan.html#subjekViewTab3

4. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1923–94.

5. GBD 2017 Mortality Collaborators. Global, regional, and national age-sex-specific mortality and life expectancy, 1950-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1684–735.

6. GBD 2017 Causes of Death Collaborators. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1736–88.

7. GBD 2017 DALYs and HALE Collaborators. Global, regional, and national disability-adjusted life-years (DALYs) for 359 diseases and injuries and healthy life expectancy (HALE) for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 10;392(10159):1859–922.

8. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1789–858.

9. Population Health Metrics Research Consortium (PHMRC) [Internet]. Institute for Health Metrics and Evaluation. 2014 [cited 2018 Nov 7]. Available from: http://www.healthdata.org/population-health-metrics-research-consortium-phmrc

Chapter 1

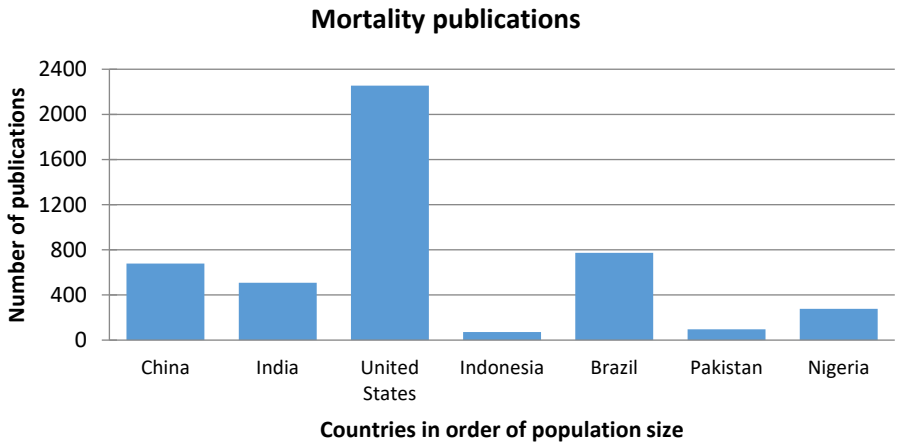
Age-specific and sex-specific all-cause mortality in 34 Indonesian provinces, 1990-2015

Introduction

All-cause mortality is the foundation for many important population-level health indicators, but it can be difficult to quantify accurately if no complete recording of deaths exists. These numbers are not only useful for assessing differences in health in terms of premature death, but they are also a crucial primary input for the necessary subsequent step of identifying the causes of these differences as well as the opportunities for prevention and intervention.

Indonesia has been understudied in terms of published estimates of its population's mortality. Other than the Global Burden of Diseases, Injuries, and Risk Factors study (GBD) (1,2), there have been several publications about all-cause mortality in Indonesia (Supplementary Table 1), though not as many as a country with a population this large might warrant (Figure 1). Of the 66 publications, 38 focus solely on infant or child mortality, 9 only on maternal conditions, 3 on both maternal and infant/child mortality, 1 on the elderly, and 15 publications covering all ages—often without any age or sex pattern. Over a third of these publications describe Indonesia as a whole, and all the other publications describe one sub-population or geography (certain hospitals, a city, urban slums, etc.). None provide mortality estimates for multiple subnational locations using systematic, comparable methods and data, much less by age, sex, and year.

Figure 1. The number of peer-reviewed journal articles containing the country name and "death" or "mortality" in their titles available in PubMed for the largest countries in the world. PubMed search criteria (country[Title] AND (mortality[Title] OR death[Title])



The Indonesian national statistics office, BPS, publishes the mortality results from national surveys as they become available for infants and children, as well as population, fertility, and migration estimates. However, they do not produce a publicly-available full set of age- and sex-specific mortality rates,

neither at the national nor more local level, nor do they interpolate in order to have a full time series of these data.(3)

Indonesia's size and diversity across the 6,000 inhabited islands(4) require more detailed estimates to assess the trends and levels of mortality across geography. Because the government decentralization of Indonesia in 1999 gave more political and economic autonomy to individual provinces and there is a wealth of survey data at that level, provinces are the unit of analysis in this study.(5)

Currently, publicly-available estimates of all-cause mortality in Indonesia rely on a single data source rather than combining sources, are not at the subnational level, and/or are not age/sex-specific (Supplementary Table 1). These estimates are also not reported with any uncertainty around them, though much uncertainty exists in the raw data and modeling procedures. This study quantifies annual age/sex-specific all-cause mortality estimates with uncertainty for all Indonesian provinces using all relevant data sources.

Methods

Overview

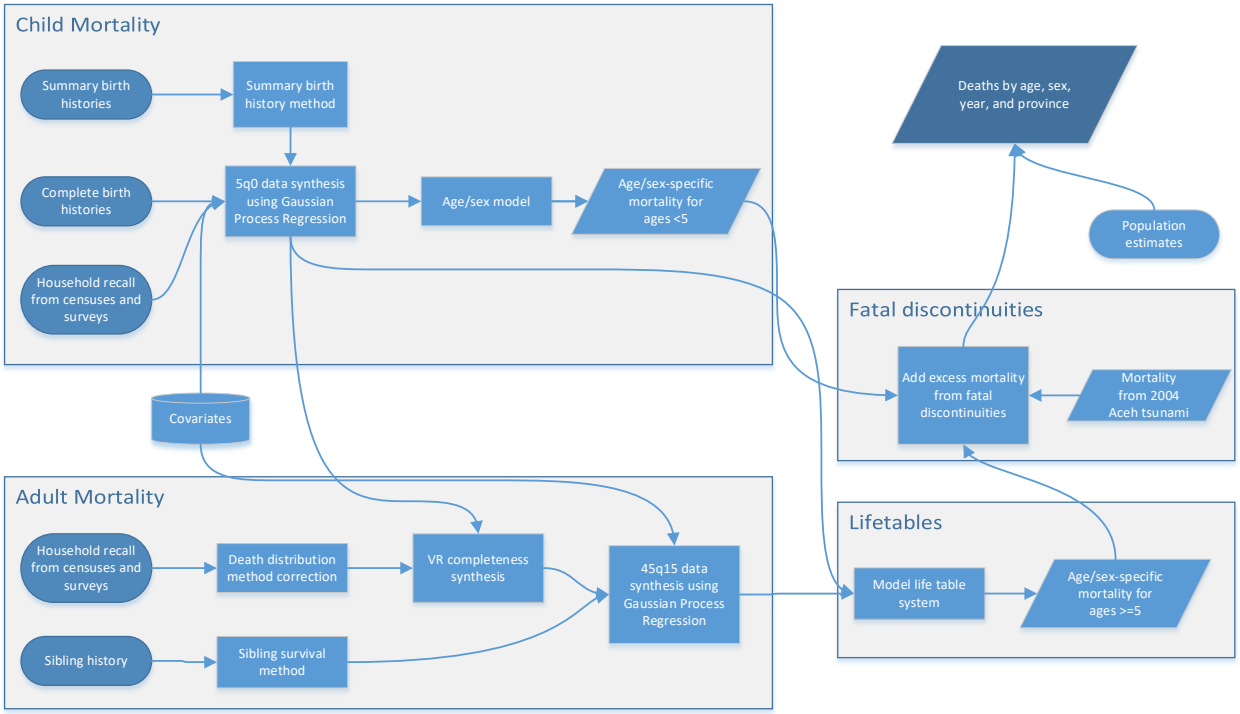
In the absence of a vital registration system, the estimation of all-cause mortality rates in Indonesia has to rely on complete birth histories, summary birth histories, reported household deaths, and sibling histories collected in surveys and censuses.

Since adult mortality questions cover a much wider age range than children, age misreporting or heaping can be an issue because the respondent may not know the exact age at death of the deceased person in question and would then be likely to round to the nearest multiple of 5. For this reason and to prevent implausible age trends due to small sample size or non-represented age groups in the raw data, it is useful to first estimate the probability of dying between age 15 and 60, and then to obtain the fine age pattern using a model life table. Model life tables define a relationship between age and mortality levels, derived from observed high-quality age-specific mortality rates in demographically similar populations.

The estimation process in this study has four main components: estimating under-5 mortality, estimating adult mortality, estimating age-specific mortality using model life tables, and adding excess mortality due to fatal discontinuities (Figure 2). The first two components include identifying data sources, extracting the relevant metrics from these data while taking into account measurable data

biases, and data synthesis methods. The third and fourth components use these child and adult mortality estimates along with a model life table system to generate annual mortality rate estimates for all 34 provinces, 19 age groups (neonatal, postneonatal, 1-4, and 5-year age groups up to 80+), and two sexes, from 1990 to 2015. Each component propagates uncertainty from sampling error, estimated non-sampling error, missing data, and model parameters.

Figure 2. Estimation process for all-cause mortality



This analysis adheres to the new Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER) proposed by the World Health Organization (WHO) and others.(6) Supplementary Table 2 shows the precise ways in which I have adhered to each element of the GATHER agreement.

Geographic units and time period estimated

Over the estimation period, eight provinces have split into two, creating eight new provinces and resulting in a total of thirty-four provinces (Table 1). To account for changes in province boundaries over

time, data were extracted at the district level and assigned to the province that they belong to as of 2016.

Table 1. List of provinces created since 1973 and their original province aggregate before creation.

Province	formerly part of	year created
North Maluku	Maluku	1999
Bangka–Belitung Islands	South Sumatra	2000
Banten	West Java	2000
Gorontalo	North Sulawesi	2000
Special Region of West Papua	Papua	2003
Riau Islands Province	Riau	2004
West Sulawesi	South Sulawesi	2004
North Kalimantan	East Kalimantan	2013

Data were analyzed to generate annual estimates from 1990 to 2015 for each province. For the primary national and subnational results, I have focused on trends over the past 25 years, from 1990 to 2015, and detailed findings in 2015.

Data source summary

Data on birth histories, household deaths, sibling histories, and population were extracted from available periodic population surveys, censuses, and intercensal surveys (Table 2). The under-5 mortality database consists of data from complete and summary birth histories, and adult mortality was informed by data from household deaths and sibling histories.

Table 2. Number of provinces covered by each mortality data source for Indonesia by type. SBH = summary birth history, CBH = complete birth history, HH = household deaths, SIBS = sibling survival, POP = population.

Survey name*	SBH	CBH	HH	SIBS	POP
1971 Census					18
1980 Census	34				34
1990 Census	32				34
2000 Census	33	34	27		33
2010 Census	33				34
SUPAS 1985	34		national		34
SUPAS 1995	34	33			34
SUPAS 2005		33	22		33
SUSENAS 1992	30				
SUSENAS 1993	30				

SUSENAS 1994	30			
SUSENAS 1995	29			
SUSENAS 1996	30			
SUSENAS 1997				
SUSENAS 1998	30			
SUSENAS 1999	30			
SUSENAS 2000	28		17	
SUSENAS 2001	33			
SUSENAS 2002	29			
SUSENAS 2003	34			
SUSENAS 2004	34		15	
SUSENAS 2005	33			
SUSENAS 2006	34			
SUSENAS 2007	33		21	
SUSENAS 2008	34			
SUSENAS 2009	34			
SUSENAS 2010	34			
SUSENAS 2011	34			
SUSENAS 2012	34			
SUSENAS 2013	34			
Family Life Survey 1993	13	16		15
Family Life Survey 1997	13	16		14
Family Life Survey 2000	17	22		16
Family Life Survey 2007	18	20		
Family Life Survey East	7	7		
DHS 1987	20	22		
DHS 1991	27	34		
DHS 1994	27	34		31
DHS 1997	27	34		33
DHS 2002-2003	26	29		28
DHS 2007	33	34		34
DHS 2012	33	34		34

**SUPAS = Intercensal Population Survey; SUSENAS = National Socioeconomic Survey; DHS = Demographic and Health Surveys*
Note: Different surveys sometimes contained different combinations of questions about mortality, even within the same survey series or among different provinces. For example, DHS 1997 contained CBH responses for 34 provinces but SBH responses for only 27 provinces.

Complete birth history microdata and ${}_5q_0$ computation

Complete birth histories rely on administering surveys to women who have given birth. The questionnaires ask about all living and deceased children, including date of birth, survival status, and date of death. These modules are included in many routine survey series, including the Demographic and Health Surveys (DHS), censuses, and many national survey programs in Indonesia. I downloaded and used microdata that have individual-level survey responses as opposed to using tabulated results.

Microdata from complete birth histories allow for direct calculation of death numbers and probabilities of death in the under-5 age group. Surveys were pooled by series, as in GBD,(7) grouping observations from all DHS complete birth history questionnaires from a province into one full set of observations and all census observations from multiple survey years into another full set of observations to give annual estimates of under-5 mortality.

Under-5 age-sex patterns

In the absence of vital registration systems, complete birth history surveys can be used to obtain age-sex patterns of mortality in under-5 age groups. For all complete birth history microdata sources, I applied direct estimation methods to obtain probabilities of death for each of the under-5 age groups: neonatal, late neonatal, and postneonatal. Within each survey, if each observation is a child recalled by a mother, observations were grouped into 5-year groups in time to provide a data point of probability of death for each of the under-5 age-sex groups. Recall was cut off 15 years before the survey, limiting data points estimated from the survey to the 15 years prior. All of these estimates were then in the database for estimating the age-sex pattern of under-5 mortality.

Summary birth history microdata and ${}_5q_0$ computation

Summary birth history (SBH) questionnaires are a shorter alternative to complete birth histories. Instead of asking in detail about each child, summary birth histories simply ask women how many children they have given birth to and how many of those children have died. The questionnaires are shorter and are more easily attached to other surveys. Often, censuses and household surveys contain summary birth histories. For this study, I have complied all available SBH data with micro-level data that enables us to apply the updated summary birth history method that leads to more accurate and timely assessment of under-5 mortality rate. I applied the combined summary birth history method from Rajaratnam et al. to create estimates of under-5 mortality rates from microdata on SBH from surveys and censuses.(8)

Household recall of deaths

Household recall is ascertained from large survey series including DHS, Family Life Survey, the census, and intercensal surveys (Table 2). A survey series must include a module asking about the number of deaths of household members within a given recall time period. These survey series must be considered geographically representative, include survey weights (if applicable), and include the sex and age (either current or at death) of all household members**.**

Completeness Assessment: Death Distribution Methods and completeness estimates synthesis

Household recall of deaths may not accurately capture all adult deaths because of recall issues like age misreporting. It is important to assess the quality, or in other words, the completeness, of available household death data. Demographers have long been applying a suite of death distribution methods (DDMs) including generalized growth balance (GGB), synthetic extinct generation (SEG), and a combined approach (GGBSEG) to assess completeness.(9–13) These methods compare the age distribution of the population recorded in two surveys with the age distribution of deaths recorded between these two surveys and attempt to estimate completeness of the data. Recent modifications of these DDM methods provide estimates of completeness that, based on careful simulation studies, are more accurate and robust than traditional methods; nevertheless, these methods generate completeness estimates with substantial uncertainty intervals.(14) For the GBD study, the process for estimating completeness of survey death data for adults is based on estimates of adult completeness from three death distribution methods updated by Murray and colleagues in 2010 as well as information about child completeness.(14) These two sources of information were combined to generate a series of estimates of source- and province-specific adult death registration completeness from 1970 to 2015. The underlying assumption of this process is that completeness of systems will change gradually, and consequently, assessments of completeness for a given year should be informed by DDM estimates for prior and future years in that province. Further, completeness is likely to be similar among provinces within a region in Indonesia, and I can inform estimates of completeness with levels of completeness estimated for provinces in the same region by borrowing strength over space as well as time. To do this, I used a two-stage model, whereby I first predicted adult completeness based on child completeness and then used a spatial-temporal regression model to incorporate information about adult completeness from the application of DDM methods. Child completeness was calculated as the ratio of observed child mortality in the source with household recall (DHS, FLS, census, or intercensal survey) to

estimated child mortality for the given source, province, and year. For a particular province-source, estimates of child completeness were only available for years where that data source was present, but a complete time series of child completeness estimates was produced, conditional on how many years of data the source covered within the province. If a source within a province covered only one, two, or three years of data, a constant level of child completeness at the level of the mean of those years that were available was assumed for the entire time series. However, for province-sources with more than three years of data, Loess regression was used to fill in the time series. In order to be prevent implausible out-of-sample estimates of child completeness, instead of using the Loess predictions to forecast and backcast I applied the child completeness of the first observation to all years preceding it, and the child completeness of the last observation to all years following it. Additionally, when there was a gap of more than five years I linearly interpolated between the observations on either side of this gap instead of using the Loess predictions to fill in these years.

Completeness data synthesis

Once I obtained a full series of under-5 completeness estimates for each province-source, I fit the model described in the equation:

$$log_{10}\left(c_{i,s,t}^{adult}\right) = \alpha + \beta_1 * log_{10}\left(c_{i,s,t}^{child}\right) + \gamma_1^R + \gamma_2^R * log_{10}\left(c_{i,s,t}^{child}\right) + \eta_{i,s} + \xi_{i,s,t}$$

Where $c_{i,s,t}^{adult}$ is completeness of adult deaths recording in province *i*, source *s*, at time *t*;

$c_{i,s,t}^{child}$ is completeness of child deaths recording in province *i*, source *s*, at time *t*;

γ terms are random effects at the region (R) level;

$\eta_{i,s}$ is a random effect at the province and source level; and

$\xi_{i,s,t}$ is an error term.

Here, "region" is defined as the groupings of provinces within Indonesia based on geography and sociodemographic similarities (Table 3).

This model relates adult completeness to under-5 completeness and includes region- level random effects to allow for differences in both the average level of adult completeness and the relationship between child and adult completeness at this level. The province-source random effect captures the fundamental difference in level of completeness between different data sources. A $\log_{10}$ transformation was employed to make over- and under-completeness symmetric (e.g., 50% complete and 200%

complete are symmetric around 0 when log10 transformed) and to simplify calculation of the variance of completeness estimates in $\log_{10}$ space, which was needed for the adult mortality estimation process. Also, to avoid allowing outlying DDM-derived estimates of adult completeness from unduly influencing the predictions from the model in the equation above, for any given set of three DDM estimates (GGB, SEG, GGBSEG) calculated from a single pair of censuses, the estimate that was furthest from 1

Table 3. Province groupings into regions within Indonesia.

Region	Province
Sumatra	Aceh Sumatera Utara Sumatera Barat Riau Jambi Sumatera Selatan Bengkulu Lampung Bangka Belitung Kepulauan Riau
Java	DKI Jakarta Jawa Barat Jawa Tengah DI Yogyakarta Jawa Timur Banten
Lesser Sunda Islands	Bali Nusa Tenggara Barat Nusa Tenggara Timur
Kalimantan	Kalimantan Barat Kalimantan Tengah Kalimantan Selatan Kalimantan Timur Kalimantan Utara
Sulawesi	Sulawesi Utara Sulawesi Tengah Sulawesi Selatan Sulawesi Tenggara Gorontalo Sulawesi Barat
Maluku Islands	Maluku Maluku Utara
Western New Guinea	Papua Barat Papua

(i.e., complete) in either direction was excluded. For each province-source, $\log_{10}(c_{i,s,t})$ was predicted from coefficients estimated in the model above and child completeness using the following equation.

$$log_{10}\left(c_{i,s,t}^{adult}\right) = \hat{\alpha} + \hat{\beta}_1 * log_{10}\left(c_{i,s,t}^{child}\right) + \hat{\gamma}_1^R + \hat{\gamma}_2^R * log_{10}\left(c_{i,s,t}^{child}\right)$$

Not every province could be used to fit this model as DDM cannot be applied in some cases due to lack of appropriate census data. However, because the coefficients used for prediction were at the region level, predictions from this model could be generated for all provinces where estimates of child completeness were available.

For sources that only include household death recall, I set an arbitrary value of 1 for the first stage values of child completeness. However, it should be noted that the predictions based on this set value by no means reflect the true completeness of adult age groups and it changed once I applied the spatial-temporal regression in the second-stage estimation. In the second stage, I calculated the residuals from the first stage and apply spatial-temporal smoothing to these residuals. The predicted residuals were then added back onto the first-stage predictions, generating the second-stage predictions. Spatial-temporal smoothing was carried out in the same way as in the adult mortality estimation process (described below) with three modifications: the λ and ζ parameters were set to 2.0 and 0.95; only the fixed effect local regression variant was used; and the residuals were not held constant out of sample. The variance of the completeness estimates was also calculated, as this information was utilized in the adult mortality estimation process. To do this, I approximated the variance based on the median absolute deviation (MAD) compared to the second-stage estimates. I calculated variances at the regional level. Then, for each province-source-year, I generated 10,000 simulations from a normal distribution with mean equal to the second-stage prediction for that year and variance calculated as just described. I exponentiated the 10,000 simulations and found the mean, which serves as the final prediction for completeness. Before exponentiating the simulations, the variance of the simulations was calculated, and used as the variance of the completeness estimates in the adult mortality estimation process. The final completeness estimates were used to adjust, where appropriate, the corresponding province-source-years before these data were used in the adult mortality estimation process. For a small number of data points, completeness could not be estimated using the procedure described above due to a lack of appropriate census data, and the original growth balance method was the only viable option.(11)

Sibling survival histories and ${}_{45}q_{15}$ computation

Data for sibling survival histories were taken from large survey series in which respondents were asked about the status of their siblings, alive and dead. To generate estimates of sibling survival, each questionnaire contains a module with a full accounting of all siblings (children born to the same woman) of all respondents, along with data on the year of death (if applicable), sex, and age at death or year of birth. Additionally, the surveys must not have significant missingness in terms of responses to the sibling survival history module.

In Indonesia where sources including vital registration and household death recall are nonexistent or scarce, sibling histories provide important information on the levels and trends of adult mortality rates. The sibling survival technique employed by this study is based on the adapted Obermeyer et al methods(15) used by the GBD study(16), which includes a few improvements to the original methods: (1) use of appropriate survival weights that account for the study design; (2) implementation of a correction to account for the mortality experience of families not represented because none of the siblings were alive and eligible to respond to the survey; and (3) refinements for adjusting for recall bias. Selection bias refers to the underrepresentation of high mortality sibships in the sample population—sibships with higher rates of mortality are less likely to be represented in the survey because fewer of them are likely to have survived to be selected into the sample. A method to correct for this underrepresentation, proposed by Gakidou and King(17), incorporates a sibship-level weight, Wj = Bj /Sj, where Bj is the original sibship size and Sj is the number of siblings in sibship j who survive to the time of the survey. When each observation in the dataset being analyzed was at the sibship level, this Gakidou-King (GK) weight was used to compute a weighted average of the proportions of siblings deceased as reported by each respondent. In the absence of any sibships where all siblings have died, this correction algebraically corrected for the underrepresentation of high-mortality sibships in the survey sample. When the dataset was expanded to the sibling level (i.e., one observation for each sibling as opposed to sibship), the number of observations listed in the dataset for each sibship corresponded to the original sibship size, Bj, and so the numerator of Wj was already accounted for. The resulting sibling-level weight is therefore Wi = 1/Sj for sibling i in sibship j.(16) Since the analysis reported here was carried out at the sibling level, I used Wi rather than Wj . Also, the number of surviving siblings in the family was also tailored to the eligibility criteria for respondents of the given survey.(18) In applying Gakidou and King's elucidation of the survivorship correction, Sj/Bj represents the probability that a sibling in sibship j survived and was eligible to be selected in the survey. For Demographic and Health Surveys (DHS),

respondents must be women between the ages of 15 and 49 and so the Sj in this case would be the number of surviving women in a sibship j who were between the ages of 15 and 49 at the time of the survey. In this analysis, the value of Sj was chosen to be consistent with the eligibility criteria of each survey. The sampled population excluded sibships in which there were not any eligible siblings to respond to the surveys; thus, I cannot report on the mortality experiences of these siblings. The zero-survivor correction estimated the number of sibling deaths that were missing from the sample by age and sibship size, and then added these siblings to the observed sample before calculating age-specific mortality rates. This correction was applied to sibships with one or two females. The correction used the relationship between the true number of sibships with one (or two) females and the cumulative probability of dying before the time of the survey to estimate the number of missing sibling deaths. For one-sibling sibships,

$$K^1_{obs} = K^1_{true} * (1 - {}_aq^1_0)$$

$$K^1_{miss} = K^1_{true} * {}_aq^1_0$$

Where: K^1_{obs} is the number of sibships with one sister that are observed in the sampled population;

K^1_{true} is the true number of sibships with one sister in the population;

K^1_{miss} is the number of sibships with one sister that are not represented in the sampled population due to zero-survivor bias;

${}_aq^1_0$ is the cumulative probability of death for five-year age group *a*

$(1 - {}_aq^1_0)$ is the probability that the sister has survived to the time of the survey.

From these two equations, it follows that the number of sibships with only one sister that were not represented in the population due to zero-survivor bias is equal to:

$$K^1_{miss} = \frac{K^1_{obs}}{1 - {}_aq^1_0} * {}_aq^1_0$$

I multiplied this estimate of the number of missing sibships by the number of females in the sibship (which in this case was one) to get an estimate of the number of females in each age group that were missing from the sample because they had died. I then expanded this number so that I had one observation per missing sibling, assigned birth and death dates to these missing siblings based on the

distribution in the observed siblings, and appended them to the existing dataset. This process was also carried out for families with two sisters:

$$K^2_{obs} = K^2_{true} * (1 - {}_aq^1_0 * {}_aq^2_0)$$

$$K^2_{miss} = K^2_{true} * {}_aq^1_0 * {}_aq^2_0$$

$$\therefore K^2_{miss} = \frac{K^2_{obs}}{1 - {}_aq^1_0 * {}_aq^2_0} * {}_aq^1_0 * {}_aq^2_0$$

Where: K^2_{obs} is the number of sibships with two sisters that are observed in the sampled population;

K^2_{true} is the true number of sibships with two sisters in the population;

K^2_{miss} is the number of sibships with two sisters that are not represented in the sampled population due to zero-survivor bias;

${}_aq^1_0$ is the cumulative probability of death for the first sister in five-year age group *a*; and

${}_aq^2_0$ is the cumulative probability of death for the second sister in five-year age group *a*.

If there was only one sister within the 15 to 49 age range, the equations were different than above because the second sister did not contribute to the probability of the sibship being observed in the sample.

$$K^2_{obs} = K^2_{true} * (1 - {}_aq^1_0)$$

$$K^2_{miss} = K^2_{true} * {}_aq^1_0 * {}_aq^2_0 + K^2_{true} * {}_aq^1_0 * (1 - {}_aq^2_0)$$

$$\therefore K^2_{miss} = \frac{K^2_{obs}}{1 - {}_aq^1_0} * {}_aq^1_0 * {}_aq^2_0 * \frac{K^2_{obs}}{1 - {}_aq^1_0} * {}_aq^1_0 * (1 - {}_aq^2_0)$$

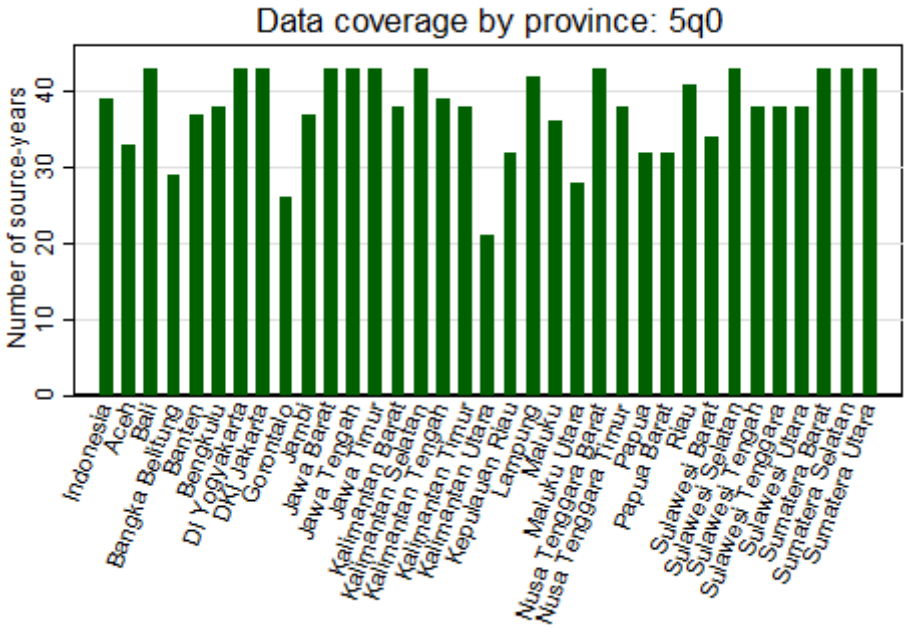
This analysis took into account time prior to the survey in the logistic regression to model mortality with the updated GBD method for recall bias adjustment.(16) After ${}_{45}q_{15}$ was estimated for each of the four surveys, the estimates were combined and paired up for all periods where they overlap. This overlap occurred when there were at least two surveys carried out in the same province within 15 years of each other. I estimated adult mortality from sibling histories for three five-year periods prior to the survey date.(7)

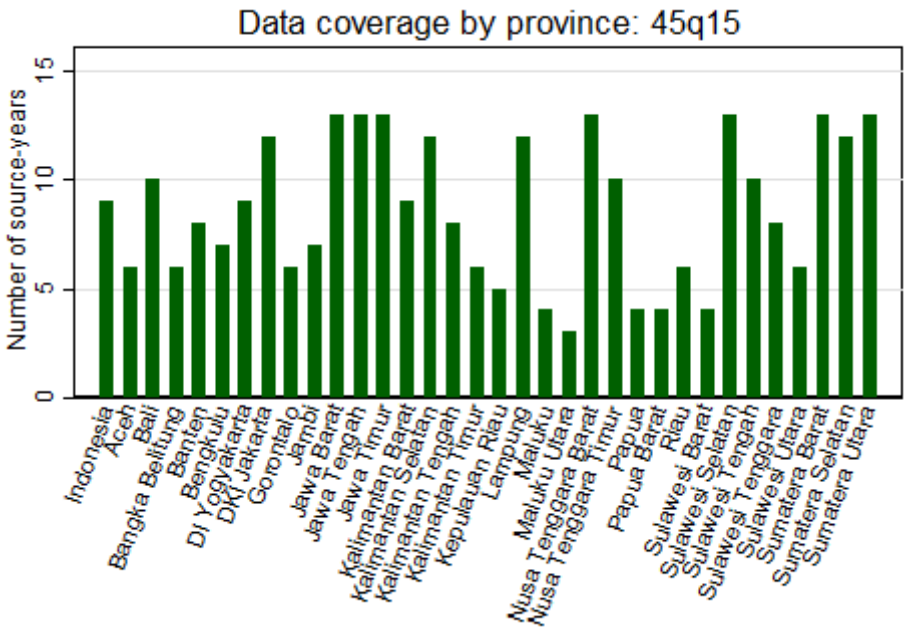
Upper and lower uncertainty intervals were also derived. The coefficient on recall period represents the effect of recall bias and was used to adjust the ${}_{45}q_{15}$ estimates to account for that bias.

Final mortality database

Our final database contained 25,609 measurements, consisting of 22,792 measurements from summary birth histories, 1,557 from complete birth histories, 157 from household deaths, and 1,103 from sibling histories. 229 of the total measurements were classified as outliers (and were subsequently excluded from the analysis) on the basis of examination of country plots and in some cases because of known biases in the data. There are several important quality-control steps in reviewing mortality data and estimates. For child mortality data, I excluded all summary birth histories from the East Family Life Survey, which were all implausibly high compared to other available data and complete birth histories from the same survey. Any data points with zero deaths were also excluded from the analysis because the sample sizes were large enough and mortality high enough that these were assumed implausible. For adult mortality data, I excluded data points that were both substantially inconsistent with the other sources of information and resulted in an implausible time trend for the same province that cannot be explained by a known mortality shock. Generally, I favored the inclusion of data points rather than their exclusion. For all analyses, data from the 2000 census were excluded for Aceh because conflict in the province at the time prevented quality primary data collection (19). Overall, I produced yearly estimates of under-5 mortality from 24,148 empirical measurements and adult mortality from 1,232 data points across the 34 Indonesian provinces (Figure 3).

Figure 3. Data coverage, represented as the number of source-years of data by province for ${}_5q_0$ and ${}_{45}q_{15}$ estimation.





Births and population numbers

At the national level, estimates of live births come from the World Population Prospects 2015 (WPP 2015).(16) For provinces, I multiplied age-specific fertility rates obtained from GBD by the female population for a given province-year, and then scaled these births to the national estimates obtained from GBD 2015.(16)

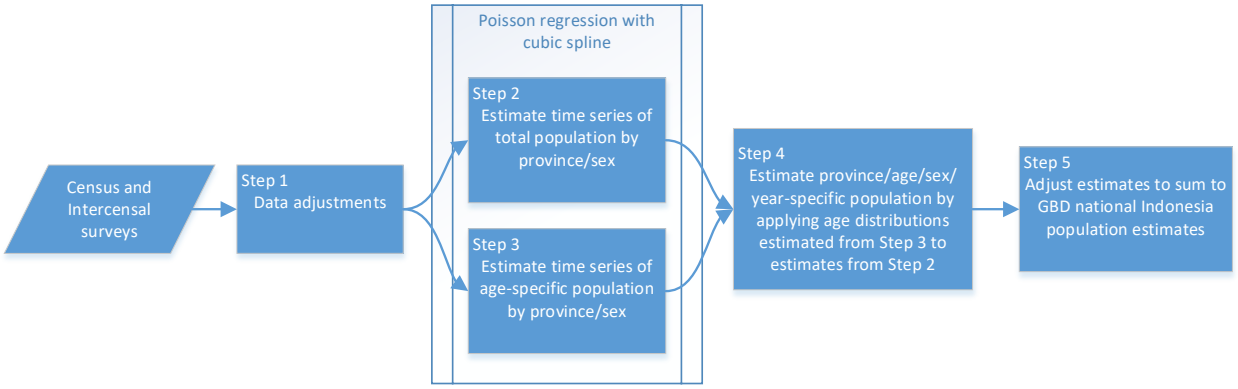
Population estimates were interpolated from census estimates by year for each province/age/sex using the following steps after conducting census raw data adjustments (Figure 4).

Two adjustments were made to implausible raw data values. First, Banten age 98 from census 2000 had unreasonably high population numbers for both males and females, likely from common use of "98" as "unknown" response. This population outlier was replaced by the average population of ages 97 and 99 by sex. Another anomaly was that Papua and West Papua both showed zero population for age 75-79 females in the 1985 census, which was adjusted to the mean population of age groups 70-74 and 80-84.

In order to compare and adjust census totals to sum to national population estimates obtained from GBD 2015(16), the census population numbers were adjusted for any missing provinces. This applied to 2 provinces in 1990 (Papua Barat and Papua), 3 in 2000 (Aceh, Papua Barat, and Papua), and 3 in 2005 (Aceh, Sulawesi Barat, and Papua Barat). I applied the mean proportion of the national population for

each of these missing provinces from the two nearest censuses. For example, Aceh was missing in censuses 2000 and 2005, and the proportions of the national population in Aceh were averaged from years 1995 and 2010 to adjust the sum of province populations in 2000 and 2005 to represent the whole country.

Figure 4. Steps to estimate subnational population numbers for Indonesia by province/age/sex/year using microdata from cesnsuses and intercensal surveys.



I summed the populations by province/year, and then proportionally adjusted these total province populations to sum to the total national GBD-estimated population (Figure 4 Step 2). Then annual total province populations were estimated by province/sex using a Poisson regression with a cubic spline with knots at years with data for that province/sex/year.

$$Population_{p,s} = \beta_0 + f(year)$$

Where *p* is province, *s* is sex, and *f(year)* is a piecewise cubic polynomial function with knots at years observed in the data for each province/sex. These estimates were adjusted proportionally to sum to the total national GBD-estimated population for Indonesia by year and sex.

Separately, I estimated the annual age-specific population in each province/sex using the same method of cubic interpolation (Figure 4 Step 3).

$$Population_{p,s,a} = \beta_0 + f(year)$$

Where *p* is province, *s* is sex, *a* is the GBD age group, and *f(year)* is a piecewise cubic polynomial function with knots at years observed in the data for each province/sex/age group.

Because SUPAS 2015 data were not yet available at the time of analysis, the census 2010 age distribution was applied to 2011-2015 estimates by province/year/sex.

These interpolated age distributions were applied to the interpolated total province populations to estimate detailed population numbers by province/year/age/sex (Figure 4 Step 4). Again these were adjusted proportionally as needed to sum to national GBD estimates by year/age/sex by multiplying them by the national GBD estimates divided by the sum of province-level estimates by year/age/sex (Figure 4 Step 5).

Education covariate

Age-specific education, maternal education, and lag-distributed income were used as covariates in the child and adult mortality models below. A time series of maternal education by province was created using all available province-representative available survey data.

Data sources and extraction

To estimate the average number of years of education by province, 1119 province-years of data were extracted from 36 surveys, resulting in 37,340 province/year/age/sex-specific data points (Table 4).

Table 4. Number of provinces extracted from data sources for education covariate estimation.

Survey name*	# Provinces
1990 Census	15
2000 Census	34
2010 Census	34
SUPAS 1985	34
SUPAS 1995	34
SUPAS 2005	33
SUSENAS 1992	30
SUSENAS 1993	30
SUSENAS 1994	30
SUSENAS 1995	29
SUSENAS 1996	30
SUSENAS 1997	30
SUSENAS 1998	30
SUSENAS 1999	30
SUSENAS 2000	28
SUSENAS 2001	32
SUSENAS 2002	33
SUSENAS 2003	34
SUSENAS 2004	34
SUSENAS 2005	33
SUSENAS 2006	34
SUSENAS 2007	34
SUSENAS 2008	34
SUSENAS 2009	34
SUSENAS 2010	34
SUSENAS 2011	34
SUSENAS 2012	34
SUSENAS 2013	34
MICS 2000	25
Family Life Survey East 2012	7
DHS 1991	33
DHS 1994	34
DHS 1997	34
DHS 2002-2003	29
DHS 2007	34
DHS 2012	34

**SUPAS = Intercensal Population Survey; SUSENAS = National Socioeconomic Survey; DHS = Demographic and Health Surveys; MICS = Multiple Indicator Cluster Surveys*

Most survey-years have unique question/response combinations about education, and mapping among them is necessary in order to compare the average education levels achieved. In most cases, four questions are asked about education of respondents, three of these questions to those who have attended at least some schooling:

1) What is the attendance status of the respondent? (never attended, currently attending, completed education)

2) What is the highest grade completed (up to grade 8)?

3) What level of education are they currently attending?

4) What is the highest level of education attended?

The number of years attributed to each possible response given their current school attendance status (Table 5) was derived from profiles of education in Indonesia both from the World Education News and Reviews and from the United Nations International Centre for Technical and Vocational Education and Training.(20,21)

Table 5. Number of education years assigned to each education level currently attending or completed.

Level	Currently attending	Completed education
Not completed	Highest grade completed	Highest grade completed
Primary school/SD/SDLB	5	6
Paket A	5	6
"Ibtidaiyah" (Islamic)	5	6
Junior high/ Vocational/SD/SDLB	8	9
Paket B	8	9
"Tsanawiyah" (Islamic)	8	9
Senior High school/SMA/SMLB	11	12
Vocational high school/SMK	11	12
Paket C	11	12
"Aliyah" (islamic)	11	12
Diploma I/II/D1/D2	13	13.5
Diploma I/D1	12.5	13
Diploma II/D2	13.5	14
Diploma III/bachelor/D3/Sarjana Muda	14.5	15
Diploma IV/graduate/D4/S1	15.5	16
Master/PhD/S2/S3	17.5	18.5
Master/S2	17	18
PhD/S3	18	19

The final database for estimating education in Indonesia contains 37,339 data points by province, year, age, and sex. Of these, 1,311 were excluded as outliers because their implausible values given surrounding data created unlikely trends over time or age.

Estimation methods

First, I ran a simple mixed effects linear model on the data with fixed effects on age group, sex, and year, and random effects on survey, region, and province:

$$education_{p,y,a,g} = \beta_1 age_1 + \cdots \beta_n age_n + \beta_{n+1} sex + \beta_{n+2} year + \gamma_s + \gamma_r + \gamma_p$$

Where *p* is province, *y* is year, *a* is 5-year age group, *g* is sex, *s* is survey, and *r* is region. The first two terms represent indicator variables for each 5-year age group in order to allow a flexible age trend. The last three terms represent the random effects on survey, region, and province, and predictions were made using the following equation, with γ_p set to zero for any provinces with no observed data.

$$education_{p,y,a,g} = \hat{\beta}_1 age_1 + \cdots \hat{\beta}_n age_n + \hat{\beta}_{n+1} sex + \hat{\beta}_{n+2} year + \hat{\gamma}_r + \hat{\gamma}_p$$

The predictions from the linear model were then run through a spacetime model and Gaussian process regression in order to borrow strength across geography and time, especially in the absence of data, while allowing the final predictions to more closely follow the data when present. These two processes are described in detail in the Under-5 mortality section below.

Results

Random effects from the linear model of average years of education are shown in Table 6 and Table 7. The province with the highest location random effect on both region and province (and thereby the highest initial estimated average years of education) is the national capital, DKI Jakarta. The province with the lowest estimated level is Papua Barat, one of the poorest provinces.

Table 6. Location random effects on region and province for linear model of average years of education.

Province	Region RE	Province RE
Aceh	0.415	0.093
Sumatera Utara		0.436
Sumatera Barat		0.738
Riau		0.241
Jambi		0.036
Sumatera Selatan		0.295
Bengkulu		-0.206
Lampung		-0.357
Bangka Belitung		-0.250
Kepulauan Riau		-0.233
DKI Jakarta	1.378	0.965
Jawa Barat		0.549
Jawa Tengah		0.080
DI Yogyakarta		0.656
Jawa Timur		0.115
Banten		0.264
Bali	0.359	0.640
Nusa Tenggara Barat		0.279
Nusa Tenggara Timur		-0.234
Kalimantan Barat	-0.358	-0.751
Kalimantan Tengah		-0.015
Kalimantan Selatan		0.329
Kalimantan Timur		0.241
Kalimantan Utara		-0.486
Sulawesi Utara	-0.052	0.611
Sulawesi Tengah		-0.060
Sulawesi Selatan		-0.364
Sulawesi Tenggara		-0.552
Gorontalo		0.851
Sulawesi Barat		-0.586
Maluku	-0.053	0.412
Maluku Utara		-0.514
Papua Barat	-0.690	-0.912
Papua		-0.404

Table 7. Survey random effects for linear model of average years of education.

survey	RE
CENSUS	0.707
DHS	-0.245
EFLS	0.238
MICS	-0.163
SUPAS	-0.334
SUSENAS	-0.203

For use in modeling child and adult mortality, average maternal education across age was calculated from these results by population-weighting the estimated years of education among women aged 15-49 by province and year (Table 8).

Table 8. Average years of education among women aged 15-49 by province.

	Average years of maternal education					
	1990	**1995**	**2000**	**2005**	**2010**	**2015**
Aceh	5.92	6.93	7.97	8.50	8.72	8.94
Sumatera Utara	6.45	7.35	8.23	8.75	8.94	9.16
Sumatera Barat	6.44	7.24	8.08	8.70	9.05	9.40
Riau	5.76	6.62	7.57	8.21	8.46	8.76
Jambi	5.18	5.99	6.86	7.50	7.88	8.30
Sumatera Selatan	5.33	6.12	6.91	7.50	7.84	8.16
Bengkulu	5.48	6.25	7.10	7.78	8.15	8.63
Lampung	4.89	5.68	6.55	7.23	7.68	8.16
Bangka Belitung	5.49	5.89	6.41	6.96	7.48	7.92
Kepulauan Riau	6.26	7.07	8.37	8.97	9.16	9.51
DKI Jakarta	8.03	8.92	9.70	10.03	10.15	10.43
Jawa Barat	5.12	6.14	7.08	7.57	7.89	8.11
Jawa Tengah	4.53	5.70	6.65	7.10	7.59	7.96
DI Yogyakarta	6.11	7.18	8.23	8.98	9.52	10.03
Jawa Timur	4.30	5.57	6.78	7.29	7.68	7.97
Banten	4.68	5.66	6.88	7.71	8.09	8.44
Bali	4.67	5.89	7.29	7.94	8.25	8.70
Nusa Tenggara Barat	3.34	4.31	5.37	6.11	6.54	7.03
Nusa Tenggara Timur	4.69	5.62	6.38	6.83	7.14	7.56
Kalimantan Barat	3.36	4.46	5.74	6.51	6.84	7.24
Kalimantan Tengah	5.45	6.23	7.16	7.75	8.01	8.34
Kalimantan Selatan	5.02	5.89	6.85	7.56	7.87	8.26
Kalimantan Timur	5.63	6.69	7.73	8.57	8.88	9.32
Kalimantan Utara	5.42	6.07	6.95	7.66	8.16	8.62
Sulawesi Utara	6.89	7.81	8.67	9.27	9.47	9.74
Sulawesi Tengah	5.86	6.63	7.31	7.83	8.11	8.54
Sulawesi Selatan	4.77	6.19	7.42	7.90	8.07	8.46
Sulawesi Tenggara	5.04	6.24	7.21	7.79	8.13	8.61
Gorontalo	7.46	7.52	7.36	7.50	7.77	8.16
Sulawesi Barat	4.63	5.39	6.08	6.66	7.05	7.60
Maluku	6.53	7.19	7.93	8.52	8.90	9.31
Maluku Utara	5.55	6.18	7.06	7.71	8.18	8.63
Papua Barat	2.02	3.34	5.28	6.83	7.81	8.50
Papua	4.62	4.96	5.25	5.32	5.34	5.46

Lag-distributed income covariate

Lag-distributed income (LDI) was calculated from an extrapolated time series of GDP for all provinces.

Data sources and extraction

Three time series of Gross Domestic Product (GDP) by province were obtained from the World Bank Indonesia Database for Policy and Economic Research: total GDP based on expenditure from 1990 to

2011 for most provinces, total GDP excluding oil and gas at current price from 2000 to 2011, and total GDP including oil and gas at current price from 2000 to 2011.(22) GDP based on expenditure is the only GDP time series for Indonesian provinces that precedes the financial crisis in 1997-1998 and can show its economic impact. The latter two time series were extracted in order to have information to inform lag-distributed income (LDI) without oil and gas by year for each province, in order to obtain an income time series without the influence of oil and gas since those incomes are not representative of the province population as a whole.

All three time series were converted from millions of rupiah to international dollars using the International Monetary Fund World Economic Outlook's time series of national currency per current international dollar for Indonesia.(23) The three series were then divided by province population to obtain per capita values. No data is currently available for North Kalimantan.

Estimation methods

I ran five models to create complete time series of GDP per capita based on expenditure, which had the longest time series of raw data, and chose the model with the most plausible results as the final model. First, I directly regressed province-level GDP on the national GDP obtained from GBD 2015 and predicted using the complete national series.(16) The second model is a Receiver Operating Characteristic (ROC) model to forecast and backcast GDP. Third, I directly modeled the difference between subnational and national GDP in log space. Next, I modeled the percent of national lag-distributed GDP that each province represents and recalculated LDI per capita after predictions using two approaches: 1) using the average proportion for the years lacking GDP data, and 2) using year-specific percent of national for all years by first backcasting and forecasting province-level GDP using the province-level trend.

For regressions on GDP level, I included a covariate indicating years after the financial crisis of 1998, and I included a covariate indicating the year 1998 for the ROC model to allow for a very different growth rate that year. Results from the regressions were used to estimate any province-year lacking raw data; the raw data was retained in the final time series when available. The most plausible model of GDP per capita is the ROC model, giving reasonable backcasting and forecasting estimates from 1950 to 2015 for all provinces.

In order to create a GDP per capita time series without oil and gas, I first calculated the proportion of GDP due to oil and gas from the two comparable raw data sets of GDP at current price with and without

oil and gas from 2000 to 2011. I then predicted this proportion to equal the average proportion of years 2000-2004 for years before 2000, and the average proportion of years 2005-2011 for years after 2011. This prevented the proportion of GDP due to oil and gas from nearing implausibly high or low values for any province. This proportion time series was then applied to the GDP per capita estimated to obtain GDP without oil and gas per capita.

Results for GDP per capita and GDP without oil and gas per capita were then converted to LDI per capita and LDI without oil and gas per capita by lag distributing the previous ten years of GDP for the current year of LDI. Because North Kalimantan had no raw data, it was assumed to have the same final time series of GDP per capita and LDI per capita with and without oil and gas as East Kalimantan, the province from which it split.

These estimates were then raked to sum to the national GDP and LDI values obtained from GBD 2015 using the below equations.(16)

$$GDP_p = GDP_p^* * \frac{GDP_{Indonesia}}{\sum_{p=1}^{34} GDP_p^*}$$

$$LDI_p = LDI_p^* \frac{LDI_{Indonesia}}{\sum_{p=1}^{34} LDI_p^*}$$

Where GDP_p and LDI_p represent the final province-specific GDP and LDI, GDP_p^* and LDI_p^* represent the province-specific GDP and LDI before raking, and $GDP_{Indonesia}$ and $LDI_{Indonesia}$ are the national-level GDP and LDI.

Results

The provinces with the consistently highest GDP per capita are Riau (largely due to oil and gas), Kepulauan Riau (due somewhat to the smaller population), Kalimantan Timur, and Kalimantan Utara (both with large oil and gas revenue) (Table 9).

Table 9. GDP and LDI per capita by province in International Dollars.

	Total GDP per capita						GDP without oil/gas per capita					
	1990	**1995**	**2000**	**2005**	**2010**	**2015**	**1990**	**1995**	**2000**	**2005**	**2010**	**2015**
Aceh	7248	7519	6701	6437	6270	7401	4025	4175	3329	4007	5227	5509
Sumatera Utara	3593	4909	4487	5737	7733	9708	3558	4860	4417	5694	7672	9633
Sumatera Barat	2828	4252	4025	4703	6571	8205	2828	4252	4025	4703	6571	8205
Riau	18447	14429	18871	14609	22644	29922	8408	6577	6091	8309	14177	17424
Jambi	2371	3231	2974	4110	6308	8472	2013	2743	2620	3364	5285	6965
Sumatera Selatan	4894	5102	4502	5798	7687	9797	3430	3576	3049	3750	5479	6769
Bengkulu	2327	3127	2331	3146	3813	4989	2327	3127	2331	3146	3813	4989
Lampung	1834	2706	2616	2768	5120	6874	1793	2645	2579	2667	5061	6746
Bangka Belitung	4845	5557	4788	6522	7638	9941	4630	5311	4576	6229	7453	9653
Kepulauan Riau	20485	22688	18876	15391	15654	19135	18321	20291	15235	14051	14537	17620
DKI Jakarta	9447	16902	20414	23613	33645	43276	9408	16831	20298	23507	33500	43077
Jawa Barat	3939	5256	4098	4840	6560	7872	3757	5013	3868	4609	6279	7495
Jawa Tengah	2603	3494	2764	3548	4988	6127	2405	3228	2616	3073	4384	5337
DI Yogyakarta	2226	4338	3213	3664	4829	5956	2226	4338	3213	3664	4829	5955
Jawa Timur	3071	4334	4356	5407	7589	9683	3063	4323	4339	5395	7557	9653
Banten	4422	5010	4265	4511	5129	7091	4422	5010	4265	4511	5129	7091
Bali	3716	5670	4096	4835	6093	7729	3716	5670	4096	4835	6093	7729
Nusa Tenggara Barat	1356	2105	2379	2952	3541	3964	1353	2101	2374	2946	3534	3956
Nusa Tenggara Timur	1252	1779	1537	1669	2133	2652	1252	1779	1537	1669	2133	2652
Kalimantan Barat	2911	4352	3869	4026	5011	6070	2911	4352	3869	4026	5011	6070
Kalimantan Tengah	3404	5939	4565	5280	7016	9068	3404	5939	4565	5280	7016	9068
Kalimantan Selatan	3083	4755	4326	4449	5643	7177	3014	4649	4206	4378	5577	7081
Kalimantan Timur	19502	20634	25248	30439	33069	42205	7923	8383	9255	11499	19357	20501
Kalimantan Utara	19502	20634	25248	30439	33069	42205	7923	8383	9255	11499	19357	20501
Sulawesi Utara	2709	4474	3996	4270	5954	7523	2706	4468	3993	4265	5947	7514
Sulawesi Tengah	1922	2869	3211	3576	4968	6990	1893	2825	3162	3563	4865	6883
Sulawesi Selatan	2437	3386	3311	3318	5335	7125	2432	3379	3303	3310	5325	7110
Sulawesi Tenggara	2085	2536	2428	3167	4608	5870	2080	2530	2422	3159	4597	5856
Gorontalo	1287	1507	1326	1814	2810	3568	1287	1507	1326	1814	2810	3568
Sulawesi Barat	2135	2477	2158	2121	3419	4400	2135	2477	2158	2121	3419	4400
Maluku	4435	5481	1797	1755	1908	2515	4414	5455	1789	1748	1904	2507
Maluku Utara	2308	2539	2098	1404	1886	2303	2308	2539	2098	1404	1886	2303
Papua Barat	6320	7588	6845	4491	10892	20712	4623	5551	4872	3080	6622	13629
Papua	7120	10754	12718	12962	11575	9819	7120	10754	12718	12962	11575	9819

Table 9. GDP and LDI per capita by province in International Dollars. (continued)

	Total LDI per capita						LDI without oil/gas per capita					
	1990	**1995**	**2000**	**2005**	**2010**	**2015**	**1990**	**1995**	**2000**	**2005**	**2010**	**2015**
Aceh	6543	3633	7208	4003	7140	3894	6649	3792	6487	4590	6882	5228
Sumatera Utara	3136	3106	4144	4104	4868	4816	5168	5120	6434	6383	8546	8480
Sumatera Barat	2461	2461	3406	3406	4268	4268	4423	4423	5464	5464	7234	7234
Riau	16423	7486	16091	7334	14891	6331	15390	7404	18567	10642	26262	15431
Jambi	2052	1743	2675	2272	3134	2678	3588	3016	4959	4077	7264	5975
Sumatera Selatan	4319	3027	4807	3369	4981	3472	5016	3466	6537	4522	8682	6059
Bengkulu	2050	2050	2641	2641	2736	2736	2718	2718	3342	3342	4397	4397
Lampung	1586	1550	2147	2099	2752	2693	2769	2693	3718	3644	5837	5738
Bangka Belitung	4275	4085	5064	4840	5442	5201	5882	5621	6859	6641	8796	8553
Kepulauan Riau	18435	16487	21091	18863	21920	19305	17520	15708	15521	14257	17458	16108
DKI Jakarta	8104	8070	12589	12536	17868	17788	21850	21759	27554	27434	37498	37326
Jawa Barat	3469	3309	4441	4236	4700	4475	4399	4197	5552	5287	7082	6746
Jawa Tengah	2283	2109	2981	2754	3205	2973	3192	2902	4141	3627	5446	4753
DI Yogyakarta	1942	1942	3160	3160	3790	3790	3547	3547	4155	4155	5299	5299
Jawa Timur	2666	2659	3609	3599	4301	4289	4837	4825	6265	6246	8464	8436
Banten	3928	3928	4598	4598	4884	4884	4580	4580	4756	4756	6235	6235
Bali	3275	3275	4603	4603	5035	5035	4675	4675	5258	5258	6824	6824
Nusa Tenggara Barat	1175	1173	1684	1681	2142	2138	2609	2604	3193	3187	3666	3659
Nusa Tenggara Timur	1101	1101	1477	1477	1634	1634	1628	1628	1839	1839	2350	2350
Kalimantan Barat	2564	2564	3508	3508	4254	4254	4039	4039	4412	4412	5438	5438
Kalimantan Tengah	2975	2975	4488	4488	5406	5406	5084	5084	5958	5958	7941	7941
Kalimantan Selatan	2705	2644	3823	3738	4615	4508	4536	4441	4856	4785	6318	6236
Kalimantan Timur	17162	6972	19490	7918	22013	8761	25256	10141	30973	14593	38135	19291
Kalimantan Utara	17162	6972	19490	7918	22013	8761	25256	10141	30973	14593	38135	19291
Sulawesi Utara	2364	2361	3445	3441	4512	4507	4214	4209	4910	4904	6596	6589
Sulawesi Tengah	1663	1638	2277	2242	3140	3092	3481	3436	4136	4062	5963	5862
Sulawesi Selatan	2125	2120	2822	2816	3347	3340	3412	3405	4140	4132	6097	6085
Sulawesi Tenggara	1818	1813	2220	2214	2537	2531	2778	2771	3759	3750	5149	5136
Gorontalo	1122	1122	1358	1358	1488	1488	1608	1608	2196	2196	3111	3111
Sulawesi Barat	1872	1872	2243	2243	2436	2436	2210	2210	2677	2677	3837	3837
Maluku	4095	4076	4905	4882	4415	4394	2311	2300	1791	1785	2249	2242
Maluku Utara	2085	2085	2370	2370	2447	2447	1833	1833	1588	1588	2050	2050
Papua Barat	5436	3977	6741	4931	7568	5512	5724	4148	6606	4482	16098	10234
Papua	6275	6275	8498	8498	12036	12036	11633	11633	11329	11329	9963	9963

Child mortality $_5q_0$

Under-5 mortality was estimated using GBD 2015(16) methodology as follows. Logit-linear regression was used to estimate $_5q_0$, with a random effect on province, fixed effects on source type to adjust to the reference type (CBH from censuses and SUPAS), fixed effects on income and average years of maternal education, and a random effect on the slope for province.

$$logit(_5q_0)_{i,t} = \alpha^c + (\beta_1^c + \gamma_{i,1}^c) * Edu_{i,t} + (\beta_2^c + \gamma_{i,2}^c) * LDI_{i,t} + \gamma_{i,3}^c + \varepsilon^c$$

Where: *c* indicates parameters and variables specific to child age-groups (0-4);

α^c is a fixed effect on source type across provinces

$Edu_{i,t}$ is maternal education in province *i* at time *t*;

$LDI_{i,t}$ is lagged distributed income in province *i* at time *t*;

γ_i are province random effects;

ε is an error term.

Predictions were made using the following equation without random effects. The residuals from the above regression were smoothed using Loess regression and different space-time weights for in-province and in-country data (Figure 5).

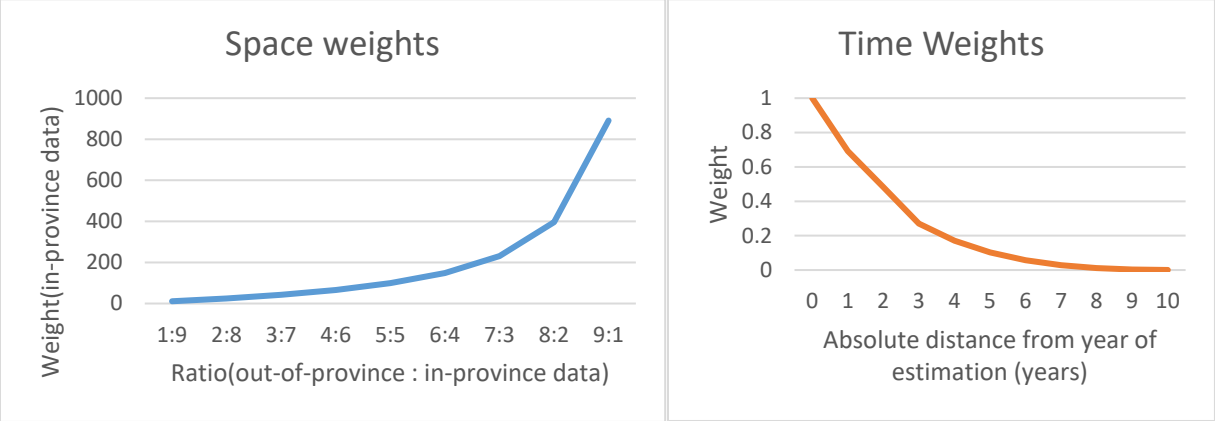
$$logit(_5q_0)_{i,t} = \alpha^c + \beta_1^c * Edu_{i,t} + \beta_2^c * LDI_{i,t}$$

The space weight ζ gives 99% of the weight to in-province residuals and 1% of the weight to out-of-province residuals. Data closer in time were given more weight than those farther in time using a modified tricubic window:

$$w_t = (1 - (\frac{|r_t - r_{est}|}{1 + \text{argmax}_t|r_t - r_{est}|})^\lambda)^3$$

Where r_t is the residual of the year of interest, r_{est} is the residual of the residual being weighted, and $\text{argmax}_t|r_t - r_{est}|$ is the maximum distance observed between the year of interest and a residual. The λ parameter dictates how quickly the weight approaches zero with increased distance in time. For all provinces, λ was 0.8.

Figure 5. Space weights from ratio of in-country to out-of-country data, and time weights dependent on distance from year of estimation, used for residual smoothing of initial child mortality estimates.



After adding the smoothed residuals to the linear predictions, the estimates were used as the mean function for Gaussian process regression (GPR).

GPR Model

As in GBD(16), the third stage of the prediction method was a Gaussian process regression (GPR) based on the model given in the below equation where μ_t is the true $\log_{10}({}_5q_0)$ at time t, f(t) is the baseline mortality risk, and S_t captures excess mortality due to war and disasters. S_t was estimated independently of f(t) as described in a later section. M and C describe the Gaussian process, giving the mean and covariance, respectively.

$$\mu_t = f(t) + S_t$$
$$f(t) \sim GP(M, C)$$

Gaussian process regression is a method of Bayesian inference. I specified a prior distribution for f(t), and a likelihood function which describes the data generation process; the specified prior distributions and likelihood function are described in subsequent sections. I then used Markov Chain Monte Carlo (MCMC) to approximate the posterior distribution of f(t) which also incorporates information from the observed empirical estimates of adult mortality. An MCMC chain of length 5000 was produced; the first 3000 samples were discarded and the remaining 2000 were thinned by a factor of 2 for a total of 1000 simulations retained. The reported best estimates and confidence intervals were generated from the mean and the 2.5th and 97.5th percentiles of the 1000 samples, respectively.

GPR Priors

The prior distribution of f(t) can be described in terms of the mean prior—the prior for M—and the covariance prior—the prior for C. I utilized the second stage predictions as the mean prior and used a Matérn covariance function to describe the covariance prior. This covariance function incorporates three parameters: the amplitude, which controls the amount by which realizations of the Gaussian process distribution can deviate from the mean function, the scale, which controls the distance (in time) over which the function is correlated, and the degree of differentiability, which influences the smoothness of the samples from the Gaussian process.

I set the differentiability to 2.0 in Indonesia. I used a higher differentiability than GBD uses for countries with complete vital registration data because I don't want the final estimates to be overly influenced by individual data points that may be less reliable. The parameter selection of amplitude and scale are described below.

GPR Likelihood:

The likelihood describes the probability of observing the data given a particular set of parameters. As shown in the below equation, I used a normal (Gaussian) model for describing the probability of observing a particular value of $\log_{10}({}_5q_0)$ where the mean is given by f(t) and the variance by V_t, the data variance.

$$log_{10}\left({}_5q_{0_t}\right) \sim Normal(f(t), V_t)$$

Data variance was calculated for each empirical observation of ${}_5q_0$ using either the sampling variance that was calculated in the complete birth history process or the estimated mean variance of the residuals from an out-of-sample predictive validity analysis of the summary birth history methods.(24)

Parameter selection

I selected prior values for the amplitude and scale based on model performance as assessed by out-of-sample predictive validity. For spacetime smoothing parameters in the ${}_5q_0$ analysis, I set the condition that λ must be 0.8 and that ζ be 0.99 for Indonesian provinces. For GPR, I tested six values of the scale—5, 10, 15, 20, 25, and 30—and five values of the squared amplitude— 0.2, 0.6, 1.0, 3.0, and 5.0 times the mean squared error of the residuals from the second stage prediction model—for a total of 30 combinations of parameters, following GBD methods.(7) The out-of-sample predictive validity was

assessed for each parameter combination by repeatedly dividing the data available into training and testing sets, fitting the model on the training sets and comparing the predictions to the corresponding data in the testing set.

I divided the data into testing and training sets 100 times. Data were divided as follows: for each region, a number N between 5 and 10 was sampled and the most recent N years of data for Indonesia were assigned to the testing set. Then data were additionally divided by region: a number M between 5 and 10 was sampled, a year Y within the data was sampled, and the data M years before and after year Y were assigned to the testing data set for each region. Data for the testing sets will likely overlap because the 100 iterations of this data division are independent. Any data that were not selected for the testing set were included in the training set.

For each testing and training division, the second stage model was fit on the training data. Then, the third stage GPR model was also fit on the training data using each combination of scale and squared amplitude values tested for a total of 30 sets of predictions. The testing data were matched to the predictions in the corresponding province and year for each set of predictions. For each match I calculated the absolute relative error of the prediction compared to the empirical estimate in the testing set. I also classified each empirical estimate in the testing set as being covered or not covered by the 95% uncertainty interval of each corresponding GPR prediction.

Once this procedure was carried out for all 30 parameter combinations over all 100 testing and training divisions of the data, I calculated the mean absolute relative error (MARE) and the mean coverage for each combination of spacetime and GPR parameters across by region because all locations have some data across the estimation time period (Table 10). The ideal set of parameters would produce estimates with low mean absolute relative error and mean coverage close to 0.95. I calculated a loss metric which incorporates both the coverage and the absolute relative error into a single measure to assess performance, and the parameter combination with the lowest resulting loss function defined below was selected for each region: for ${}_5q_0$, scale 30 and squared amplitude 5.0 for all regions.

If coverage <= 0.95, loss = MARE + ((1-coverage) - 0.05) / 5

If coverage > 0.95, loss = MARE + (0.05 – (1-coverage)) / 1

Table 10. Out-of-sample predictive validity results: Mean absolute relative error (MARE) and coverage for ${}_5q_0$ estimation parameter combination with lowest loss metric by region.

Region	MARE	Coverage
${}_5q_0$		
Indonesia	1.881	0.942
Java	1.138	0.976
Kalimantan	1.852	0.940
Lesser Sunda Islands	0.500	0.982
Maluku Islands	1.308	0.819
Sulawesi	0.511	0.975
Sumatra	1.650	0.972
Western New Guinea	4.163	0.833

All model fits for ${}_5q_0$ are shown in Supplementary Figure 1.

This process was repeated with national-level data to obtain estimates for Indonesia. To ensure consistency between the subnational estimates and national estimates of ${}_5q_0$, I rescaled the subnational estimates to the national level by population-weighting to get an implied national estimate from the subnational estimates. I first created a scalar of the national-level estimate from GPR to the aggregated subnational estimates (median 0.9997, IQR 0.9734-1.0269), and then multiplied all of the subnational estimates by this scalar to obtain the scaled estimates. I considered national-level estimates to be more reliable, so I chose this strategy of subnational scaling.

Adult mortality ${}_{45}q_{15}$

Adult mortality was estimated using the same method as under-5 mortality, with no reference category since neither household deaths nor sibling histories represent gold standard measures of mortality. Logit-linear regression was used to estimate ${}_{45}q_{15}$, with a random effect on province, fixed effects on income and sex-specific average years of education among adults, and a random effect of slope on province.

$$logit({}_{45}q_{15})_{i,t} = \alpha^a + \beta_1^a * Edu_{i,t}^a + \beta_2^a * LDI_{i,t} + \gamma_i^a + \varepsilon^a$$

Where: *a* indicates parameters and variables specific to adult age-groups (15-59);

$Edu_{i,t}$ a is population-weighted education for adult age-groups in province *i* at time *t*;

$LDI_{i,t}$ is lagged distributed income in province *i* at time *t*;

γ_i are province random effects;

ε is an error term.

The linear estimates were smoothed and analyzed with GPR as described above for ${}_5q_0$ to produce updated estimates with uncertainty intervals, with a few analytical differences.

Parameter selection

For spacetime parameters in the ${}_{45}q_{15}$ analysis, I tested all combinations of λ (0.6, 0.7, 0.8, 0.9) and ζ (0.7, 0.8, 0.9, 0.99). For GPR parameters, I tested all combinations of scale from 15 to 20 (15.0, 17.5, 20.0) and squared amplitude of 2.0. For each combination—16 for spacetime and 3 for GPR for a total of 48 combinations—across all 100 iterations, I calculated the MARE of the estimates compared to the raw data in the testing data sets and mean coverage as the percent of the empirical data covered by the 95% uncertainty interval of the GPR estimates (Table 11). The parameter combination with the lowest resulting loss function defined below was selected: scale 20, amplitude 2.0, λ 0.8, and ζ 0.99 for all data.

If coverage <= 0.95, loss = MARE + ((1-coverage)-0.05) / 5

If coverage > 0.95, loss = MARE + (0.05 – (1-coverage)) / 1

Adult mortality data is sparse compared to child mortality data, so ${}_{45}q_{15}$ parameter selection occurs over Indonesia as a whole instead of by region.

Table 11. Out-of-sample predictive validity results: Mean absolute relative error (MARE) and coverage for ${}_{45}q_{15}$ estimation parameter combination with lowest loss metric.

Region	MARE	Coverage
${}_{45}q_{15}$		
All regions	0.243	0.997

Gaussian process regression

The GPR priors were slightly different for the ${}_{45}q_{15}$ process than for ${}_5q_0$. I tested two values of the scale—15 and 20—and one value of the squared amplitude— 2 times the mean squared error of the residuals from the second stage prediction model—for a total of 2 combinations of parameters. The

values of scale and squared amplitude were limited to these values because of the sparse data with influential sibling histories in Indonesia.(16)

Another difference is that the method for calculating the data variance for adult mortality depended on the type of data, as in GBD.(16) For estimates derived from household recall data assessed with death distribution methods, I wanted to include not only sampling variance but also the non-sampling variance that arises from uncertainty in the completeness estimate. I calculated the variance of 45m15 using p(1-p)/N where N is the appropriate population size for the given survey aged 15 to 59 years and p is the mortality rate 45m15. I then transformed this to the variance of ${}_{45}q_{15}$ in normal space using the delta method and then to logit space using the equation:

$$Var_{logit(45q15)} = \left(\frac{1}{45m15 * (1 - 45m15)}\right)^2 * Var_{45q15}$$

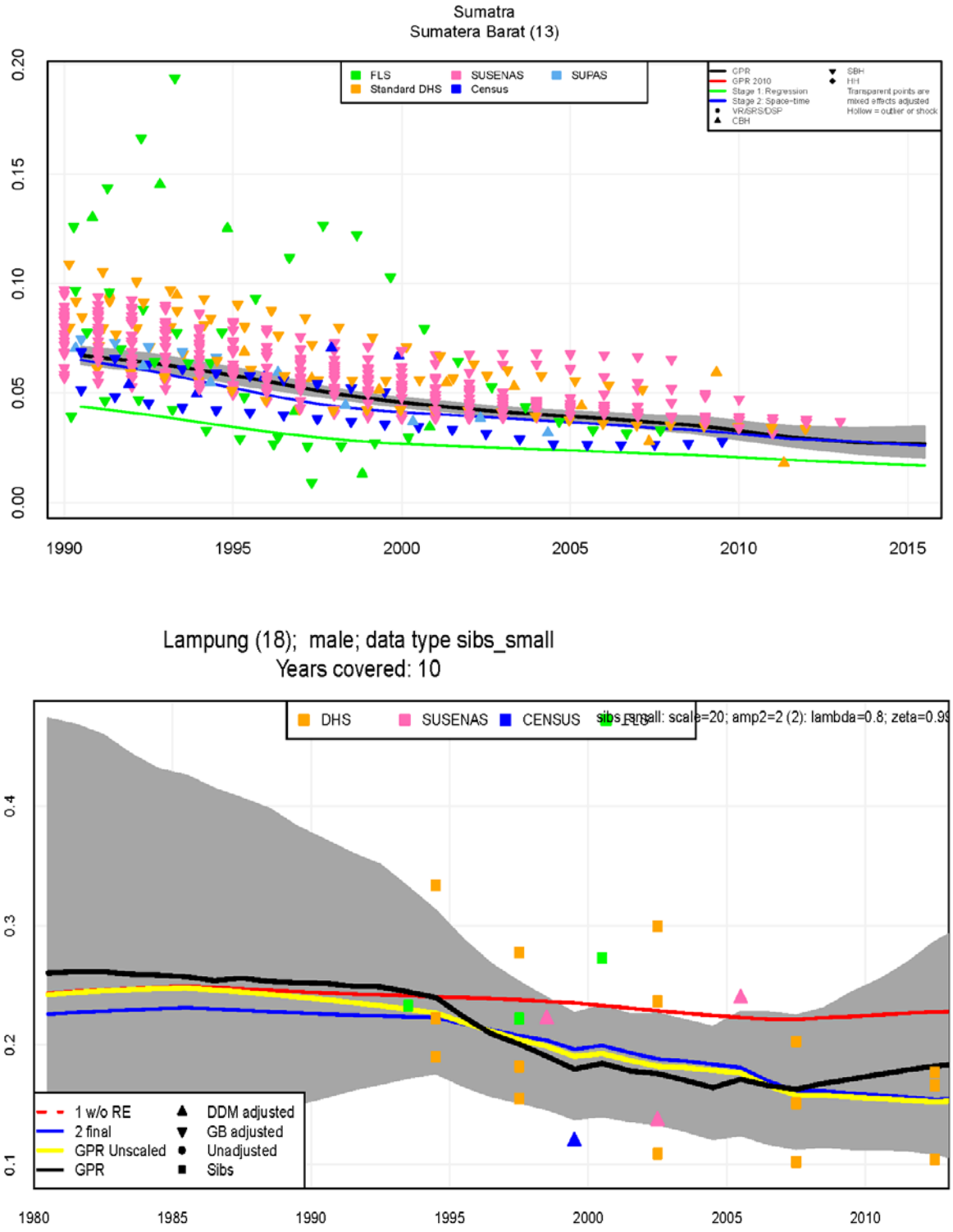
For these data, the total data variance was given by the sum of the sampling variance and the variance of the completeness estimate.

Also, for estimates from household recall data where completeness was assessed by the growth balance method only, the highest data variance generated by the above procedures within the same region was assigned. Finally, for estimates derived from sibling history data, the mean absolute deviation (MAD) estimator of the variance was calculated with reference to the first stage predictions.

$$\theta_{r,s}^2 = (1.4826 * \mathrm{MAD}_{r,s})^2 = 1.4826 * \mathrm{median}(\left|\log_{10}({}_{45}q_{15}^{\mathrm{observed}}) - \log_{10}({}_{45}q_{15}^{\mathrm{predicted}})\right|)^2$$

Figure 6 shows two examples of how the models fit the data for ${}_{5}q_{0}$ and ${}_{45}q_{15}$ for Sumatera Barat and Lampung, respectively. All model fits for ${}_{45}q_{15}$ are shows in Supplementary Figure 2.

Figure 6. Examples of ${}_5q_0$ and ${}_{45}q_{15}$ model fits to data in Sumatera Barat and Lampung, respectively.



FLS = Family Life Survey; DHS = Demographic and Health Survey; SUSENAS = National Socio-Economic Survey; SUPAS = Intercensal Population Survey

Age-specific mortality rate estimation

Under-5 deaths

Once the ${}_5q_0$ and ${}_{45}q_{15}$ estimates were created, child deaths in more detailed age groups were estimated by age and sex. I used a two-stage modeling process to generate sex-specific estimates of neonatal (days 0 to 27), postneonatal (the remainder of the first year), and childhood (ages 1 to 4) mortality.

Sex-splitting model

First, the ratio of male to female probability of death under age 5 (${}_5q_0$) was estimated for each province i in region j in year t using data from complete birth histories converted to mortality risks by sex. The data were ordered by observed ${}_5q_0$, and categorized into 20 evenly sized bins. Then, the model was fit to the data as described in the equation below.

$$\left(\frac{Male_{5q0}}{Female_{5q0}}\right)_{jit} = \beta + \gamma_{5q0\,bin} + \gamma_j + \gamma_i + \varepsilon_{jit}$$

The ratio was predicted by nested region and province random effects γi and γj, a random effect on the ${}_5q_0$ bin, and an intercept term, β. A Loess regression was then used to smooth the estimated $\gamma_{5q0\,bin}$ on ${}_5q_0$, creating a continuous $\gamma'_{5q0\,bin}$. Then, the equation below was used to predict the ratio of male to female ${}_5q_0$:

$$\left(\frac{Male_{5q0}}{Female_{5q0}}\right)_{jit} = \hat{\beta} + \gamma'_{5q0\,bin} + \hat{\gamma}_j + \hat{\gamma}_i$$

The male and female ${}_5q_0$ values were found using the system of equations that includes the model above and equation below, where r_{birth} was the sex-ratio at birth.

$${}_5q_0 = \left(\frac{1}{1 + r_{birth}}\right) * (female\,{}_5q_0) + \left(\frac{r_{birth}}{1 + r_{birth}}\right) * (male\,{}_5q_0)$$

Age-splitting model

Age-specific models were then fit for each age group on sex-specific data from complete birth histories. A separate model was fit for each age group yielding four models for each sex: neonatal, postneonatal, infant, and child. The log of the probability that an under-5 death occurred in a given age

group was modeled instead of the mortality risk, simplifying the scaling process and restricting risks to be between 0 and 1. In addition, two new covariates were included to improve model fit. First, I included the maternal education covariate that was also used in the ${}_5q_0$ first-stage model. Second, I used the completeness of the source-specific ${}_5q_0$ estimate for the data-point used in the regression. This completeness was calculated by taking the source-specific ${}_5q_0$ point estimate and dividing by the final ${}_5q_0$ estimate from GPR. The functional form of the model is below.

$$\log\left(\Pr(death\ at\ age\ y|u5\ death)_{jit}\right) = \beta_1 + \beta_2 * Mat.Ed._{it} + \beta_3 * Completeness_{sit} + \gamma_{5q0\ bin} * \gamma_j + \gamma_i + \varepsilon_{jit}$$

Similar to the sex model, the sex-specific age prediction used ${}_5q_0$ bins and smooths the random effect on the bin using ${}_5q_0$. The prediction equation for age y in province in region j at time t is seen below, with nested random effects on province ($\hat{\gamma}_i$) and region ($\hat{\gamma}_j$), an intercept term ($\hat{\beta}_1$), a smoothed random effect on ${}_5q_0$ bin ($\hat{\gamma}'_{5q0\ bin}({}_5q_{0\ jit})$), a coefficient on maternal education ($\hat{\beta}_2$), and a coefficient on completeness ($\hat{\beta}_3$):

$$\log\left(\Pr(death\ at\ age\ y|u5\ death)_{jit}\right) = \hat{\beta}_1 + \hat{\beta}_2 * Mat.Ed._{it} + \hat{\beta}_3 * 1 + \hat{\gamma}'_{5q0\ bin}({}_5q_{0\ jit}) + \hat{\gamma}_j + \hat{\gamma}_i$$

Note that for prediction, the completeness coefficient was multiplied by 1 instead of a source-specific completeness, as I sought to predict based on a hypothetically complete source. Once each of these predictions was made by age group, they were rescaled such that the probabilities of death in the Neonatal, Postneonatal, and 1-4 year age groups aggregate to the ${}_5q_0$ estimates from the under-5 model.

Under-5 age-sex splitting model application

First, the results of the sex model were applied to ${}_5q_0$ estimates, yielding sex-specific ${}_5q_0$ estimates. Once age-sex-specific predictions of the log conditional probability of death were made, these were exponentiated and rescaled so that they some to 1. First, the under-1 and 1-4 conditional probabilities were scaled to add to 1. Then, the neonatal and post neonatal conditional probabilities were scaled to the under-1 conditional probability. Then, the probabilities of death were calculated so that they properly aggregate to the final ${}_5q_0$ prediction. For example, to calculate the probability of death in the neonatal age group, the rescaled conditional probability of neonatal death given under-5 death was multiplied by the probability of under-5 death. Then, to obtain the probability of death in the postneonatal age group, the rescaled conditional probability of death in the postneonatal age group

given under-5 death was multiplied by the probability of under-5 death and then divided by the probability of survival to the beginning of the age group, and so on. Equations below represent this process, where *nn* represents neonatal and *pnn* represents postneonatal.

$$q_{nn} = \Pr(death\ in\ nn \mid u5\ death) * {}_5q_0$$

$$q_{pnn} = \Pr(death\ in\ pnn \mid u5\ death) * {}_5q_0/(1 - q_{nn})$$

The rest of the older age groups were also calculated in this manner, yielding probabilities of death in each of the under-5 age-sex groups.

Under-5 death number estimation, assigning under-5 deaths to age-sex groups

To estimate the number of under-5 deaths, I ran an estimation process that ages birth cohorts through my estimated probabilities of death. This process separated the yearly birth numbers for each location into week-sized cohorts and aged each of these cohorts through the mortality estimates in week-long steps to estimate the number of person-years and deaths in each of the neonatal, postneonatal, and 1-4 years age groups.

Over-5 deaths

The number of deaths by age and sex was estimated for all other age groups using a model life table system based on empirical life tables and population estimates.

Until now, the GBD life table database that feeds into the model life table system has not included any Indonesia-specific empirical life tables. For this study, life tables derived from the following household survey sources were assessed for addition to the GBD life table database: Census 2000, SUPAS 1985, SUPAS 2005, SUSENAS 2000, SUSENAS 2004, and SUSENAS 2007; Supplementary Figure 3 shows the log of mortality rates by age for each of these life tables.

Each of these sources was assessed both nationally and subnationally for completeness using a suite of death distribution methods including generalized growth balance (GGB), and synthetic extinct generations (SEG), and a combination of the two (GGBSEG).(9–13,16) The province-specific life table from a survey was excluded if any age group was not represented in population, if the survey was estimated by GGB/SEG to be less than 90% complete, if the corresponding ${}_{45}q_{15}$ exceeded 0.8, or if any life table did not conform to Gompertz law of mortality from age 30 to 80. This last exclusion criterion was applied to ensure that mortality rates tend to increase exponentially with age after childhood. This restriction excluded one life table from Sulawesi Selatan for females because of implausible jumps in

adulthood, and it excluded any life tables where the absolute value of any of the residuals exceeds 0.5 for a simple linear regression on the log mortality on age over this age range by province/source/sex/year.(25) This restriction prevents any implausible jumps in the mortality rate in log space.

Life tables from provinces with a sample size smaller than 6 million were combined with life tables from the same province within a ten-year window by summing deaths and population numbers to create an aggregate life table for a given province-sex with year averaged across those combined. All non-excluded resulting life tables were used in standard life table generation below.

Standard life tables were generated by first calculating the Mahalanobis distance for ${}_5q_0$ and ${}_{45}q_{15}$ between each empirical life table and the estimates from this analysis by province/sex/year.(26)

$$D_M^i\left(Q^i\right) = \sqrt{(Q^i - O)^T S^{-1} (Q^i - O)}$$

Where *O* is a multivariate vector representing the mean entry parameters ${}_5q_0$ and ${}_{45}q_{15}$ in logit space. Q^i is a multivariate vector that corresponds to the logit of ${}_5q_0$ and ${}_{45}q_{15}$ of an empirical life table *i* in the life table database. S^{-1} represents the covariance matrix, which is included because ${}_5q_0$ and ${}_{45}q_{15}$ are correlated in logit space. If they were not correlated, this would simplify to the Euclidean distance.

Sets of empirical life tables were created for each province/year/sex by combining the top 200 most similar empirical life tables as determined by the ranked Mahalanobis distance of ${}_5q_0$ and ${}_{45}q_{15}$ for each province/year/sex. These sets of selected life tables were averaged together to generate standard life tables, using weights across time and space, ensuring that those nearer in geography and year were given more weight. The weights were obtained from GBD 2015, which were derived by calculating the reciprocal of the squared means of the summed differences in ${}_nq_x$ in logit space between every pair of life tables in the GBD life table database.(16) This weighted average of empirical life tables results in the formation of a standard reference life table for each province/year/sex.

These standard life tables were used to generate annual age/sex-specific mortality rates by province for all age groups over age 5 using the following equation, where quantities with superscript *s* are from the standard life table, and those with *c* are from estimates of child and adult mortality above.

$$logit_n q_x^c = logit_n q_x^s + \hat{\beta}_x^1 * (logit_5 q_0^c - logit_5 q_0^s) + \hat{\beta}_x^2 * (logit_{45} q_{15}^c - logit_{45} q_{15}^s)$$

The age-specific coefficients β^1 and β^2 were obtained from GBD and were estimated from the following equation using country-time specific and region specific standards for each life table not affected by HIV/AIDS in the GBD life table database (Table 12).(16)

$$logit_n q_x^c - logit_n q_x^s = \beta_x^1 * (logit_5 q_0^c - logit_5 q_0^s) + \beta_x^2 * (logit_{45} q_{15}^c - logit_{45} q_{15}^s) + \xi_x$$

The coefficients β_x^1 and β_x^2 were age-specific coefficients that vary with age x and are limited to certain age groups in order to prevent implausible age trends when ${}_5q_0$ and ${}_{45}q_{15}$ change in opposite directions. In the equation for age group 40-44, for example, β_x^1 would be zero, and only the β_x^2 term and error term would inform the above model. These coefficients determine how much the estimated mortality age pattern deviates from the standard life table and from linearity.

Table 12. Model life table coefficients from GBD 2015.(76)

	$\beta_x^1({}_5q_0)$		$\beta_x^2({}_{45}q_{15})$	
Age	**Male**	**Female**	**Male**	**Female**
0	0.984	0.977	-	-
1-4	1.023	1.057	-	-
5-9	0.814	0.763	-	-
10-14	0.464	0.360	0.389	0.514
15-19	0.156	0.148	0.777	0.783
20-24	0.053	0.148	0.807	0.853
25-29	0.039	0.132	0.766	0.917
30-34	0.023	0.109	0.791	0.912
35-39	-	-	0.880	1.080
40-44	-	-	0.912	0.986
45-49	-	-	0.93	0.922
50-54	-	-	0.903	0.871
55-59	-	-	0.857	0.855
60-64	-	-	0.792	0.841
65-69	-	-	0.78	0.857
70-74	-	-	0.785	0.898
75-79	-	-	0.777	0.898
80-84	-	-	0.675	0.807

Fatal discontinuities

Deaths from conflict and natural disasters are considered fatal discontinuities that add mortality to the background mortality estimates. In Indonesia, a tsunami in 2004 impacted mortality in Aceh. To reflect the effects of this natural disaster, I added estimates of the number of deaths attributable to the tsunami with uncertainty to the age/sex-specific mortality estimates for Aceh in 2004 to give final mortality estimates. These were added separately from the mortality estimation process because no province-level input mortality survey data for 2004 exists for Aceh for either child or adult mortality.

Total deaths from the 2004 tsunami primarily affecting Aceh were extracted from Indonesia Disaster Data and Information (BIDI).(27) The age and sex pattern of these deaths was obtained from Doocy 2007, the only published paper containing age/sex-specific mortality rates from the 2004 tsunami.(28) The study conducted household surveys among internally displaced people in nine of the most affected districts in Aceh. For this analysis, I assumed that the survivorship bias in the data was negligible and that households with no survivors experienced the same age and sex pattern of mortality as the deaths recorded in the surveys.

The high level of uncertainty inherent in disaster mortality data was accounted for in two ways. First, the total number of deaths from BIDI was converted to a normal distribution of 1000 draws, with 166,561 deaths as the mean and 20% of that as the standard deviation, or 33,312.2 deaths. Second, 1000 estimates of deaths were drawn from a binomial distribution defined by the age/sex-specific mortality rates and corresponding sample sizes from Doocy 2007; these were then divided by sample size and multiplied by province population numbers to get province-level draws of deaths by age and sex. The purpose of that step is to adjust the mortality rates for any differences in the makeup of the surveyed population versus the province population. Next, I calculated the proportion of deaths observed in each age/sex group by draw and multiplied this by the 1000 draws of total deaths to obtain final estimates of age/sex-specific deaths with uncertainty attributable to the 2004 tsunami affecting Aceh.

These deaths were then added to the all-cause age/sex-specific death estimates for Aceh in 2004 to create the final estimates of mortality in Indonesia by province that include the fatal discontinuity in Aceh 2004.

Validation

To validate this modeling approach, I conducted out-of-sample predictive validity for ${}_5q_0$ and ${}_{45}q_{15}$. I divided the data into training and testing datasets 100 times. Various spacetime and GPR parameters were tested for ${}_5q_0$ and ${}_{45}q_{15}$ by fitting the models on the training datasets and seeing how well the models predicted the empirical data in the testing datasets by calculating the absolute relative error and coverage. The best performing set of parameters in terms of mean absolute relative error and coverage were selected ; the resulting mean absolute relative error and mean coverage are shown in Table 10 and Table 11 above. I present results only for the best performing combinations of parameters for ${}_5q_0$ and ${}_{45}q_{15}$.

To test sensitivity to the life table database, I held out one life table at a time, then all of the life tables from each region, and then all 24 life tables from Indonesia. I estimated life expectancy at birth and total deaths for each holdout (Table 13).

Table 13. Sex-specific life expectancy at birth and total deaths estimated for each life table holdout scenario compared to overall results.

	Life expectancy at birth			Total Deaths		
Omitted life tables	**1990**	**2000**	**2015**	**1990**	**2000**	**2015**
Female						
All 5 female life tables (Census 1999, Census 2004)	66.95	69.31	73.52	643,430	731,764	687,389
Jawa Barat (Census 2004)	67.05	68.55	73.89	637,361	698,084	668,979
Jawa Barat (Census 1999)	67.10	69.01	73.94	639,015	708,603	671,726
Jawa Tengah (Census 1999)	67.00	68.51	73.86	641,874	714,444	673,345
Jawa Timur (Census 1999)	67.07	69.03	73.91	638,848	701,700	670,611
Jawa Timur (Census 2004)	67.06	68.58	73.91	638,788	706,203	670,314
FEMALE RESULTS	**67.18**	**68.18**	**73.40**	**638,420**	**704,943**	**667,852**
Male						
All 9 male life tables (Census 1999, Census 2004, SUSENAS 2005)	64.91	67.43	70.16	691,343	867,466	779,645
DKI Jakarta (Census 2004)	64.67	64.13	70.24	685,203	816,211	775,215
Jawa Barat (Census 1999)	64.72	64.30	70.23	688,693	867,373	775,409
Jawa Barat (Census 2004)	64.68	64.14	70.27	689,902	883,982	773,924
Jawa Tengah (Census 2004)	64.68	64.14	70.25	690,565	884,989	775,446
Jawa Tengah (Census 1999)	64.73	64.46	70.24	688,127	872,341	774,626
Jawa Timur (Census 2004)	64.66	64.13	70.23	689,512	883,352	773,034
Kalimantan Barat (Census 1999)	64.62	63.88	70.17	690,342	884,944	774,498
Kalimantan Selatan (SUSENAS 2005)	64.68	64.16	70.26	687,511	864,192	774,376
Sulawesi Selatan (Census 2004)	64.67	64.13	70.24	690,585	885,612	775,788
MALE RESULTS	**64.60**	**64.20**	**69.13**	**681,570**	**751,281**	**777,810**

Aggregation

The estimation processes for ${}_5q_0$ and ${}_{45}q_{15}$ do not enforce consistency between province-level estimates and national estimates for Indonesia. To ensure consistency, I rescaled the subnational age/sex-specific mortality rate estimates to the national level by creating a scalar ratio of the national-level mortality rate estimates from GPR to the population-weighted aggregated subnational estimates. I then multiplied all of the subnational estimates by this scalar to obtain scaled province-level mortality rate estimates. I considered the national-level estimates to be more reliable because there more national than subnational data covers the time period of interest for Indonesia.

Uncertainty analysis

Uncertainty intervals have been estimated at each step of this mortality estimation process, to account for uncertainty from data sample sizes, data source adjustments, spacetime and GPR model specifications, and model life table specifications. This was accomplished by producing 1000 draws of child mortality rate, adult mortality rate, age-specific mortality rate, and death numbers for each province by sex for all years covered by each step from the posterior distribution in the estimation process. This process propagated uncertainty into the final quantities of interest.

Results

From 1990 to 2015, life expectancy at birth for Indonesia increased by 8.2% for males and 10.1% for females, to 70.14 years and 73.81 years, respectively. Table 14 details the life expectancy at birth by province and sex for 1990, 2000, and 2015 coarse changes over time. Detailed annual life expectancy can be seen in Supplementary Table 3. Life expectancy at age 50, shown in Supplementary Table 4, was 25.7 for males (95% UI 25.1-26.4) and 28.0 for females (95% UI 27.6-28.4) in 2015, increases of 6.4% and 11.1%, respectively. Mortality has improved in children under 5 also, with the probability of death declining by over 60% from 0.054 in 1990 to 0.020 in 2015 for both sexes, with males and females observing similar declines (Supplementary Table 5). In fact, age and sex-specific mortality rates have declined in every age group from 1990 to 2015 in Indonesia, with the largest relative improvements observed in children, which can be visualized in Supplementary Figure 4. Females achieved faster

declines than men in almost every age group, with reductions of almost 20% or greater through adulthood to age group 70-74.

At the province level, life expectancy at birth improved for all provinces from 1990 to 2015. In 2015, males and females differ in terms of provinces with the best and worst life expectancy at birth. For males, the provinces with the highest life expectancy (72-74 years) in 2015 are Kalimantan Utara, Jawa Barat, Kepulauan Riau, and DKI Jakarta, and those with the lowest in 2015 (65-66 years) are Kalimantan Selatan, Sulawesi Tengah, Nusa Tenggara Barat, and Papua. For females, however, the best provinces (76-78 years) are Kalimantan Utara, Jawa Barat, Jawa Timur, and DKI Jakarta, while the worst (64-65 years) are Maluku Utara, Papua Barat, Sulawesi Barat, and Gorontalo. Kalimantan Utara, the newest province in Indonesia, has high life expectancy due to estimates of low adult mortality. Notable are the provinces with the largest gap in life expectancy between males and females: in Maluku Utara, male life expectancy is 7.61 years greater than females due at least in part to the over twice as high ${}_{45}q_{15}$ observed in sibling survival data for three separate DHS surveys in that province for females (Supplementary Figure 2, Supplementary Table 6). Kapulauan Riau has life expectancy for males that is almost four years greater than that for females in 2015 consistent with a similar gap in ${}_{45}q_{15}$ between males and females in the sibling survival data for 2002, 2007, and 2012.

Female life expectancy is over 6 years greater than that of males in both Sumatera Utara and Jawa Timur, where male ${}_{45}q_{15}$ is much greater than female ${}_{45}q_{15}$ in 2015. The biggest absolute improvements in life expectancy from 1990 to 2015 for both males and females were found in Nusa Tenggara Barat, Sulawesi Barat, Jambi, Kalimantan Barat, and Jawa Barat, with both sexes experiencing an increase of at least 6.5 years in life expectancy.

There were an estimated 1,445,662 deaths in Indonesia in 2015 (UI 1,390,446.1-1,499,115.9), 46.2% of which were female (Table 15), compared to 1,524,484 deaths estimated by GBD (UI 1,468,784.2-1,581,326.83, 47% female).(29) This is an overall increase of about 125,000 deaths since 1990. The number of deaths reduced by almost 160,000 in people under 25 years old, even with a population expanding by over 15 million in those age groups over the same period of time (population numbers not shown). The population age 25 and over increased by almost 61 million people and observed just over 370,000 more deaths in 2015 than in 1990. There is substantial uncertainty around all of these estimates, especially because of the sparseness and variable consistency of the sibling survival and household death data in the ${}_{45}q_{15}$ estimation step.

The largest absolute decreases in deaths in specific age groups from 1990 to 2015 are observed in children in the Java region (Supplementary Table 7): age groups postneonatal and ages 1-4 in Jawa Barat, Jawa Timur, and Jawa Tengah all declined by about 10,000 to 18,000 deaths each; the neonatal age group in these provinces saw declines by 2,500 to 9,500 deaths. Every province saw declines in deaths among children from 2000 to 2015 with a few exceptions: Sulawesi Utara had just 13 more neonatal deaths, Riau had 572 more infant (neonatal and postneonatal) deaths, Bangka Belitung had 233 more infant deaths, and Papua Barat had 291 more infant deaths. Jawa Barat and Jawa Timur, the provinces with the largest and fastest-growing under 5 populations in 2015 (from 3.9 to 4.6 million and from 3.0 to 3.3 million children, respectively), saw the largest drops in child deaths under age 5 by 41,143 and 26,355 deaths, respectively. Jawa Tengah, the third largest province in terms of under 5 population with 3.1 million children (up almost 150,000 children population since 2000), saw 7,571 fewer child deaths from 2000 to 2015, a decrease of just 13.5% in neonates compared to 60.0% and 66.7% drops in Jawa Timur and Jawa Barat neonatal deaths. Other provinces with small relative declines in neonatal deaths (<10%) from 2000 to 2015 are Sulawesi Tenggara, Bali, Sulawesi Tengah, Sumatera Selatan, DKI Jakarta, and Kalimantan Tengah. Supplementary Figure 5 displays the change in age-specific mortality rate in log space from 2000 to 2015 by sex for each province and Indonesia. For young adults below age 30, female mortality rates tended to drop faster than male from 2000 to 2015, while many provinces experienced much greater drops in mortality for males than females for the older population aged 60-90. The middle-aged and very elderly (over age 90) tended to see the smallest declines for most provinces.

Table 14. Life expectancy at birth with 95% uncertainty by province and sex in 1990, 2000, 2015

	Male			Female		
	1990	2000	2015	1990	2000	2015
Sumatera						
Aceh	66.67 (65.04, 68.2)	67.29 (65.77, 68.8)	69.44 (67.04, 72.05)	67.19 (65.69, 68.86)	67.83 (66.18, 69.39)	70.34 (68.49, 72.12)
Sumatera Utara	63.73 (62.19, 65.03)	64.77 (63.13, 66.19)	67.28 (65.14, 69.54)	66.14 (64.93, 67.53)	69.49 (68.22, 70.75)	73.74 (72.09, 75.34)
Sumatera Barat	63.58 (62.31, 64.94)	65.42 (64.16, 66.64)	67.66 (65.43, 69.94)	64.82 (63.46, 66.46)	68.76 (67.45, 70.23)	72.89 (71.29, 74.44)
Riau	66.34 (64.87, 67.99)	69.3 (67.99, 70.74)	70.89 (67.95, 73.18)	65.63 (64.13, 67.22)	67.89 (66.49, 69.18)	72.85 (71.09, 74.51)
Jambi	62.94 (61.33, 64.46)	65.87 (64.24, 67.38)	70.51 (68.09, 73.01)	62.71 (61.19, 64.16)	65.66 (64.1, 67.21)	71.56 (69.7, 73.29)
Sumatera Selatan	64.64 (63.36, 65.98)	66.91 (65.68, 68.37)	69.7 (66.94, 72.27)	67.62 (66.07, 69.31)	70.07 (68.61, 71.54)	73.5 (71.82, 75.41)
Bengkulu	63.78 (62.45, 65.22)	64.46 (63, 65.84)	68.56 (66.12, 70.92)	60.26 (58.59, 61.95)	62.42 (60.91, 64.11)	68.38 (66.32, 70.41)
Lampung	64.27 (62.92, 65.63)	67.61 (66.15, 68.96)	71.53 (68.96, 73.61)	68.57 (66.8, 70.23)	70.09 (68.61, 71.68)	74.07 (72.51, 75.66)

Bangka Belitung	63.6	65.74	67.06	63.41	65.95	69.08
	(62.16, 65.01)	(64.15, 67.37)	(64.71, 69.44)	(61.81, 65.06)	(64.3, 67.66)	(67.13, 70.99)
Kepulauan Riau	65.71	70.55	73.08	65.68	67.56	69.68
	(64.07, 67.08)	(69.14, 72.12)	(70.11, 75.21)	(64.02, 67.24)	(65.84, 69.07)	(67.55, 71.73)
Java						
DKI Jakarta	70.37	70.1	72.08	69.9	73.07	75.56
	(69.02, 71.86)	(68.38, 71.56)	(68.74, 74.21)	(68.59, 71.47)	(71.72, 74.41)	(73.79, 77.39)
Jawa Barat	65.99	69.93	73.41	69.22	72.67	76.64
	(64.76, 67.3)	(68.68, 71.13)	(71.5, 75.27)	(67.88, 70.53)	(71.44, 73.75)	(74.92, 78.16)
Jawa Tengah	66.6	67.1	70.4	69.42	70.49	74
	(65.44, 68.01)	(65.75, 68.33)	(68.23, 72.65)	(67.93, 70.9)	(69.31, 71.61)	(72.59, 75.5)
DI Yogyakarta	68.94	69.93	70.55	68.54	70.66	73.52
	(67.32, 70.28)	(68.36, 71.58)	(68.33, 72.94)	(67.02, 69.95)	(69.08, 72.07)	(71.64, 75.15)
Jawa Timur	64.07	66.51	70.26	68.14	71.07	76.6
	(62.96, 65.14)	(65.35, 67.59)	(68.1, 72.56)	(66.75, 69.6)	(69.88, 72.42)	(75.06, 78.01)
Banten	62.08	63.69	67.21	66.84	68.04	71.63
	(60.58, 63.69)	(62.15, 65.12)	(64.98, 69.35)	(65.18, 68.33)	(66.69, 69.68)	(70.07, 73.14)
Lesser Sunda Islands						
Bali	65.32	67.84	71.16	67.12	69.69	73.27
	(63.77, 66.85)	(66.5, 69.37)	(68.5, 73.42)	(65.71, 68.51)	(68.39, 70.98)	(71.75, 74.77)
Nusa Tenggara Barat	56.39	59.87	66.33	57.94	63.26	69.48
	(54.95, 57.77)	(58.43, 61.31)	(63.72, 68.89)	(56.32, 59.71)	(61.67, 64.73)	(67.59, 71.46)
Nusa Tenggara Timur	62.25	64.09	68.37	61.81	65.41	70.65
	(60.75, 63.64)	(62.81, 65.39)	(66.09, 70.55)	(60.19, 63.34)	(64.05, 66.72)	(69, 72.5)
Kalimantan						
Kalimantan Barat	63.85	66.04	70.56	62.87	66.83	71.36
	(62.5, 65.23)	(64.53, 67.53)	(68.17, 72.99)	(61.43, 64.44)	(65.31, 68.3)	(69.6, 73.09)
Kalimantan Tengah	65.64	69.09	69.87	63.29	67.17	70.91
	(64.17, 67.06)	(67.69, 70.45)	(67.43, 72.47)	(61.51, 65.04)	(65.72, 68.69)	(69.13, 72.55)
Kalimantan Selatan	59.68	61.23	64.75	60.72	62.93	68.92
	(58.17, 61.07)	(59.62, 62.72)	(62.28, 67.06)	(59.22, 62.31)	(61.36, 64.46)	(66.86, 70.79)
Kalimantan Timur	65.22	63.35	67.14	63.27	64.81	70.43
	(63.86, 66.58)	(61.7, 65.05)	(64.84, 69.41)	(61.76, 64.84)	(63.19, 66.23)	(68.02, 72.26)
Kalimantan Utara	68.85	70.3	74.34	71.88	73.41	77.99
	(67, 70.45)	(68.61, 71.99)	(72.08, 76.42)	(70.23, 73.35)	(71.78, 75.08)	(76.1, 79.45)
Sulawesi						
Sulawesi Utara	63.32	65.26	69.03	63.76	65.54	70.06
	(61.7, 65.15)	(63.83, 66.82)	(66.73, 71.55)	(62.13, 65.36)	(63.96, 67.33)	(68.21, 71.9)
Sulawesi Tengah	59.28	61.49	66.09	59.28	63.2	67.63
	(57.63, 60.82)	(59.87, 62.85)	(63.56, 68.5)	(57.71, 60.87)	(61.66, 64.61)	(65.32, 69.51)
Sulawesi Selatan	63.3	65.61	69.22	65.02	68.52	73.1
	(61.99, 64.66)	(64.31, 66.89)	(66.71, 71.73)	(63.56, 66.78)	(67.15, 69.99)	(71.39, 74.65)
Sulawesi Tenggara	65.08	64.87	67.46	60.45	63.14	68.97
	(63.69, 66.66)	(63.41, 66.28)	(65.01, 69.64)	(58.72, 62.13)	(61.49, 64.86)	(66.98, 70.75)
Gorontalo	66.89	70.08	71.5	61.59	61.54	65
	(65.28, 68.36)	(68.65, 71.49)	(69.28, 73.66)	(60.03, 63.28)	(59.77, 63.3)	(62.79, 67.02)
Sulawesi Barat	59.61	62.63	67.46	56.12	59.56	64.83
	(58.15, 61.02)	(61.16, 64.13)	(64.79, 69.89)	(54.52, 57.81)	(58.01, 61.16)	(62.66, 66.88)
Maluku Islands						
Maluku	63.22	64.73	67.11	60.92	62.24	65.87
	(61.75, 64.71)	(63.27, 66.35)	(64.25, 69.6)	(59.24, 62.45)	(60.55, 63.97)	(63.66, 68.05)
Maluku Utara	66.28	67.93	71.28	58.99	59.62	63.67
	(64.6, 67.95)	(66.36, 69.54)	(69.11, 73.58)	(57.13, 60.91)	(57.82, 61.48)	(61.39, 65.75)
Western New Guinea						
Papua Barat	66.58	66.84	69.35	59.32	59.73	63.69
	(64.83, 68.45)	(65.22, 68.37)	(66.91, 71.53)	(57.5, 61.14)	(58.06, 61.45)	(61.44, 65.72)

Papua	65.84 (64.41, 67.56)	65.39 (63.91, 66.97)	66.5 (63.6, 68.68)	61.13 (59.56, 62.81)	61.72 (60.17, 63.55)	66.06 (64.04, 68.17)
Indonesia	64.81 (64.37, 65.24)	66.86 (66.42, 67.29)	70.14 (69.29, 70.98)	67.04 (66.53, 67.52)	69.6 (69.16, 70.03)	73.81 (73.21, 74.36)

Supplementary Figure 6 shows the age distributions of deaths for each province for 1990, 2000, and 2015 for coarse age groups. Notable and desirable is the simultaneous decline in the proportion of child deaths and the increase in the proportion of elderly deaths in almost every province. Exceptions to this are Bangka Belitung, where children under 5 did not see improvements from 2000 to 2015, but 5-14 year-olds and 70-79 year- olds did; Kepulauan Riau and Gorontalo, where the age distribution above age 50 appears almost identical from 1990 to 2015; Jawa Barat, Banten, and Kalimantan Timur, where the age distribution above age 70 appears the same from 2000 to 2015; and Sulawesi Utara, where deaths under 5 account for a slightly greater proportion of deaths in 2015 than in 2000.

Indonesian age-specific death estimates follow a similar age pattern to GBD 2016 estimates, as shown in Supplementary Figure 7. Supplementary Figure 8 shows a comparison of life expectancy at birth for Indonesia with GBD 2016 estimates, highlighting a dip in 2004 due to the tsunami that devastated Aceh.

Table 15. Mortality rate per 1000 and number of deaths in thousands with 95% uncertainty by age and sex in 2015 for Indonesia

	Mortality Rate (per 1000 population)		**Deaths**	
Age (years)	**Male**	**Female**	**Male**	**Female**
<1	27.08 (22.97, 31.1)	21.76 (18.81, 25.12)	72891.6 (61830.2, 83712.5)	56098.8 (48488.4, 64757)
1-4	1.40 (1.14, 1.7)	1.13 (.94, 1.36)	14507.6 (11896.6, 17625.6)	11264.8 (9330.8, 13619.8)
5-9	0.9 (.81, 1)	0.53 (.48, .6)	11601.4 (10510.3, 12888.5)	6504.9 (5855.2, 7276.3)
10-14	0.87 (.8, .94)	0.50 (.46, .56)	10911.2 (10066.4, 11846.3)	6017.2 (5437.9, 6696.1)
15-19	1.46 (1.27, 1.68)	0.83 (.73, .94)	16719.3 (14514.3, 19226.1)	9180.5 (8114, 10458.9)
20-24	1.99 (1.71, 2.34)	0.94 (.8, 1.1)	21276.4 (18232.6, 24970.8)	10160.8 (8709.2, 11863)
25-29	2.38 (2.05, 2.78)	1.18 (.98, 1.42)	27328 (23577.6, 31999.6)	13587.2 (11272.7, 16355.9)
30-34	2.55 (2.17, 3.04)	1.45 (1.18, 1.78)	27435.5 (23369.3, 32698.7)	15414.2 (12550.6, 18898.4)

	3.17	2.14	32034.7	21129.5
35-39	(2.66, 3.83)	(1.72, 2.68)	(26913.3, 38772.6)	(16938.9, 26477.9)
	4.41	3.06	39775.5	27053.9
40-44	(3.69, 5.37)	(2.48, 3.82)	(33222, 48439.6)	(21904.7, 33767.9)
	6.28	4.65	47781.1	35071.8
45-49	(5.19, 7.68)	(3.82, 5.71)	(39521.7, 58500.9)	(28802.1, 43099.4)
	9.51	6.83	60423	41891.7
50-54	(7.95, 11.6)	(5.65, 8.36)	(50511.2, 73698.3)	(34637.1, 51281.5)
	13.63	10.02	64979	43569.7
55-59	(11.34, 16.58)	(8.26, 12.3)	(54057.6, 79025.6)	(35905.5, 53492.9)
	22.73	15.69	72117.7	52634.8
60-64	(19.32, 27.22)	(13.09, 18.95)	(61318.8, 86366.2)	(43937.5, 63580.1)
	31.65	22.46	76096.9	58934.8
65-69	(26.63, 37.83)	(18.72, 27.42)	(64017.9, 90962.2)	(49117.2, 71945.7)
	49.62	36.05	82243.6	74494.5
70-74	(41.78, 58.85)	(30.14, 43.96)	(69256.3, 97540.7)	(62282.9, 90839.1)
	67.04	54.83	60911.6	67955.2
75-79	(55.77, 80.6)	(45.78, 66.8)	(50670.2, 73227.2)	(56733.4, 82793)
	135.33	130.53	94375.7	137349.3
80+	(120.74, 151.66)	(117.1, 147.14)	(84200.4, 105766.6)	(123214.7, 154832)
	6.43	5.38	833409.8	688313.6
All ages	(5.6, 7.49)	(4.65, 6.29)	(725802.3, 971633.3)	(594119.8, 804177.8)

Discussion

Life expectancy at birth has increased in all provinces in Indonesia from 1990 to 2015. These improvements are in large part due to declining child mortality in all provinces, with probability of death before age 5 decreasing by 50%-85% in more than two-thirds of the provinces over this time period. Concurrently, adult mortality decreased in every province except in Aceh for females, and for 26 of the 34 provinces for males, with an average relative decrease of 28% and 24%, respectively, in these provinces. Uncertainty is very wide around the adult mortality estimates due to data quality and sparseness, but the data that are available do not show as consistent or drastic a decline as those in child mortality.

This analysis quantifies province-level mortality rates over time with 95% uncertainty using all available data sources, and there were several measurement challenges throughout this study. First, new province boundaries over the years cause complexity in data extraction and reporting of estimates. Consistent predictions are now made for all current provinces over time by mapping past data to current

province boundaries, overcoming this obstacle. This allows newer provinces to observe their own trends over time, even before they existed as separate entities. However, even with this detailed mapping, the newest province North Kalimantan (created in 2013) has little-to-no raw data for many indicators, except for child mortality, and is heavily dependent on its neighbors.

Also, the absence of a vital registration system leads to sparse data for adult mortality: only household deaths and sibling histories are available, and both are noisy and uncertain. The planned expansion of the Sample Registration System will help fill these gaps and provide a more consistent source of mortality data for Indonesia. Not much can be done to respond to data sparseness except taking care to create appropriate uncertainty intervals, which has been done here by incorporating both sampling error of the data points and uncertainty in the estimation process itself. Specifically, for sibling history data for ${}_{45}q_{15}$, I used strategies to mitigate biases (survivor bias, zero-surviving sibship bias, recall bias). This is sufficient under the assumption that adult mortality in a sibship is not correlated with sibship size. In terms of life tables, eleven of the fourteen included empirical life tables from Indonesia here are from provinces on Java. True, these provinces also represent a population majority of the country, but the underrepresentation of smaller provinces causes their mortality age patterns to be heavily dependent on those observed on Java.

The large uncertainty around estimates reduces confidence in the interpretation and application of these results, as it should. It highlights the need for more data, particularly better capture and reporting of adult deaths. Uncertainty intervals are likely actually underestimated, because uncertainty is not incorporated for population, births, empirical life tables, or the covariates. Data are sparse for the standard life table generation and for the LDI covariate in the 1990s, and the Indonesian life tables are based on household death data which is prone to recall bias. Lastly, the raw data accuracy for the 2004 Aceh tsunami is difficult to assess because data are more difficult to obtain during conflict and disaster, and the age and sex structure of this fatal discontinuity is based on a survey of surviving refugees from the disaster, which may not be representative of those who died.

These mortality estimates are similar to national estimates from GBD, converging over time (Supplementary Figures 7 and 8), with life expectancy estimates within a year of each other in 2015.(29) The age distributions of deaths are also similar between these estimates and GBD (Supplementary Figure 9).

Most of the machinery behind this analysis corresponds to GBD methods, with some important differences. First, I generated several input time series from scratch, including births, population, years of education, and LDI. Population was interpolated using a period method, applying cubic splines across years, but a cohort-component projection method as conducted in GBD 2017 would be a more thorough approach that would enable and ensure consistency among population, fertility, migration, and mortality trends over time.(30)

Additionally, the Indonesia-specific life tables included in this analysis likely has a large impact on differences in estimates because GBD estimates in Indonesia do not yet utilize any Indonesia life tables. I also accounted for spatial similarities among neighboring provinces by adding a hierarchical level of "region" between provinces and the national level. This regional effect is included as a nested random effect with province in the completeness data synthesis, child sex-splitting, child age-splitting, and education models. Region also informs the GPR parameter combinations for the ${}_5q_0$ estimation. Another major difference between this analysis and GBD is that GBD estimates HIV mortality separately and creates mortality tables including and excluding HIV deaths. This is an important step in locations with substantial HIV epidemics, where HIV can drastically change the age patterns of mortality from what is observed in life tables. However, in Indonesia, HIV accounts for only 0.49% of total deaths in 2015, compared to 11.15% in sub-Saharan Africa.(16)

If and when the Sample Registration System expands in Indonesia, mortality estimation can improve and reduce uncertainty in light of additional data. With recent (1999) decentralization, provinces can use these estimates to help inform health policy decisions and to catalyze investigations into why some provinces are experiencing better health than others. These mortality estimates can also feed into other province-level analyses such as assessing causes of death and risk factor exposures and effects.

References

1. GBD 2013 Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015 Jan 10;385(9963):117–71.

2. Wang H, Dwyer-Lindgren L, Lofgren KT, Rajaratnam JK, Marcus JR, Levin-Rector A, et al. Age-specific and sex-specific mortality in 187 countries, 1970-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012 Dec 15;380(9859):2071–94.

3. Population [Internet]. BPS Statistics Indonesia; 2015. Available from: http://www.bps.go.id/Subjek/view/id/12#subjekViewTab3|accordion-daftar-subjek1

4. The World Factbook — Central Intelligence Agency [Internet]. [cited 2017 Jan 30]. Available from: https://www.cia.gov/library/publications/the-world-factbook/geos/id.html

5. Ahmad E, Hofman B. Indonesia: Decentralization-Opportunities and Risks [Internet]. 2000 [cited 2017 Jan 30]. Available from: http://siteresources.worldbank.org/INTINDONESIA/Resources/Decentralization/Opportunities_and_Risks_A1.pdf

6. Stevens GA, Alkema L, Black RE, Boerma JT, Collins GS, Ezzati M, et al. Guidelines for Accurate and Transparent Health Estimates Reporting: the GATHER statement. PLOS Med. 2016 Jun 28;13(6):e1002056.

7. GBD 2013 Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet Lond Engl. 2015 Jan 10;385(9963):117–71.

8. Rajaratnam JK, Marcus JR, Flaxman AD, Wang H, Levin-Rector A, Dwyer L, et al. Neonatal, postneonatal, childhood, and under-5 mortality for 187 countries, 1970-2010: a systematic analysis of progress towards Millennium Development Goal 4. Lancet Lond Engl. 2010 Jun 5;375(9730):1988–2008.

9. Hill K. Estimating census and death registration completeness. Asian Pac Popul Forum. 1987 May;1(3):8–13, 23–4.

10. Bennett NG, Horiuchi S. Estimating the Completeness of Death Registration in a Closed Population. Popul Index. 1981;47(2):207–21.

11. BRASS W, COALE AJ, DEMENY P, HEISEL DF, LORIMER F, ROMANIUK A, et al. Methods of Analysis and Estimation. In: Demography of Tropical Africa [Internet]. Princeton University Press; 1968 [cited 2017 Jan 12]. Available from: http://www.jstor.org/stable/j.ctt183pzx0

12. Preston S, Coale AJ, Trussell J, Weinstein M. Estimating the Completeness of Reporting of Adult Deaths in Populations That Are Approximately Stable. Popul Index. 1980;46(2):179–202.

13. Preston S, Hill K. Estimating the completeness of death registration. Popul Stud. 1980 Jul;34(2):349–66.

14. Murray CJL, Rajaratnam JK, Marcus J, Laakso T, Lopez AD. What can we conclude from death registration? Improved methods for evaluating completeness. PLoS Med. 2010 Apr 13;7(4):e1000262.

15. Obermeyer Z, Rajaratnam JK, Park CH, Gakidou E, Hogan MC, Lopez AD, et al. Measuring adult mortality using sibling survival: a new analytical method and new results for 44 countries, 1974-2006. PLoS Med. 2010 Apr 13;7(4):e1000260.

16. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet Lond Engl. 2016 Oct 8;388(10053):1459–544.

17. Gakidou E, King G. Death by survey: estimating adult mortality without selection bias from sibling survival data. Demography. 2006 Aug;43(3):569–85.

18. Masquelier B. Adult mortality from sibling survival data: a reappraisal of selection biases. Demography. 2013 Feb;50(1):207–28.

19. Indonesia - Sensus Penduduk 2000 - Gambaran [Internet]. [cited 2017 Jan 18]. Available from: http://microdata.bps.go.id/mikrodata/index.php/catalog/82

20. Education in Indonesia - WENR [Internet]. [cited 2017 Mar 16]. Available from: http://wenr.wes.org/2014/04/education-in-indonesia

21. UNESCO-UNEVOC World TVET Database [Internet]. [cited 2017 Mar 16]. Available from: http://www.unevoc.unesco.org/go.php?q=World+TVET+Database&ct=IDN

22. Indonesia Database for Policy and Economic Research [Internet]. The World Bank; [cited 2015 May 28]. Available from: http://databank.worldbank.org/data/reports.aspx?source=1266

23. World Economic Outlook [Internet]. International Monetary Fund; [cited 2016 Aug 25]. Available from: http://www.imf.org/en/Publications/WEO/Issues/2017/04/04/world-economic-outlook-april-2017

24. Rajaratnam JK, Tran LN, Lopez AD, Murray CJL. Measuring under-five mortality: validation of new low-cost methods. PLoS Med. 2010 Apr 13;7(4):e1000253.

25. Gompertz B. On the Nature of the Function Expressive of the Law of Human Mortality, and on a New Mode of Determining the Value of Life Contingencies. Philos Trans R Soc Lond. 1825;115:513–83.

26. Mahalanobis PC. On the generalised distance in statistics. Proc Natl Inst Sci India. 1936;2(1):49–55.

27. Data Dan Informasi Bencana Indonesia [Internet]. [cited 2017 Aug 30]. Available from: http://dibi.bnpb.go.id/data-kependudukan/bencana-vs-kependudukan

28. Doocy S, Rofi A, Moodie C, Spring E, Bradley S, Burnham G, et al. Tsunami mortality in Aceh Province, Indonesia. Bull World Health Organ. 2007 Apr;85(4):273–8.

29. GBD 2016 Mortality Collaborators. Global, regional, and national under-5 mortality, adult mortality, age-specific mortality, and life expectancy, 1970-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Lond Engl. 2017 Sep 16;390(10100):1084–150.

30. GBD 2017 Population and Fertility Collaborators. Population and fertility by age and sex for 195 countries and territories, 1950-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Lond Engl. 2018 10;392(10159):1995–2051.

List of Tables

List of Supplementary Tables

List of Figures

List of Supplementary Figures

Chapter 2

A systematic analysis of cause-specific mortality in 34 Indonesian provinces in 2014

Introduction

Until 2014, knowledge about causes of death in Indonesia depended on sparse and inconsistently-collected hospital data, self-report questions in surveys about a household or community, and expert knowledge from local leadership ((1,2)). The first collection of a nationally-representative sample of verbal autopsy deaths in 2014 makes a more systematic and comparable set of cause of death estimates possible.

Verbal autopsy (VA) is a method of determining the cause of death for an individual for whom there is no death certificate, without needing an autopsy or medical record review. Many of these deaths that VA captures occur outside the formal health system and therefore do not have death certificates with the underlying causes of death. Trained interviewers ask someone familiar with the deceased about signs and symptoms leading up to the death, using a structured questionnaire. Standardized analytics or trained physicians then assign a cause of death to the deceased using the responses from the interview. VA has become an important method to assess the mortality landscape in countries that lack vital registration systems and have scarce or poor-quality data on the causes of death.(3,4)

The Indonesian Ministry of Health first implemented VA in select sites in 2007, in order to assess sites with higher tuberculosis mortality rates. The number of sites has gradually increased since then, and VA collection in a nationally-representative sample of over 43,000 deaths was conducted in 2014, and the collection continues to scale up as part of a Sample Registration System. These verbal autopsies were assigned causes of death by a trained physician within two months of data collection. For a country with over 250 million people, this collection of physician-coded verbal autopsies (PCVA) has been a massive effort and a huge step toward better understanding the health of the country's residents.

In order to understand the distribution of causes of death in verbal autopsy data, the causes must be grouped to form a cause list that fulfills two criteria: relevance to the stakeholders (policy makers, physicians, communities), and acceptable accuracy of the cause of death assignments at that level of detail. "Acceptable" here is an arbitrary threshold that is important to describe in detail when working with verbal autopsies in order to convey this aspect of the confidence and uncertainty of any resulting estimates. Few studies have attempted to assess the accuracy of PCVA by cause group (4–14). The largest, most comprehensive study to assess PCVA performance against gold standard diagnoses (clinical diagnostic criteria) is that conducted by the Population Health Metrics Research Consortium (PHMRC), which collected 12,542 PCVA's and their corresponding gold standard cause of death assignment from four countries: Mexico, Tanzania, the Philippines, and India (5). No study has utilized this information to inform the cause list used for cause of death analysis in published literature (PUBMED search for any articles citing PHMRC PCVA validation study (5)), until this analysis.

This study is comprised of four aims:

1. To create an analytical cause list hierarchy that optimizes validity and policy relevance of the cause groups;
2. To aggregate all available verbal autopsy data in Indonesia and map all deaths to this cause list;

3. To estimate cause fractions by province/age/sex for all cause groups, along with measures of predictive validity for these estimates; and
4. To calculate cause-specific mortality rates, deaths, and years of life lost for each province/age/sex at each level in the analytical cause list hierarchy after adjusting cause fraction estimates to sum to 100%.

Methods

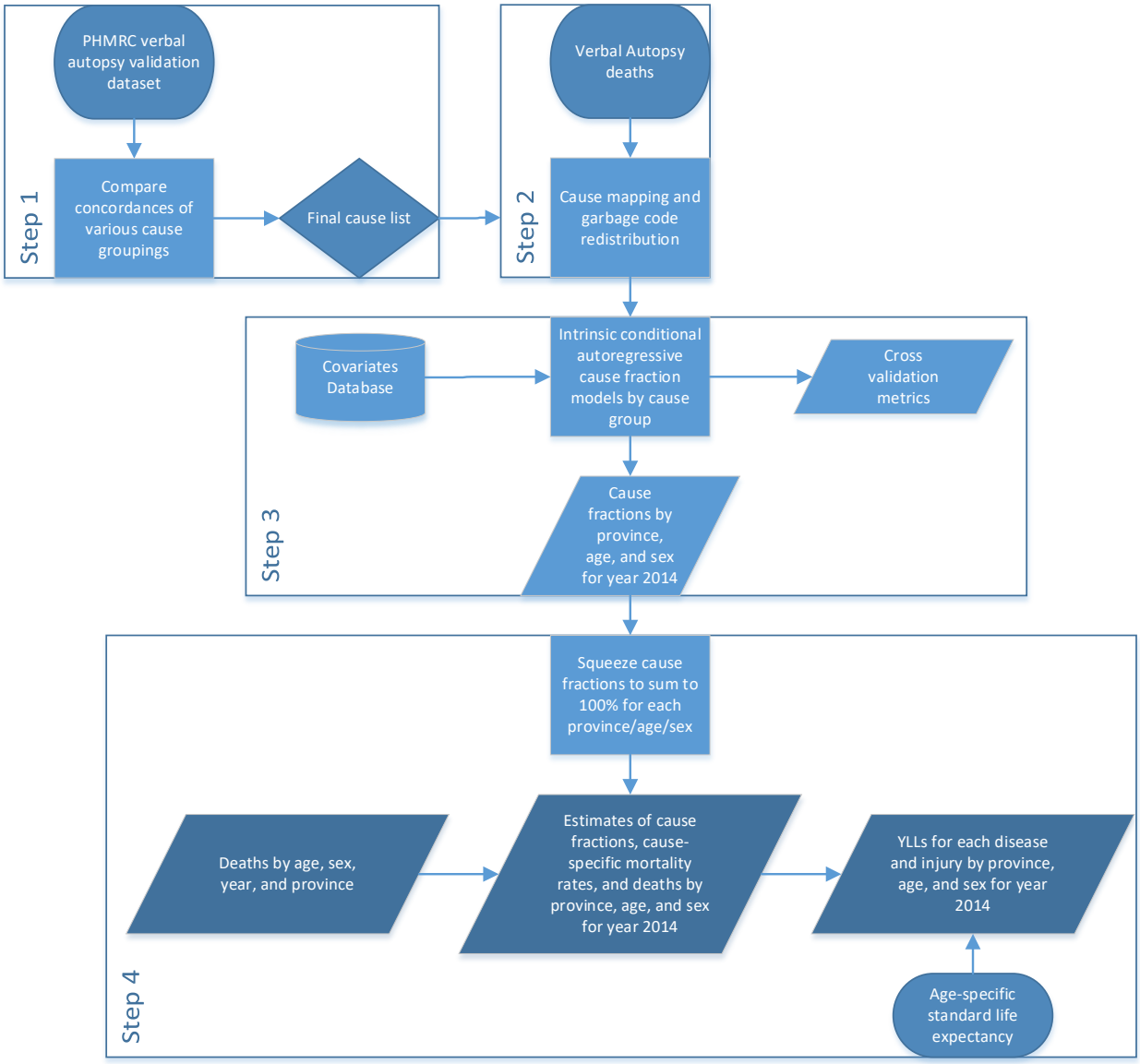
This study is comprised of four steps (Figure 1). First, I chose the 3-tier analytical cause list that performed best in terms of concordance within the PHMRC PCVA validation datasets. Second, the Indonesia PCVA causes of death were mapped to the cause list resulting from the first step. Third, I applied small area estimation models that incorporate spatial autocorrelation via an intrinsic conditional autoregressive (ICAR) random effect as well as fixed effects to each cause of death and calculated predictive validity metrics to assess model performance. Fourth, the cause fraction estimates were proportionally adjusted to sum to 100% at each level of the cause list hierarchy and then applied to all-cause mortality rates to obtain cause-specific mortality rates, which were then multiplied by the standard life expectancy to obtain years of life lost (YLLs) by cause. Data were analyzed to generate cause-specific mortality and YLL estimates with uncertainty for 2014 by age and sex for all 34 provinces in Indonesia.

Step 1: Cause List Hierarchy Selection

The PCVA data for Indonesia contain over 130 distinct cause of death assignments. However, many of these causes of death are either too detailed to be useful in analysis (57 of the 133 causes have fewer than 50 deaths nationwide concentrated in populous provinces, making spatial estimation difficult), or known to have questionable data accuracy when compared with gold standard diagnoses (5). To address the former disadvantage, PCVA deaths can be aggregated into more analytically useful cause groups, such as aggregating all homicide deaths into one category regardless of the method used, to result in data with larger sample sizes across the stratification groups (province, age, sex). The data accuracy issue is more difficult to address because of a lack of precedence in published literature and scarce studies on the validity of physician-certified verbal autopsies. This analysis presents a method to create an analytical cause list that both acknowledges and, more importantly, addresses PCVA data accuracy issues based on current literature.

In this analysis, a PCVA validation dataset informed the cause list for this analysis based on observed concordances between cause of death assignment and the true cause of death according to gold standard diagnoses. This validation dataset was compiled for a validation assessment of PCVA performance, conducted by the Population Health Metrics Research Consortium (PHMRC) in four countries (4–6). This consortium consisted of collaborating scientists from various areas of expertise, such as medicine, epidemiology, demography, and biology, from eleven institutions across the United States, India, Tanzania, the Philippines, and Australia (15).

Figure 1. Analytical steps for cause of death estimation



The PHMRC study defined clinical diagnostic criteria as the gold standard for a multisite sample of 12,542 verbal autopsies gathered from Bohol in the Philippines, Mexico City in Mexico, Dar es Salaam and Pemba Island in Tanzania, and Andhra Pradesh and Uttar Pradesh in India. Separate questionnaires were used for three age modules—stillbirths and neonates, children 1 month to 11 years, and "adults" 12 years and older—and each death was assigned both a physician-certified cause of death and a gold-standard cause of death based on clinical diagnostic criteria, resulting in a validation dataset of 7,836 adults, 2,075 children, 1,629 neonates, and 1,002 stillbirths. The target list of causes for the PHMRC analysis consisted of 34 for adults, 21 for children, and 10 for neonates, excluding stillbirths. The study created 500 test-train dataset pairs from the universe of 12,542 cases, covering a range of cause compositions to ensure assessment metrics were independent of the cause distribution (5). Each pair was created by first randomly splitting the data without replacement into 75%/25% training and test datasets by cause. Then the data in the test datasets were resampled with replacement to have the

same number of cases for adult, child, neonatal, and stillbirth deaths as the collected validation dataset. This resampling with replacement ensured that the cause distributions in the test datasets were not correlated with those in the training datasets (4). I created fourteen cause list variations based on these PHMRC validation datasets split into three levels of detail: Level 1, with 3-5 adult cause groups; Level 2, with 7-19 adult cause groups; and Level 3, with 23-34 adult cause groups. These cause list variations include cause groups based on policy relevance and similar symptomology at varying degrees of detail in each Level. Level 3 represents the most detailed level, Level 2 the somewhat detailed level, and Level 1 the coarsest level.

In order to create a cause list appropriate for PCVA in Indonesia, I calculated concordance as a measure of accuracy using the PHMRC PCVA validation dataset for each of these cause lists, detailed below. The best-performing cause lists in terms of concordances formed a 3-tier analytical hierarchy for the subsequent subnational cause of death estimation in Indonesia.

To obtain the concordances for each cause list, I calculated average percent concordance by cause and age module across the 500 PHMRC validation datasets described above, defined as the percentage of deaths in a cause group that was correctly assigned to that cause of death. My goal was to obtain a useful hierarchy of cause groups that depends not only on policy relevance, but also on the validity of those cause of death assignments.

Level 1

Level 1 causes of death represent the coarsest grouping with the highest observed percent concordances, that can logically inform further cause group detail. For this level, I tested three groupings, which I have named “A,” “B,” and “C” for reference in tables. Cause grouping A consists of the same three high-level groupings used in GBD, cause grouping B breaks out maternal conditions as a separate group, and cause grouping C breaks out both maternal conditions and respiratory diseases as separate groups. The grouping chosen for Level 1 is cause grouping A, which has the highest observed concordances for all cause groups (Supp Table 1, Figure 2).

Level 2

Next, I calculated percent concordances for five cause groupings (referred to as D, E, F, G, and H) to form a mid-tier level between the coarse Level 1 groups and the most detailed Level 3 groups. Respiratory diseases presented a challenge because they tend to be difficult to capture accurately in PCVA data, and they are also critical conditions to monitor and address because of their high burden in Indonesia (2). I tested various combinations of diseases that can have common respiratory symptomology (Table 1) to find the cause grouping structure that would result in the highest observed percent concordances, thereby conveying the most accurate estimates for these diseases. AIDS was included in this list because it can have similar respiratory symptoms to the other respiratory diseases and also predisposes the individual to respiratory disease comorbidity such at tuberculosis due to an impaired immune system (16).

Table 1. Comparison of common symptoms across respiratory diseases in the PHMRC validation dataset, with sources in parentheses.

Symptom	AIDS(17)	Pneumonia(18)	Lung Cancer(19)	COPD(20)	Asthma(21)	TB(22)	Other Respiratory Diseases(23–27)
chest pain or tightness		X	X	X	X	X	X
cough		X	X	X	X	X	X
coughing up green or bloody sputum		X	X	X	X	X	
fast breathing and shortness of breath		X	X	X	X		X
fever	X	X				X	X
headache	X						
lung infections	X						X
muscle aches and joint pain	X						X
Nausea, vomiting, or diarrhea		X					
night sweats	X					X	
rapid heart beat		X				X	X
shaking and chills		X					X
skin rash	X						
sore throat	X						
swollen glands	X						
tiredness	X	X	X	X		X	
unexplained weight loss	X		X			X	X
weakness		X	X			X	
wheezing			X	X	X		X
other (malnutrition, infertility, arthritis, cyanosis, anxiety)							X

Cause grouping E has separate cause groups for infectious respiratory diseases and chronic respiratory diseases. Cause grouping H breaks down the infectious respiratory diseases by specific disease (pneumonia and tuberculosis). Cause groupings D, F, and G combine HIV/AIDS, pneumonia, lung cancer, COPD, asthma, tuberculosis, and other respiratory diseases into one group representing all respiratory diseases with common symptomatology (Table 2), to be broken down into further detail in Level 3. This grouping of all respiratory diseases attempts to create the most accurate envelope possible for respiratory diseases so that they can be broken down more in Level 3 with a higher overall level of accuracy.

The concordance for “All respiratory diseases” is at least 10 percentage points higher—in cause groupings D, F, G—than separating respiratory diseases into chronic and infectious or more detail—

cause groupings E, H (Table 2; Supp Table 1: Level 2), confirming that grouping them together will lead to more accurate estimates for both infectious and chronic respiratory diseases.

Table 2. Percent concordances and number of deaths in the PHMRC validation dataset to inform cause list groupings: subset of respiratory diseases shown here. The selected cause list grouping for is boxed in red. The full list of concordances by cause group is in Supplementary Table 1.

Grouping	Module	Cause group	Deaths (#)	Percent concordance (%)
D	Adult	All respiratory diseases	458	67.6%
D	Child	All respiratory diseases	35	55.6%
E	Adult	Chronic respiratory diseases	336	53.3%
E	Adult	Infectious respiratory diseases	89	50.7%
E	Child	Infectious respiratory diseases	35	54.4%
F	Adult	All respiratory diseases	667	73.1%
F	Child	All respiratory diseases	83	63.7%
G	Adult	All respiratory diseases	667	72.5%
G	Child	All respiratory diseases	83	63.4%
H	Adult	HIV/AIDS	77	69.2%
H	Adult	Lower respiratory infections	89	41.0%
H	Adult	Chronic respiratory diseases	203	55.6%
H	Adult	Tuberculosis	74	67.3%
H	Child	HIV/AIDS	24	50.0%
H	Child	Lower respiratory infections	35	54.8%
H	Child	Other respiratory diseases	1	0.0%
H	Child	Tuberculosis	4	0.0%

After excluding groups with fewer than ten deaths represented, cause list F has the highest proportion of groups with concordances of at least 60% (20 of the 26 groups have concordances >=60%), and with the fewest groups with concordances less than 50% (F has only 2, H has 7, G has 6, D has 3, and E has 3), and with the highest overall mean and median observed concordances of 70.1% and 69.0%, respectively (Table 3). For cause grouping F, two cause groups have percent concordances lower than 50%: falls, which is 49.2%, and other noncommunicable diseases in children including cardiovascular diseases, which is 29.2%. The latter is quite low for children, but it is also somewhat rare among children (estimated 15.7% of all deaths for ages 1-14 in GBD (2)). Among adults in whom this cause group is more common, the percent concordance is much higher: 80.9%. For these reasons, I chose cause grouping F for Level 2 of my analytical hierarchy (Figure 2).

Table 3. Mean and median concordances across cause groups for each Level 2 grouping. The selected grouping F is denoted by a red box.

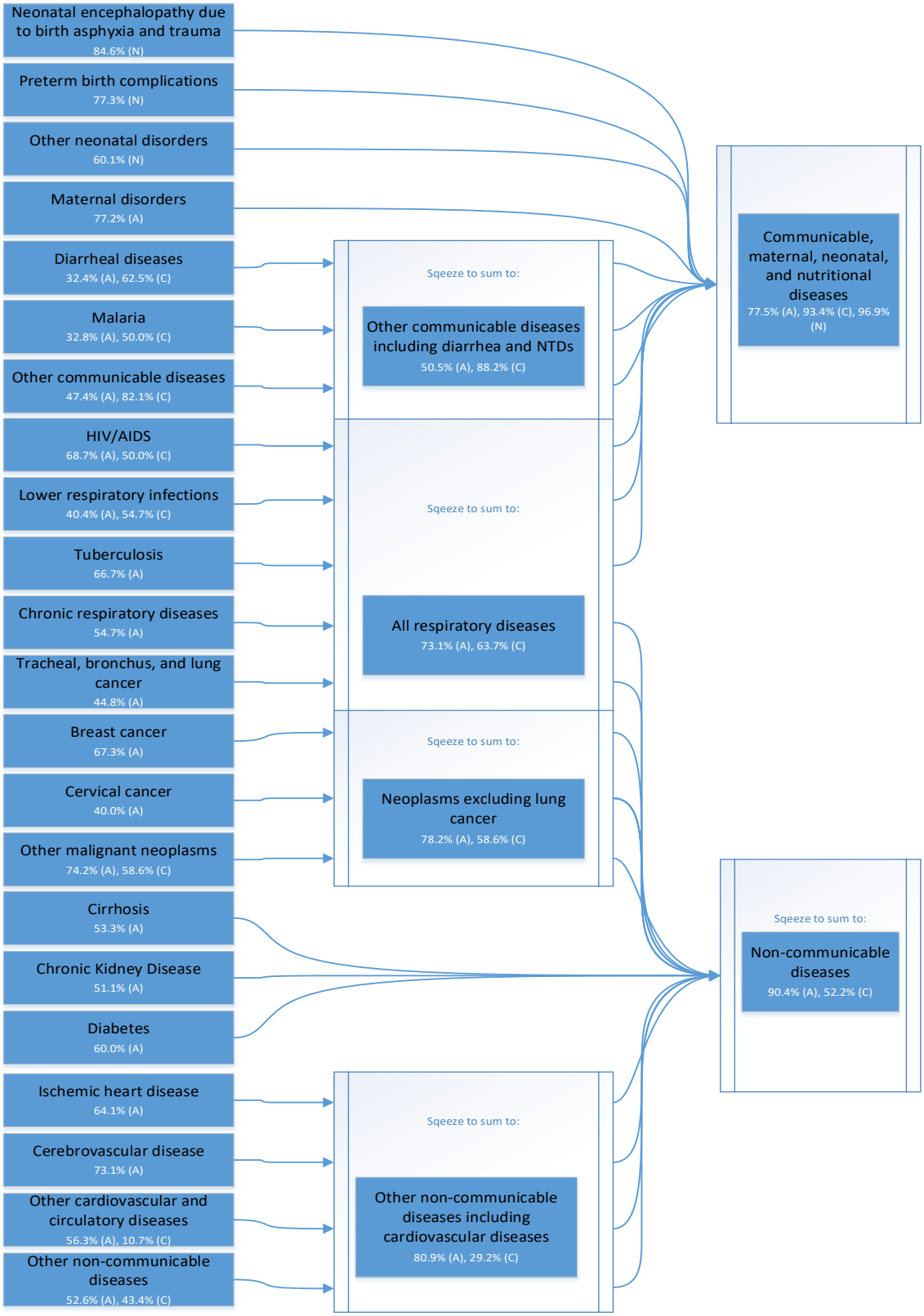
	Observed Concordances							
	Adults		Children		Neonates		**Overall**	
Grouping	mean	median	mean	median	mean	median	**mean**	**median**
D	68.8%	69.0%	63.8%	61.9%	73.9%	81.6%	**67.9%**	**68.3%**
E	64.6%	63.6%	63.4%	61.9%	73.1%	81.7%	**65.7%**	**65.3%**
F	66.0%	67.4%	73.6%	76.8%	77.4%	81.0%	**70.1%**	**69.0%**
G	61.3%	63.6%	64.4%	63.4%	76.8%	80.6%	**64.4%**	**65.4%**
H	61.4%	61.7%	63.9%	60.1%	76.8%	80.7%	**64.0%**	**61.7%**

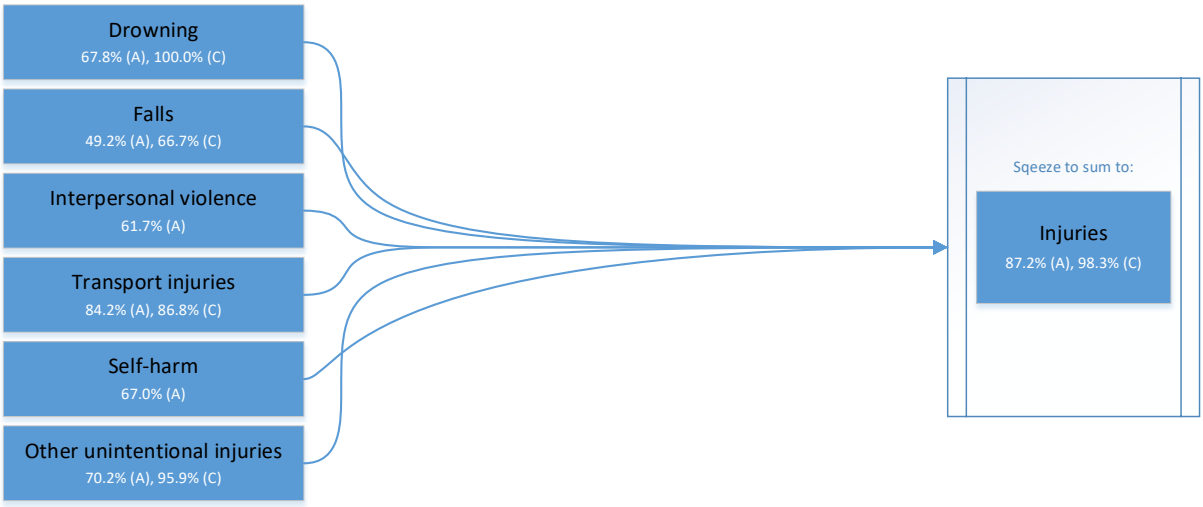
Level 3

Level 3 represents the most detailed level of cause groups to be estimated. I tested six different cause lists to find one with a minimum percent concordance of 40% for all causes among cause groups that have at least 90 observed deaths in Indonesia in the PHMRC dataset. I rejected the cause lists that split chronic respiratory diseases further because very little of the Indonesian PCVA data has that level of specificity: just 6% of chronic respiratory deaths are assigned a more specific cause of death after garbage code redistribution, which is not enough to reliably inform a more detailed cause breakdown. I also rejected cause lists that split maternal conditions into more specific causes like obstructed labor and hemorrhage because 34% of maternal deaths in the Indonesian PCVA data are non-specific, and the remaining data with specific causes show a very different cause distribution than GBD 2016(2) (hemorrhage/hypertension/other 37%/51%/13% data vs. 33%/12%/55% in GBD). This suggests that there may be a loss of cause distribution information in the non-specific 34% of maternal deaths. These non-specific deaths are not likely evenly distributed across hemorrhage, hypertension, and other, but rather I suspect that they contain more "other" deaths and fewer hypertension deaths, which would align more with the GBD estimates. Regardless, the observed proportions are so different from GBD 2016 that I rejected their inclusion in the cause list for this analysis.

I selected cause grouping J for Level 3 of the analytical hierarchy (Supp Table 1, Figure 2). It has concordances of at least 40% for all causes represented by more than 90 deaths, and only two cause groups have concordances less than 40% and more than ten deaths: malaria among adults (32.8%) and other cardiovascular and circulatory diseases among children (10.7%). The former is paired with a much higher percent concordance observed for malaria among children (50%). The latter is rare among children (<1% of deaths under age 5, 25.3% of deaths age 15-49 estimated by GBD 2016 (2)) and paired with a much higher percent concordance among adults (64.1% for IHD, 73.1% for cerebrovascular disease, and 56.3% for other cardiovascular and circulatory diseases).

Figure 2. Analytical cause list hierarchy resulting from the assessment of PCVA performance in PHMRC validation datasets, with corresponding concordance values for each cause group for adults (A), children (C), and neonates (N) where at least 5 deaths were represented in the PHMRC dataset (excludes chronic respiratory diseases and tuberculosis in children).





Step 2: VA data prep and garbage code redistribution

All available verbal autopsy data for Indonesia were combined from 2006 to 2014 by province, age, sex, and cause in order to maximize the power of the cause fraction estimates, for a total sample of 64,064 deaths (Table 4). RISKESDAS (Population Health Basic Health Research) is a national household survey conducted in 2007-2008 which covered all major health indicators such as household consumption, smoking habits, various aspects of health services, and health status, including verbal autopsies for deaths that occurred in the previous 12 months (28). The RISKESDAS verbal autopsy instruments were adapted from the WHO World Health Survey (29) and consisted of questionnaires for three age groups: neonatal (0-28 days), infant and young child (29 days-4 years), and older child and adult (5 years and older), with a cause of death assigned by one trained physician.

The Indonesian Mortality Registration System Strengthening Project (IMRSSP) was created to improve the completeness of death registration and to formalize the recording of causes of death using verbal autopsies for deaths at home (30). It consisted of sentinel sites around the country that scaled up over time to cover more provinces. The verbal autopsy questionnaire was an Indonesian adaptation of draft versions of international standard VA questionnaires created by the World Health Organization (3,30), with the trained local health center physician assigning the cause of death (31–34). The IMRSSP verbal autopsy instruments consisted of questionnaires for three age groups: neonatal (0-28 days), infant and young child (29 days-4 years), and older child and adult (5 years and older).

The Sample Registration System (SRS) collects information on births, deaths, and causes of death via verbal autopsies in a sample area that, in the 2014 nationally-representative sample, covered 128 subdistricts across 30 provinces. The selection of sites in 2014 was based on the Village Potency Survey (PODES) sampling scheme. The WHO Standard Verbal Autopsy instruments were administered by trained paramedical personnel, with the diagnosis assigned by two trained physicians. Physicians assigned causes of death for the verbal autopsies from all of these sources based on ICD-10 rules and coding system. The instruments consisted of modules for three age groups: neonatal (0-28 days), infant and child (29 days-11 years), and adult (12 years and older) (35).

Table 4. Number of deaths by source, year, and province.

Province	RISKESDAS 2006-2007	IMRSSP 2009-2011	IMRSSP 2011	SRS 2012	SRS 2013	SRS 2014	TOTAL
Aceh	1	0	0	87	24	285	**397**
Sumatera Utara	1	0	2,742	421	102	1,760	**5,026**
Sumatera Barat	79	212	1,563	80	0	404	**2,338**
Riau	0	0	0	119	56	597	**772**
Jambi	41	0	0	45	30	140	**256**
Sumatera Selatan	108	0	1,404	283	8	894	**2,697**
Bengkulu	0	0	0	44	0	144	**188**
Lampung	0	0	0	54	26	405	**485**
Bangka Belitung	0	0	0	39	0	171	**210**
Kepulauan Riau	0	0	0	0	0	179	**179**
DKI Jakarta	0	0	0	0	0	3,498	**3,498**
Jawa Barat	5	2	3,359	0	116	8,689	**12,171**
Jawa Tengah	374	3,875	0	0	18	8,137	**12,404**
DI Yogyakarta	0	0	0	0	0	568	**568**
Jawa Timur	35	0	280	0	4	9,529	**9,848**
Banten	0	0	2	0	0	1,689	**1,691**
Bali	0	923	0	0	0	723	**1,646**
Nusa Tenggara Barat	80	0	734	0	0	1,041	**1,855**
Nusa Tenggara Timur	1	0	817	0	0	379	**1,197**
Kalimantan Barat	0	0	0	0	0	167	**167**
Kalimantan Tengah	74	0	0	0	0	53	**127**
Kalimantan Selatan	111	0	0	0	0	149	**260**
Kalimantan Timur	72	0	0	0	0	399	**471**
Kalimantan Utara	22	0	0	0	0	0	**22**
Sulawesi Utara	105	0	0	0	0	310	**415**
Sulawesi Tengah	68	0	0	0	0	83	**151**
Sulawesi Selatan	151	0	1,894	0	2	803	**2,850**
Sulawesi Tenggara	0	0	0	0	0	16	**16**
Gorontalo	1	1,760	0	0	0	0	**1,761**
Sulawesi Barat	0	0	1	0	4	185	**190**
Maluku	15	0	0	0	0	0	**15**
Maluku Utara	0	0	0	0	0	15	**15**
Papua Barat	0	0	0	0	0	0	**0**
Papua	0	0	0	0	0	178	**178**
TOTAL	**1,344**	**6,772**	**12,796**	**1,172**	**390**	**41,590**	**64,064**

RISKESDAS = Indonesia Basic Health Research

IMRSSP = Indonesia Mortality Registration System Strengthening Project

SRS = Sample Registration System

Each of these deaths contains a cause of death assignment using ICD-10 classification by two trained physicians and a third in case of a tie, and they were then grouped into a cause list of 28 causes, determined from Step 1 above (Supplementary Table 2). The cause list mapped directly to that derived from Step 1, with one difference: age ranges estimated for were extended for almost all cause groups to correspond to the age ranges used in the GBD cause list, rather than being restricted to three age modules in the PHMRC data (Table 5).

Table 5. Age ranges used for analysis and prediction of each cause group.

Cause Group	Analytical level	Age start	Age end
Group I: Communicable, maternal, neonatal, and nutritional diseases	1	0	80+
Group II: Non-communicable diseases	1	0	80+
Group III: Injuries	1	0	80+
Maternal disorders	2	10	50
Neonatal encephalopathy due to birth asphyxia and trauma	2	0	1
Preterm birth complications	2	0	1
Other neonatal disorders	2	0	1
Other communicable diseases including diarrhea and NTDs	2	0	80+
All respiratory diseases	2	0	80+
Neoplasms except lung cancer	2	0	80+
Other non-communicable diseases including cardiovascular diseases	2	0	80+
Diabetes mellitus	2	0	80+
Chronic kidney disease	2	PNN	80+
Cirrhosis	2	1	80+
Drowning	2	0	80+
Falls	2	0	80+
Interpersonal violence	2	0	80+
Self-harm	2	10	80+
Transport injuries	2	0	80+
Other unintentional injuries	2	0	80+
Diarrheal diseases	3	0	80+
HIV/AIDS	3	PNN	80+
Lower respiratory infections	3	0	80+
Malaria	3	0	80+
Tuberculosis	3	PNN	80+
Other communicable diseases	3	0	80+
Tracheal, bronchus, and lung cancer	3	15	80+
Breast cancer	3	15	80+
Cervical cancer	3	15	80+
Other malignant neoplasms	3	0	80+
Cerebrovascular disease	3	0	80+
Ischemic heart disease	3	PNN	80+
Other cardiovascular and circulatory diseases	3	0	80+
Chronic respiratory diseases	3	1	80+
Other non-communicable diseases	3	0	80+

Some ICD codes are considered "garbage codes" because they represent intermediate rather than underlying causes of death, such as sepsis or kidney failure; they are uninformative, such as senility or abdominal pain; or they are unspecified codes within a disease grouping, such as unspecified cancer or injury. Garbage code deaths were redistributed to plausible causes of death according to the garbage code redistribution methods used in GBD(2), and then deaths were aggregated into the cause groups in the shorter cause list for this analysis. For example, the garbage code redistribution strategy redistributes deaths coded to R54 ("Age-related physical debility") to over 50 diseases ranging from chronic kidney disease or diabetes to neglected tropical diseases using fixed proportions. Redistribution packages use fixed proportions, a uniform proportional method, custom regression models, or fractional reassignment of a death assigned to multiple causes (2).

Step 3: iCAR model specifications

Model choice

Given the small sample sizes in the verbal autopsy data for some of the provinces (12 provinces have fewer than 200 deaths, with four provinces having fewer than twenty deaths), the GBD 2016 methods may not be the appropriate strategy to estimate cause fractions subnationally for Indonesia. That method of using mixed effects models of the logit of the cause fraction and log of the rate, then smoothing the residuals with spatiotemporal regression, and finalizing estimates with GPR(2) may be too sensitive to some of the extreme outliers that are observed in the raw cause fraction data: cause fractions of 100%, for instance. I propose using a model developed specifically for small area estimation to deal with these small sample sizes: an intrinsic conditional autoregressive model (iCAR).

Bayesian hierarchical models have been widely used for modeling with sparse data or rare outcomes (36,37). The first step of the causes of death methodology for GBD is similar to these models, which generally use covariates and a set of random effects to borrow strength from grouped areas (38). Conditional autoregressive (CAR) models model the random effects using conditional autoregressive prior distributions. CAR models explicitly account for neighborhood structure by specifying that neighboring provinces are likely to be more similar than non-neighboring provinces (38). This method has more flexibility than nested random effects because it allows for more nuanced relationships between neighboring provinces than a rigid hierarchy.

Here I utilize integrated nested Laplace approximations (INLA) in R (www.r-inla.org) (39) to model cause fractions using a Besag-York-Mollie (BYM) specification, such that the spatially structured residual is an intrinsic conditional autoregressive (ICAR) model described by Besag et al. in 1991 (40). INLA is a novel approach to fitting data that is computationally much faster than traditional Markov Chain Monte Carlo (MCMC) simulation techniques because it uses an approximation for inference rather than intensive simulations (39).

Specifically, for these cause fraction models, the number of deaths due to a given cause in each province *p* is modeled as

$$y_p \sim Binomial(N, \lambda_p)$$

Where N is the sample size (total number of deaths) and λ_p is the cause fraction, with the equation

$$logit(\lambda_p) = \alpha + \boldsymbol{\beta X_p} + \upsilon_p + \nu_p$$

where α is the intercept estimating the average cause fraction in all provinces, $\boldsymbol{\beta X_p}$ represents the fixed effects (covariates and age) included in the model, and υ_p and ν_p are two province-specific random effects with normal distributions. With the BYM specification, υ_p is the spatially structured residuals using a matrix of defined neighbors for each province (41):

$$\upsilon_p | \upsilon_{j \sim p} \sim Normal(m_p, s_p^2)$$

$$m_p = \frac{\sum_{j \sim p} w_j \upsilon_j}{\sum_{j \sim p} w_j} \qquad \text{and} \qquad s_p^2 = \frac{\sigma_\upsilon^2}{\sum_{j \sim p} w_j}$$

where w_j is the weight of each province defined as a neighbor of the p-th one, and $j \sim p$ is the set of neighbors of p (38). This specification contains a more complex definition of "neighbors" than the binary yes or no to sharing a border (38,40).

The final term, ν_p, represents the unstructured residuals for the provinces, defined as an independent and identically distributed mean zero normal distribution (41), given by:

$$\nu_p \sim Normal(0, \sigma_\nu^2)$$

The spatially structured residual term (υ_p) depends on a matrix of neighbors for the provinces (Supplementary Table 3), where the adjacency relationship between each pair of provinces is set to a number between 0 (completely unrelated) and 1 (strong relationship). Four criteria define this matrix:

1. Direct land neighbor: Adjacency = 1 if the two provinces share a land border (not including corner neighbors)
2. Indirect land neighbor: Adjacency = 0.5 if the two provinces share a neighbor by land borders (i.e., two provinces are on the same island with one province in between them)
3. Direct water neighbor: Adjacency = 0.5 if the two provinces share a border over water, if the distance between the geographical centers of the provinces is less than 400 miles.
4. Not a neighbor: Adjacency = 0 for all other province-pairs.

Supplementary Table 4 contains the structured spatial effects (4a) and the unstructured spatial residuals (4b) by province.

Age group was also included, and the age pattern was accounted for by incorporating a second-order random walk on age group:

$$y_k = 2y_{k-1} - y_{k-2} + u_k$$

$$u_k \sim \text{Normal}(0, \sigma_u^2)$$

A second-order random walk imposes dependence of an estimate at age group *k* on the trend of the previous two age groups. This method penalizes deviations from the linear trend of the previous two age groups. It thus smooths the age pattern while allowing fluctuations by age for more complex age trends. For HIV/AIDS and chronic conditions at young ages, indicator variables for ages under 5 and ages 5-19 were also included to allow for more age variation in the younger ages than the random walk affords. For neonatal conditions, age was included in the model only as three indicator variables—neonatal, postneonatal, and 1-4—instead of the random walk.

Sex was included as a fixed effect in every model except cervical cancer and maternal conditions. Other fixed effects included province-specific covariate combinations for each cause of death, which were chosen based on the following criteria:

1. Based on at least one recent (since 2005) subnational source covering at least half of the 34 provinces
2. Education (age/sex-specific for most models, maternal education for neonatal conditions, diarrhea, and LRI) and the natural log of LDI were included in every model. These two covariates were modeled in Aim 1. Sociodemographic index was excluded from models in consideration of these inclusions.
3. Precedence: Selected in the top five submodels of the logit cause fraction CODEm covariate selection process for GBD 2016, ranked by model predictive validity, if available

Several cause- or covariate-specific choices were also made in addition to the applied criteria above. One covariate common in GBD 2016 CODEm models that was excluded based on criteria 1 above was alcohol consumption because for Indonesia, it was based on a single study of just 9,995 people across ten provinces. As a proxy for alcohol consumption, I included in models for those cause groups a binary covariate indicating whether the majority of each province is Muslim because alcohol consumption is generally forbidden in Islam. Second, the age-standardized fertility rate was included as a covariate for breast cancer and cervical cancer, and the covariate values for males were set to zero so that it would not influence the male breast cancer results. Third, instead of LDI per capita, I used LDI per capita excluding oil and gas, in an effort to better capture the economic level of the general population of each province and exclude the income isolated to oil and gas businesses. The estimated effects of all fixed effects used for each cause group is shown in Supplementary Table 5a, along with the age-level random effects in Supplementary Table 5b.

Validation

Model performance for each cause of death was assessed using four metrics. First, leave-one-out predictive validity was calculated to convey the models' performance when predicting for a single province/age/sex observation at a time. To summarize the performance, I calculated the mean logarithmic score (42) for each model based on the conditional predictive ordinate (CPO)(43–46), defined as:

$$\mathrm{CPO}_i = \Pr(\mathrm{Y_i} = \mathrm{y_i} \mid \mathbf{y_{-i}})$$

$$\overline{LS_i} = -\frac{\sum_{i=1}^{n} \log(\mathrm{CPO}_i)}{n}$$

Where CPO_i designates the cross-validated leave-one-out predictive probability mass at the observed value y_i, and i is the observation number from 1 to n. The subscript $-i$ in $\mathbf{y}_{-i}$ indicates that observation i is removed. A small CPO value indicates an observation $\mathrm{y_i}$ that is unexpected given the posterior model fit excluding that observation (47).

The logarithmic score (LS) is a strictly proper scoring rule used widely in Bayesian statistics to assess model prediction accuracy (42,48,49), and the mean of the logarithmic score is a robust tool for model assessment and is not sensitive to sample size (49,50). The closer to zero the logarithmic score is, the better the prediction accuracy. The leave-one-out cross validation approach is the repeated holdout method taken to its extreme, by holding out one observation at a time in n total holdouts. Calculating the scores in this approach can be extremely time-consuming with large datasets, but the INLA approach creates leave-one-out CPO values as a by-product of the model computations (51), which makes this approach quick and feasible on a personal computer (39,48,52–54). The mean logarithmic score thus conveys the relative predictive performance of each cause-specific model of a single province/age/sex group when that single data point is missing. When LS is closer to zero, the model predictions are more accurate in predicting for a single province/age/sex group when data for that group are missing.

The performance of each cause of death model was also evaluated using in-sample and two types of out-of-sample (OOS) cross validation. The first OOS method created 10 holdouts of 20% of the data, randomly selected. The second OOS method created 33 holdouts of one entire province at a time. These province holdouts were performed to assess the performance of the models in predicting cause fractions in Papua Barat, which has no verbal autopsy data in this dataset. The performance tests conducted on each of these holdouts include the root mean squared error (RMSE) of the estimated cause fractions, bias of the estimated cause fractions, and coverage of the predicted 95% uncertainty interval.

The root mean squared error (RMSE) was calculated using the equation:

$$RMSE = \sqrt{\frac{\sum_{p,s,a}(\widehat{CF}_{p,s,a} - CF_{p,s,a})^2}{N}}$$

Where p is province, s is sex, a is age, $\widehat{CF}$ is the predicted cause fraction, CF is the observed cause fraction in the data, and N is the number of province/age/sex-specific observations in the data.

Mean bias was calculated for each cause of death model using the following equation.

$$\overline{Bias} = \frac{\sum_{p,s,a}(\widehat{CF}_{p,s,a} - CF_{p,s,a})}{N}$$

Where p is province, s is sex, a is age, $\widehat{CF}$ is the predicted cause fraction, CF is the observed cause fraction in the data, and N is the number of province/age/sex-specific observations in the data.

Finally, I calculated coverage of the cause-specific models by creating a simulated verbal autopsy dataset 1consisting of 1000 binomial draws conditional on the model prediction draws and corresponding raw data sample sizes for each prediction.

$$simulated\ VA\ deaths_{p,a,s,i} | CF\ prediction_{p,a,s,i} \sim Binomial(N_{p,a,s}, CF\ prediction_{p,a,s,i})$$

Where p represents province; a represents age; s represents sex; i represents the draw number (from 1 to 1000); $simulated\ VA\ deaths_{p,a,s,i}$ represents the newly created draw i of a simulated verbal autopsy dataset; $CF\ prediction_{p,a,s,i}$ represents the draw i of the cause fraction predictions; and $N_{p,a,s}$ is the sample size observed in the raw data. I then calculated the 95% uncertainty interval of these prediction draws and calculated coverage as the proportion of input verbal autopsy data points that fell within these uncertainty intervals by cause group.

Step 4: Final estimates of cause fractions, mortality rates, deaths, and YLLs

After 1000 draws of the cause fraction estimates were generated from the posterior predicted distributions of the ICAR model described above for each province/age/sex, these cause fractions were proportionally adjusted to sum to 100% at the draw level using the analytical hierarchy (Figure 2). Specifically, Group I, Group II, and Group III were proportionally adjusted to sum to 100%. Simultaneously, the constituents of Other communicable diseases including diarrhea and NTDs, All respiratory diseases, Other non-communicable diseases including cardiovascular diseases, and Neoplasms except lung cancer were adjusted to sum to 100% within each of these parent cause groups. Then those adjusted constituents were readjusted along with all other detailed cause groups to sum to their parent Group I, Group II, and Group III adjusted cause fractions.

The resulting analytical hierarchy was utilized as follows: cause fractions were estimated for each cause group in Level 1, Level 2, and Level 3 (Steps 2 and 3 in Figure 1) and adjusted in a modified top-down approach. Level 3 cause fractions were proportionally "squeezed" to sum to the aggregated Level 2 cause fraction estimates. Then the Level 1 cause fractions were squeezed to sum to 100%, and the adjusted Level 3 cause fractions were squeezed into the adjusted coarse Level 1 cause fractions. This method ensures that the detailed cause group cause fractions sum to 100% overall by province, age, and sex, and that the adjustments utilize cause groupings with the best concordances according to the PHMRC validation datasets by incorporating the Level 2 cause groups. Level 2 cause groups were not squeezed directly into Level 1 cause groups (as in a top-down approach) because the Level 2 cause group "All respiratory diseases" contains detailed causes in both Group I and Group II Level 1 cause groups and so cannot be squeezed as such into either.

Calculate final estimates of cause-specific mortality and YLLs

Cause-specific mortality rates (CSMR) were calculated by multiplying the resultant cause fractions by the all-cause mortality rates estimated in Aim 1, across 1000 posterior draws to propagate uncertainty through analyses, including all sources of sampling error, non-sampling error, and model parameter estimation incorporated into both the all-cause mortality rate and cause fraction draws. CSMR was multiplied by population to obtain the number of deaths by province, age, and sex. Years of life lost due to premature mortality (YLLs) were calculated by multiplying the number of estimated deaths by the reference life expectancy at the average age of death in each age group. This average age of death by age group was obtained from all-cause mortality life table estimates by location and sex in 2014 in Aim 1 using the following equation.

$$AD_{x,l,s} = x + a_{x,l,s}$$

Where $AD_{x,l,s}$ represents the average age at death for the age interval beginning at age x, and $a_{x,l,s}$ is the average number of years lived in that age interval.

The reference life expectancy by age was constructed by GBD from the lowest observed mortality rates in each age in any population of at least five million people (Table 6).(55) The reference life expectancy was linearly interpolated across age to obtain reference life expectancy for the average ages at death using the equation:

$$e_{AD_{x,l,s}} = \frac{e_{x+n_x} - e_x}{(n_x)} * \left(AD_{x,l,s} - x\right) + e_x$$

Where $e_{AD_{x,l,s}}$ is the reference life expectancy at the average age of death within the age group beginning at age x, e_x is the reference life expectancy at age x, and n_x represents the interval length of that age group. YLLs were then calculated using estimated deaths and these life expectancies at average age of death using the equation:

$$YLL_{l,x,s,c} = d_{l,x,s,c} e_{AD_{x,l,s}}$$

Where $d_{l,x,s,c}$ represents the estimated number of deaths due to cause group c in 2014 for age group x and sex s in location l. All quantities were aggregated across province to obtain national-level estimates by cause group, age, and sex for year 2014.

Table 6. GBD 2015 standard life expectancy table

Age	e_{age}
0	86.6
1	85.8
5	81.8
10	76.8
15	71.9
20	66.9
25	62.0
30	57.0
35	52.1
40	47.2
45	42.4
50	37.6
55	32.9
60	28.3
65	23.8
70	19.4
75	15.3
80	11.5
85	8.2
90	5.5
95	3.7
100	2.6
105	1.6
110	1.4

Results

Cause of death models that performed well in terms of logarithmic score, RMSE, and bias both in-sample and out-of-sample were HIV/AIDS, malaria, lung cancer, breast cancer, and all injuries except transport injuries (Table 7). Models with larger LS, RMSE, and bias tended to be the aggregate cause groups with corresponding larger cause fractions: Communicable, maternal, neonatal, and nutritional diseases; All respiratory diseases; Non-communicable diseases; Other NCDs and CVD; Other non-communicable diseases; and Injuries. All neonatal-associated causes of death seem to have performed poorly in terms of bias. In general, causes with high cause fractions like aggregate cause groups have larger values of these validation metrics because of the impact of scale; cause groups with larger cause fractions overall have larger residuals and therefore larger resulting LS, RMSE, and bias values.

Also, the raw data include more values of 100% in provinces or age groups with very small sample sizes, whereas those with lower cause fractions like the injury cause groups rarely contained cause fraction values of 100% (Supplementary Figure 1), which may also contribute to the larger error for more common causes of death. Interestingly, the in-sample and out-of-sample metrics tend to be similar across cause groups, which may be in part due to the large amount of noise in the raw data.

Table 7. Mean logarithmic scores (LS), root mean squared error (RMSE), bias, and coverage by cause group, according to type of cross validation. Cells highlighted in red represent higher RMSE or LS values, or bias or coverage values that diverge from 0 and 0.95, respectively. *LOO=leave-one-out cross validation for N holdouts (where N is the number of observations), IS=in-sample, OOS 20%=average across 10 holdouts of 20% of the data randomly selected, and OOS Prov=average across 33 holdouts of an entire province's data excluded.*

	LS	RMSE			Bias			Coverage		
Cause group	**LOO**	**IS**	**OOS20%**	**OOSProv**	**IS**	**OOS20%**	**OOSProv**	**IS**	**OOS20%**	**OOSProv**
Communicable, maternal, neonatal, and nutritional diseases	2.222	0.186	0.184	0.186	0.0033	0.0030	0.0033	0.937	0.938	0.937
Diarrheal diseases	1.327	0.095	0.097	0.095	-0.0009	-0.0012	-0.0009	0.951	0.951	0.952
HIV/AIDS	0.575	0.042	0.041	0.042	-0.0005	-0.0005	-0.0004	0.928	0.935	0.931
Lower respiratory infections	1.352	0.100	0.098	0.100	-0.0015	-0.0016	-0.0015	0.961	0.959	0.961
Malaria	2.390	0.051	0.055	0.051	-0.0015	-0.0017	-0.0014	0.962	0.967	0.963
Tuberculosis	1.672	0.099	0.100	0.099	0.0027	0.0027	0.0027	0.960	0.961	0.960
Maternal disorders	0.803	0.109	0.100	0.109	0.0000	0.0018	0.0000	0.934	0.945	0.941
Neonatal encephalopathy due to birth asphyxia and trauma	0.940	0.131	0.134	0.131	-0.0048	-0.0049	-0.0048	0.974	0.978	0.978
Preterm birth complications	1.473	0.118	0.116	0.118	0.0052	0.0047	0.0053	0.987	0.985	0.987
Other neonatal disorders	4.384	0.088	0.090	0.089	0.0103	0.0096	0.0100	0.968	0.975	0.969
Communicable diseases other than resp maternal neonatal	1.749	0.146	0.152	0.148	-0.0017	-0.0049	-0.0025	0.957	0.937	0.951
Other communicable diseases	1.539	0.119	0.120	0.119	-0.0007	-0.0006	-0.0007	0.958	0.959	0.958
All respiratory diseases	2.270	0.160	0.160	0.160	0.0001	-0.0002	0.0001	0.937	0.938	0.937
Non-communicable diseases	1.902	0.185	0.183	0.185	0.0014	0.0011	0.0014	0.950	0.949	0.950
Tracheal, bronchus, and lung cancer	0.982	0.033	0.034	0.033	0.0006	0.0005	0.0006	0.959	0.960	0.958
Breast cancer	1.046	0.031	0.032	0.031	0.0036	0.0037	0.0036	0.938	0.940	0.940
Cervical cancer	2.333	0.073	0.076	0.073	-0.0029	-0.0032	-0.0029	0.974	0.970	0.973
Other malignant neoplasms	1.575	0.074	0.075	0.074	0.0030	0.0028	0.0030	0.971	0.972	0.971
Neoplasms	1.646	0.091	0.093	0.091	0.0044	0.0046	0.0045	0.961	0.960	0.959
Cerebrovascular disease	1.593	0.115	0.116	0.115	0.0002	-0.0002	0.0002	0.965	0.965	0.964
Ischemic heart disease	2.141	0.094	0.095	0.094	0.0014	0.0028	0.0017	0.978	0.982	0.982
Other cardiovascular and circulatory diseases	1.447	0.055	0.053	0.055	-0.0005	-0.0004	-0.0005	0.956	0.957	0.958
Diabetes mellitus	2.602	0.081	0.080	0.081	0.0012	0.0014	0.0012	0.955	0.958	0.953
Chronic kidney disease	1.307	0.054	0.054	0.054	0.0015	0.0015	0.0015	0.973	0.973	0.972
Cirrhosis	1.414	0.088	0.090	0.088	-0.0008	-0.0012	-0.0008	0.965	0.967	0.966
Chronic respiratory diseases	1.531	0.074	0.073	0.074	0.0013	0.0016	0.0012	0.965	0.968	0.966
Other NCDs and CVD	2.151	0.179	0.178	0.179	-0.0015	-0.0019	-0.0015	0.947	0.953	0.951
Other non-communicable diseases	1.874	0.131	0.129	0.131	-0.0022	-0.0017	-0.0022	0.966	0.965	0.965
Injuries	1.758	0.142	0.142	0.142	0.0002	-0.0002	0.0002	0.963	0.961	0.963
Drowning	0.584	0.054	0.056	0.054	0.0017	0.0013	0.0017	0.977	0.973	0.976
Falls	0.866	0.056	0.056	0.056	-0.0013	-0.0015	-0.0013	0.966	0.965	0.967
Interpersonal violence	0.382	0.045	0.045	0.045	-0.0016	-0.0014	-0.0015	0.957	0.958	0.959
Self-harm	0.555	0.040	0.039	0.040	0.0004	0.0006	0.0004	0.960	0.957	0.954
Transport injuries	1.259	0.094	0.095	0.094	0.0032	0.0030	0.0032	0.977	0.976	0.976
Other unintentional injuries	1.034	0.071	0.072	0.071	-0.0016	-0.0016	-0.0016	0.970	0.969	0.969

In Indonesia, the top three causes of deaths and YLLs in 2014 were the same for males and females: cerebrovascular disease, ischemic heart disease, and other noncommunicable diseases (Table 8, Supplementary Tables 6 and 7). According to the ICD-10 codes of the redistributed data mapped to my cause list, the cause group "other noncommunicable diseases" was dominated by digestive diseases (60.7% of deaths in this cause group for males and 61.8% for females), along with urinary diseases,

congenital defects, hemoglobinopathies, endocrine disorders, and neurological disorders (Supplementary Table 8).

Also highly ranked for both sexes in terms of deaths were tuberculosis, chronic respiratory diseases, diabetes, and diarrhea (Table 8). Transportation injuries were the fifth highest cause of YLLs for males, whereas that cause group was ranked the 14th out of 28 cause of YLLs for females. The cause-specific mortality rate (CSMR) due to injuries overall was over twice as high among males as among females: 55 per 100,000 for males and 21 per 100,000 for females (Supplementary Table 6).

Table 8. Rankings of causes of death and YLLs by sex for Indonesia. (blue=noncommunicable diseases, red=communicable/maternal/neonatal/nutritional disorders, green=injuries)

Rank	Male Deaths	Male YLLs	Female Deaths	Female YLLs
1	Cerebrovascular disease	Cerebrovascular disease	Cerebrovascular disease	Cerebrovascular disease
2	Ischemic heart disease	Ischemic heart disease	Ischemic heart disease	Other noncommunicable diseases
3	Other noncommunicable diseases	Other noncommunicable diseases	Other noncommunicable diseases	Ischemic heart disease
4	Tuberculosis	Tuberculosis	Diabetes	Diabetes
5	Chronic respiratory diseases	Transport injuries	Diarrhea	Other communicable diseases
6	Other communicable diseases	Other communicable diseases	Tuberculosis	Tuberculosis
7	Diabetes	Chronic respiratory diseases	Chronic respiratory diseases	Neonatal preterm birth
8	Transport injuries	Other malignant neoplasms	Other cardiovascular diseases	Diarrhea
9	Diarrhea	Lower respiratory infections	Other communicable diseases	Lower respiratory infections
10	Other malignant neoplasms	Neonatal encephalopathy due to birth asphyxia and trauma	Lower respiratory infections	Other malignant neoplasms

The top four causes of death in 2014 for male children younger than 5 years in Indonesia, accounting for over 59% of deaths, were neonatal encephalopathy, preterm birth complications, other communicable diseases, and lower respiratory infections. For female children younger than 5, the top four causes of death, accounting for 62% of deaths, were preterm birth complications, other non-communicable diseases, diarrheal diseases, and other communicable diseases. Cause-specific mortality rates were over 5% higher in males in this age group for almost all causes with at least 500 deaths observed nationally, except diarrhea (the only communicable disease higher in females) by 12.7% and other non-communicable diseases by 3.7%.

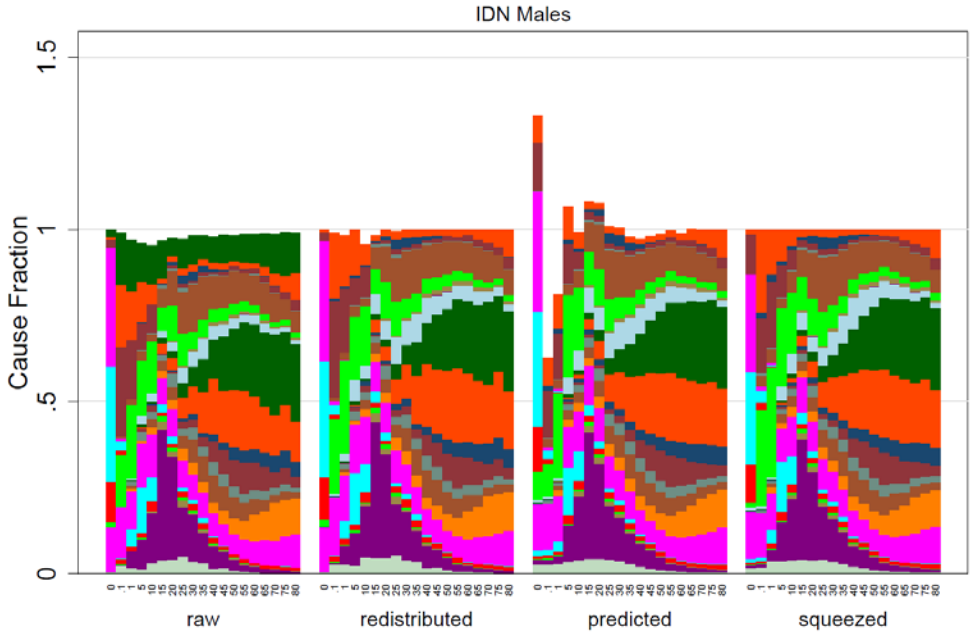
Deaths due to chronic kidney disease occurred at similar rates among males and females (<3% difference). Communicable, neonatal, and nutritional diseases were 20.1% higher in males overall, led by other neonatal disorders (46.9% higher in males), neonatal encephalopathy (43.1% higher in males), and lower respiratory infections (36.8% higher in males). For noncommunicable diseases, cancer mortality rates were 10.2% higher in males than females. Mortality rates due to injury were a 2.7 times (170%) higher in males than females, especially for drowning and transportation deaths: both 3.2 times (220%) higher in males less than 5 years old.

For children aged 5-14 years old, the top cause of death among males and females was other communicable diseases—which includes causes such as intestinal infections, meningitis, dengue, nutritional deficiencies, sexually transmitted diseases, and hepatitis—accounting for just over one of five deaths among males and females in this age group (Figure 3, Supplementary Table 11). Other top causes included transport injuries, other non-communicable diseases, and lower respiratory diseases for both sexes, in addition to drowning for males, and diarrhea for females. Female CSMR was higher for cardiovascular disease and diabetes, the latter of which was 48% higher in females but accounted for fewer than 200 deaths nationwide for both sexes in this age group. Males and females had less than 5% difference between male and female mortality rates for diarrhea, chronic kidney disease, neoplasms, and other non-communicable diseases. All other causes had higher mortality rates among males. Mortality rates due to communicable, maternal, and nutritional diseases were 32.5% higher among males, with both lower respiratory infections and other communicable diseases over 40% higher in males than females. For noncommunicable diseases, cirrhosis CSMR was almost twice as high among males as females, and the mortality rate due to chronic respiratory diseases was 2.3 times (130%) higher for males. As with children under five, injuries again were a much bigger cause of death among males in this age group, with rates 2.5 times (150%) higher than observed in females and responsible for one in three male deaths (and one in five female deaths). Drowning rates were 2.6 times higher in males, and transportation rates were 2.7 times higher in males.

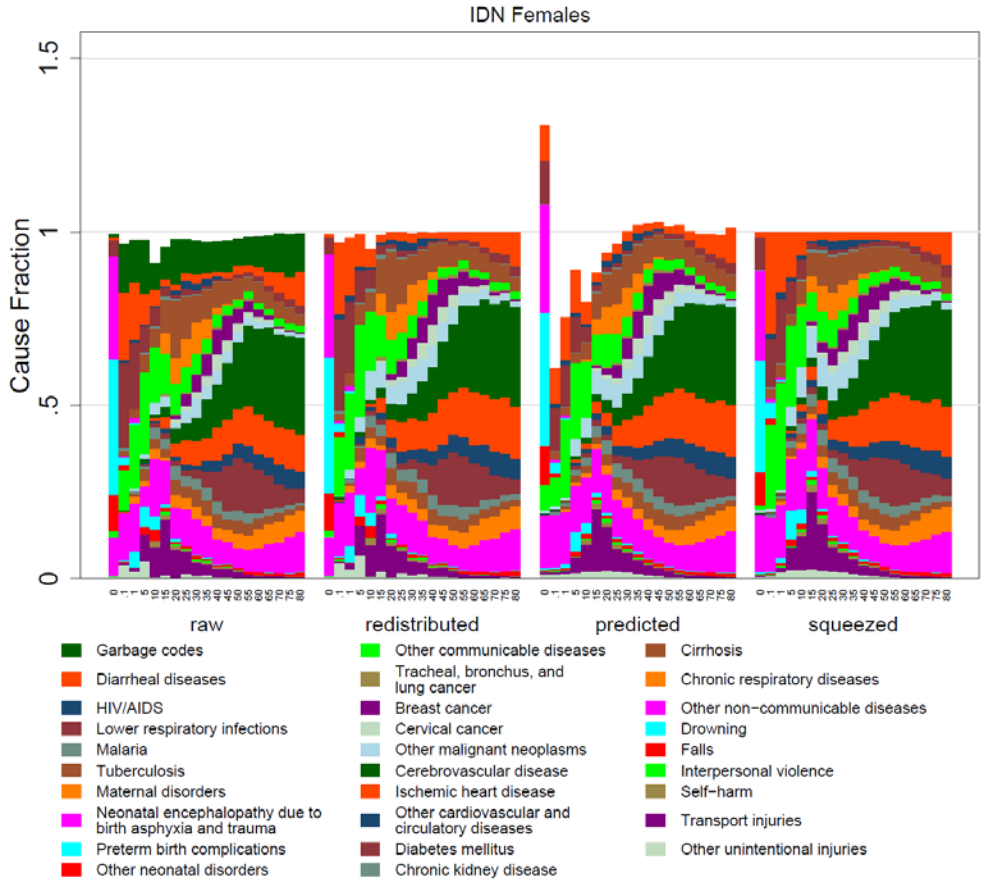
Among adults 15-49 years old, stroke, ischemic heart disease, other non-communicable diseases, and tuberculosis were among the top five causes of death for both males and females. The number one cause of death in males was transport injuries, responsible for 12.4% of male deaths in 2014. If all cardiovascular diseases were aggregated into one cause group, they accounted for 24% of both male and female deaths in this age group. Notable among females, breast cancer and cervical cancer combined account for 11,432 deaths (9.6% of all female deaths), and maternal conditions claimed 4,504 lives. This age group observed a substantial shift from mortality being dominated by communicable diseases in ages under 5, to being dominated by noncommunicable diseases (57.0% of deaths for males, 67.6% for females) in ages 15-49. As with the younger age groups, injuries were a major cause of death among males, accounting for 36,761 male deaths in this age group, resulting from mortality rates approximately three times higher than those for females.

Figure 3. Cause distribution for Indonesia 2014 by age for a) Males and b) Females.
"Raw" = Raw data grouped into cause groups, including garbage codes. "Redistributed" = data after garbage codes were redistributed to cause groups. "Predicted" = initial predictions from cause fraction models. "Squeezed" = final cause fraction predictions after squeezing estimates to 100%.

a)



b)



The shift toward noncommunicable diseases as the dominant cause of death continued in age groups 50-69 and 70 and older, accounting for 80.1% of male and 83.5% of female deaths for ages 50-69, and 78.3% of male and 80.1% of female deaths for ages 70 and older. The oldest ages experienced a slight increase in mortality with age due to communicable diseases because of approximately hundred-fold increases in diarrhea mortality rates from 4.8/100,000 in males and 3.9/100,000 in females for ages 15-49, to 429.9/100,000 in males and 502.3/100,000 in females for ages 70 and older. Transport injuries and falls caused the most deaths among injury deaths for both males and females for ages 50 and older. Transport injuries were responsible for 14% of male deaths age 5-49 in Indonesia in 2014.

At the subnational level, cerebrovascular disease and ischemic heart disease (IHD) were among the top three causes of YLLs for 23 of the 34 provinces (Figure 4), with the high age-standardized CSMR for cerebrovascular disease found in Maluku Utara, Gorontalo, and Sulawesi Barat, and for ischemic heart disease in Kalimantan Timur, Banten, and Sumatera Selatan (Figure 4, Supplementary Figures 2 and 3). Cerebrovascular disease age-standardized CSMR was higher for females than males in twenty provinces, and IHD in only five (Supplementary Table 9). The most pronounced sex differences for cerebrovascular disease were found in Maluku Utara, Gorontalo, and Papua Barat, where female age-standardized CSMR was over twice the rate for males. For IHD, male age-standardized CSMR was over twice the rate for females in Jawa Timur and Sumatera Utara. The lowest cerebrovascular disease CSMR for males and females were observed in DKI Jakarta, Sulawesi Selatan, Jawa Barat, and Kalimantan Utara, whereas the lowest IHD CSMR were found in Lampung, Kalimantan Utara, Papua, and DI Yogyakarta. DKI Jakarta had the sixth highest CSMR for IHD among males and the lowest male CSMR for cerebrovascular disease in the country.

In fact, Kalimantan Utara had low CSMR for many causes in large part due to low all-cause mortality rates estimated in Aim 1 (Aim 1 Appendix Tables 13 and 16) resulting from low observed adult mortality in its sibling survival data. It is unclear without further data collection for both all-cause mortality and causes of death how accurate the notable differences were between Kalimantan Utara and its neighbor and former province-partner (they were combined as one province until 2013) Kalimantan Timur.

HIV/AIDS and drowning were ranked fifth and eighth, respectively, in terms of YLLs for Papua, but they were much lower in the rankings elsewhere (Figure 4). Transport injuries was the second highest cause of YLLs in Papua, the highest ranking for that cause across provinces. Diarrheal diseases, ranked thirteenth for Indonesia, entered the top seven causes for Jawa Tengah and Gorontalo. Tuberculosis was the top cause of YLLs for Nusa Tenggara Timur, Kalimantan Selatan, Maluku, and Papua. Neonatal disorders—preterm birth complications and neonatal encephalopathy—were highly ranked in Riau and Bangka Belitung. The capital DKI Jakarta saw preterm birth complications and diabetes in the top five causes of YLLs, and falls were more highly ranked in DKI Jakarta than any other province. Maternal conditions were notably more highly ranked in Kepulauan Riau than any other province: 16th compared to 25th at the national level.

Figure 4. Ranking of leading causes of years of life lost (YLLs) by province. Causes in the figure are ordered according to the national ranking. Ranks are color shaded to indicate relative rank and highlight differences across provinces.

Cause group	Indonesia	Aceh	Sumatera Utara	Sumatera Barat	Riau	Jambi	Sumatera Selatan	Bengkulu	Lampung	Bangka Belitung	Kepulauan Riau	DKI Jakarta	Jawa Barat	Jawa Tengah	DI Yogyakarta	Jawa Timur	Banten	Bali	Nusa Tenggara Barat	Nusa Tenggara Timur	Kalimantan Barat	Kalimantan Tengah	Kalimantan Selatan	Kalimantan Timur	Kalimantan Utara	Sulawesi Utara	Sulawesi Tengah	Sulawesi Selatan	Sulawesi Tenggara	Gorontalo	Sulawesi Barat	Maluku	Maluku Utara	Papua Barat	Papua
Cerebrovascular disease	1	1	1	2	2	1	2	1	1	1	1	3	2	1	1	1	2	1	1	2	1	1	2	1	1	1	2	3	1	1	1	2	1	2	3
Ischemic heart disease	2	3	3	3	3	3	1	3	3	3	3	1	1	3	3	2	1	2	4	8	3	2	4	2	4	3	5	1	4	6	8	4	4	8	11
Other non-communicable diseases	3	2	2	1	1	2	3	2	2	2	2	2	4	2	2	3	4	3	2	4	2	3	3	3	2	2	1	2	2	3	2	3	2	1	4
Tuberculosis	4	4	7	7	10	4	4	7	5	9	8	6	3	4	10	5	3	11	3	1	4	6	1	7	11	8	6	5	3	2	7	1	3	4	1
Other communicable diseases	5	9	6	5	5	6	6	4	4	8	4	8	5	5	11	7	5	9	6	5	5	4	6	9	5	10	4	4	7	5	5	5	7	3	6
Transport injuries	6	6	4	6	7	5	5	6	7	5	5	7	6	14	4	8	6	14	8	11	6	7	8	5	7	5	10	6	9	11	9	12	5	6	2
Diabetes mellitus	7	8	5	11	11	11	11	5	9	7	7	5	7	10	9	4	9	8	13	15	13	5	9	4	6	4	13	11	6	9	10	9	8	10	14
Chronic respiratory diseases	8	7	9	4	13	7	13	8	11	13	17	13	8	11	7	9	15	6	9	7	10	13	15	10	16	7	8	8	12	7	6	6	6	13	12
Preterm birth complications	9	17	12	10	4	12	9	10	8	4	6	4	13	6	13	11	8	5	5	13	14	10	10	14	3	11	9	13	13	14	15	24	22	9	23
Other malignant neoplasms	10	12	14	12	12	13	14	13	15	12	10	9	12	8	6	6	12	4	11	6	8	14	12	6	8	6	14	10	15	13	11	10	9	12	9
Lower respiratory infections	11	5	11	8	8	9	10	14	10	10	11	10	9	15	16	14	10	7	7	3	7	9	7	13	12	17	3	7	8	8	4	7	12	5	7
Neonatal encephalopathy due to birth asp	12	16	8	9	6	14	7	11	6	6	9	11	10	9	15	16	7	10	10	9	11	8	5	19	14	16	7	9	5	10	3	15	16	7	10
Diarrheal diseases	13	10	10	15	9	8	12	9	12	17	15	15	15	7	8	12	13	12	14	14	15	12	14	16	13	18	11	12	11	4	13	8	11	14	15
Cirrhosis	14	13	13	14	16	15	15	16	13	15	13	12	14	13	12	10	11	13	12	10	12	11	13	11	15	14	16	15	14	12	16	13	10	15	13
Other cardiovascular and circulatory disea	15	15	15	13	18	10	8	15	14	11	14	17	11	12	5	13	14	15	15	19	9	18	11	8	18	12	15	16	16	19	14	11	14	17	17
Chronic kidney disease	16	11	16	16	17	16	17	12	16	16	12	16	16	16	14	15	16	16	18	17	16	17	18	12	10	9	18	17	18	16	19	14	13	21	20
Other unintentional injuries	17	14	18	17	14	17	16	17	17	14	23	24	17	17	20	17	22	17	16	16	18	15	17	15	9	19	17	19	17	20	17	19	17	16	19
Other neonatal disorders	18	26	17	18	15	19	19	20	18	18	19	18	20	21	22	24	17	23	17	12	19	16	16	18	17	13	12	14	10	15	12	17	20	11	22
Breast cancer	19	18	21	19	20	20	20	19	20	23	20	19	21	19	19	18	18	24	19	21	21	20	21	17	24	15	20	20	20	18	20	16	15	19	24
Drowning	20	19	19	20	19	18	18	21	19	19	25	25	18	20	23	23	21	25	21	18	17	19	19	24	21	21	19	18	19	17	18	18	19	20	8
Falls	21	22	20	21	22	21	21	23	21	20	22	14	19	18	17	20	20	19	20	20	22	22	22	21	20	24	22	21	21	24	23	23	21	26	16
HIV/AIDS	22	27	28	27	26	28	28	28	24	27	18	20	25	23	25	19	19	20	22	25	26	26	28	23	25	26	26	26	22	26	27	21	26	18	5
Cervical cancer	23	21	26	24	24	23	22	25	26	25	24	23	23	22	21	22	25	18	27	23	24	25	23	20	23	20	24	24	23	23	22	22	24	22	26
Tracheal, bronchus, and lung cancer	24	24	25	23	23	25	23	22	23	26	21	21	24	24	18	21	24	21	28	28	23	27	25	22	19	22	28	23	28	28	28	28	28	28	28
Maternal disorders	25	20	24	26	21	24	25	24	25	21	16	22	22	25	26	26	23	27	23	24	20	21	24	25	26	28	21	28	27	22	21	26	25	23	18
Self-harm	26	23	23	22	27	27	24	26	22	28	27	27	26	26	24	25	26	22	25	26	28	28	27	27	27	23	27	22	26	25	24	25	23	27	27
Interpersonal violence	27	25	27	25	25	26	26	27	28	24	28	26	27	27	27	27	27	26	26	27	27	24	26	26	22	25	23	25	25	27	25	27	27	25	25
Malaria	28	28	22	28	28	22	27	18	27	22	26	28	28	28	28	28	28	28	24	22	25	23	20	28	28	27	25	27	24	21	26	20	18	24	21

* Neonatal encephalopathy due to birth asphyxia and trauma

** Other cardiovascular and circulatory diseases

Female breast cancer age-standardized CSMR was greater than 30 per 100,000 in Maluku Utara, Maluku, Sulawesi Utara, and Papua Barat, and rates were lowest below 10 per 100,000 in Sumatera Utara, Lampung, Jawa Barat, and Kalimantan Utara. For cervical cancer, rates were highest (>18 per 100,000) in Papua Barat, Sulawesi Barat, and Sulawesi Utara, and lowest (<5 per 100,000) in Sulawesi Selatan, Sumatera Barat, Jawa Barat, and Lampung. Other malignant neoplasms, ranked tenth overall for YLLs in Indonesia, had large sex discrepancies in Kalimantan Utara—with male age-standardized CSMR three times as high as that for females—and Sumatera Utara, Jawa Timur, and Kalimantan Timur—with male rates approximately twice as high as female rates. Female age-standardized CSMR for other malignant neoplasms was over 15% higher than male rates in four provinces (Gorontalo, Maluku Utara, Sulawesi Barat, and Papua Barat), whereas male rates were at least 15% higher than female rates in 23 provinces.

Figure 5 displays province-level CSMR by age and sex with color-shaded rows to highlight differences across province and cause. Each row is shaded from green (relatively low CSMR) to red (relatively high CSMR) independent of other rows to facilitate identifying how provinces perform compared to the national CSMR level for each age/sex group by cause. For example, with diarrheal diseases, Sumatera Utara had higher male CSMR and similar female CSMR compared to levels for Indonesia, and its

neighbor Sumatera Barat had similar male CSMR and lower female CSMR compared to national levels. Also, the high CSMR in Sulawesi Utara for breast cancer, cervical cancer, and other malignant neoplasms were a striking red block across age and sex. Many observations could be made from these results, but I focus here on some of the most prominent. Papua performed poorly for HIV/AIDS, malaria, tuberculosis, maternal disorders, and all injuries other than self-harm and other unintentional injuries; however, it experienced lower CSMR than Indonesia for chronic kidney disease, lung cancer, ischemic heart disease, neonatal disorders, and diarrheal diseases. The lowest rates of neonatal disorders were observed in Aceh for both males and females. Interestingly, Aceh had the highest or second highest CSMR due to lower respiratory infections (LRI) for every aggregated age group over 5 and was among the lowest LRI CSMR for children under 5.

Interesting observations can be made for geographical neighbors here. All provinces on Java and neighbor Bali experienced low rates of malaria compared to most other provinces. Sumatera Selatan and Bengkulu had opposite patterns of cardiovascular diseases although they are land-adjacent neighbors; the former had low rates for stroke and high for IHD and other cardiovascular/circulatory diseases, and Bengkulu had high rates for stroke and rates for IHD and other cardiovascular/circulatory diseases more similar to Indonesia. In addition to Sulawesi Utara, high CSMR for other malignant neoplasms was found in the neighboring islands of Bali, Nusa Tenggara Barat, and Nusa Tenggara Timur, especially among children. Overall, CSMR due to injuries was high in Maluku, Papua, and Papua Barat, along with especially high CSMR for self-harm in Bali, transport injuries in Yogyakarta, and other unintentional injuries in Bangka Belitung across age and sex.

Figure 5. Heatmap of cause-specific mortality rates per 100,000 by province for age groups 0-4, 5-14, 15-49, 50-69, and 70+. Causes in the figure are ordered according to cause groups. Each row is color shaded to highlight differences in CSMR across provinces. For example, if a column contains many red-shaded cells, then that province has higher CSMR than the CSMR in many provinces for many cause groups (such as Papua for HIV/AIDS, tuberculosis, and several injuries).

Cause group		Age	Indonesia	Aceh	Sumatera Utara	Sumatera Barat	Riau	Jambi	Sumatera Selatan	Bengkulu	Lampung	Bangka Belitung	Kepulauan Riau	DKI Jakarta	Jawa Barat	Jawa Tengah	DI Yogyakarta	Jawa Timur	Banten	Bali	Nusa Tenggara Barat	Nusa Tenggara Timur	Kalimantan Barat	Kalimantan Tengah	Kalimantan Selatan	Kalimantan Timur	Kalimantan Utara	Sulawesi Utara	Sulawesi Tengah	Sulawesi Selatan	Sulawesi Tenggara	Gorontalo	Sulawesi Barat	Maluku	Maluku Utara	Papua Barat	Papua
Communicable/maternal/NN/nutritional diseases*	Male	0-4	548.5	208.4	590.2	709.0	763.6	463.6	686.8	503.7	454.9	821.8	422.0	659.6	353.0	639.9	440.6	394.8	615.2	642.5	983.9	697.0	394.1	546.5	893.0	211.6	347.2	593.9	1,187.0	755.5	758.4	783.0	1,079.4	470.6	252.1	899.4	534.3
	Male	5-14	62.4	71.2	72.0	67.2	42.2	60.4	49.9	64.2	54.9	53.5	47.2	47.3	54.2	53.6	42.5	59.0	94.8	43.4	81.9	87.1	68.3	61.2	121.7	60.5	33.1	40.8	90.2	69.1	76.2	75.8	66.2	116.3	62.5	63.5	129.8
	Male	15-49	31.8	33.6	30.8	29.9	22.6	29.9	26.1	28.4	27.8	23.9	29.0	23.2	36.0	26.6	16.8	24.3	35.6	22.2	41.3	51.9	33.1	29.8	42.8	24.4	21.1	20.7	48.5	32.4	47.9	57.1	39.0	56.0	46.5	45.8	63.8
	Male	50-69	223.3	269.5	247.0	241.2	148.3	222.6	181.4	236.3	201.2	181.1	163.2	156.7	191.0	199.8	175.2	220.4	332.6	172.3	316.8	340.6	276.6	226.2	430.1	187.9	120.7	139.4	307.3	288.6	270.1	277.8	247.8	418.1	222.2	215.6	424.4
	Male	70+	1,316.6	1,633.5	1,507.7	1,433.8	965.2	1,393.8	1,114.4	1,524.9	1,272.5	1,109.0	998.3	898.9	1,166.3	1,187.5	1,123.4	1,301.5	1,912.7	1,065.6	1,757.0	1,867.2	1,551.3	1,421.1	2,356.5	1,148.9	770.8	844.1	1,764.3	1,715.9	1,694.0	1,606.8	1,484.3	2,276.3	1,376.7	1,273.9	2,259.8
	Female	0-4	454.5	170.3	504.6	593.0	645.4	392.9	577.8	416.2	381.5	700.2	354.5	547.8	289.8	523.1	367.7	320.0	508.0	531.8	811.6	580.6	334.2	456.6	758.6	179.4	280.1	488.3	987.7	616.2	609.5	668.4	879.0	397.9	212.2	776.6	483.5
	Female	5-14	38.1	56.9	31.5	33.3	28.0	47.4	27.9	59.4	33.7	39.5	53.7	25.3	30.9	31.4	29.6	25.4	51.7	30.1	52.4	60.1	56.3	48.8	73.4	38.5	17.1	33.2	67.1	40.6	54.9	125.9	78.2	114.0	114.5	100.5	122.8
	Female	15-49	24.0	26.7	22.4	21.8	17.3	23.7	19.7	23.6	20.8	19.1	24.4	17.2	25.4	19.2	12.9	18.3	26.2	16.9	31.4	39.0	25.3	23.7	33.1	19.2	14.8	16.5	39.6	24.8	38.5	52.5	34.6	49.4	45.0	42.9	59.0
	Female	50-69	164.0	257.4	141.1	153.0	125.1	209.9	129.0	250.3	158.8	157.7	231.1	110.9	139.8	146.5	137.8	122.7	236.9	145.4	253.2	283.1	257.7	214.6	327.6	149.2	84.1	126.9	282.5	207.1	251.6	490.2	330.8	463.3	430.3	370.7	445.0
	Female	70+	1,170.4	1,680.6	1,159.4	1,220.8	1,026.0	1,539.9	1,032.4	1,788.1	1,223.8	1,154.2	1,499.3	904.2	1,064.3	1,045.6	1,072.8	936.5	1,606.1	1,086.8	1,720.5	1,899.8	1,728.6	1,572.9	2,151.2	1,067.8	697.6	893.3	1,874.8	1,553.3	1,831.7	2,759.0	2,091.4	2,746.9	2,485.0	2,251.1	2,672.3
Diarrheal diseases	Male	0-4	76.4	32.0	113.8	72.3	121.5	86.2	118.3	73.2	65.9	106.1	52.3	84.4	44.2	97.3	73.9	56.6	68.4	58.6	122.7	78.6	44.0	74.2	111.2	19.5	38.7	70.2	173.3	91.0	105.7	180.2	136.5	84.1	33.6	112.3	52.2
	Male	5-14	4.82	6.17	8.18	4.05	4.00	5.81	4.29	6.10	5.17	3.57	2.71	2.75	3.18	5.72	6.93	5.27	5.90	3.18	4.31	4.15	3.85	4.37	8.14	3.74	2.85	3.27	6.81	4.41	5.44	8.74	3.53	7.35	3.92	3.42	3.36
	Male	15-49	5.39	6.81	7.38	3.85	4.39	6.50	5.13	6.01	5.39	3.98	4.25	3.42	5.03	5.51	4.69	4.35	5.33	3.44	6.11	7.19	4.45	4.71	7.46	3.50	3.13	3.64	8.69	4.61	9.27	14.69	5.38	10.61	9.17	7.20	5.75
	Male	50-69	32.0	36.4	44.0	30.0	25.3	36.3	25.5	43.1	34.1	23.3	22.3	14.6	20.4	37.3	48.2	37.2	36.1	29.3	33.6	30.1	30.0	30.3	44.6	22.9	24.4	19.3	38.1	35.0	32.2	40.9	25.7	39.1	24.2	21.1	14.3
	Male	70+	429.9	455.7	571.8	442.3	349.2	490.2	342.7	608.1	457.1	350.7	307.5	186.4	307.1	459.7	558.0	462.8	486.8	389.3	451.7	445.6	400.7	455.2	592.2	345.8	313.3	269.7	516.0	521.5	496.4	473.2	400.1	506.3	392.2	308.6	214.7
	Female	0-4	85.7	36.5	128.5	88.2	142.5	91.2	139.7	89.4	78.2	124.8	51.9	92.1	47.7	108.6	73.7	57.8	75.4	80.7	137.6	91.7	50.5	79.0	127.1	24.5	40.6	81.8	205.4	101.7	116.4	201.8	155.7	98.6	43.1	131.2	69.0
	Female	5-14	3.85	6.28	4.48	2.79	2.65	6.26	3.23	7.78	4.34	2.71	3.18	1.89	2.46	4.39	6.38	3.13	4.51	3.09	3.87	3.89	4.06	4.41	7.10	3.12	1.73	3.60	6.39	3.63	5.79	17.05	5.29	10.63	10.89	8.02	4.92
	Female	15-49	5.34	6.99	6.86	3.73	4.41	6.60	5.05	6.32	5.25	4.22	4.71	3.39	4.79	5.10	4.48	4.35	5.28	3.48	6.15	7.14	4.61	4.98	7.57	3.72	2.87	3.78	9.15	4.69	9.61	16.43	6.23	11.84	11.02	8.62	7.30
	Female	50-69	36.5	56.4	39.2	29.7	32.1	55.5	29.8	73.9	42.2	33.4	44.9	16.7	22.5	42.3	53.8	32.2	43.5	35.3	49.2	42.6	46.2	45.9	60.3	29.4	22.1	24.7	55.5	40.5	54.4	98.7	57.8	68.8	81.4	59.8	26.8
	Female	70+	502.3	638.6	570.5	501.2	477.9	708.1	447.3	943.4	576.6	506.0	576.3	287.9	361.9	524.4	639.5	444.0	586.5	479.4	662.7	611.4	627.6	674.8	780.2	421.0	334.5	346.0	705.4	612.0	766.9	1,037.2	760.5	786.2	946.7	706.3	364.0
HIV/AIDS	Male	0-4	6.35	0.93	2.99	3.10	4.65	2.04	3.31	1.50	2.55	5.96	11.14	11.57	2.53	6.24	3.14	8.24	9.06	9.82	15.69	7.05	2.48	3.62	2.47	1.63	3.58	3.83	7.20	4.08	9.06	4.02	9.62	7.30	2.23	22.77	71.27
	Male	5-14	4.79	3.94	3.10	2.42	2.13	1.76	1.38	1.65	2.87	3.25	8.80	6.66	1.91	3.32	3.24	8.54	8.03	9.44	5.80	4.29	3.06	3.31	2.66	6.57	2.53	2.39	4.84	2.84	5.41	1.22	3.75	6.94	3.33	7.80	37.40
	Male	15-49	0.89	0.42	0.35	0.31	0.31	0.26	0.25	0.21	0.41	0.41	1.70	1.01	0.53	0.61	0.37	1.27	1.29	1.35	1.32	1.06	0.52	0.42	0.31	0.63	0.49	0.35	0.66	0.41	1.33	0.42	0.70	1.37	0.94	2.24	12.00
	Male	50-69	1.41	1.00	0.78	0.60	0.51	0.44	0.33	0.41	0.74	0.79	2.81	1.72	0.47	0.92	1.00	2.81	2.24	3.16	1.66	1.13	0.85	0.90	0.65	1.60	0.67	0.60	1.09	0.76	1.37	0.29	0.94	1.98	0.91	2.55	16.04
	Male	70+	4.05	2.71	2.07	1.61	1.53	1.29	1.03	1.14	2.11	2.23	8.64	5.37	1.57	2.76	2.95	8.45	6.86	8.82	4.84	3.39	2.41	2.59	1.76	4.13	1.89	1.79	2.85	2.24	4.45	0.89	2.67	5.81	2.80	7.37	44.35
	Female	0-4	5.04	0.82	2.35	2.58	3.81	1.60	2.90	1.37	2.16	5.13	7.53	8.51	1.86	4.97	2.28	5.93	6.91	10.64	12.91	5.94	2.03	2.77	2.27	1.44	2.69	3.12	6.65	3.13	7.44	3.40	8.40	6.28	2.02	20.72	61.02
	Female	5-14	2.50	2.68	1.07	1.04	0.78	1.24	0.67	1.29	1.55	1.33	5.58	2.72	0.95	1.65	2.12	3.16	3.91	5.55	3.17	2.59	1.94	2.08	1.54	3.36	0.89	1.84	3.00	1.55	3.85	1.86	3.45	7.06	6.11	11.55	31.85
	Female	15-49	0.63	0.31	0.23	0.21	0.21	0.19	0.17	0.16	0.28	0.31	1.27	0.66	0.34	0.40	0.26	0.85	0.86	0.96	0.92	0.75	0.36	0.31	0.23	0.46	0.33	0.27	0.52	0.29	1.00	0.36	0.61	1.14	0.85	2.02	10.38
	Female	50-69	1.09	1.07	0.47	0.41	0.50	0.47	0.27	0.47	0.63	0.77	4.50	1.48	0.38	0.73	0.84	1.68	1.90	2.79	1.61	1.11	0.94	0.93	0.54	1.41	0.50	0.63	1.13	0.60	1.56	0.59	1.42	2.64	2.03	5.23	20.90
	Female	70+	3.41	2.72	1.52	1.34	1.61	1.39	0.97	1.25	1.97	2.17	13.13	5.60	1.46	2.35	2.50	5.81	6.11	8.28	4.97	3.60	2.68	2.76	1.59	3.67	1.66	1.94	3.01	2.00	4.93	1.66	3.74	7.60	5.04	13.62	58.29
Lower respiratory infections	Male	0-4	99.4	45.3	100.3	141.8	128.7	92.4	124.2	79.0	76.8	125.4	60.1	114.6	67.6	103.7	59.2	59.0	104.1	113.0	206.6	167.3	79.9	85.7	173.2	33.3	42.6	86.3	270.9	156.6	138.3	159.2	248.4	98.3	46.7	192.8	116.1
	Male	5-14	3.77	10.95	5.30	5.02	2.90	4.24	2.78	3.90	4.62	3.65	1.94	2.76	2.57	2.63	2.21	3.05	3.84	3.50	4.14	5.83	4.94	4.44	8.65	5.47	1.42	2.85	10.82	6.11	4.23	3.13	6.39	6.68	3.27	3.31	4.19
	Male	15-49	5.98	9.77	5.64	6.07	4.21	5.94	4.76	4.64	5.50	4.28	4.34	4.84	6.64	4.10	2.72	3.78	5.75	4.92	8.10	11.94	7.03	5.20	9.26	5.60	2.64	3.94	13.06	7.43	9.69	8.98	10.02	11.57	8.84	9.24	10.04
	Male	50-69	23.9	65.3	27.5	27.3	17.2	26.0	15.9	24.6	29.4	21.1	15.9	14.6	14.7	18.3	18.7	24.7	26.2	34.4	34.3	36.7	36.1	26.8	47.4	30.6	9.2	14.1	56.1	41.4	27.9	15.8	38.5	37.5	19.7	20.8	23.0
	Male	70+	194.8	505.0	227.2	201.9	157.3	218.0	137.5	205.5	239.0	171.6	146.5	125.2	130.8	149.1	166.5	195.3	238.6	270.4	264.2	294.8	275.6	218.0	379.5	264.9	79.0	114.1	423.1	338.4	277.9	128.6	302.3	300.0	182.6	178.3	186.0
	Female	0-4	72.6	34.3	74.5	105.3	95.7	68.3	94.4	58.3	57.3	95.3	42.1	81.3	47.8	73.0	41.3	40.3	74.2	85.7	153.6	127.2	60.0	62.4	135.6	25.5	29.2	63.0	215.1	114.1	101.3	121.4	192.9	76.3	35.6	152.3	95.3
	Female	5-14	1.96	6.86	1.81	1.96	1.20	2.88	1.30	2.91	2.35	1.63	1.40	1.25	1.29	1.31	1.44	1.14	1.84	2.31	2.35	3.33	3.29	2.65	4.76	2.78	0.54	1.83	6.37	3.09	2.82	3.65	5.67	5.92	5.51	4.88	3.78
	Female	15-49	4.14	7.14	3.75	4.15	2.91	4.34	3.29	3.55	3.76	3.15	3.24	3.20	4.25	2.67	1.89	2.58	3.82	3.48	5.72	8.39	4.93	3.75	6.55	4.04	1.71	2.90	10.05	5.24	7.18	7.37	8.36	9.33	7.71	7.94	8.73
	Female	50-69	21.2	71.8	18.1	18.8	16.9	30.1	13.9	29.5	26.3	21.4	28.0	14.0	13.4	15.9	18.1	16.1	24.4	34.2	34.6	37.4	41.3	29.0	46.9	30.0	7.8	13.5	58.2	35.4	34.6	27.1	61.9	47.4	48.3	45.4	31.0
	Female	70+	196.2	551.0	194.3	188.1	180.0	271.7	151.2	256.4	243.8	202.0	251.4	175.0	148.2	147.0	175.1	155.1	234.5	291.5	294.5	333.9	347.1	261.9	413.4	270.3	86.3	132.4	480.7	329.6	342.3	244.1	471.2	387.9	395.2	360.2	273.6
Malaria	Male	0-4	2.51	0.66	5.12	0.95	1.88	5.11	3.21	8.80	2.19	15.28	4.07	0.20	0.17	0.23	0.12	0.11	0.79	0.36	9.70	10.49	3.27	5.97	9.96	0.50	0.81	2.61	9.17	3.79	5.25	11.94	11.08	7.71	5.95	10.55	10.02
	Male	5-14	1.42	1.52	4.54	0.69	0.77	3.85	1.32	8.11	1.82	5.36	2.58	0.12	0.15	0.15	0.19	0.11	0.87	0.30	3.64	5.64	2.84	3.91	8.17	1.37	0.65	1.44	3.92	1.93	2.95	4.77	2.86	7.23	6.76	3.07	5.36
	Male	15-49	0.29	0.24	0.56	0.09	0.11	0.66	0.24	1.20	0.31	0.92	0.50	0.01	0.03	0.02	0.01	0.01	0.11	0.04	0.79	1.49	0.55	0.61	1.13	0.15	0.10	0.22	0.73	0.32	0.73	1.53	0.70	1.56	2.35	0.99	1.54
	Male	50-69	3.35	3.74	11.95	2.32	2.05	10.04	3.50	22.95	4.78	15.11	7.77	0.34	0.40	0.37	0.43	0.29	2.02	0.82	8.50	16.94	8.39	12.53	20.98	3.44	2.16	4.54	10.33	6.46	6.62	11.93	8.79	17.82	18.10	7.98	13.22
	Male	70+	13.3	14.8	47.7	12.0	8.4	43.3	15.4	100.0	20.6	72.2	33.3	1.55	1.93	1.39	1.24	1.16	7.97	2.91	37.6	77.2	37.3	61.8	87.6	14.0	9.1	21.5	43.7	27.3	28.2	49.5	44.1	81.7	91.2	41.2	73.9
	Female	0-4	2.08	0.52	4.11	0.81	1.51	3.98	2.75	8.08	1.90	13.41	2.77	0.15	0.13	0.18	0.08	0.08	0.61	0.38	7.31	8.85	2.70	4.58	8.82	0.46	0.68	2.27	8.03	2.96	4.15	9.64	9.33	7.00	5.48	9.69	9.43
	Female	5-14	0.84	1.00	1.56	0.34	0.31	2.45	0.63	6.15	0.98	2.32	1.81	0.05	0.07	0.07	0.11	0.04	0.38	0.15	1.75	3.41	1.65	2.34	4.44	0.66	0.25	1.15	2.38	1.06	2.01	7.49	2.59	6.85	11.52	4.21	3.87
	Female	15-49	0.22	0.17	0.36	0.06	0.08	0.49	0.17	0.97	0.22	0.72	0.41	0.01	0.02	0.02	0.01	0.01	0.08	0.03	0.55	1.08	0.41	0.47	0.85	0.11	0.07	0.17	0.57	0.23	0.55	1.38	0.62	1.35	2.24	0.95	1.37
	Female	50-69	2.21	2.79	5.33	1.25	1.29	7.00	1.96	18.39	2.86	10.69	8.04	0.18	0.22	0.19	0.20	0.12	1.10	0.42	5.45	11.98	5.98	9.50	12.82	1.92	0.94	3.65	7.89	3.50	4.97	19.81	9.42	18.60	30.15	11.96	12.97
	Female	70+	8.12	9.55	21.76	6.56	5.48	28.92	8.61	68.47	11.79	44.25	32.70	0.85	1.04	0.69	0.57	0.48	4.38	1.65	24.74	55.47	26.74	43.53	50.80	7.57	4.28	15.47	30.81	15.99	19.61	61.37	40.03	79.73	107.26	55.52	68.34
Tuberculosis	Male	0-4	11.4	1.9	7.1	9.5	10.6	9.2	17.1	6.2	5.4	12.1	5.6	8.9	11.5	11.9	3.5	5.3	14.4	3.4	34.7	23.8	8.2	7.5	17.7	1.6	4.5	7.3	17.1	13.0	19.3	43.8	24.3	19.2	6.5	26.2	37.9
	Male	5-14	31.2	33.5	32.4	32.8	19.8	30.6	27.3	27.8	24.5	24.0	18.8	24.3	32.0	25.3	18.7	24.2	51.6	15.9	48.1	50.3	35.8	26.2	69.5	27.9	11.1	19.7	43.4	33.7	43.4	42.2	34.4	69.3	35.0	34.0	62.9
	Male	15-49	3.48	2.06	1.92	2.19	1.60	2.74	2.95	1.99	2.07	1.88	1.85	1.71	5.27	2.59	1.02	1.94	4.66	1.13	6.49	7.55	3.91	2.04	5.08	1.44	1.23	1.53	3.51	2.96	6.04	9.47	4.08	7.83	5.61	5.35	12.05
	Male	50-69	127.6	135.5	131.5	128.9	77.4	120.7	112.8	106.3	97.6	93.0	78.6	108.5	127.0	107.4	81.0	112.7	216.2	69.5	196.6	220.2	156.3	113.7	272.0	97.5	41.2	80.5	165.8	156.3	175.4	186.1	139.9	290.1	139.3	139.2	333.7
	Male	70+	526.7	550.4	539.7	547.5	341.2	521.3	525.6	440.5	413.1	383.0	334.7	516.5	597.3	434.4	288.0	466.6	959.1	232.2	819.7	869.4	639.3	494.5	1,114.4	369.2	172.3	350.8	625.3	588.0	750.4	875.4	567.9	1,255.5	601.1	629.4	1,643.0
	Female	0-4	9.44	1.71	5.82	8.22	8.82	7.55	15.73	6.08	4.84	11.03	3.86	6.73	8.73	9.71	2.60	3.98	11.47	3.94	29.20	20.85	6.86	6.01	17.51	1.49	3.63	6.31	16.88	10.30	16.60	38.05	22.64	17.70	5.99	25.97	32.74
	Female	5-14	13.67	18.72	9.65	12.54	6.66	17.05	10.94	17.15	10.54	8.88	10.39	8.64	13.71	10.38	10.06	7.17	19.69	7.14	19.05	26.21	18.77	13.67	33.19	11.54	3.24	13.44	23.33	15.87	24.36	59.84	26.2	56.86	50.26	39.31	39.21
	Female	15-49	2.51	1.58	1.31	1.56	1.14	2.11	2.13	1.69	1.47	1.50	1.48	1.17	3.52	1.80	0.76	1.32	3.19	0.84	4.64	5.61	2.84	1.56	3.83	1.09	0.86	1.22	2.90	2.20	4.73	8.96	3.84	6.98	5.58	5.31	10.68
	Female	50-69	72.4	92.7	57.7	65.9	49.2	81.5	62.5	75.0	54.1	60.2	88.5	63.5	79.7	56.5	41.5	44.7	119.6	37.4	111.5	150.6	106.1	80.3	161.2	53.8	19.4	65.6	119.7	84.1	118.8	305.6	138.5	283.5	214.6	193.9	317.3
	Female	70+	327.4	363.0	281.7	337.2	251.8	396.4	331.8	312.1	250.2	254.5	386.5	359.2	436.7	244.6	154.5	207.7	580.0	150.6	529.5	717.3	489.9	377.5	720.8	223.6	104.8	318.8	497.4	383.6	528.5	1,283.5	569.9	1,340.2	820.9	928.1	1,780.6
Maternal disorders	Female	5-14	6.47	11.61	6.44	4.85	11.02	7.43	4.72	10.36	5.02	16.26	21.60	5.79	4.84	5.05	2.48	3.49	8.62	4.15	12.04	10.28	14.10	11.02	7.81	8.49	4.15	3.60	13.11	4.50	5.51	16.87	19.99	8.46	11.13	13.32	20.52
	Female	15-49	0.13	0.10	0.09	0.15	0.12	0.09	0.09	0.10	0.12	0.14	0.16	0.31	0.12	0.13	0.09	0.14	0.13	0.12	0.21	0.16	0.11	0.09	0.17	0.20	0.15	0.10	0.14	0.09	0.13	0.19	0.08	0.11	0.18	0.23	0.21
	Female	50-69	0.29	0.19	0.11	0.16	0.26	0.30	0.17	0.37	0.30	0.50	0.55	0.19	0.15	0.35	0.17	0.33	0.31	0.37	1.31	0.30	0.58	0.23	0.39	0.46	0.41	0.09	0.23	0.17	0.30	0.26	0.39	0.15	0.45	0.50	0.39
NN enceph due to birth asphyxia/trauma**	M	0-4	101.8	47.5	113.0	137.7	132.1	67.9	121.2	104.9	99.9	163.8	66.8	84.2	69.0	134.7	100.9	64.6	134.3	118.3	148.0	106.4	78.7	93.5	214.5	31.5	35.3	88.6	203.2	134.1	150.2	102.6	229.9	72.2	53.0	138.2	85.8
	F	0-4	71.1	31.0	82.1	94.5	86.5	51.2	76.9	66.9	67.5	113.7	54.1	59.2	51.2	89.8	76.6	45.3	99.8	70.6	97.8	75.7	58.1	71.4	156.9	21.5	23.8	60.9	131.2	96.8	103.2	69.6	155.6	48.5	35.0	100.9	71.8
Preterm birth complications	M	0-4	100.7	32.9	86.8	126.2	146.4	69.9	111.2	101.4	89.7	177.1	88.7	174.6	61.4	136.5	108.5	107.6	112.7	202.1	202.6	79.0	61.8	76.2	135.8	53.2	106.8	107.5	133.3	87.9	85.9	66.7	71.5	21.3	29.4	105.6	22.7
	F	0-4	93.9	29.0	85.1	116.5	131.7	71.6	97.6	87.2	81.4	167.2	94.2	162.9	60.4	122.7	106.3	99.4	110.6	163.6	179.1	77.2	62.0	78.8	131.4	49.7	96.1	101.2	118.0	85.7	80.2	62.5	65.6	19.8	26.6	105.3	26.0
Other neonatal disorders	M	0-4	42.6	16.4	49.0	55.6	57.1	40.8	47.9	42.5	30.8	56.0	31.0	39.7	17.9	30.2	30.1	14.1	53.6	45.3	64.2	95.9	39.4	71.0	88.4	35.7	31.3	124.6	156.7	105.7	128.3	79.1	129.2	64.3	38.1	115.5	30.6
	F	0-4	29.0	10.4	34.3	36.7	37.1	30.1	30.2	26.8	20.2	37.5	24.0	27.3	12.5	19.2	21.7	9.4	37.6	26.7	40.5	68.4	28.5	53.7	60.9	24.7	21.1	88.7	102.1	74.7	88.2	54.0	84.8	46.2	25.7	84.2	25.0
Other communicable diseases	Male	0-4	107.3	30.9	112.1	162.0	160.7	90.0	140.3	86.3	81.5	160.0	102.2	141.5	78.8	119.1	61.3	79.1	117.9	91.7	179.7	128.4	76.4	128.7	139.7	34.8	83.6	103.1	216.1	159.3	116.4	135.5	218.9	96.2	36.8	175.6	107.8
	Male	5-14	16.4	15.1	18.5	22.2	12.6	14.2	12.9	16.6	15.9	13.7	12.4	10.8	14.4	16.5	11.3	17.8	24.6	11.2	15.9	17.0	17.8	18.9	24.6	15.5	14.6	11.1	20.4	20.1	14.7	15.8	15.3	18.8	10.2	12.0	16.7
	Male	15-49	15.8	14.3	15.0	17.3	11.9	13.8	12.8	14.4	14.1	12.4	16.4	12.2	18.5	13.8	8.0	12.9	18.4	11.3	18.5	22.6	16.6	16.8	19.6	13.1	13.5	11.1	21.8	16.7	20.9	22.0	18.1	23.1	19.6	20.8	22.5
	Male	50-69	35.1	27.5	31.3	52.1	25.9	29.1	23.4	38.9	34.5	27.8	35.9	17.0	28.2	35.5	25.9	42.7	49.9	35.1	42.2	35.4	45.0	41.9	44.5	31.8	43.1	20.3	35.8	48.7	26.7	22.8	34.0	31.6	20.0	23.9	24.2
	Male	70+	147.9	105.0	119.2	228.5	107.5	119.7	92.3	169.7	140.6	129.4	167.7	63.9	127.6	140.1	106.8	167.2	213.4	162.1	179.0	176.8	195.9	189.0	181.0	150.8	195.2	86.2	153.4	238.5	136.6	79.2	167.2	127.0	106.7	109.0	97.9
	Female	0-4	85.5	26.0	87.9	140.1	137.8	67.3	117.6	72.1	68.0	132.2	74.0	109.6	59.4	95.1	43.3	57.7	91.4	89.6	153.7	104.8	63.5	97.9	117.9	30.1	62.3	81.0	184.4	126.8	92.0	108.1	183.9	77.5	32.7	146.4	93.2
	Female	5-14	8.78	9.70	6.52	9.79	5.38	10.10	6.42	13.77	8.97	6.38	9.73	4.96	7.63	8.57	6.99	7.25	12.81	7.67	10.19	10.41	12.44	12.59	14.56	8.49	6.31	7.76	12.50	10.88	10.60	19.15	14.98	18.18	19.06	19.18	18.65
	Female	15-49	11.1	10.4	9.8	11.9	8.5	9.9	8.8	10.8	9.7	9.1	13.1	8.5	12.4	9.1	5.4	9.0	12.9	8.0	13.2	15.9	12.1	12.6	13.9	9.6	8.8	8.1	16.3	12.0	15.3	17.8	14.9	18.6	17.4	17.9	20.3
	Female	50-69	30.3	32.5	20.3	36.8	24.9	35.0	20.4	52.6	32.5	30.9	56.7	15.0	23.5	30.5	23.3	27.6	46.1	34.9	49.5	39.2	56.5	48.8	45.6	32.3	32.9	18.7	39.8	42.9	36.9	38.1	61.3	42.2	53.3	54.0	35.5
	Female	70+	133.0	115.7	89.7	186.3	109.2	133.4	92.5	206.5	139.5	145.3	239.3	75.6	115.1	126.6	100.6	123.4	194.6	155.4	204.2	178.2	234.7	212.4	184.4	141.7	166.0	78.7	157.4	210.1	169.6	131.1	246.1	145.3	209.9	187.3	127.4

Cause group	Sex	Age	Indonesia	Aceh	Sumatera Utara	Sumatera Barat	Riau	Jambi	Sumatera Selatan	Bengkulu	Lampung
Non-communicable diseases	Male	0-4	104.2	37.8	108.9	124.0	173.0	79.9	153.0	106.1	89.0
		5-14	152.2	153.3	183.9	171.7	129.3	134.1	143.1	169.9	132.3
		15-49	20.2	20.6	20.3	18.8	18.9	17.7	21.4	21.0	19.1
		50-69	1,245.0	1,387.9	1,510.2	1,450.3	1,066.3	1,163.9	1,246.3	1,390.6	1,111.4
		70+	5,604.7	6,336.9	6,928.1	6,642.0	5,130.3	5,495.3	5,817.0	6,671.2	5,269.0
	Female	0-4	106.1	38.2	114.1	127.8	177.0	84.2	160.4	110.5	92.5
		5-14	115.7	165.7	105.9	116.4	109.3	135.9	104.8	202.4	108.0
		15-49	18.9	20.4	18.2	17.1	17.8	17.6	20.1	22.2	17.8
		50-69	981.7	1,454.1	946.7	1,028.7	984.0	1,178.6	964.1	1,592.2	961.1
		70+	5,201.6	7,003.3	5,560.5	5,976.3	5,810.1	6,364.3	5,590.2	8,192.6	5,387.6
Neoplasms	Male	0-4	5.85	1.33	4.28	8.00	7.59	3.32	6.23	4.43	3.30
		5-14	21.6	14.9	18.2	23.3	16.8	16.5	16.1	21.1	13.0
		15-49	3.92	2.99	3.00	3.85	3.28	2.90	3.63	3.30	2.77
		50-69	92.6	74.3	77.2	102.3	79.3	74.4	75.3	96.4	59.4
		70+	205.0	158.6	168.5	216.4	190.5	164.7	164.6	234.8	142.8
	Female	0-4	5.31	1.19	4.00	7.29	6.82	3.16	5.87	4.17	3.04
		5-14	28.6	33.1	20.0	31.4	26.2	29.6	23.2	43.6	20.6
		15-49	3.76	3.00	2.78	3.77	3.14	2.97	3.42	3.59	2.63
		50-69	108.0	120.3	71.1	121.0	103.4	107.1	92.1	145.2	73.7
		70+	221.9	211.5	146.1	258.0	217.0	204.3	194.0	270.0	149.4
Tracheal, bronchus, and lung cancer	Male	15-49	2.59	2.76	2.63	2.31	3.00	2.56	1.75	5.12	2.63
		50-69	22.4	26.4	22.3	17.3	32.7	22.6	18.8	42.8	23.2
		70+	60.9	66.7	57.9	43.2	93.7	59.8	47.5	126.6	70.8
	Female	15-49	1.10	1.68	0.87	0.84	1.71	1.53	0.77	3.77	1.31
		50-69	6.12	8.36	4.61	4.30	10.08	8.12	4.35	17.31	7.04
		70+	15.3	16.7	12.5	11.1	27.4	19.3	11.6	40.1	17.5
Breast cancer	Male	15-49	0.18	0.12	0.18	0.26	0.12	0.11	0.12	0.14	0.07
		50-69	1.01	0.90	1.13	1.57	0.85	0.77	0.89	0.83	0.44
		70+	1.58	1.39	1.99	2.92	1.45	1.26	1.59	1.42	0.72
	Female	15-49	10.3	12.4	7.5	15.0	9.7	9.5	8.8	16.9	7.4
		50-69	35.6	42.4	28.3	56.8	36.5	30.9	34.4	50.3	24.0
		70+	41.6	48.4	33.6	74.4	43.7	35.2	42.2	53.8	26.9
Cervical cancer	Female	15-49	6.11	8.39	5.12	3.99	5.02	7.53	4.48	7.66	3.66
		50-69	24.3	32.8	14.7	14.2	21.2	30.4	17.6	29.3	13.2
		70+	27.8	32.2	24.3	16.8	26.1	33.4	18.8	31.2	14.9
Other malignant neoplasms	Male	0-4	5.85	1.33	4.28	8.00	7.59	3.32	6.23	4.43	3.30
		5-14	18.8	12.0	15.4	20.7	13.7	13.8	14.2	15.8	10.3
		15-49	3.92	2.99	3.00	3.85	3.28	2.90	3.63	3.30	2.77
		50-69	69.2	47.0	53.7	83.5	45.8	51.0	55.6	52.8	35.7
		70+	142.5	90.6	108.6	170.3	95.4	103.7	115.5	106.8	71.3
	Female	0-4	5.31	1.19	4.00	7.29	6.82	3.16	5.87	4.17	3.04
		5-14	11.0	10.6	6.5	11.5	9.8	11.1	9.1	15.3	8.3
		15-49	3.76	3.00	2.78	3.77	3.14	2.97	3.42	3.59	2.63
		50-69	42.0	36.7	23.5	45.7	35.7	37.7	35.8	48.2	29.5
		70+	137.2	114.3	75.7	155.7	119.9	116.4	121.4	145.0	90.0
Cardiovascular diseases	Male	0-4	2.95	1.01	2.71	2.98	4.94	2.39	6.64	3.73	2.53
		5-14	63.5	69.5	73.4	62.5	56.9	56.1	73.0	74.3	56.8
		15-49	2.12	2.22	1.89	1.45	2.12	1.88	3.48	2.57	1.97
		50-69	690.7	821.2	784.8	720.0	621.6	659.6	781.2	807.6	642.9
		70+	3,343.2	3,903.9	3,944.6	3,473.3	3,225.2	3,259.3	3,849.4	4,170.5	3,208.5
	Female	0-4	3.47	1.17	3.24	3.41	5.57	2.90	7.96	4.49	3.09
		5-14	40.6	61.9	36.2	34.0	40.1	50.3	44.7	75.1	39.6
		15-49	2.42	2.65	2.09	1.68	2.29	2.31	3.85	3.32	2.29
		50-69	511.6	799.8	468.3	478.0	545.2	650.9	564.8	853.7	519.5
		70+	3,177.3	4,354.5	3,274.3	3,240.9	3,704.6	3,959.7	3,751.7	5,180.2	3,338.5
Cerebrovascular disease	Male	0-4	1.36	0.62	1.47	1.10	2.53	1.12	2.09	2.08	1.31
		5-14	24.8	32.9	30.1	21.0	22.5	26.6	21.0	35.3	28.7
		15-49	0.77	1.11	0.81	0.43	0.86	0.77	0.87	1.19	0.88
		50-69	329.6	454.2	379.7	299.0	295.1	362.1	286.3	445.8	370.7
		70+	1,703.1	2,265.9	2,040.3	1,501.6	1,641.5	1,860.8	1,491.0	2,418.4	1,912.4
	Female	0-4	1.61	0.72	1.78	1.33	2.90	1.37	2.55	2.48	1.60
		5-14	19.3	35.5	18.3	13.9	20.4	27.5	15.9	42.1	22.5
		15-49	0.89	1.35	0.93	0.53	0.98	0.96	0.99	1.55	1.03
		50-69	286.8	520.5	269.5	234.1	321.5	405.9	249.3	546.9	333.5
		70+	1,822.7	2,864.5	1,932.2	1,609.0	2,221.7	2,468.1	1,696.8	3,357.8	2,159.3
Ischemic heart disease	Male	0-4	0.38	0.10	0.37	0.30	0.67	0.20	1.00	0.29	0.17
		5-14	32.1	31.6	37.5	32.7	29.4	21.5	41.5	31.5	21.4
		15-49	0.67	0.60	0.59	0.40	0.67	0.38	1.14	0.61	0.39
		50-69	290.6	311.6	345.3	317.5	275.4	211.3	382.2	288.1	201.4
		70+	1,243.7	1,330.3	1,549.9	1,383.9	1,273.9	906.9	1,706.3	1,312.4	891.0
	Female	0-4	0.49	0.14	0.48	0.43	0.75	0.26	1.46	0.44	0.27
		5-14	15.3	19.8	13.9	13.3	14.1	13.4	19.7	22.3	10.7
		15-49	0.76	0.70	0.64	0.48	0.64	0.47	1.31	0.79	0.48
		50-69	158.7	207.4	153.1	160.2	160.4	143.6	212.7	205.8	115.5
		70+	928.5	1,076.4	1,014.7	1,043.1	1,036.2	842.1	1,345.0	1,184.9	707.4

Cause group	Sex	Age	Bangka Belitung	Kepulauan Riau	DKI Jakarta	Jawa Barat	Jawa Tengah	DI Yogyakarta	Jawa Timur	Banten	Bali	Nusa Tenggara Barat	Nusa Tenggara Timur	Kalimantan Barat	Kalimantan Tengah	Kalimantan Selatan
Non-communicable diseases	Male	0-4	184.0	86.2	124.7	63.6	147.6	84.8	87.1	103.8	139.1	181.6	95.8	65.4	103.7	113.0
		5-14	177.3	118.7	132.9	118.2	158.9	153.8	172.3	185.0	149.9	174.7	141.7	132.3	140.8	201.3
		15-49	21.7	19.7	15.5	21.5	21.5	13.4	19.2	19.5	18.4	25.4	21.7	18.1	18.8	17.1
		50-69	1,501.4	950.7	1,065.8	970.5	1,234.8	1,318.4	1,333.7	1,473.1	1,178.4	1,471.5	1,303.2	1,206.3	1,217.8	1,639.1
		70+	6,834.0	4,328.5	4,767.8	4,532.4	5,510.5	5,950.4	5,971.5	6,403.7	5,223.6	6,195.8	5,389.3	5,192.7	5,804.6	6,874.9
	Female	0-4	198.2	87.7	126.2	63.9	146.6	87.2	86.1	105.5	147.4	181.5	99.4	68.1	107.7	123.4
		5-14	172.8	164.5	89.2	84.6	118.5	139.6	94.8	128.8	125.1	139.6	127.0	138.4	141.7	155.0
		15-49	22.0	20.6	14.3	18.7	19.4	12.9	17.6	17.8	17.5	23.7	20.6	17.1	18.7	16.8
		50-69	1,416.8	1,453.1	806.2	767.3	976.3	1,092.0	806.2	1,141.6	1,046.2	1,283.9	1,194.8	1,218.0	1,258.9	1,348.8
		70+	7,288.0	6,908.7	4,749.1	4,277.8	5,074.3	5,801.6	4,465.3	5,737.1	5,611.4	6,503.3	5,978.2	6,109.4	6,747.6	6,515.8
Neoplasms	Male	0-4	7.04	3.27	6.72	2.29	8.27	4.94	6.30	5.55	14.13	13.72	13.38	3.43	4.16	6.86
		5-14	19.6	15.7	22.3	13.3	22.4	31.0	29.8	23.7	39.2	24.9	32.9	19.4	16.8	31.3
		15-49	3.32	2.99	3.11	3.21	4.33	2.87	4.84	3.79	5.70	5.96	7.02	3.54	2.95	3.51
		50-69	85.2	89.9	103.4	62.6	82.8	131.1	114.9	101.1	156.0	88.4	157.2	89.8	77.8	131.9
		70+	185.5	214.6	256.2	140.4	177.5	284.0	265.2	217.6	333.9	172.7	300.7	193.8	177.2	262.7
	Female	0-4	6.80	2.93	5.95	2.07	7.28	4.55	5.54	5.02	13.49	11.98	12.63	3.16	3.84	6.77
		5-14	33.6	36.6	23.4	16.1	30.1	41.7	28.0	31.9	46.1	38.9	49.1	33.7	26.1	39.4
		15-49	3.39	3.14	2.82	2.82	4.03	2.85	4.45	3.56	5.62	5.91	7.45	3.42	2.83	3.58
		50-69	112.7	132.6	95.7	62.1	108.9	144.6	107.1	117.9	179.0	149.1	230.7	130.0	98.9	150.0
		70+	202.8	242.2	221.4	127.3	215.0	274.2	235.4	222.2	386.2	290.9	479.3	242.7	202.3	279.0
Tracheal, bronchus, and lung cancer	Male	15-49	2.43	3.46	3.52	1.50	2.00	6.52	3.50	2.66	5.80	1.70	1.68	3.14	2.17	3.02
		50-69	22.2	49.5	36.3	12.0	14.6	43.7	28.8	33.7	44.7	14.3	14.2	30.7	24.6	25.5
		70+	58.0	132.8	109.4	29.2	40.7	116.8	87.5	94.5	110.8	38.8	30.4	80.9	64.9	63.7
	Female	15-49	1.44	3.64	1.41	0.55	0.81	3.10	1.03	1.24	2.60	0.70	0.70	1.93	1.35	1.25
		50-69	7.34	26.36	9.64	3.31	4.28	14.73	6.23	8.36	14.92	4.09	4.45	11.00	7.28	6.90
		70+	17.3	65.6	29.5	9.1	10.4	34.1	16.7	21.1	35.9	9.3	10.5	25.2	18.7	15.8
Breast cancer	Male	15-49	0.13	0.10	0.20	0.11	0.21	0.25	0.23	0.20	0.28	0.21	0.23	0.13	0.14	0.20
		50-69	0.82	0.81	1.57	0.82	0.83	0.98	0.91	1.29	1.14	0.98	1.41	0.70	0.98	1.32
		70+	1.32	1.17	2.88	1.25	1.29	1.11	1.66	2.02	1.40	1.44	1.70	1.06	1.68	1.84
	Female	15-49	10.3	11.6	9.5	5.1	10.6	14.1	9.6	13.8	12.4	16.2	13.9	10.3	10.8	12.7
		50-69	34.3	32.3	38.9	18.9	34.4	38.5	31.9	47.4	35.6	56.0	59.2	34.4	39.8	43.4
		70+	32.7	32.1	52.4	21.0	38.6	33.7	40.7	51.8	42.7	63.1	67.8	37.1	53.0	46.4
Cervical cancer	Female	15-49	7.96	7.36	3.70	3.42	7.19	7.35	6.94	4.75	12.65	4.68	10.73	7.22	5.45	9.08
		50-69	24.4	37.2	15.1	13.6	25.6	29.6	27.4	20.2	59.2	22.2	51.7	31.8	22.3	36.5
		70+	27.4	44.0	21.8	19.7	26.2	29.6	30.0	25.1	66.4	18.2	45.5	31.4	28.8	35.1
Other malignant neoplasms	Male	0-4	7.04	3.27	6.72	2.29	8.27	4.94	6.30	5.55	14.13	13.72	13.38	3.43	4.16	6.86
		5-14	17.1	12.2	18.6	11.7	20.2	24.3	26.1	20.8	33.1	23.0	31.0	16.1	14.5	28.1
		15-49	3.32	2.99	3.11	3.21	4.33	2.87	4.84	3.79	5.70	5.96	7.02	3.54	2.95	3.51
		50-69	62.2	39.6	65.5	49.8	67.4	86.4	85.1	66.2	110.2	73.1	141.6	58.3	52.3	105.1
		70+	126.3	80.6	143.9	109.9	135.5	166.1	176.1	121.1	221.6	132.5	268.6	111.9	110.6	197.2
	Female	0-4	6.80	2.93	5.95	2.07	7.28	4.55	5.54	5.02	13.49	11.98	12.63	3.16	3.84	6.77
		5-14	13.8	14.0	8.8	7.1	11.5	17.1	10.4	12.1	18.4	17.4	23.7	14.3	8.5	16.4
		15-49	3.39	3.14	2.82	2.82	4.03	2.85	4.45	3.56	5.62	5.91	7.45	3.42	2.83	3.58
		50-69	46.7	36.7	32.1	26.4	44.7	61.8	41.6	41.9	69.4	66.7	115.3	52.8	29.5	63.1
		70+	125.3	100.6	117.7	77.6	139.8	176.8	148.0	124.2	241.2	200.3	355.6	149.1	101.7	181.8
Cardiovascular diseases	Male	0-4	5.95	3.07	3.73	2.12	4.48	2.25	2.20	3.36	3.01	3.87	1.75	1.74	2.90	3.44
		5-14	78.5	52.7	54.5	56.3	67.3	56.1	70.4	81.9	49.8	64.0	39.1	54.2	60.4	75.6
		15-49	2.66	2.63	1.83	2.91	2.27	1.24	1.76	2.30	1.44	1.72	1.02	1.77	2.07	1.66
		50-69	872.1	539.3	557.6	569.4	711.7	703.9	740.7	857.6	580.0	783.4	558.6	690.4	701.9	852.1
		70+	4,295.6	2,729.7	2,798.6	2,796.4	3,403.6	3,454.8	3,597.2	4,089.1	2,896.3	3,489.5	2,563.3	3,097.6	3,581.4	3,898.3
	Female	0-4	7.40	3.40	4.14	2.45	5.22	2.70	2.51	3.94	3.96	4.56	2.24	2.08	3.42	4.40
		5-14	67.1	63.5	30.3	35.4	43.4	45.7	31.2	47.2	36.5	40.6	28.7	50.2	48.7	49.9
		15-49	3.24	3.23	1.99	3.09	2.56	1.49	1.96	2.58	1.73	2.10	1.36	2.08	2.38	2.07
		50-69	778.1	827.2	390.4	440.0	535.7	567.2	406.8	620.6	508.2	615.9	493.1	670.3	604.6	657.9
		70+	4,712.1	4,581.1	2,863.7	2,797.5	3,224.4	3,550.4	2,700.4	3,687.4	3,309.5	3,642.7	3,054.1	3,779.9	3,949.7	3,827.7
Cerebrovascular disease	Male	0-4	2.77	1.54	1.76	0.87	2.19	1.02	1.11	1.42	1.51	1.82	0.91	0.67	1.41	1.23
		5-14	32.0	19.9	15.9	18.4	30.4	24.0	28.8	28.0	16.8	26.8	15.7	20.4	23.0	28.7
		15-49	1.00	1.05	0.62	0.90	0.91	0.47	0.69	0.75	0.50	0.66	0.41	0.56	0.78	0.49
		50-69	424.6	261.2	204.4	240.2	374.6	361.5	366.6	367.8	259.0	404.2	276.8	319.0	319.9	384.2
		70+	2,215.0	1,426.1	1,103.7	1,254.6	1,881.4	1,913.7	1,879.7	1,888.8	1,423.7	1,885.1	1,367.0	1,507.4	1,735.8	1,850.3
	Female	0-4	3.46	1.77	2.04	1.03	2.54	1.21	1.27	1.69	1.82	2.18	1.14	0.81	1.67	1.53
		5-14	32.3	30.0	11.7	13.9	22.9	22.5	15.7	20.1	15.9	21.5	14.1	22.9	22.2	21.6
		15-49	1.22	1.34	0.71	0.97	1.04	0.56	0.78	0.85	0.60	0.82	0.54	0.67	0.91	0.61
		50-69	435.1	473.7	180.8	211.1	324.3	337.3	239.0	323.6	276.8	383.5	287.1	367.0	327.6	341.8
		70+	2,713.5	2,678.1	1,387.2	1,374.8	1,986.4	2,198.1	1,616.6	1,962.4	1,851.9	2,271.1	1,802.1	2,081.2	2,173.1	2,020.5
Ischemic heart disease	Male	0-4	0.70	0.42	0.76	0.37	0.42	0.15	0.28	0.39	0.37	0.45	0.26	0.18	0.43	0.26
		5-14	37.4	27.6	34.5	32.2	29.2	23.2	35.4	44.5	28.7	30.8	20.5	25.9	31.7	33.2
		15-49	0.77	0.83	0.78	1.10	0.59	0.27	0.57	0.73	0.57	0.49	0.36	0.48	0.69	0.39
		50-69	352.7	228.5	310.8	265.9	260.6	240.2	311.9	398.2	274.3	309.3	240.9	279.2	319.1	318.8
		70+	1,536.9	1,015.3	1,436.8	1,170.8	1,102.2	993.7	1,364.1	1,695.3	1,207.5	1,244.2	978.4	1,111.2	1,464.5	1,265.3
	Female	0-4	1.01	0.41	0.90	0.45	0.55	0.18	0.34	0.51	0.61	0.60	0.38	0.24	0.53	0.38
		5-14	24.6	24.3	15.6	16.6	13.6	13.3	11.7	19.2	15.9	13.3	11.5	17.6	20.1	15.9
		15-49	0.96	0.95	0.86	1.20	0.65	0.31	0.62	0.83	0.64	0.59	0.47	0.57	0.79	0.47
		50-69	241.6	259.2	174.5	173.5	137.2	126.5	123.0	209.7	174.2	158.2	158.5	190.8	207.4	172.8
		70+	1,368.3	1,360.0	1,206.1	1,052.1	777.5	714.3	774.4	1,181.2	1,072.2	905.2	941.8	1,030.1	1,298.7	945.1

Cause group	Sex	Age	Kalimantan Timur	Kalimantan Utara	Sulawesi Utara	Sulawesi Tengah	Sulawesi Selatan	Sulawesi Tenggara	Gorontalo	Sulawesi Barat	Maluku	Maluku Utara	Papua Barat	Papua
Non-communicable diseases	Male	0-4	53.4	92.1	157.3	175.1	108.0	128.4	98.3	182.3	59.5	38.1	132.1	58.5
		5-14	244.5	107.8	180.3	174.4	128.4	170.8	97.6	143.1	167.4	125.1	125.8	152.3
		15-49	25.0	19.7	22.8	23.3	15.4	28.0	20.4	22.9	20.6	23.3	22.1	18.8
		50-69	1,711.2	860.7	1,422.0	1,423.9	1,270.7	1,429.5	895.4	1,313.4	1,455.9	1,115.0	1,070.2	1,287.7
		70+	7,635.2	4,076.2	6,526.0	6,136.1	5,518.4	6,332.6	4,108.3	5,866.4	6,143.4	5,076.7	4,827.1	5,625.8
	Female	0-4	56.7	92.8	161.4	186.0	108.3	128.4	103.5	188.1	63.9	39.4	148.5	63.9
		5-14	190.8	66.8	196.3	168.7	101.4	157.1	233.7	220.8	218.5	296.5	249.1	159.6
		15-49	24.6	17.2	23.0	24.2	14.7	28.4	24.5	26.4	23.4	29.5	27.4	21.6
		50-69	1,434.2	626.8	1,451.4	1,457.4	998.3	1,430.7	1,876.8	1,909.6	1,816.3	2,321.4	1,985.2	1,502.1
		70+	7,382.3	3,691.9	7,434.3	7,083.7	5,403.3	7,300.6	7,889.5	8,867.3	8,331.4	9,716.7	9,148.1	7,191.2
Neoplasms	Male	0-4	2.98	5.05	14.08	9.24	7.54	6.74	3.79	11.03	3.62	1.93	8.06	3.49
		5-14	40.5	19.5	36.1	22.5	21.0	19.9	8.5	18.4	22.6	14.6	19.4	21.9
		15-49	5.21	3.89	6.41	4.28	3.42	4.97	2.92	4.65	4.33	4.02	4.56	3.98
		50-69	146.7	92.0	157.2	85.7	104.0	73.2	37.4	81.8	108.4	71.4	87.5	100.5
		70+	308.1	232.9	367.1	165.1	206.4	142.9	77.6	162.9	200.0	138.8	176.2	192.7
	Female	0-4	2.83	4.61	13.07	8.84	6.74	6.00	3.60	10.15	3.55	1.76	8.35	3.44
		5-14	49.8	15.4	61.9	39.1	28.4	36.1	45.6	56.8	59.7	66.0	64.5	35.2
		15-49	5.06	3.08	6.48	4.68	3.46	5.32	3.80	5.81	5.18	5.41	5.96	4.63
		50-69	162.6	59.8	225.1	143.6	119.7	125.7	153.1	208.9	230.0	217.4	223.8	165.5
		70+	313.7	131.2	473.1	253.3	242.4	224.9	227.1	362.0	413.1	318.9	394.0	284.8
Tracheal, bronchus, and lung cancer	Male	15-49	5.06	4.15	2.78	2.15	3.13	1.81	0.53	1.30	1.14	1.01	1.35	1.03
		50-69	41.2	37.3	22.1	16.4	26.5	18.5	5.0	10.9	11.4	9.8	14.9	17.7
		70+	104.0	126.2	65.0	36.1	61.7	47.0	12.7	24.9	23.8	22.3	31.5	36.5
	Female	15-49	2.39	1.58	1.67	1.14	1.24	0.99	0.70	1.10	0.79	1.35	1.56	0.61
		50-69	11.46	9.50	8.40	5.78	7.11	5.60	3.61	5.41	4.43	7.04	8.72	6.31
		70+	26.8	38.3	23.1	12.0	16.8	12.8	7.3	10.5	9.5	13.4	18.5	14.5
Breast cancer	Male	15-49	0.36	0.11	0.42	0.24	0.22	0.24	0.07	0.16	0.25	0.15	0.17	0.18
		50-69	2.00	0.81	2.87	1.48	1.45	1.27	0.53	1.35	2.03	1.45	1.50	1.64
		70+	3.12	1.17	5.64	2.14	1.82	1.66	0.98	1.81	2.88	1.98	2.46	2.78
	Female	15-49	18.2	4.6	25.7	16.0	12.6	14.5	19.2	19.4	24.7	29.0	25.0	11.5
		50-69	51.4	14.4	102.1	58.6	48.5	45.2	68.8	64.6	97.2	90.2	82.6	53.1
		70+	59.0	17.1	139.3	65.4	56.1	46.7	63.2	72.4	119.4	77.3	95.2	63.2
Cervical cancer	Female	15-49	12.32	4.62	18.17	8.76	3.76	7.37	10.56	14.38	13.06	8.55	13.37	7.30
		50-69	52.5	22.0	50.2	31.5	16.3	32.4	27.6	61.7	49.5	39.2	57.4	56.8
		70+	69.0	31.1	93.0	34.6	15.8	41.5	28.5	51.9	42.7	43.3	57.9	64.6
Other malignant neoplasms	Male	0-4	2.98	5.05	14.08	9.24	7.54	6.74	3.79	11.03	3.62	1.93	8.06	3.49
		5-14	35.1	15.2	32.9	20.1	17.7	17.8	7.9	17.0	21.2	13.5	17.9	20.7
		15-49	5.21	3.89	6.41	4.28	3.42	4.97	2.92	4.65	4.33	4.02	4.56	3.98
		50-69	103.5	53.9	132.2	67.8	76.0	53.4	31.9	69.6	95.0	60.2	71.1	81.1
		70+	201.0	105.5	296.5	126.9	142.8	94.2	63.9	136.2	173.3	114.5	142.3	153.4
	Female	0-4	2.83	4.61	13.07	8.84	6.74	6.00	3.60	10.15	3.55	1.76	8.35	3.44
		5-14	16.8	4.6	16.3	13.2	10.8	13.2	15.2	21.9	21.1	27.1	24.6	15.8
		15-49	5.06	3.08	6.48	4.68	3.46	5.32	3.80	5.81	5.18	5.41	5.96	4.63
		50-69	47.3	13.9	64.5	47.7	47.8	42.5	53.1	77.2	78.9	81.0	75.1	49.3
		70+	158.9	44.7	217.7	141.3	153.7	123.8	128.2	227.2	241.5	184.8	222.4	142.5
Cardiovascular diseases	Male	0-4	1.86	3.22	4.16	4.37	1.89	2.93	1.56	4.42	1.31	0.75	2.90	1.28
		5-14	100.3	44.5	66.6	64.2	44.8	62.1	37.9	53.4	61.9	45.2	44.1	58.4
		15-49	3.16	2.44	1.92	1.96	1.01	2.19	1.20	1.80	1.52	1.58	1.65	1.39
		50-69	927.5	485.8	725.5	725.0	629.1	734.4	478.7	664.6	731.5	561.0	522.1	662.0
		70+	4,672.5	2,592.6	3,694.6	3,307.9	2,909.1	3,591.4	2,192.0	3,125.7	3,245.4	2,681.8	2,511.6	2,942.5
	Female	0-4	2.15	3.63	4.88	5.45	2.30	3.43	1.98	5.39	1.58	0.91	3.71	1.66
		5-14	65.0	20.4	56.9	53.2	29.4	48.0	74.6	70.4	63.0	92.8	72.4	55.4
		15-49	3.52	2.32	2.32	2.61	1.29	2.86	1.93	2.74	2.13	2.65	2.55	2.04
		50-69	714.0	281.6	644.8	698.4	468.9	699.1	916.7	956.7	836.7	1,161.1	910.5	733.1
		70+	4,534.3	2,168.3	4,179.5	3,974.2	2,970.0	4,238.2	4,290.4	5,042.5	4,452.7	5,544.5	4,928.4	4,000.8
Cerebrovascular disease	Male	0-4	0.80	1.93	2.33	1.94	0.81	1.45	0.97	2.20	0.48	0.38	1.32	0.67
		5-14	35.3	19.6	32.4	26.2	13.5	26.5	19.9	24.9	22.5	20.4	17.8	34.1
		15-49	1.05	1.14	0.89	0.72	0.30	0.89	0.63	0.75	0.47	0.66	0.62	0.67
		50-69	402.8	259.3	397.0	347.2	243.1	374.5	280.0	365.8	329.5	305.3	261.5	412.7
		70+	2,215.2	1,463.5	2,103.1	1,689.8	1,243.2	1,977.8	1,334.0	1,800.6	1,526.7	1,557.3	1,328.1	1,857.5
	Female	0-4	0.95	2.19	2.74	2.40	0.95	1.71	1.22	2.62	0.59	0.46	1.68	0.90
		5-14	28.6	10.8	31.0	25.3	11.2	24.5	44.5	38.4	28.0	49.3	36.6	35.9
		15-49	1.24	1.10	1.10	0.97	0.38	1.17	1.00	1.14	0.70	1.13	0.99	1.02
		50-69	383.6	175.0	385.7	387.0	224.0	427.5	594.0	600.8	434.2	712.6	536.9	494.1
		70+	2,506.1	1,344.2	2,525.7	2,242.7	1,464.4	2,662.2	2,847.3	3,179.4	2,310.9	3,496.7	2,899.1	2,654.1
Ischemic heart disease	Male	0-4	0.19	0.41	0.34	0.50	0.38	0.28	0.20	0.46	0.16	0.06	0.32	0.10
		5-14	51.5	21.7	27.6	29.9	28.0	28.7	15.8	22.6	29.5	19.8	20.4	17.0
		15-49	0.94	0.77	0.45	0.51	0.44	0.56	0.32	0.43	0.36	0.38	0.42	0.22
		50-69	409.2	194.3	256.0	291.8	337.5	288.4	171.4	227.1	281.9	193.4	195.5	169.6
		70+	1,787.6	939.0	1,157.5	1,163.2	1,404.0	1,226.3	707.5	936.7	1,108.2	787.6	820.7	663.8
	Female	0-4	0.25	0.49	0.46	0.74	0.50	0.39	0.29	0.64	0.22	0.10	0.44	0.15
		5-14	24.8	7.6	18.2	18.6	14.7	16.2	23.5	20.7	21.3	29.5	23.0	11.9
		15-49	1.02	0.73	0.54	0.69	0.56	0.76	0.52	0.66	0.53	0.66	0.62	0.34
		50-69	223.2	85.3	179.5	205.4	194.0	185.9	249.2	226.2	239.5	301.7	239.1	143.3
		70+	1,323.7	667.7	1,119.5	1,105.4	1,169.5	1,049.2	1,085.3	1,140.6	1,222.2	1,330.6	1,247.7	774.8

Cause group	Sex	Age	Indonesia	Aceh	Sumatera Utara	Sumatera Barat	Riau	Jambi	Sumatera Selatan	Bengkulu	Lampung	Bangka Belitung	Kepulauan Riau	DKI Jakarta	Jawa Barat	Jawa Tengah	DI Yogyakarta	Jawa Timur	Banten	Bali	Nusa Tenggara Barat	Nusa Tenggara Timur	Kalimantan Barat	Kalimantan Tengah	Kalimantan Selatan	Kalimantan Timur	Kalimantan Utara	Sulawesi Utara	Sulawesi Tengah	Sulawesi Selatan	Sulawesi Tenggara	Gorontalo	Sulawesi Barat	Maluku	Maluku Utara	Papua Barat	Papua
Other cardiovascular/ circulatory diseases	Male	0-4	1.22	0.29	0.87	1.57	1.73	1.06	3.56	1.36	1.05	2.48	1.11	1.21	0.88	1.87	1.08	0.81	1.56	1.14	1.60	0.58	0.89	1.07	1.95	0.88	0.87	1.49	1.93	0.70	1.20	0.39	1.77	0.68	0.31	1.26	0.51
		5-14	6.70	4.92	5.76	8.86	5.04	8.02	10.56	7.52	6.79	9.08	5.24	4.08	5.75	7.65	8.93	6.16	9.43	4.22	6.37	2.89	7.87	5.75	13.74	13.58	3.24	6.66	8.10	3.35	6.94	2.18	5.96	9.86	5.08	5.80	7.35
		15-49	0.69	0.52	0.49	0.62	0.59	0.73	1.48	0.77	0.70	0.90	0.76	0.43	0.91	0.77	0.50	0.51	0.82	0.37	0.57	0.26	0.73	0.60	0.78	1.17	0.53	0.57	0.73	0.26	0.75	0.25	0.62	0.69	0.54	0.62	0.50
		50-69	70.4	55.4	59.9	103.5	51.1	86.2	112.7	73.6	70.9	94.7	49.5	42.4	63.3	76.5	102.2	62.1	91.6	46.7	69.8	41.0	92.1	62.9	149.2	115.5	32.2	72.6	86.0	48.6	71.5	27.4	71.7	120.2	62.4	65.1	79.7
		70+	396.5	307.7	354.4	587.8	309.8	491.7	652.1	439.7	405.1	543.7	288.2	258.1	371.1	420.0	547.4	353.5	505.1	265.0	360.2	217.9	479.1	381.2	782.7	669.6	190.2	434.1	454.9	261.8	387.3	150.4	388.4	610.5	337.0	362.8	421.2
	Female	0-4	1.37	0.32	0.98	1.66	1.92	1.27	3.95	1.58	1.22	2.92	1.23	1.21	0.97	2.12	1.31	0.90	1.74	1.53	1.78	0.73	1.03	1.22	2.49	0.96	0.96	1.67	2.32	0.85	1.32	0.48	2.13	0.77	0.35	1.60	0.61
		5-14	6.05	6.57	4.06	6.71	5.53	9.40	9.12	10.66	6.42	10.25	9.26	3.03	4.91	6.90	9.87	3.91	7.85	4.76	5.87	3.15	9.77	6.33	12.36	11.65	1.99	7.74	9.28	3.51	7.41	6.67	11.28	13.77	14.03	12.84	7.74
		15-49	0.76	0.60	0.52	0.66	0.66	0.88	1.55	0.98	0.78	1.06	0.95	0.43	0.92	0.87	0.62	0.55	0.89	0.50	0.69	0.35	0.85	0.68	0.99	1.26	0.49	0.68	0.95	0.34	0.93	0.40	0.95	0.91	0.87	0.94	0.68
		50-69	66.2	72.0	45.6	83.8	63.3	101.4	102.8	101.0	70.6	101.4	94.4	35.2	55.4	74.2	103.5	44.9	87.3	57.1	74.2	47.5	112.6	69.6	143.3	107.2	21.3	79.5	106.0	50.9	85.8	73.5	129.7	163.0	146.9	134.5	95.7
		70+	426.1	413.6	327.4	588.9	446.7	649.4	709.9	637.5	471.8	630.2	543.0	270.4	370.6	460.5	638.0	309.3	543.8	385.4	466.4	310.2	668.6	477.9	862.1	704.4	156.5	534.3	626.2	336.1	526.8	357.8	722.5	919.6	717.1	781.6	571.9
Diabetes mellitus	Male	0-4	0.37	0.09	0.66	0.40	0.56	0.21	0.44	0.33	0.26	0.65	0.36	0.72	0.17	0.39	0.24	0.34	0.42	0.59	0.64	0.29	0.15	0.31	0.45	0.27	0.30	0.59	0.65	0.38	0.77	0.24	0.75	0.18	0.11	0.48	0.16
		5-14	10.4	7.8	20.0	10.2	8.6	7.3	8.5	10.5	7.3	13.1	9.2	14.0	7.2	8.7	8.8	12.6	13.6	10.1	10.3	6.5	5.9	8.4	13.9	23.1	6.7	12.5	11.9	7.5	18.1	4.8	10.1	9.8	6.7	8.8	8.0
		15-49	0.50	0.40	0.89	0.39	0.48	0.36	0.48	0.49	0.41	0.64	0.64	0.66	0.48	0.41	0.27	0.50	0.55	0.45	0.55	0.32	0.30	0.43	0.43	0.94	0.47	0.55	0.60	0.33	1.20	0.36	0.62	0.42	0.45	0.58	0.36
		50-69	124.8	108.9	241.7	128.6	111.0	96.5	115.9	131.3	92.4	172.4	116.7	176.2	89.5	99.7	110.8	140.8	164.1	118.1	128.2	90.9	80.4	113.9	168.5	253.0	88.2	155.2	145.4	108.6	219.9	64.7	137.6	127.8	91.7	115.0	101.8
		70+	273.3	237.3	567.3	287.8	262.9	215.9	266.1	303.2	209.1	391.6	273.3	432.1	204.9	219.4	238.1	320.7	352.2	262.3	263.2	183.8	167.9	266.1	349.2	573.7	211.9	371.6	300.6	224.7	481.1	140.1	296.8	258.0	195.2	255.6	209.0
	Female	0-4	0.51	0.13	0.84	0.58	0.58	0.30	0.61	0.58	0.44	1.01	0.40	0.96	0.27	0.50	0.29	0.43	0.51	0.63	0.94	0.39	0.20	0.92	0.78	0.40	1.24	1.11	1.02	0.54	0.83	0.42	0.76	0.30	0.15	0.88	0.30
		5-14	11.1	13.8	15.2	10.3	8.2	10.2	8.9	21.3	10.6	18.9	14.5	12.7	8.0	8.7	10.5	9.9	12.4	9.3	12.2	7.4	8.4	23.1	17.5	25.4	13.4	24.3	17.2	8.8	18.8	20.4	16.4	18.9	21.9	27.0	14.3
		15-49	0.74	0.68	1.11	0.60	0.53	0.53	0.66	0.97	0.71	1.02	0.81	0.91	0.70	0.53	0.35	0.73	0.72	0.52	0.91	0.48	0.41	1.41	0.81	1.46	1.85	1.15	1.06	0.53	1.53	0.83	0.83	0.82	0.89	1.34	0.84
		50-69	137.7	182.9	192.4	136.7	118.8	134.1	125.1	250.6	136.3	234.3	212.9	175.8	108.3	104.9	116.7	121.1	168.9	118.2	171.3	113.0	113.1	297.3	223.6	291.7	182.2	277.4	224.1	127.2	253.6	243.9	220.0	244.7	269.4	338.5	215.9
		70+	373.4	444.9	592.3	401.9	347.7	353.6	365.2	661.2	387.6	616.1	522.3	579.0	305.8	277.7	301.0	358.5	441.1	331.5	449.6	296.1	279.4	913.2	572.0	829.4	681.8	820.1	558.3	347.0	665.3	504.2	508.6	580.1	555.3	828.0	523.7
Chronic kidney disease	Male	0-4	2.87	1.00	2.78	3.04	5.20	2.54	3.50	3.35	2.17	5.52	2.87	2.97	1.50	4.67	2.23	2.29	2.35	3.14	5.17	3.85	1.59	3.53	2.74	1.03	3.64	5.72	5.22	3.49	3.39	3.46	5.19	2.32	1.20	3.22	1.32
		5-14	9.15	10.46	9.66	9.25	9.14	8.80	7.30	14.01	7.68	12.22	9.75	7.62	6.39	11.59	12.12	9.71	10.90	10.22	9.20	8.68	7.34	10.41	11.37	14.42	10.48	14.97	9.82	7.50	9.89	5.47	6.93	11.04	9.43	6.63	5.04
		15-49	1.39	1.62	1.23	1.10	1.48	1.34	1.23	1.96	1.35	1.77	1.70	0.90	1.33	1.85	1.10	1.26	1.28	1.36	1.54	1.48	1.20	1.60	1.14	1.59	2.08	2.07	1.56	1.08	1.84	1.43	1.33	1.45	1.92	1.22	0.76
		50-69	46.2	57.0	48.7	49.7	45.7	46.0	39.4	68.9	38.6	62.5	41.7	38.4	32.4	56.6	62.0	45.2	50.5	46.5	42.2	52.8	39.5	56.6	59.6	60.7	51.1	82.8	50.3	46.1	46.3	32.7	37.8	59.1	50.5	32.5	28.2
		70+	141.3	170.7	151.0	156.0	145.5	141.3	124.5	215.4	120.4	191.0	127.1	125.4	102.1	168.9	171.9	139.0	143.5	133.2	118.1	143.9	112.6	181.9	166.8	175.9	161.0	272.3	139.3	127.2	126.9	99.8	109.8	164.4	142.9	97.2	82.3
	Female	0-4	2.95	1.08	2.97	3.67	5.01	2.55	4.27	3.92	2.60	6.43	2.34	3.24	1.53	4.59	1.89	2.18	2.39	3.62	5.12	3.83	1.59	3.42	2.93	1.28	3.86	6.21	6.00	3.19	3.72	3.52	5.25	2.75	1.37	3.68	1.58
		5-14	6.52	11.01	5.51	6.72	6.85	8.30	5.43	14.89	6.49	11.62	12.39	5.13	4.68	7.65	8.80	4.75	7.07	6.03	6.17	6.41	6.92	9.08	7.32	10.75	5.53	14.76	9.01	5.18	9.12	13.01	9.61	13.87	20.49	11.06	5.10
		15-49	1.33	1.61	1.17	1.19	1.29	1.36	1.26	2.05	1.37	1.90	1.69	0.94	1.27	1.62	0.96	1.18	1.23	1.11	1.47	1.42	1.14	1.54	1.07	1.67	1.79	2.10	1.68	1.01	2.06	1.72	1.56	1.81	2.50	1.55	1.00
		50-69	32.7	54.4	28.7	36.9	36.6	41.1	29.7	65.9	32.2	54.6	56.2	28.4	24.8	37.9	38.8	24.1	35.3	29.3	30.6	39.4	34.8	46.4	37.5	45.4	27.6	76.4	46.4	29.9	42.1	65.7	47.6	71.6	92.0	50.1	32.6
		70+	117.5	175.1	111.3	145.6	142.4	147.9	114.8	226.0	120.3	183.6	178.6	116.3	92.5	132.0	125.4	92.2	120.8	107.7	108.6	140.6	118.2	177.6	125.0	160.0	115.9	288.8	151.3	107.8	138.3	177.5	149.3	232.4	246.6	164.4	110.3
Cirrhosis	Male	0-4	1.12	0.22	1.45	1.47	1.66	0.78	1.17	0.87	0.83	1.66	0.76	1.39	0.64	1.61	0.73	0.78	1.36	0.92	2.28	2.22	0.47	1.05	1.33	0.39	0.60	1.29	1.80	1.28	1.40	1.21	1.29	1.02	0.39	2.21	1.14
		5-14	16.3	12.2	20.1	19.5	11.1	11.8	11.5	15.0	15.7	17.1	10.8	13.0	12.3	18.2	12.9	17.7	23.2	12.5	23.9	24.2	14.4	15.3	28.0	19.9	8.2	15.0	17.4	16.2	20.2	11.5	13.3	18.8	14.0	13.6	17.8
		15-49	2.30	1.79	2.62	2.38	1.76	1.64	1.73	2.01	2.37	2.16	2.06	1.96	2.32	2.46	1.38	2.02	2.64	1.77	3.32	3.61	1.98	2.18	2.60	2.37	1.61	2.00	2.55	2.05	3.68	2.30	2.11	2.58	2.81	2.68	2.14
		50-69	76.3	60.8	86.2	92.5	51.8	58.1	54.6	71.9	74.5	79.7	49.5	52.2	55.4	83.4	67.3	80.5	106.5	62.9	121.6	133.2	76.1	72.9	129.5	80.3	39.6	64.4	76.1	87.8	93.5	53.8	65.4	88.8	70.2	65.0	76.6
		70+	191.0	146.0	214.4	215.4	136.2	143.2	132.9	186.0	187.1	199.6	131.9	129.0	137.1	209.3	185.8	201.2	260.4	173.9	276.9	322.0	177.7	184.5	296.8	208.7	110.8	163.4	176.6	217.0	245.8	122.2	156.6	195.8	177.2	156.5	154.0
	Female	0-4	0.69	0.16	0.76	0.88	1.06	0.43	0.74	0.52	0.50	1.05	0.42	0.70	0.33	0.99	0.39	0.44	0.75	0.82	1.99	1.59	0.33	0.70	1.21	0.23	0.40	0.67	1.39	0.83	0.87	0.81	1.12	0.53	0.22	1.57	0.55
		5-14	7.37	7.75	6.58	6.77	5.47	7.35	4.72	11.14	7.77	9.86	9.03	4.53	5.22	8.27	7.36	5.55	9.69	7.18	12.11	13.12	9.45	9.62	13.71	8.42	3.13	8.39	9.97	7.89	10.88	15.91	12.42	13.22	20.74	16.53	12.15
		15-49	1.23	0.99	1.31	1.21	0.91	0.93	0.86	1.23	1.27	1.19	1.19	0.92	1.13	1.30	0.80	1.05	1.36	1.07	1.91	2.25	1.08	1.32	1.58	1.20	0.85	1.14	1.56	1.25	2.07	1.69	1.41	1.58	2.09	1.90	1.41
		50-69	40.8	43.3	34.1	38.2	31.9	41.5	26.9	58.6	44.1	50.0	52.3	23.7	28.3	46.2	42.6	31.9	56.2	45.4	76.4	86.9	55.9	50.6	76.0	42.8	20.3	37.9	52.1	50.4	65.5	70.6	68.4	66.3	103.1	82.0	57.5
		70+	127.0	116.9	114.9	121.5	103.9	117.5	86.9	170.8	140.0	149.4	143.2	83.6	88.5	143.9	145.1	107.0	162.1	150.1	215.1	257.2	150.2	156.6	213.8	132.2	76.7	117.5	142.0	158.9	200.4	166.1	170.9	161.4	247.6	198.8	130.6
Chronic respiratory diseases	Male	0-4	2.17	0.42	2.66	4.42	3.07	2.18	3.05	2.36	1.35	3.24	1.01	1.42	1.21	2.80	1.21	1.16	1.69	1.60	6.09	4.85	0.96	1.66	1.73	0.63	1.61	3.01	5.25	2.77	3.01	4.82	5.35	2.27	1.08	5.91	3.42
		5-14	9.92	12.38	15.84	22.81	7.44	11.50	8.46	12.75	8.98	10.79	4.95	6.79	6.73	8.76	9.74	8.29	8.02	11.42	15.04	16.41	9.30	9.13	14.12	16.24	5.95	11.85	23.38	13.54	12.89	9.10	18.28	13.69	11.59	12.71	12.30
		15-49	1.73	1.56	2.07	2.98	1.30	1.85	1.73	2.10	1.63	1.61	1.04	0.84	1.67	1.58	1.03	1.22	1.26	1.45	3.29	3.12	1.63	1.37	1.40	1.62	1.55	1.76	3.17	1.90	2.94	3.11	3.56	2.20	2.84	2.73	2.27
		50-69	109.9	137.1	150.0	225.6	69.6	118.8	95.6	115.4	93.7	108.9	49.4	74.9	85.8	96.1	111.4	96.3	88.0	123.3	164.0	217.4	107.1	94.4	155.6	127.7	49.6	123.2	216.2	174.2	129.4	111.0	202.8	174.7	139.1	142.3	173.3
		70+	760.0	894.5	1,048.4	1,439.9	551.6	841.4	710.3	861.3	665.9	765.0	412.7	592.5	638.6	657.5	747.8	688.1	661.6	792.8	1,021.4	1,292.7	707.6	709.4	1,037.9	901.7	382.4	876.9	1,271.9	1,057.9	869.6	763.4	1,232.3	1,107.6	912.2	960.3	1,208.6
	Female	0-4	1.04	0.24	1.11	2.29	1.46	0.99	1.59	1.14	0.63	1.66	0.41	0.57	0.47	1.25	0.46	0.49	0.71	1.07	3.90	2.67	0.50	0.80	1.21	0.31	0.80	1.23	3.34	1.27	1.54	2.43	3.92	1.04	0.48	3.72	1.32
		5-14	5.34	10.26	6.49	11.11	4.45	8.23	4.46	10.68	5.40	6.93	4.50	2.84	3.21	4.46	5.83	2.95	3.97	5.74	8.59	9.96	6.66	5.52	7.09	7.76	1.93	7.91	15.70	7.45	8.74	16.59	20.62	12.97	19.57	16.27	7.44
		15-49	0.74	0.68	0.84	1.35	0.54	0.84	0.71	1.04	0.68	0.73	0.47	0.32	0.62	0.64	0.45	0.48	0.50	0.64	1.44	1.50	0.69	0.58	0.62	0.69	0.58	0.81	1.58	0.86	1.39	1.78	2.07	1.16	1.72	1.63	1.16
		50-69	61.8	105.4	67.3	116.1	50.7	84.2	54.0	97.4	58.5	67.7	57.8	38.9	41.7	53.6	64.4	41.3	52.4	73.7	106.9	139.5	77.8	60.7	83.9	70.9	20.7	79.1	155.9	96.2	98.8	172.0	212.3	152.2	191.0	170.9	132.3
		70+	470.3	669.7	556.3	923.5	442.5	648.9	458.2	730.5	456.5	494.6	435.6	372.6	350.9	396.7	464.9	339.3	416.4	548.1	783.4	1,014.0	564.2	498.6	608.4	542.5	189.1	638.3	1,012.6	707.7	705.7	1,046.1	1,312.4	1,055.2	1,106.8	1,210.5	1,019.8
Other non-communicable diseases	Male	0-4	88.8	33.7	94.3	103.7	150.0	68.4	132.0	91.1	78.6	159.9	74.9	107.7	55.7	125.4	73.2	74.0	89.0	115.7	149.9	69.4	57.1	90.1	96.4	46.2	77.7	128.5	148.5	90.6	110.1	83.2	154.3	48.8	32.7	109.4	47.7
		5-14	21.2	26.1	26.7	24.2	19.4	22.0	18.3	22.2	22.8	26.0	15.6	14.8	16.0	22.0	23.1	23.9	23.8	16.6	27.3	13.9	21.9	20.4	27.0	30.0	12.4	23.4	25.2	17.8	27.6	20.3	22.6	29.6	23.5	20.7	28.8
		15-49	8.24	9.97	8.60	6.69	8.46	7.71	9.14	8.56	8.64	9.53	8.62	6.23	9.52	8.63	5.47	7.60	7.66	6.26	9.00	5.15	7.70	8.24	6.33	10.12	7.69	8.10	9.13	5.58	11.21	9.06	8.87	8.12	9.69	8.65	7.89
		50-69	104.5	128.6	121.5	131.6	87.1	110.4	84.4	99.1	109.9	120.7	64.3	63.0	75.4	104.6	132.0	115.4	105.3	91.5	143.8	93.0	123.1	100.3	141.9	115.3	54.4	113.7	125.4	120.8	132.7	117.3	123.4	165.7	131.1	105.7	145.4
		70+	690.9	825.9	834.1	853.2	618.5	729.5	569.3	700.0	735.3	805.7	439.2	434.1	513.0	674.3	868.1	760.0	679.2	631.3	854.1	582.9	735.5	704.2	863.1	794.6	384.6	780.0	774.7	776.3	875.0	713.4	782.3	972.2	828.6	669.8	836.7
	Female	0-4	92.1	34.2	101.1	109.7	156.5	73.9	139.3	95.7	82.2	173.8	77.8	110.7	56.8	126.8	76.9	74.5	92.1	123.8	153.0	76.0	60.3	94.6	106.1	49.5	78.3	134.2	159.9	93.5	112.0	90.7	161.5	54.2	34.5	126.5	55.1
		5-14	16.3	27.9	16.0	16.1	18.0	21.9	13.4	25.8	17.5	24.9	24.0	10.4	12.0	16.0	19.8	12.4	16.6	14.2	21.0	12.3	23.0	19.5	20.1	23.8	7.1	22.1	24.6	14.3	25.5	47.5	34.6	36.8	55.0	41.3	29.9
		15-49	8.63	10.83	8.92	7.35	9.07	8.67	9.29	9.97	8.85	10.54	10.06	6.41	9.12	8.70	5.98	7.73	7.82	6.84	9.92	6.14	8.30	8.66	7.09	10.99	6.71	8.98	11.03	6.31	13.13	12.72	11.98	10.71	14.24	12.48	10.48
		50-69	89.2	148.0	85.0	101.8	97.5	119.7	71.6	120.7	96.7	119.4	114.0	53.4	62.2	89.0	117.7	73.9	90.2	92.4	133.9	92.3	136.1	100.4	120.0	106.9	34.7	110.7	136.9	106.1	145.8	254.8	195.8	214.8	287.5	209.4	165.2
		70+	714.3	1,030.7	765.2	885.0	852.0	932.5	619.6	953.9	795.3	929.5	805.7	512.6	515.3	684.7	940.6	632.5	687.1	778.4	1,013.0	736.9	974.8	849.7	889.8	870.3	328.9	917.1	992.1	869.6	1,127.9	1,478.2	1,321.6	1,436.5	1,697.0	1,424.0	1,121.3
Injuries	Male	0-4	40.8	14.8	56.0	69.3	74.8	40.2	61.0	41.5	29.1	81.2	29.7	48.7	18.4	39.7	64.0	25.0	35.3	57.9	75.4	50.1	26.7	46.3	65.1	18.6	34.2	64.6	85.0	71.4	53.8	39.4	95.0	31.3	21.2	83.2	51.9
		5-14	52.3	61.3	75.8	73.6	51.4	56.3	58.1	63.0	46.7	78.4	35.9	37.0	33.7	45.0	64.7	48.9	58.5	47.3	74.1	64.1	55.3	58.1	85.3	75.3	39.7	66.8	68.6	69.2	65.9	39.2	72.6	61.4	57.9	54.4	98.4
		15-49	26.1	28.5	32.2	32.6	26.2	27.5	27.9	27.6	22.8	31.1	23.9	21.2	22.5	21.6	26.4	20.4	23.5	25.8	35.3	36.2	25.9	27.6	32.1	30.9	24.3	31.3	37.2	31.0	40.1	28.1	38.1	33.0	41.7	39.8	47.5
		50-69	71.5	82.9	94.9	100.5	73.7	81.7	79.7	93.3	64.9	110.7	52.2	39.7	38.8	63.8	123.2	73.2	74.7	86.6	125.8	83.1	86.9	80.9	111.9	99.6	63.5	86.6	81.9	108.5	86.6	41.3	105.4	69.4	78.9	72.3	118.3
		70+	232.1	263.4	295.6	289.9	251.3	265.4	245.0	318.1	211.5	356.4	182.5	112.2	121.0	205.9	498.6	228.4	236.5	330.9	375.3	266.3	266.8	259.0	327.0	336.4	235.4	263.7	255.1	378.8	324.3	120.5	339.5	200.7	281.2	227.5	288.2
	Female	0-4	15.1	5.7	21.2	27.5	29.6	14.3	23.2	14.5	10.4	30.6	10.7	17.5	6.3	14.9	24.7	9.2	12.3	21.8	30.4	17.8	10.0	16.6	25.0	6.8	11.5	23.3	31.2	26.6	19.2	14.5	36.1	10.6	7.8	30.2	18.6
		5-14	17.5	25.1	18.7	19.9	18.8	24.0	18.0	30.7	14.9	31.9	21.4	11.0	10.4	14.2	26.1	11.8	17.1	19.3	27.4	23.5	24.8	25.3	28.4	25.8	11.9	28.2	26.8	22.7	26.2	28.9	47.2	30.2	57.3	48.1	59.7
		15-49	10.6	12.4	13.2	14.0	11.2	11.7	11.4	12.4	8.8	13.6	10.7	8.4	8.0	8.4	13.0	8.3	8.8	11.0	14.5	14.2	10.4	11.9	13.4	13.1	9.4	14.6	15.8	13.7	17.2	12.9	18.6	14.9	22.9	20.0	24.1
		50-69	29.9	44.4	29.2	33.9	33.7	44.9	31.1	57.6	27.9	55.0	41.1	15.7	15.2	27.0	63.6	23.5	30.1	46.2	60.3	38.1	48.0	42.2	49.4	46.2	27.1	39.7	40.0	45.1	49.6	32.9	81.9	39.8	89.0	71.0	67.5
		70+	121.7	156.3	128.7	137.0	141.8	164.1	130.6	217.4	115.3	221.4	145.1	68.0	61.8	109.9	318.7	99.7	110.1	197.4	209.8	138.3	164.9	156.4	175.8	176.0	127.5	143.0	141.0	190.5	209.4	105.7	266.3	116.8	297.5	204.0	175.0
Drowning	Male	0-4	10.5	3.0	13.9	14.9	17.8	13.4	19.6	10.7	6.9	17.0	6.8	3.8	5.0	10.9	12.2	5.0	10.2	11.5	15.6	14.2	9.4	12.3	21.2	3.8	4.2	13.2	20.2	22.7	15.3	15.9	29.2	11.4	6.0	23.0	22.5
		5-14	3.11	3.07	3.45	2.92	2.73	4.77	4.93	3.82	2.81	4.76	1.31	0.34	1.92	3.09	1.35	2.05	3.20	1.64	3.56	5.74	6.14	4.64	7.42	2.49	1.18	2.89	4.40	5.44	4.74	6.19	6.55	5.22	3.95	3.27	14.84
		15-49	8.90	9.11	10.02	9.19	8.69	11.39	11.35	9.93	7.61	9.99	6.86	2.27	7.64	7.91	6.01	5.66	7.91	8.01	10.24	14.54	11.41	11.00	13.46	8.42	5.48	8.69	13.39	11.82	15.57	14.67	15.22	15.03	16.68	14.74	23.17
		50-69	2.18	2.01	1.92	1.84	2.01	3.78	3.46	3.15	2.17	3.26	1.15	0.12	1.10	2.41	1.56	1.87	2.18	2.06	4.39	3.05	5.61	2.98	4.79	1.76	0.99	1.47	2.28	4.13	3.32	2.65	4.71	2.75	2.72	2.26	7.71
		70+	13.0	12.5	12.1	9.2	13.5	22.8	19.8	21.4	12.9	19.9	7.9	0.5	6.0	13.5	15.7	10.5	12.5	17.6	23.0	18.3	30.4	17.3	26.5	13.3	8.0	9.0	14.3	31.6	28.8	11.4	29.0	13.8	19.5	14.4	26.0
	Female	0-4	3.27	0.96	4.48	4.99	6.23	3.96	6.36	2.91	1.99	4.92	2.17	1.12	1.49	3.55	4.06	1.54	3.06	3.28	5.58	4.10	3.03	3.52	6.61	1.13	1.03	3.89	5.76	7.32	4.54	5.01	9.24	3.10	1.85	6.46	7.00
		5-14	1.00	0.96	0.69	0.55	0.84	1.84	1.32	1.60	0.74	1.63	0.72	0.09	0.53	0.88	0.50	0.46	0.89	0.69	1.43	1.65	2.59	1.76	2.17	0.79	0.30	0.86	1.34	1.38	1.59	3.07	3.58	2.12	3.52	2.77	10.86
		15-49	3.38	3.56	3.89	3.66	3.52	4.58	4.40	3.91	2.68	3.86	2.86	0.78	2.56	2.84	2.69	2.16	2.79	2.95	3.97	5.19	4.41	4.37	5.18	3.22	1.77	3.61	5.04	4.95	6.22	6.15	6.68	6.15	8.16	6.38	11.51
		50-69	1.17	1.38	0.69	0.65	1.10	2.84	1.71	2.67	1.17	2.04	1.13	0.05	0.51	1.24	1.17	0.74	1.13	1.60	3.00	1.66	4.26	1.93	2.86	1.15	0.64	0.63	1.30	2.17	2.89	1.69	4.92	1.69	3.99	3.01	5.18
		70+	8.31	9.32	6.62	5.10	8.75	17.42	14.54	18.94	8.94	16.66	6.67	0.32	3.62	8.66	13.42	5.83	6.67	12.08	16.21	9.56	22.66	12.72	18.66	8.52	6.17	5.15	8.79	17.83	23.98	9.90	26.77	7.70	25.30	14.00	17.18

Cause group		Age	Indonesia	Aceh	Sumatera Utara	Sumatera Barat	Riau	Jambi	Sumatera Selatan	Bengkulu	Lampung	Bangka Belitung	Kepulauan Riau	DKI Jakarta	Jawa Barat	Jawa Tengah	DI Yogyakarta	Jawa Timur	Banten	Bali	Nusa Tenggara Barat	Nusa Tenggara Timur	Kalimantan Barat	Kalimantan Tengah	Kalimantan Selatan	Kalimantan Timur	Kalimantan Utara	Sulawesi Utara	Sulawesi Tengah	Sulawesi Selatan	Sulawesi Tenggara	Gorontalo	Sulawesi Barat	Maluku	Maluku Utara	Papua Barat	Papua
Falls	Male	0-4	4.97	1.10	5.07	6.69	6.86	3.01	2.96	3.25	2.46	6.69	5.73	25.17	2.25	5.28	11.63	2.79	5.99	7.58	7.52	6.47	2.16	3.16	4.65	2.32	3.03	4.10	6.50	6.41	4.55	3.88	7.57	3.24	2.37	5.19	5.83
		5-14	4.04	3.21	3.90	4.12	3.27	3.23	2.16	3.54	2.87	5.25	3.46	7.93	2.53	4.59	5.23	3.66	5.55	3.87	5.23	6.74	3.73	3.42	4.84	5.16	2.61	2.66	4.08	4.30	3.86	3.82	4.55	4.48	4.51	2.22	10.38
		15-49	1.98	1.62	1.84	2.05	1.70	1.38	0.92	1.55	1.33	1.99	2.74	6.54	1.66	1.88	2.68	1.49	2.26	2.57	2.38	3.35	1.40	1.49	1.55	2.45	1.90	1.35	2.17	1.71	2.38	1.88	2.00	2.36	3.23	1.82	3.51
		50-69	16.7	13.7	14.1	16.5	14.4	14.8	9.3	17.0	12.5	22.0	16.3	20.0	9.2	19.4	31.5	17.2	22.4	24.3	31.1	25.6	19.3	13.8	18.3	20.7	12.7	8.9	13.9	20.6	17.4	11.1	20.1	15.2	18.7	9.7	29.9
		70+	95.7	81.4	89.6	87.1	92.3	87.6	54.9	108.9	73.9	127.9	96.6	78.6	50.3	101.6	242.9	93.6	118.6	166.4	156.8	132.6	98.5	80.6	99.9	135.9	86.3	54.7	82.2	139.0	126.3	50.0	117.3	75.9	120.0	58.7	114.8
	Female	0-4	2.06	0.45	2.19	3.02	3.09	1.20	1.27	1.23	0.95	2.63	2.39	10.04	0.90	2.28	5.26	1.15	2.44	2.91	3.39	2.50	0.91	1.22	1.94	0.93	1.02	1.63	2.48	2.74	1.78	1.63	3.11	1.21	0.97	1.99	2.41
		5-14	1.66	1.51	1.08	1.19	1.40	1.74	0.79	2.20	1.07	2.60	2.58	2.80	0.94	1.82	2.81	1.12	2.09	2.14	2.69	2.78	2.16	1.76	1.95	2.23	0.92	1.18	1.74	1.60	1.81	2.92	3.48	2.56	5.61	2.53	7.98
		15-49	0.93	0.80	0.87	1.02	0.84	0.68	0.43	0.79	0.58	0.97	1.45	3.04	0.71	0.86	1.53	0.71	1.01	1.20	1.14	1.50	0.64	0.72	0.73	1.21	0.80	0.70	1.04	0.86	1.16	1.00	1.09	1.22	2.03	1.00	2.04
		50-69	9.62	10.36	6.08	7.34	8.96	11.76	5.25	15.14	7.49	14.95	17.03	9.76	4.90	11.01	23.78	7.72	12.36	18.60	21.07	15.15	14.77	9.90	11.90	14.01	8.00	4.96	9.03	12.10	14.69	9.66	21.82	10.73	29.01	13.65	21.88
		70+	64.4	64.4	53.8	56.0	67.1	71.5	42.2	100.0	53.2	108.1	91.1	56.1	32.9	67.1	198.5	54.0	68.2	120.9	112.5	81.1	78.0	64.7	73.2	91.8	63.9	38.2	57.7	88.1	104.6	51.6	118.1	50.9	158.8	66.7	84.2
Interpersonal violence	Male	0-4	1.82	1.01	2.48	2.85	3.96	1.79	2.56	2.16	1.71	5.99	0.73	0.92	0.46	1.15	0.91	1.10	0.78	4.00	5.35	3.09	1.33	3.17	3.08	0.85	2.54	2.90	6.20	3.56	3.01	1.60	6.41	1.55	1.30	5.12	4.15
		5-14	1.61	3.37	2.48	2.24	2.18	2.06	1.83	2.65	1.82	4.25	0.70	0.64	0.55	0.88	0.88	1.42	0.88	3.36	3.24	2.77	1.98	3.51	3.25	2.76	2.68	2.03	4.10	2.14	3.04	1.07	3.15	3.03	2.91	3.11	4.35
		15-49	0.40	0.77	0.48	0.47	0.51	0.45	0.42	0.53	0.48	0.92	0.18	0.13	0.18	0.22	0.12	0.32	0.16	0.79	0.89	0.85	0.46	0.77	0.55	0.47	0.82	0.47	1.08	0.50	0.82	0.41	0.91	0.59	0.87	0.92	1.08
		50-69	1.61	3.64	2.90	2.63	2.45	2.31	2.22	2.89	1.83	4.74	0.59	0.56	0.52	0.82	0.96	1.37	0.79	3.15	2.92	2.80	2.17	4.23	3.70	2.78	2.85	2.64	4.34	2.73	2.97	1.14	3.63	2.91	3.01	3.22	5.47
		70+	2.13	5.27	4.32	4.14	3.47	3.31	3.52	4.08	2.66	6.55	0.71	0.67	0.74	1.02	1.14	1.90	0.90	3.61	3.81	3.19	2.85	6.18	4.83	3.30	3.88	4.36	5.73	3.68	3.53	1.58	5.10	3.75	3.83	4.71	7.54
	Female	0-4	1.24	0.67	1.77	2.09	2.70	1.23	1.85	1.47	1.12	4.13	0.49	0.58	0.31	0.79	0.68	0.75	0.53	2.78	3.59	2.01	0.91	2.07	2.38	0.59	1.56	2.03	4.24	2.45	1.99	1.11	4.57	1.06	0.84	3.71	2.70
		5-14	0.93	2.44	1.03	1.18	1.35	1.43	0.96	2.19	0.99	2.91	0.72	0.29	0.28	0.47	0.59	0.55	0.41	1.81	1.72	1.76	1.40	2.48	1.80	1.53	1.24	1.67	2.76	1.26	2.02	1.82	3.43	2.56	4.63	3.98	3.09
		15-49	0.32	0.64	0.38	0.38	0.41	0.38	0.34	0.48	0.36	0.81	0.16	0.10	0.13	0.17	0.12	0.25	0.12	0.63	0.68	0.65	0.35	0.64	0.46	0.40	0.60	0.43	0.91	0.42	0.69	0.40	0.89	0.55	0.95	0.95	0.99
		50-69	0.82	2.35	1.18	1.29	1.42	1.41	1.11	1.99	0.96	2.85	0.53	0.23	0.26	0.39	0.50	0.53	0.34	1.64	1.46	1.49	1.29	2.66	1.83	1.35	1.24	2.02	2.72	1.36	1.67	1.60	3.24	2.24	3.76	3.75	3.85
		70+	1.10	3.21	1.93	2.24	2.13	2.01	1.85	2.59	1.43	3.66	0.58	0.25	0.37	0.47	0.55	0.75	0.40	2.00	1.97	1.90	1.69	3.85	2.43	1.65	1.81	3.35	3.73	2.05	2.01	1.76	4.28	2.96	3.91	5.17	5.27
Self-harm	Male	5-14	2.56	4.29	3.96	3.34	1.30	2.34	2.43	4.09	4.08	2.88	1.06	0.36	1.26	2.13	3.46	2.24	2.90	9.09	4.51	4.19	2.06	2.48	3.81	1.74	0.86	3.72	3.45	4.42	3.66	1.70	4.53	5.09	4.10	2.72	2.50
		15-49	0.52	0.81	0.63	0.56	0.24	0.43	0.49	0.69	0.92	0.52	0.18	0.05	0.36	0.45	0.39	0.42	0.45	1.65	1.00	1.05	0.43	0.44	0.55	0.22	0.19	0.66	0.69	0.87	0.76	0.56	1.09	0.77	0.90	0.57	0.45
		50-69	4.22	7.43	7.62	6.40	2.34	4.17	4.83	6.95	6.56	5.18	1.36	0.54	1.97	3.14	5.88	3.36	4.05	13.19	5.95	6.90	3.55	4.84	7.04	2.73	1.42	8.20	5.99	9.33	5.65	3.08	8.52	7.97	6.75	4.45	5.30
		70+	10.10	18.95	20.01	18.42	5.73	10.56	13.70	17.35	16.97	12.32	2.75	1.13	5.05	6.72	11.47	8.09	7.91	25.61	13.52	13.25	7.91	12.59	16.05	5.49	3.25	24.03	13.77	21.14	11.22	7.58	20.97	18.11	14.44	11.28	13.38
	Female	5-14	1.21	2.67	1.40	1.55	0.68	1.36	1.08	2.85	1.89	1.67	0.89	0.14	0.56	0.97	1.94	0.73	1.12	4.06	1.96	2.27	1.20	1.45	1.77	0.80	0.34	2.66	2.00	2.23	2.07	2.63	4.14	3.67	5.45	2.82	1.31
		15-49	0.34	0.56	0.39	0.38	0.16	0.30	0.32	0.54	0.56	0.38	0.14	0.03	0.21	0.30	0.31	0.26	0.28	1.04	0.63	0.66	0.28	0.31	0.38	0.15	0.11	0.50	0.49	0.62	0.53	0.52	0.94	0.62	0.93	0.53	0.35
		50-69	1.64	3.69	2.44	2.54	1.03	1.87	1.85	3.61	2.60	2.35	0.93	0.16	0.76	1.13	2.22	0.98	1.32	4.99	2.14	2.80	1.51	2.33	2.62	0.98	0.44	5.13	2.92	3.56	2.29	3.66	5.66	4.90	6.36	3.83	2.80
		70+	4.02	8.90	7.12	8.09	2.79	4.96	5.56	8.39	6.93	5.15	1.77	0.32	1.97	2.33	4.01	2.41	2.72	10.84	5.33	6.34	3.58	6.15	6.18	2.09	1.09	15.28	7.12	9.40	4.72	6.78	13.49	11.89	11.09	10.05	7.39
Transport injuries	Male	0-4	9.22	2.41	15.51	18.89	16.06	7.84	12.44	7.71	5.50	13.93	9.01	15.19	4.63	7.74	22.67	5.97	9.36	7.42	17.32	7.26	4.72	6.44	12.26	5.32	6.06	18.41	14.05	17.22	10.01	4.89	19.10	4.91	3.72	18.27	11.11
		5-14	33.2	33.4	51.7	49.7	32.5	34.3	36.4	36.4	26.6	44.1	25.8	26.6	23.3	27.3	47.1	31.6	40.3	18.5	46.2	33.4	32.7	29.7	50.8	52.6	21.3	44.7	37.4	44.6	38.7	20.0	43.1	33.1	32.3	32.9	59.2
		15-49	11.3	10.9	15.8	16.4	11.4	10.6	11.1	10.8	9.1	12.2	12.2	11.8	10.5	8.6	15.2	9.8	11.0	8.1	16.2	11.5	9.3	9.0	12.1	16.1	10.2	16.1	13.6	13.3	15.2	7.4	14.6	10.5	14.8	16.3	16.9
		50-69	34.1	32.5	51.8	54.0	36.1	39.7	41.3	41.1	28.0	46.5	27.5	17.3	20.0	27.2	70.0	36.1	37.2	23.8	61.2	27.8	40.1	30.4	53.4	55.3	25.7	47.4	32.8	55.9	38.3	14.4	49.7	25.8	31.6	35.9	59.0
		70+	73.9	72.9	117.2	113.0	84.7	89.2	93.4	96.4	62.0	103.0	59.9	28.4	41.2	53.9	185.4	76.3	75.5	57.8	122.9	55.2	83.0	66.6	110.7	130.5	68.1	112.1	72.7	134.2	96.0	26.3	110.8	50.0	75.1	85.9	99.3
	Female	0-4	2.86	0.75	4.95	6.29	5.31	2.34	4.02	2.20	1.59	4.18	2.77	4.43	1.36	2.44	7.58	1.80	2.81	2.21	5.81	2.09	1.48	1.86	4.02	1.63	1.59	5.50	4.15	5.42	2.96	1.52	6.10	1.39	1.12	5.54	3.29
		5-14	10.1	11.6	12.0	12.3	11.0	13.5	10.6	15.6	7.5	15.9	14.3	7.4	6.8	7.8	17.4	7.0	10.9	6.4	15.6	10.8	13.5	11.6	15.6	16.8	5.9	17.0	13.0	13.5	14.0	13.0	25.6	14.0	28.0	27.2	32.8
		15-49	4.3	4.3	6.2	6.7	4.5	4.2	4.3	4.5	3.2	4.9	5.2	4.3	3.5	3.1	7.2	3.7	3.8	2.9	6.0	4.0	3.4	3.5	4.8	6.5	3.6	7.2	5.3	5.5	6.0	3.2	6.7	4.4	7.6	8.0	8.0
		50-69	11.9	15.0	13.9	15.7	14.2	18.8	14.2	21.9	10.2	19.7	17.8	5.1	6.6	9.3	30.3	9.7	12.2	10.5	24.5	10.3	18.6	13.5	20.3	22.2	9.5	18.5	13.6	19.9	18.9	9.1	33.3	12.3	30.0	31.6	28.2
		70+	28.6	34.5	39.9	41.3	36.3	42.2	39.7	49.8	26.0	46.8	35.6	10.0	15.7	20.1	83.9	24.1	24.7	24.6	50.2	20.7	37.6	30.8	45.6	52.0	27.6	49.5	31.4	52.1	45.7	17.5	67.0	22.4	58.9	64.7	46.9
Other unintentional injuries	Male	0-4	14.3	7.3	19.0	26.0	30.1	14.1	23.4	17.6	12.5	37.6	7.5	3.6	6.0	14.6	16.6	10.2	8.9	27.4	29.6	19.1	9.1	21.2	23.9	6.3	18.4	26.0	38.1	21.6	21.0	13.1	32.7	10.2	7.8	31.7	8.4
		5-14	7.78	14.02	10.32	11.28	9.49	9.61	10.30	12.57	8.45	17.21	3.57	1.09	4.17	7.03	6.71	7.92	5.62	10.85	11.35	11.33	8.78	14.27	15.17	10.55	11.02	10.80	15.18	8.31	11.95	6.42	10.68	10.47	10.07	10.16	7.08
		15-49	3.00	5.25	3.40	3.94	3.63	3.23	3.57	4.08	3.33	5.46	1.75	0.47	2.19	2.55	1.99	2.70	1.69	4.71	4.64	4.98	2.89	4.85	3.89	3.26	5.72	4.10	6.29	2.80	5.38	3.17	4.28	3.71	5.24	5.49	2.35
		50-69	12.7	23.6	16.6	19.1	16.4	17.0	18.6	22.2	13.9	29.1	5.3	1.3	6.0	11.0	13.3	13.2	8.1	20.1	20.3	17.0	16.2	24.7	24.7	16.3	19.8	17.9	22.6	15.9	19.0	9.0	18.7	14.7	16.1	16.8	10.9
		70+	37.3	72.4	52.3	58.1	51.7	52.0	59.6	70.1	43.0	86.7	14.6	2.9	17.6	29.2	41.9	38.0	21.1	59.9	55.2	43.7	44.2	75.8	69.1	48.0	65.8	59.6	66.4	49.3	58.4	23.6	56.5	39.2	48.4	52.5	27.2
	Female	0-4	5.67	2.85	7.79	11.08	12.28	5.52	9.71	6.67	4.73	14.75	2.92	1.33	2.27	5.84	7.14	3.96	3.45	10.63	11.98	7.11	3.62	7.95	10.08	2.49	6.34	10.25	14.57	8.68	7.94	5.25	13.07	3.86	2.98	12.51	3.21
		5-14	2.65	5.90	2.54	3.17	3.51	4.18	3.25	6.34	2.71	7.17	2.21	0.31	1.31	2.29	2.80	1.93	1.66	4.15	4.04	4.17	3.95	6.26	5.15	3.68	3.22	4.84	5.94	2.77	4.73	5.47	6.99	5.25	10.08	8.82	3.69
		15-49	1.39	2.54	1.56	1.90	1.72	1.57	1.66	2.11	1.44	2.75	0.88	0.20	0.90	1.12	1.09	1.25	0.73	2.22	2.09	2.19	1.29	2.36	1.88	1.59	2.48	2.15	3.05	1.37	2.60	1.70	2.35	1.94	3.19	3.15	1.27
		50-69	4.81	11.66	4.94	6.41	6.99	8.24	6.93	12.25	5.45	13.15	3.66	0.39	2.19	3.97	5.66	3.82	2.76	8.85	8.13	6.74	7.57	11.85	9.82	6.56	7.27	8.47	10.34	6.02	9.19	7.24	12.97	7.90	15.88	15.24	5.59
		70+	15.3	36.0	19.3	24.3	24.7	26.0	26.7	37.7	18.8	41.1	9.4	1.0	7.2	11.2	18.3	12.6	7.4	26.9	23.5	18.8	21.3	38.2	29.7	19.9	27.0	31.5	32.2	21.0	28.3	18.2	36.6	21.0	39.4	43.3	14.1

Discussion

This analysis has produced cause of death estimates for Indonesia at the subnational level using an analytical cause list generated based on the observed cause-specific performance of PCVA data in the largest PCVA validation dataset in existence (5). As of December 2018, I could not find any published literature with a similar use of the PHMRC validation data. This analysis highlights the opportunity to apply similarly-derived cause lists for VA analysis in other locations rather than the current strategy of relying on default cause lists that contain cause groups that favor detail over the reliability of cause of death assignment. The PHMRC-based cause list approach presents a way to create a cause list analytically based on the estimated reliability of cause-of-death assignments in that study. This prevents the inclusion of cause groups with low concordance, which thus can prevent stakeholders from using cause of death estimates based on unreliable VA data.

I used all available verbal autopsy data in Indonesia and cause-specific ICAR models with low computational effort to generate detailed estimates and uncertainty intervals for 28 causes of death by province, age, and sex. These methods can be implemented by anyone with access to R and the INLA package (both free), without the need for a computer cluster system or long periods of code runtime. Each cause-specific model took approximately fourteen seconds to run on a personal computer while running other applications, and 2.4 minutes per model when producing 1000 posterior draws in addition to summary statistics.

Prediction comparisons

Comparison with estimates from other sources can help validate the plausibility of these estimates, and GBD produces the only comparable estimates by year, age, and sex in published literature.(2) The overall correlation observed in log-transformed CSMR between GBD and my age/sex-specific estimates for Indonesia was 0.957, and 0.945 for log-transformed YLLs per capita (Supplementary Table 10). The following causes had correlations below 0.9 for log(CSMR): other malignant neoplasms, drowning, malaria, HIV/AIDS, and self-harm. Comparisons of national-level model fits to the data with those from GBD(2) show that despite lower correlations between these and GBD estimates for drowning, HIV/AIDS, and self-harm log(CSMR), both models follow trends observed in the data for males and females (Supplementary Figure 4). For malaria, GBD estimates were greater than the data for both males and females, over twice the data values for some age groups. This deviation from the data could be due to the influence of covariates or of neighboring countries in the same region and super region as Indonesia, or other custom model specifications. I also included maternal conditions in these comparison figures because of the importance of this cause of death and the disparity in estimates despite its correlation greater than 0.9 (correlation=0.919). This analysis included only verbal autopsy data in Indonesia, and GBD included additional maternal mortality data from sibling histories, which drove GBD estimates higher than this analysis' predictions.

Because of differences in modeling approaches, some variation in estimates is expected. For example, GBD imposes nested location random effects, so that all provinces are nested with equal weight under

Indonesia, which is nested under the region Southeast Asia and super region Southeast Asia, East Asia, and Oceania.(2) Indonesia is a unique country in this super region given the huge population size, diversity of population characteristics across province, the archipelago geography, and history of the country and provinces. An advantage of the ICAR method used is that it allows neighboring provinces to influence one another rather than all having equal weight as in GBD. The spatial adjacency matrix specified for my cause-specific models provides this nuanced geographical structure to inform estimates while maintaining simplicity for clarity and ease of use.

Road traffic injuries are a high profile cause of death in Indonesia with few reliable mortality estimates available. The only study other than GBD that I can compare national estimates of road traffic deaths to is a study by Jusuf et al. in 2017 (56) that compiled written crash reports collected by the National Traffic Police Corps Republic of Indonesia (Korps Lalu Lintas Kepolisian Republik Indonesia) from the Regional Police (Polisi Daerah) from 2004 to 2014. The authors found that approximately 28,000 road traffic fatalities were reported by the police in 2014, equivalent to 12 deaths per 100,000 population. My analysis estimated that road traffic injuries were responsible for almost 47,000 deaths in Indonesia in 2014, or 18 per 100,000 population (Supplementary Table 6). The deaths assessed by the Jusuf study were likely underreported because not every road traffic accident or fatality is reported to the police, and it is difficult to verify the quality of the data that are reported and compiled. Further data collection and comparison with police reporting data over time will improve estimates and facilitate observing time trends.

Limitations

Several limitations affect the strength and interpretation of my predictions, including: the cause list applied, data quality and sparseness, and aspects of the modeling strategy.

Cause list selection

First, the cause list selection is dependent on the PHMRC validation datasets of physician-certified verbal autopsies collected in four countries with different demographics and cause-specific mortality profiles than Indonesia (Supplementary Figure 5) (2). The representativeness of this PHMRC data is irrelevant if we assume that the accuracy of cause-of-death assignment for PCVA depends on the cause of death itself, not sociodemographic, epidemiological, or other contextual factors, but that assumption is difficult to validate. The PHMRC study intentionally created 500 validation datasets drawing from the collected PCVA deaths in order to negate the effect of the epidemiological cause distribution on the calculated concordance of the causes of death, and the effect of sociodemographic/cultural factors has not been quantified. Therefore, for the purpose of assessing the performance of PCVA for various cause lists, the PHMRC validation datasets make possible a quantitative, systematic, and replicable method for choosing an analytical cause list. The challenge encountered here is determining the cutoff values of concordance to use at each level of the analytical hierarchy to ensure some arbitrarily acceptable level of accuracy without losing relevant and important cause detail. For example, the highest accuracy could be attained by simply splitting the data into three cause groups: Group I (Communicable, maternal, neonatal, and nutritional diseases), Group II (Noncommunicable diseases), and Group III (Injuries). However, the loss of more detailed causes of death would drastically limit the relevance and application of those estimates to public health discourse. I attempted to settle these competing interests in this

study by choosing cutoffs at each hierarchical level that balanced both data reliability and cause of death detail. Even after this PHMRC analysis, my final cause list still contains some causes with percent concordance less than 60%, which is somewhat mitigated by the nested structure of the cause list, imposing the size of more accurate aggregated cause groups on the smaller less accurate cause groups to obtain overall more accurate results.

Data quality and sparseness

Related to the first limitation, data quality for some causes of death in Indonesian PCVA data is likely low, just as in the PHMRC PCVA data, though unquantifiable without an Indonesian-specific gold standard study. In addition, the data for this study were collected over several years by separate data collection teams, and there may be some unmeasured non-sampling error related to certain physicians or health workers involved in the data collection, processing, and cause of death assignments. Certainly this last point could lead to unknown cause of death misclassification because both RISKESDAS and IMRSSP used only one trained physician to assign the cause of death to each verbal autopsy according to available documentation, and SRS used two and a tie-breaker when needed.

In addition to data quality, sparse data is a challenge in estimating causes of death in Indonesia. Four provinces have fewer than 25 deaths with cause of death assignment: Kalimantan Utara, Sulawesi Tenggara, Maluku, and Maluku Utara. Papua Barat has none. This data scarcity forces these provinces more than others to depend on covariates and the spatial adjacency matrix to inform their cause distributions.

Even within well-represented provinces, data sparseness is observed in certain age groups, making predictions difficult. Deaths from birth to age 19 account for only approximately 7% of all PCVA deaths in Indonesia, fewer, cumulatively, than any single older age group (50-54, 55-59, . . . 80+). Further, 29 of the 34 provinces have fewer than 30 deaths represented in age group 5-9. Thus there is much more data to inform cause distributions among older adults than infants, children, and young adults. Age/sex-specific covariates are included when possible to help inform age trends where data are sparse.

Interpretation of all results should be made with care, as there are large uncertainty intervals around estimates. This uncertainty highlights the need for more data, particularly more accurate reporting of deaths and their causes in smaller provinces and in less-represented age groups. Cause of death estimation in Indonesia can improve and reduce uncertainty if additional data are collected through an expanded sample registration system.

Finally, aggregating all verbal autopsy data across years to mitigate problems caused by data sparseness may have introduced compositional bias in the results if cause compositions have changed over time because different provinces have a different mix of years represented.

The planned expansion of the Sample Registration System will help fill these data gaps and provide a more consistent source of cause of death data for Indonesia. In the meantime, the estimated uncertainty intervals reported in the Supplementary Tables attempt to capture the greater uncertainty around predictions by incorporating both sampling error of the data points and uncertainty in the estimation process itself.

Model strategy

In the model specifications, selected covariates are included as fixed effects regardless of their estimated directionality (unlike in GBD 2016) because with cause fractions, the covariate-cause fraction relationships are affected by all other causes simultaneously (as opposed to rate models). I chose not to impose directionality here because of a lack of strong evidence to do so with Indonesia's cause distribution. One could consider applying directionality based on when the covariate's slope on the rate would be expected to be, say, lower for one cause than other causes, leading to a positive cause fraction slope for that covariate. The multidimensional complexity of such an exercise across all cause group relationships prevented its exploration in this analysis and may warrant its own analysis in order to better understand the covariate relationships in these predictive models.

I aggregated all of the PCVA data for Indonesia across years from 2007 to 2014 in order to maximize age/sex-specific sample sizes within provinces and obtain more stable estimates from these larger sample sizes, especially in smaller and less-represented provinces. The main downsides of this strategy are losing the ability to estimate a trend over time, and allowing cause distributions from several years prior to influence estimates for 2014, without capturing any changes in the epidemiological profiles of provinces over that time period.

Conclusion

This analysis presents a set of comprehensive, consistent cause-specific mortality estimates for Indonesian provinces based on a cause list developed based on the reliability of physician-certified verbal autopsy cause of death assignments. With the shift of decision-making toward provinces after the recent (1999) decentralization, provinces can use these estimates to help inform health policy decisions and to catalyze investigations into differences across provinces. Until now, national estimates had limited relevance to these provincial public health discussions. Given the heterogeneity of the Indonesian population, these subnational estimates will be valuable for better understanding the health of the people and for planning current and future prevention and care of this disease burden.

Verbal autopsy data are a useful source of information about mortality in the absence of a vital registration system, but to date they have tended to be presented with detailed cause groups and occasionally disclaimers about accuracy, rather than being presented with cause groups determined by their accuracy.(57–60) The presented cause list generation process can be applied to VA data in other locations using any cause-of-death assignment approach included in the PHMRC study: physician certification, the Tariff Method, InterVA, random forests, Simplified Symptom Pattern Method, InSilicoVA, and the King and Lu method of direct estimation. Additionally, local validation studies of verbal autopsy data are rolling out and could also be used in a similar manner to inform the cause list used to interpret their collected verbal autopsy data.(61–64) Collecting verbal autopsy data is not an end in itself; it is necessary to present findings based on these data in a way that is not only relevant and detailed enough to be useful to stakeholders but also, and more importantly, faithful to the data reliability itself and at a level of detail that can incur a known level of confidence in the results.

References

1. Statistics Indonesia [Internet]. [cited 2018 Jul 26]. Available from: https://www.bps.go.id/subject/30/kesehatan.html#subjekViewTab3

2. GBD 2016 Mortality Collaborators. Global, regional, and national under-5 mortality, adult mortality, age-specific mortality, and life expectancy, 1970-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Lond Engl. 2017 Sep 16;390(10100):1084–150.

3. WHO. Verbal autopsy standards: ascertaining and attributing causes of death. Geneva: World Health Organization; 2007.

4. Murray CJ, Lopez AD, Black R, Ahuja R, Ali SM, Baqui A, et al. Population Health Metrics Research Consortium gold standard verbal autopsy validation study: design, implementation, and development of analysis datasets. Popul Health Metr. 2011;9:27.

5. Lozano R, Lopez AD, Atkinson C, Naghavi M, Flaxman AD, Murray CJ, et al. Performance of physician-certified verbal autopsies: multisite validation study using clinical diagnostic gold standards. Popul Health Metr. 2011 Aug 4;9:32.

6. Byass P. Usefulness of the Population Health Metrics Research Consortium gold standard verbal autopsy data for general verbal autopsy methods. BMC Med. 2014 Feb 4;12:23.

7. Snow RW, Armstrong JR, Forster D, Winstanley MT, Marsh VM, Newton CR, et al. Childhood deaths in Africa: uses and limitations of verbal autopsies. Lancet Lond Engl. 1992 Aug 8;340(8815):351–5.

8. Quigley MA, Armstrong Schellenberg JR, Snow RW. Algorithms for verbal autopsies: a validation study in Kenyan children. Bull World Health Organ. 1996;74(2):147–54.

9. Rodriguez L, Reyes H, Tome P, Ridaura C, Flores S, Guiscafre H. Validation of the verbal autopsy method to ascertain acute respiratory infection as cause of death. Indian J Pediatr. 1998 Aug;65(4):579–84.

10. Kahn K, Tollman SM, Garenne M, Gear JS. Validation and application of verbal autopsies in a rural area of South Africa. Trop Med Int Health TM IH. 2000 Nov;5(11):824–31.

11. Setel PW, Whiting DR, Hemed Y, Chandramohan D, Wolfson LJ, Alberti KGMM, et al. Validity of verbal autopsy procedures for determining cause of death in Tanzania. Trop Med Int Health TM IH. 2006 May;11(5):681–96.

12. Quigley MA, Chandramohan D, Setel P, Binka F, Rodrigues LC. Validity of data-derived algorithms for ascertaining causes of adult death in two African sites using verbal autopsy. Trop Med Int Health TM IH. 2000 Jan;5(1):33–9.

13. Yang G, Rao C, Ma J, Wang L, Wan X, Dubrovsky G, et al. Validation of verbal autopsy procedures for adult deaths in China. Int J Epidemiol. 2006 Jun;35(3):741–8.

14. Freeman JV, Christian P, Khatry SK, Adhikari RK, LeClerq SC, Katz J, et al. Evaluation of neonatal verbal autopsy using physician review versus algorithm-based cause-of-death assignment in rural Nepal. Paediatr Perinat Epidemiol. 2005 Jul;19(4):323–31.

15. Population Health Metrics Research Consortium (PHMRC) [Internet]. Institute for Health Metrics and Evaluation. 2014 [cited 2018 Nov 7]. Available from: http://www.healthdata.org/population-health-metrics-research-consortium-phmrc

16. Gingo MR, Morris A. Pathogenesis of HIV and the Lung. Curr HIV/AIDS Rep. 2013 Mar;10(1):42–50.

17. HIV (Human Immunodeficiency Virus) Infection | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/health-library/hw151408

18. Pneumonia | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/health-library/hw63868

19. Lung Cancer | Kellogg Eye Center | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.umkelloggeye.org/health-library/hw183816#tm1357

20. COPD & Emphysema | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/copd-emphysema

21. Asthma | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/asthma

22. Tuberculosis | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/tuberculosis

23. Cystic Fibrosis (Adults) | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/cystic-fibrosis

24. Interstitial lung disease - Symptoms and causes - Mayo Clinic [Internet]. [cited 2018 Jul 25]. Available from: https://www.mayoclinic.org/diseases-conditions/interstitial-lung-disease/symptoms-causes/syc-20353108

25. Pleural Diseases | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pleural-diseases

26. Pulmonary Fibrosis | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/pulmonary-fibrosis

27. Sarcoidosis | Michigan Medicine [Internet]. [cited 2018 Jul 25]. Available from: https://www.uofmhealth.org/conditions-treatments/pulmonary/sarcoidosis

28. Research Agency for Research and Development. RISKESDAS 2007 [Internet]. Indonesia Ministry of Health. [cited 2018 Aug 11]. Available from: http://labmandat.litbang.depkes.go.id/riset-badan-litbangkes/menu-riskesnas/menu-riskesdas/147-rkd-2007

29. WHO | World Health Survey [Internet]. WHO. [cited 2018 Oct 8]. Available from: http://www.who.int/healthinfo/survey/en/

30. Rao C, Soemantri S, Djaja S, Suhardi, Adair T, Wiryawan Y, et al. Mortality in Central Java: results from the indonesian mortality registration system strengthening project. BMC Res Notes. 2010 Dec 2;3:325.

31. NIHRD. Pengisian kuesioner autopsi verbal dalam menegakkan diangosis pemyebab kematian menurut ICD-10: Panduan untuk paramedis puskesmas.[Completion of verbal autopsy questionnaires to support the diagnosis of cause of death according to ICD-10: Guide for health centre paramedics]. Jakarta: National Institute of Health Research and Development (NIHRD)Ministry of Health Republic of Indonesia; 2008.

32. NIHRD. Pengisian sertifikat medis penyebab kematian menurut ICD-10: Panduan untuk dokter. [Completion of medical certificate of cause of death according to ICD-10: Guide for doctors]. Jakarta: National Institute of Health Research and Development, Ministry of Health, Republic of Indonesia; 2008.

33. NIHRD. Menegakkan diagnosis penyebab kematian menurut ICD-10 dari data autopsi verbal: Panduan untuk dokter. [Diagnosing causes of death from verbal autopsy data according to ICD-10: Guide for doctors]. Vol. 612.2. Jakarta: National Institute of Health Research and Development, Ministry of Health, Republic of Indonesia; 2008.

34. The Republic of Indonesia Health System Review [Internet]. Vol. 7. Asia Pacific Observatory on Health Systems and Policies; 2017 [cited 2018 Oct 8]. Available from: http://apps.who.int/iris/bitstream/handle/10665/254716/9789290225164-eng.pdf;jsessionid=125AE6CF6225BA8EDE3E1F7A49E4CA00?sequence=1

35. Widyastuti R. Sample Registration System. Center for Health Management and Humanities, Indonesia Agency for Health Research and Development, Ministry of Health; 2017.

36. Hierarchical Modeling and Analysis for Spatial Data - CRC Press Book [Internet]. [cited 2018 Nov 28]. Available from: https://www.crcpress.com/Hierarchical-Modeling-and-Analysis-for-Spatial-Data/Banerjee-Carlin-Gelfand/p/book/9781439819173

37. Bayesian Disease Mapping: Hierarchical Modeling in Spatial Epidemiology, Third Edition - CRC Press Book [Internet]. [cited 2018 Nov 28]. Available from: https://www.crcpress.com/Bayesian-Disease-Mapping-Hierarchical-Modeling-in-Spatial-Epidemiology/Lawson/p/book/9781138575424

38. Lee D. A comparison of conditional autoregressive models used in Bayesian disease mapping. Spat Spatio-Temporal Epidemiol. 2011 Jun;2(2):79–89.

39. Martino S, Rue H. Implementing Approximate Bayesian Inference using Integrated Nested Laplace Approximation : a manual for the inla program. 2008 [cited 2018 Aug 10]; Available from: /paper/Implementing-Approximate-Bayesian-Inference-using-%3A-Martino-Rue/0f9740f74ab792aff535d821ceff8b464b7a3c92

40. Besag J, York J, Mollié A. Bayesian image restoration, with two applications in spatial statistics. Ann Inst Stat Math. 1991 Mar;43(1):1–20.

41. Blangiardo M, Cameletti M, Baio G, Rue H. Spatial and spatio-temporal models with R-INLA. Spat Spatio-Temporal Epidemiol. 2013 Mar;4:33–49.

42. Good IJ. Rational Decisions. J R Stat Soc Ser B Methodol. 1952;14(1):107–14.

43. Geisser S. Predictive Inference. CRC Press; 1993. 280 p.

44. Gelfand A, Dey D, Chang H, editors. Model Determination Using Predictive Distributions with Implementation via Sampling-Based Methods | Department of Statistics. Oxf Univ Press [Internet]. 1992 [cited 2018 Aug 10]; Available from: https://statistics.stanford.edu/research/model-determination-using-predictive-distributions-implementation-sampling-based-methods

45. Congdon P. Regression Models. In: Applied Bayesian Modelling [Internet]. Wiley-Blackwell; 2003 [cited 2018 Aug 10]. p. 79–133. Available from: https://onlinelibrary.wiley.com/doi/abs/10.1002/0470867159.ch3

46. Gilks W, Richardson S, Spiegelhalter D, editors. Markov Chain Monte Carlo in Practice - CRC Press Book [Internet]. Boca Raton, FL: Chapman & Hall/CRC; 1996 [cited 2018 Aug 10]. Available from: https://www.crcpress.com/Markov-Chain-Monte-Carlo-in-Practice/Gilks-Richardson-Spiegelhalter/p/book/9780412055515

47. Pettit LI. The Conditional Predictive Ordinate for the Normal Distribution. J R Stat Soc Ser B Methodol. 1990;52(1):175–84.

48. Wang X, Ryan YY, Faraway JJ. Bayesian Regression Modeling with INLA. CRC Press; 2018. 325 p.

49. Gneiting T, Raftery AE. Strictly Proper Scoring Rules, Prediction, and Estimation. J Am Stat Assoc. 2007 Mar 1;102(477):359–78.

50. Adrion C, Mansmann U. Bayesian model selection techniques as decision support for shaping a statistical analysis plan of a clinical trial: An example from a vertigo phase III study with longitudinal count data as primary endpoint. BMC Med Res Methodol. 2012 Sep 10;12:137.

51. FAQ - The R-INLA project [Internet]. [cited 2018 Aug 10]. Available from: http://www.r-inla.org/faq#TOC-How-can-I-compute-cross-validation-or-predictive-measures-of-fit-

52. Rue H, Martino S, Chopin N. Approximate Bayesian inference for latent Gaussian models by using integrated nested Laplace approximations - Rue - 2009 - Journal of the Royal Statistical Society: Series B (Statistical Methodology) - Wiley Online Library. J R Stat Soc Ser B. 2009;71(2):319–92.

53. Mantovan P, Secchi P. Complex Data Modeling and Computationally Intensive Statistical Methods [Internet]. Mailand: Springer-Verlag; 2010 [cited 2018 Aug 10]. (Contributions to Statistics). Available from: //www.springer.com/us/book/9788847013858

54. Faraway J. INLA for linear regression [Internet]. 2017 [cited 2018 Aug 10]. Available from: http://www.maths.bath.ac.uk/~jjf23/brinla/chicago.html#conditional-predictive-ordinates-and-probability-integral-transform

55. GBD 2013 Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015 Jan 10;385(9963):117–71.

56. Jusuf A, Nurprasetio IP, Prihutama A. Macro Data Analysis of Traffic Accidents in Indonesia. J Eng Technol Sci. 2017 Apr 30;49(1):132–43.

57. Hutain J, Perry HB, Koffi AK, Christensen M, Cummings O'Connor E, Jabbi S-MB-B, et al. Engaging communities in collecting and using results from verbal autopsies for child deaths: an example from urban slums in Freetown, Sierra Leone. J Glob Health. 2019 Jun;9(1):010419.

58. Gouda HN, Hazard RH, Maraga S, Flaxman AD, Stewart A, Joseph JC, et al. The epidemiological transition in Papua New Guinea: new evidence from verbal autopsy studies. Int J Epidemiol. 2019 Mar 26;

59. Thomas L-M, D'Ambruoso L, Balabanova D. Verbal autopsy in health policy and systems: a literature review. BMJ Glob Health. 2018;3(2):e000639.

60. GBD 2017 Causes of Death Collaborators. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Lond Engl. 2018 Nov 10;392(10159):1736–88.

61. Karat AS, Maraba N, Tlali M, Charalambous S, Chihota VN, Churchyard GJ, et al. Performance of verbal autopsy methods in estimating HIV-associated mortality among adults in South Africa. BMJ Glob Health. 2018;3(4):e000833.

62. Omar A, Ganapathy SS, Anuar MFM, Khoo YY, Jeevananthan C, Maria Awaluddin S, et al. Cause-specific mortality estimates for Malaysia in 2013: results from a national sample verification study using medical record review and verbal autopsy. BMC Public Health. 2019 Jan 24;19(1):110.

63. Dehghan A, Nasirian M, Haghdoost AA, Bahramali E, Sharifi H. Validation of the verbal autopsy questionnaire for adult deaths in Iran. Med J Islam Repub Iran. 2018;32:7.

64. Tran HT, Nguyen HP, Walker SM, Hill PS, Rao C. Validation of verbal autopsy methods using hospital medical records: a case study in Vietnam. BMC Med Res Methodol. 2018 18;18(1):43.

Tables

Supplementary Tables

Figures

Supplementary Figures

Chapter 3

A comparison of four methods to calculate burden of disease attributable to fine particulate matter

Introduction

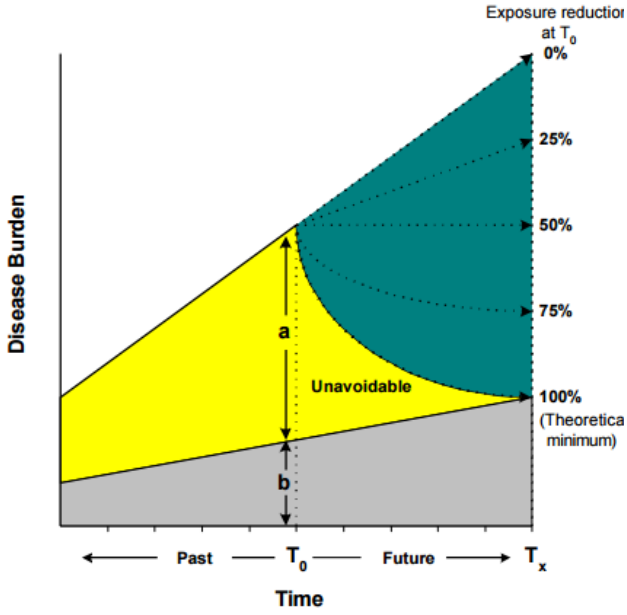
Accurate estimates of burden of disease attributable to various risk factors are a critical input to the health profile of a population. These estimates can help inform policy discussions about disease and risk factor priorities in a population for prevention and intervention.

In order to prevent future burden and better manage current burden, it is important to understand as much as possible about the full disease pathways, from distal to proximal factors, and a comparative risk assessment (CRA) is one tool to do so for part of the pathways. These risk factors can be systemic, such as water quality or access to improved sanitation; modifiable at the individual level, such as active smoking; both systemic and modifiable at the individual level, such as household air pollution which is driven both by the supply distribution of various fuel types and by individual choice; and caused by a variety of factors, such as ambient air pollution which can be driven by forest fires, dust storms, transportation exhaust and smoke, power generation pollution, or industry byproducts.

The purpose of CRA in the context of public health is to help establish priorities for communities or government agencies when allocating limited resources for reducing or preventing health burden. It is also a useful tool to educate various stakeholders about the landscape of risks and to provide information about problems that may need to be addressed in the future.(1,2) More specifically, CRA in public health assesses the attributable or avoidable burdens of disease (using a relevant metric like deaths or DALYs) due to various risk factors or in order to compare their respective magnitudes and trends. Figure 1 below describes this quantification for a single risk factor. The gray area is the underlying rate of disease burden in a population, that is unaffected by any changes in the specified risk factor. The yellow area is the disease burden attributable to past exposure to the risk factor (assuming T_0 is the present time), regardless of any changes to the risk exposure. In this example, even if the risk exposure reduced completely to the theoretical minimum (for example, all smokers quit smoking at T_0), some disease burden continues to occur because of delayed disease onset or survival of patients with chronic diseases due to the risk factor. The dark blue portion of the graph represents future disease burden attributable to a risk factor and avoidable by exposure reduction to various levels. Zero exposure reduction assumes the disease burden and risk exposure continue to follow trends prior to T_0.(3)

The World Health Organization used CRA in the first major update to the Global Burden of Disease (GBD) in 2000-2002 to assess global disease burden attributable to 26 risk factors, and again in the 2004 update for 28 risk factors.(4,5) WHO and GBD began creating separate estimates when GBD 2010 was published by the Institute for Health Metrics and Evaluation in 2012. Annual GBD publications use CRA to assess burden attributable to more than 80 risk factors at the country level (6), and the WHO produces estimates sporadically as topical reports.(7) The World Health Organization has compiled a generalizable methodology for comparative risk analysis which involves the following steps:

Figure 1. Conceptual schematic of attributable and avoidable disease burden related to a risk factor exposure over time. (3)



Burden not attributable to or avoidable with the risk factor of interest

Burden attributable to prior exposure

Burden avoidable with exposure reduction at T_0

a = disease at T_0 attributable to prior exposure
b = disease at T_0 not attributable to the risk factor (caused by other factors)
c = avoidable disease at T_x with a 50% exposure reduction at T_0
d = disease at T_x after a 50% reduction in risk factor
Attributable fraction at T_0 due to prior exposure = $a / (a + b)$
Avoidable fraction at T_x due to 50% exposure reduction at T_0 = $c / (c + d)$. In general avoidable burden at T_y due to exposure reduction at T_0 is given by the ratio of the green area to total burden at T_y. Dashed arrows represent the path of burden after a reduction at T_0. Policy choices for feasible, plausible, and cost-effective exposure reductions can be chosen from the range of distributional transitions.
Note that the burden attributable to other risk factors (grey area) may be decreasing, constant, or increasing over time. The last case is shown in the figure.

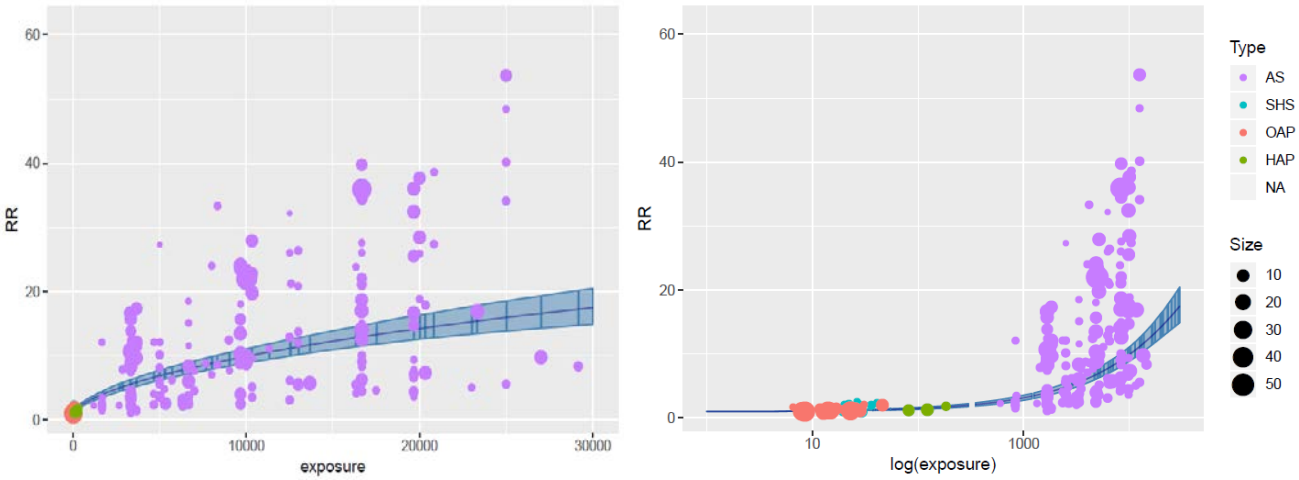
1. Select risk-outcome pairs using certain criteria. First, there must be sufficient evidence for causal effect and in order to estimate the outcome-specific effect sizes. Convincing or probable evidence is sufficient as defined by the World Cancer Research Fund grading system.(8)
2. Estimate the exposure distributions of each risk factor by geography, and also by age and/or sex where appropriate.
3. Estimate cause-effect (also known as dose-response) sizes for varying changes in exposure levels. These are usually the relative risk of outcome per unit of exposure for each selected risk-outcome pair. The effect size could be estimated on a continuous scale using integrated exposure-response curves, such as with air pollution measured by $PM_{2.5}$ concentrations. Alternatively, the effect size could be estimated for ordered categorical exposures, such as diarrhea incidence RR for non-exclusive breastfeeding.
4. Choose counterfactual exposure level. One common approach is using the theoretical minimum risk exposure level (such as zero smoking), while another option is choosing an exposure level or distribution that is actually observed in some population (such as the lowest observed active smoking prevalence rates).
5. Estimate attributable burden by calculating and applying population attributable fractions to outcome estimates.
6. Estimate uncertainty around final estimates by accounting for uncertainty at each step of the estimation process.

This analysis focuses on comparing four methods for calculating relative risks (RR) and aggregate population attributable fractions (PAF) from steps 3 and 5 above for four risk factors of exposure to

particulate matter with diameter less than 2.5 microns ($PM_{2.5}$, or fine particulate matter): ambient air pollution (AAP), household air pollution (HAP), active smoking (AS), and secondhand smoke (SHS). These risk factors were chosen because fine particulate matter is considered the main source of their related health outcomes.(6,9) Disease pathways related to $PM_{2.5}$ are not well characterized (10,11) but could include oxidative stress, pulmonary inflammation, accelerated atherosclerosis, endothelial changes, and altered cardiac autonomic function due to oxygen deprivation from the particulates traveling to alveoli.(10,12)

For step 3, AAP, HAP, AS, and SHS can all be defined in terms of $PM_{2.5}$ concentration to create integrated exposure-response (IER) curves based on studies that contain exposures and relative risks of disease morbidity and/or mortality. Cohort studies on health effects of ambient air pollution alone generally had a limited range of exposure levels, which made extrapolation of those relative risks to populations with higher exposure levels ill-advised.(13–15) Then Pope et al in 2011 proposed integrating exposure-outcome data from multiple sources of $PM_{2.5}$ exposure, such as active smoking, secondhand smoke, and ambient air pollution, into a single exposure-response curve to inform the shape of relative risks at higher exposure levels than have been included in studies examining the health risks of air pollution.(9,16) Figure 2 shows an example of an integrated exposure response curve (IER) from GBD, for lung cancer mortality based on pooling studies of AAP, HAP, AS, and SHS.(6) This method presumes that particulate size, rather than composition, is the main source of health outcomes.

Figure 2. IER curve of lung cancer mortality plotted over estimated daily exposure of $PM_{2.5}$ (ug/m^3) in normal space (left) and log space (right) to more clearly display the data at lower exposure values. Figures obtained from GBD.(6)



For these GBD IER curves, each type of exposure was converted to a concentration of $PM_{2.5}$ and then the IER curves were generated using a Bayesian MCMC approach along with a modified power function.(6,17) The exposure conversions for HAP and SHS were obtained from studies measuring $PM_{2.5}$ concentrations within households using solid cooking fuel and smoking, respectively.(6) The conversion for AS was a combination of literature and expert input.(6,18) Given an observed exposure concentration level in the population, one could extract the associated relative risk from these estimated curves to then calculate attributable burden.

All four of these risk factors affect the cumulative $PM_{2.5}$ levels experienced by subsets of a population and the resulting RRs. For example, all smokers are also exposed to AAP, and that underlying AAP $PM_{2.5}$ exposure should be taken into account when extracting corresponding RR and the theoretical minimum RR. If everyone stopped smoking, they would still be exposed to AAP and therefore their RR would not drop to 1, but rather to the level associated with their AAP exposure. GBD 2017 is the first GBD iteration to partially adjust HAP and AAP attributable burden for each other using a proportional PAF approach, but neither GBD nor WHO adjust each one of these four risk factors for every combination of the others experienced in the population. (6,19–21) Briefly, the proportion PAF approach uses the integrated exposure response to "calculate a relative risk and PAF for the combined exposure to fine particulate matter from ambient and household sources, and these are then weighted by the proportion of individuals exposed to each source" to calculate the PAF for ambient air pollution and household air pollution separately.(21)

Additionally, for step 5, these risk factors affect one another when aggregating PAFs because their exposures are all on the same scale. WHO and GBD both currently utilize a multiplicative aggregation of the PAFs for these four risk factors, with the exception of HAP and APP in GBD 2017 using the proportional PAF approach. (21) This approach ignores the common pathway to disease due to these risks through cumulative exposure to fine particulate matter, leading to overestimates of their attributable burden.

GBD does integrate many associations for other risk factors into RR and PAF calculations, referred to as "patterns of association between risk factors" (GBD Risk Factors appendix pages 24-25 (6)), which include confounding, mediation, and latent variables. I argue that there is a fourth "additive" pattern to take into consideration when calculating and aggregating PAFs, wherein risk factors interact through the same defined exposure, i.e. $PM_{2.5}$, for the risk factors chosen here, and their exposure levels can be summed.

This analysis compares four methods of calculating and aggregating population attributable fractions (PAFs) for risk factors whose primary pathway to disease is $PM_{2.5}$ concentration, including the methods used by the WHO and GBD, in an effort to further elucidate the effects of these risks on attributable burden and recommend an alternative equation to calculate that attributable burden.

Methods

The four risk factors were chosen for this analysis because their main source of health outcomes is fine particulate matter: ambient air pollution, household air pollution, active smoking, and secondhand smoke. Associated outcomes for these risk factors were chosen to create risk-outcome pairs if:

1. that outcome is associated with all four risk factors,
2. an IER curve exists in GBD for that risk-outcome pair, either by age or for all ages, and
3. the outcome corresponds with a cause of death estimated in Aim 2.

Four outcomes meet these criteria: lower respiratory infections, lung cancer, ischemic heart disease, and stroke. Excluded outcomes include breast cancer, cervical cancer, cataracts, COPD, type 2 diabetes, falls, otitis media, tuberculosis, and 44 other causes associated only with active smoking. The third

criterion only excluded type 2 diabetes because Aim 2 results do not differentiate between types 1 and 2 diabetes.

Cause of death estimates for these outcomes were obtained from Aim 2 results for Indonesia by age and sex for 2014, and all exposure estimates were obtained from the GBD database as of September 2018 (6) or generated as described below. Province-level estimates were excluded from this analysis because one set of national estimates is sufficient for this comparison of burden attribution methods and including more locations would detract from this primary analytical goal.

Rick factor exposures

Exposure for each risk factor is defined by two quantities: the proportion of the population exposed to that risk factor, and the PM2.5 concentration associated with exposure to that risk factor among those exposed. Both quantities are described for each risk factor below.

Ambient air pollution

Ambient air pollution $PM_{2.5}$ exposure is defined as the annual concentrations of $PM_{2.5}$ imputed from satellite data of atmospheric aerosol levels and atmospheric chemistry transport models, and calibrated to ground-level air particulate monitoring data in over 70 countries. After establishing this relationship, population-weighted ground-level air $PM_{2.5}$ levels were estimated by GBD for all countries as posterior distributions represented by 1,000 draws of the average PM2.5 exposure experienced across each country with uncertainty.(6) These draws for Indonesia in 2014 were used as AAP $PM_{2.5}$ exposure in this analysis.

Household air pollution

Household air pollution exposure proportion is defined as the proportion of the population that lives in a household that primarily uses solid cooking fuels (coal and biomass), which cause the majority of household air pollution.(6,17,19,22) For these analyses, other contributions by factors like candles are considered negligible compared to the levels produced by cooking with coal or biomass.(6,17,19) These data do not directly include the type of fuel used specifically for heating, which could differentially contribute to indoor air pollution.(22)

The associated $PM_{2.5}$ concentration for HAP was obtained from GBD, in the form of 1,000 draws from the estimated posterior distribution.(6) GBD collected almost 90 studies measuring $PM_{2.5}$ in households using solid cooking fuels and predicted exposure for all location-years using socio-demographic and study-level factors as covariates. This method imposes variation in PM2.5 concentration by using the sociodemographic index (SDI) as a proxy for quality of housing (e.g., ventilation) and type of solid cooking fuel. These predicted $PM_{2.5}$ concentrations were then crosswalked to values for men, women, and children (under age 25) using the ratio of each group's mean exposure to the overall mean personal exposure.(21) These ratios were based on data extracted from seven studies found in a literature review conducted by GBD.(23–30)

Active smoking

The prevalence of active smoking by age and sex was obtained from the GBD database as of September 2018 as 1,000 draws from the estimated posterior distribution. Active smoking was defined as those who currently smoke daily.(6)

In order to utilize the IER curves for AS, the $PM_{2.5}$ concentration corresponding to AS is needed. When creating the IER curves, GBD currently applies a constant $PM_{2.5}$ concentration of 667 ug/m^3 per cigarette across all active smokers, regardless of type of cigarette or different lung volumes by sex.(21) This concentration is based on work conducted by Pope et al (9,10), who assumed an average breathing rate of 18 m^3/day and inhaled dose of 12,000 ug $PM_{2.5}$ mass per cigarette when constructing IER curves. The breathing rate was selected using expert opinion, and the $PM_{2.5}$ mass from a cigarette study conducted in the United States by the National Cancer Institute on cigarettes sold in the US from the late 1970s to the early 1990s.(9,10,18)

I developed a sex-specific $PM_{2.5}$ smoking exposure concentration by converting average daily cigarette consumption per smoker to average daily $PM_{2.5}$ concentrations for smokers. This conversion is specific to Indonesia because it takes into account the average number of cigarettes smoked among active smokers in Indonesia by province, age, and sex with uncertainty; the types of cigarettes smoked in Indonesia; and their respective particulate matter emissions. It could be applied to a different population by gathering $PM_{2.5}$ mass data on the types of cigarettes smoked in that population. In countries dominated by white filtered cigarettes, the concentration used by Pope et al and GBD would be appropriate for smokers.

Here, I developed a more nuanced estimate for the daily $PM_{2.5}$ concentration experienced by active smokers, taking into account sex and types of cigarettes smoked. This concentration is defined as the average of both the $PM_{2.5}$ concentration in the puffs of cigarettes smoked and the $PM_{2.5}$ concentration for secondhand smoke, weighted by the proportion of the day spent breathing each, as in:

$$[\mathbf{PM_{2.5}}]^{\boldsymbol{AS}} = [\boldsymbol{PM2.5}]^{\boldsymbol{puff}} * \%\ \mathbf{of\ day\ puffing} + [\mathbf{PM_{2.5}}]^{\boldsymbol{SHS}} * \%\ \mathbf{of\ day\ not\ puffing} \quad \text{(Equation 1)}$$

Which can be rewritten in more detail as:

$$[\mathbf{PM_{2.5}}]^{\boldsymbol{AS}}_{\boldsymbol{p,a,s}} = [\mathbf{PM_{2.5}}]^{\boldsymbol{puff}} * \frac{\boldsymbol{Cigspd_{p,a,s} * Puffs * PuffTime}}{\frac{\mathbf{60\ sec}}{\mathbf{min}} * \frac{\mathbf{60\ min}}{\mathbf{hr}} * \frac{\mathbf{24\ hr}}{\mathbf{day}}} + [\mathbf{PM_{2.5}}]^{\boldsymbol{SHS}} * (\mathbf{1} - \frac{\boldsymbol{Cigspd_{p,a,s} * Puffs * PuffTime}}{\frac{\mathbf{60\ sec}}{\mathbf{min}} * \frac{\mathbf{60\ min}}{\mathbf{hr}} * \frac{\mathbf{24\ hr}}{\mathbf{day}}}) \quad \text{(Equation 2)}$$

where

$$[\mathbf{PM_{2.5}}]^{\boldsymbol{puff}} = \frac{\mathbf{PM_{2.5_s}}}{\boldsymbol{Puffs} * (\boldsymbol{FRC_s} + \boldsymbol{V^{puff}})} \quad \text{(Equation 3)}$$

Where p is province, a is age, s is sex, $Cigspd_{p,a,s}$ is the province/sex/age-specific distribution of the number of cigarettes smoked per day obtained from the GBD database(6), $\mathrm{PM}_{2.5_s}$ is the sex-specific $PM_{2.5}$ level, $Puffs$ is the average number of puffs per cigarette smoked, FRC_s is the functional residual

volume by sex, V^{puff} is the average volume of an inhaled puff of cigarette smoke according to the Federal Trade Commission standardized smoking practices, and $[\mathrm{PM}_{2.5}]^{SHS}$ is the $PM_{2.5}$ concentration applied to secondhand smoke. Table 1 shows the values used for each of these parameters that are constants.

Table 1. Quantities used for estimating $[\mathbf{PM2.5}]^{AS}$ among current smokers. Values in bold were constants applied to the Equations 2 and 3 above.

	Cigarette type	Sex	Value	Units	Method	Sources
$\mathrm{PM}_{2.5_s}$	White	Males, Females	12,000	ug/cig	Weighted average by proportion consumed of each type of cigarette in Indonesia (92.5% kretek, 7.5% white) among smokers	(6,18,31)
	Kretek	Males	37,000			(31,32)
		Females	29,300			
	Weighted average	**Males**	**35,125**			(33)
		Females	**28,003**			
$[\mathrm{PM}_{2.5}]^{SHS}$		Males, Females	**32.1**	ug/m^3	Average of 7 sources weighted by sample size	(34–40)
$Puffs$	White	Males, Females	9.43	Puffs/cigarette	Weighted average of two studies	(31,41)
	Kretek	Males, Females	14.90			
	Weighted average	**Males, Females**	**14.49**		Weighted average by proportion consumed of each type of cigarette in Indonesia (92.5% kretek, 7.5% white)	
FRC_s		Males	**0.0023**	m^3		(42)
		Females	**0.0018**			
V^{puff}	All	Males, Females	**0.000035**	m^3/puff	FTC standardized smoking practices	(18)
$PuffTime$	All	Males, Females	**2**	Seconds/puff	FTC standardized smoking practices	(18)

I obtained the draw-level distributions of the province/age/sex-specific estimates of the number of cigarettes smoked daily by smokers in Indonesia in 2014 from the GBD database.(6) I applied these estimates to the Equation 2 above, along with the values in Table 1, to calculate 1000 draws of average $PM_{2.5}$ concentration for active smokers.

Secondhand smoke

Secondhand smoke is defined as the proportion of the population who are nonsmokers living with at least one person who is a current daily smoker. Smokers are also exposed to secondhand smoke, both from their own cigarettes and from exposure to other smokers. The former is accounted for in the Active smoking exposure estimation, and the latter is considered a negligible additional source of $PM_{2.5}$ among smokers for this analysis. Population-representative surveys provided data on smoking status of the head of household, and other members of households were assigned smoking status based on the probability of smoking by location, age, sex, and year estimated in GBD. From those inputs, the prevalence of SHS was modeled in GBD by location, age, sex, and year, and those posterior distributions of 1000 draws are utilized in this analysis.(6)

Currently, GBD defines $PM_{2.5}$ exposure from SHS based on four studies aggregated by Semple et al. 2015, which found an average $PM_{2.5}$ concentration of 31 ug/m^3 in households with smoking.(34) This study does not report comparative $PM_{2.5}$ concentrations in nonsmoking households, so GBD does not currently adjust this 31 ug/m^3 for background fine particulate matter from sources other than SHS.

I sought to find more studies to inform this value, and so I performed a systematic literature review in PubMed on September 8, 2018 using the search criteria ("secondhand smoke" OR "environmental tobacco smoke" OR "passive smoking" OR "tobacco smoke pollution") AND ("indoors" OR "household") AND ("PM" (particulate matter) OR "PM(2.5)" OR "respirable suspended particles" OR "particulate matter" OR "CO" OR "airborne marker"), adapted from the search criteria used in a systematic review of outdoor SHS exposure performed by Sureda in 2013.(43) Exclusion criteria included studies included in the Semple 2015 study, among asthmatic populations, lacking $PM_{2.5}$ concentrations, or performed within hallways, workplaces, or the hospitality industry. From 95 search hits, seven studies provided average daily $PM_{2.5}$ concentrations within homes with at least one smoker, and four also included $PM_{2.5}$ concentrations within nonsmoking homes (Table 2). Observed [$PM_{2.5}$] in nonsmoking homes was assumed to be 10 ug/m^3 if not reported by the study (italicized values in table). The adjusted SHS [$PM_{2.5}$] is the difference between measured [$PM_{2.5}$] in smoking households and [$PM_{2.5}$] in nonsmoking households. Adjusted SHS [$PM_{2.5}$] values were then weighted by number of households N in each study to result in the weighted average of 32.15 ug/m^3.

Table 2. Studies of household secondhand smoke $PM_{2.5}$ exposure from systematic literature review. [$PM_{2.5}$] is in terms of ug/m^3.

Source	Study population	N	Observed $[PM_{2.5}]^{HH-S}$	Observed $[PM_{2.5}]^{HH-NS}$	Adjusted $[PM_{2.5}]^{SHS}$
Jedrychowski 2006 (35)	pregnant women	82	53.5	33.9	19.6
Van Deusen 2009 (36)	convenience sample	9	63	9	54
Klepeis 2013 (37)	convenience sample, low income households	34	82	*10**	72
Semple 2015 (34)	aggregate of 4 studies	93	31	*10**	21
Ratschen 2017 (38)	low income households with children under 5	205	32	*10**	22
Rice 2018 (39)	vulnerable first-time moms	47	31	9.3	21.7
Semple 2018 (40)	vulnerable first-time moms	81	48.2	10.5	37.7
				Weighted average	**32.15**

**Observed [$PM_{2.5}$] in nonsmoking homes was assumed to be 10 ug/m^3 if not reported by the study*

N = Number of households, HH-S = smoking household, HH-NS = nonsmoking household

Relative risks

For each of the risk factors in this analysis—AAP, HAP, AS, and SHS—relative risks of mortality were estimated using IER curves obtained from GBD 2017 for each outcome.(21) The specific exposures used to extract the relative risks are outlined in detail in the PAF calculations below, using four different methods.

Approach: Theoretical minimum risk exposure level

Table 3 lists the theoretical minimum risk exposure levels (TMREL) for each risk factor in this analysis. The best-case scenario was chosen for each TMREL.

Table 3. Theoretical minimum risk exposure distributions for each risk factor.

Risk factor	TMREL
Ambient air pollution	Uniform distribution between 2.4 and 5.9 ug/m^3 (6)
Household air pollution	Uniform distribution between 2.4 and 5.9 ug/m^3 (6)
Active smoking	No smoking
Secondhand smoke	No smoking → no secondhand smoke

The TMRELs for active smoking and secondhand smoke are both zero exposure to each of these risk factors because it is possible (however impractical to enforce) to have no smokers in a population. It is impossible, however, to have zero ambient or household air pollution because it is impossible to completely filter the air of all dust and normal environmental particles. The TMREL for ambient and household air pollution for this analysis, therefore, will be a uniform distribution with lower and upper bounds given by the average of the minimum and 5th percentiles of AAP cohort studies exposure distributions conducted in North America, as generated for GBD.(6)

Burden attribution

The population-attributable fraction (PAF) is defined as the percentage reduction in disease or death if there had been no (or some defined minimum level of) exposure to the risk factor. To assess risk attribution of the burden, first PAFs were calculated for each cause of death due to each relevant risk factor using the general equation:(1,3,17,23)

$$\boldsymbol{PAF} = \frac{\left(\sum_{i=1}^{n} \boldsymbol{Prev}_i * \boldsymbol{RR}_{\boldsymbol{exposure}(i)}\right) - \left(\sum_{i=1}^{n} \boldsymbol{Prev'}_i * \boldsymbol{RR'}_{\boldsymbol{exposure}(i)}\right)}{\sum_{i=1}^{n} \boldsymbol{Prev}_i * \boldsymbol{RR}_{\boldsymbol{exposure}(i)}} \quad \text{(Equation 4)}$$

Where:

- i represents each exposure category being aggregated into the PAF,
- n is the total number of i being aggregated,
- $Prev$ is the proportion of the population exposed to each i,
- $exposure(i)$ is the $PM_{2.5}$ exposure level assigned to each i,
- RR is the relative risk calculated from $exposure(i)$, and

- the prime ($'$) marking in the subtrahend of the numerator denotes the theoretical minimum population prevalence of exposure and relative risks for each i.

I used four methods to calculate risk-specific and aggregate PAFs with the above Equation 4:

1. Independent exposures (WHO method). I assumed ambient air pollution, household air pollution, and smoking exposures are independent of one another, calculated relative risks and PAFs separately for each without incorporating the others, and calculated aggregate PAFs from these risk-specific PAFs using multiplicative aggregation.
2. Proportional PAFs (GBD method). For air pollution, I assumed $PM_{2.5}$ exposures for AAP and HAP are additive on the exposure scale of IER curves, and the RR extracted for individuals exposed to HAP corresponds to the sum of AAP and HAP exposures. The resulting PAF for this subset of the population was split proportionally according to the AAP and HAP exposure levels to obtain separate PAFs for HAP and AAP. I calculated smoking PAFs as in the Independent exposures method, and I assumed air pollution and smoking exposures are independent of one another.
3. Additive exposures. I assumed $PM_{2.5}$ exposures for all four risk factors are additive on the exposure scale of IER curves. For individuals who experienced more than one $PM_{2.5}$ risk factor, I calculated PAFs by adding exposures and extracting the corresponding relative risks from the IER curves.
4. Max exposure. For individuals who experienced more than one $PM_{2.5}$ risk factor, I extracted from the IER curve the relative risk associated with the observed exposure level for the risk factor with the highest exposure level.

PAFs were calculated for each risk-outcome pair, including aggregates of the risks, using Equation 4 for all four methods. Two components were adapted to correspond with the four strategies: the risk exposure categories i that were summed over, and the exposure levels associated with those risks. To illustrate this difference across methods, the detailed equations for PAF_{AAP} are shown below for each PAF strategy.

For all methods, the aggregate all smoking PAF for AS and SHS is calculated by adding the PAFs of both risks because they are among mutually exclusive populations (smokers and nonsmokers).

$$\boldsymbol{PAF}_{\boldsymbol{all\ smoking}} = \left(\boldsymbol{PAF}_{\boldsymbol{active\ smoking}}\right) + \left(\boldsymbol{PAF}_{\boldsymbol{secondhand\ smoke}}\right) \quad \text{(Equation 5)}$$

Resulting PAFs were then multiplied by the cause-specific mortality from Aim 2 to obtain deaths of each outcome attributable to each risk factor individually and each aggregate: all smoking, all air pollution, and all $PM_{2.5}$. All calculations were performed across 1,000 draws from the exposures and relative risks in order to propagate uncertainty through the PAF and attributable deaths estimates.

Independent Exposures

The Independent Exposures method assumes that the exposures of AAP, HAP, and smoking are unrelated to one another and that, for example, if a person is exposed to AAP, HAP, and SHS, their risk of disease due to SHS $PM_{2.5}$ is not affected by their exposure to additional $PM_{2.5}$ from AAP and HAP.

This not true, given the observed curved relationship between $PM_{2.5}$ exposure and risk of disease in the IER curves. This method is commonly used across diseases because of its simplicity and validity with most diseases(44), and it is the method used by the WHO for these risk factors.(19,45,46)

The PAFs for individual risk factors AAP, HAP, AS, and SHS were calculated using the general PAF Equation 4. The number of exposure categories was two, either exposed or unexposed to that risk factor, or one in the case of AAP because the whole populations was assumed to be exposed. The PAF equation for AAP is:

$$\boldsymbol{PAF_{AAP}} = \frac{(\mathbf{1}*\boldsymbol{RR_{AAP}})-(\mathbf{1}*\boldsymbol{RR_{TMREL}})}{\mathbf{1}*\boldsymbol{RR_{AAP}}} \quad \text{(Equation 6)}$$

Where RR_{TMREL} is 1, and the 1's in the Equation 6 signify that the entire population is exposed to ambient air pollution. The combined PAFs for ambient air pollution, household air pollution, and all smoking were calculated using the multiplicative aggregation Equation 7 from previous studies (23,44):

$$\boldsymbol{PAF_{aggregated}} = \mathbf{1} - \prod_{\boldsymbol{i=1}}^{\boldsymbol{n}}(\mathbf{1} - \boldsymbol{PAF_i}) \quad \text{(Equation 7)}$$

Where i is each individual risk factor and n is the number of risk factors. Written specifically for all $PM_{2.5}$ risk factors:

$$\boldsymbol{PAF_{PM2.5}} = \mathbf{1} - (\mathbf{1} - \boldsymbol{PAF_{AAP}})(\mathbf{1} - \boldsymbol{PAF_{HAP}})\left(\mathbf{1} - \boldsymbol{PAF_{AS,SHS}}\right) \quad \text{(Equation 8)}$$

This multiplicative Equation 8 assumes that the exposures, relative risks, and resulting PAFs of the included risk factors are independent of one another. It is a computationally efficient way to approximate total attributable burden for many risk factors.(44)

Proportional PAFs

The Proportional PAFs method assumes that the exposures of all air pollution and smoking are unrelated to each another and that, similar to the Independent Exposures method, if a person is exposed to AAP, HAP, and SHS, their risk of disease due to SHS $PM_{2.5}$ is not affected by their exposure to additional $PM_{2.5}$ from AAP and HAP. However, this method does attempt to account for the relationship between AAP and HAP by adding $PM_{2.5}$ concentration levels for the two risk factors among the subset of the population who experiences both risks, before extracting the corresponding RR from the IER curves. This method was newly developed by GBD and applied to the GBD 2017 publication.(21)

The PAFs for individual risk factors AS and SHS were calculated using the general PAF Equation 8 above, similar to the Independent Exposures method. The number of exposure categories was two, either exposed or unexposed to that risk factor. The PAF equation for AS is:

$$\boldsymbol{PAF_{AS}} = \frac{(\boldsymbol{Prop_{AS}}*\boldsymbol{RR_{AS}}+(\mathbf{1}-\boldsymbol{Prop_{AS}})*\boldsymbol{RR_{TMREL}})-(\mathbf{1}*\boldsymbol{RR_{TMREL}})}{\boldsymbol{Prop_{AS}}*\boldsymbol{RR_{AS}}+(\mathbf{1}-\boldsymbol{Prop_{AS}})*\boldsymbol{RR_{TMREL}}} \quad \text{(Equation 9)}$$

The PAFs for risk factors AAP and HAP use a "proportional PAF" approach (21). For the proportion of the population not exposed to HAP, the relative risk was extracted from the IER curve for the AAP $PM_{2.5}$ exposure level.

$$PAF_{AAP\ only} = \frac{((1-Prop_{HAP})*RR_{AAP})-(1*RR_{TMREL})}{(1-Prop_{HAP})*RR_{AAP}} \quad \text{(Equation 10)}$$

For the proportion of the population exposed to both AAP and HAP, I calculated a joint relative risk from the IER curves based on the sum of exposure levels of these two risks.

$$PAF_{AAP,HAP} = \frac{((1-Prop_{HAP})*RR_{AAP}+Prop_{HAP}*RR_{AAP+HAP})-(1*RR_{TMREL})}{((1-Prop_{HAP})*RR_{AAP}+Prop_{HAP}*RR_{AAP+HAP})} \quad \text{(Equation 11)}$$

Where RR_{TMREL} is 1. I then proportioned the joint PAF based on the proportion of $PM_{2.5}$ exposure due to AAP and HAP, respectively. See Table 4 for the equations used to calculate proportional PAFs for AAP and HAP.

Table 4. AAP and HAP PAF calculations used in Proportional PAF method. Adapted from GBD 2017 documentation (21).

PAF	Population not exposed to HAP	Population exposed to HAP
AAP	PAF_{AAP}	$(Exp_{AAP}/(Exp_{AAP}+Exp_{HAP}))*PAF_{AAP+HAP}$
HAP	0	$(Exp_{HAP}/(Exp_{AAP}+Exp_{HAP}))*PAF_{AAP+HAP}$

The PAFs for air pollution and all smoking were combined using the multiplicative equation as in the Independent Exposures method to obtain the PAF for all fine particulate matter:

$$PAF_{PM2.5} = 1 - \left(1 - PAF_{AAP,HAP}\right)\left(1 - PAF_{AS,SHS}\right) \quad \text{(Equation 12)}$$

Additive Exposures

The Additive Exposures method assumes that fine particulate matter exposures are additive at the individual level. I created subpopulations within Indonesia by age and sex with every combination of these four risk factors based on their prevalence as the independent probabilities of exposure (Table 5). Active smoking and secondhand smoke are mutually exclusive, and their prevalence estimates were proportionally adjusted to sum to 1 if their sum exceeded 1 to prevent impossible values (0.7% occurrences among the 1,000 draws). For each subpopulation, I extracted the relative risk from IER curves corresponding to the sum of $PM_{2.5}$ exposures experienced by that group. For example, for smokers exposed to HAP, I summed the $PM_{2.5}$ concentrations for AS, HAP, and AAP to inform their relative risk. The AAP $PM_{2.5}$ exposure was added to all subpopulations.

Table 5. List of all risk factor combinations that subsets of the population are exposed to.

AAP only
HAP and AAP
AS and AAP
SHS and AAP
AS, HAP, and AAP
SHS, HAP, and AAP

Relative risks were extracted from cause-specific IER curves for each subpopulation, and PAFs were calculated from these added exposures and corresponding relative risks. This was repeated for each individual risk factor and for the aggregates all air pollution, all smoking, and all fine particulate matter.

In this method, the exposures were assumed to be additive to better capture the increased relative risk of death from being exposed to, say, higher levels of ambient air pollution given a certain level of household air pollution. All four risk factors were measured in terms of $PM_{2.5}$ concentration, enabling aggregation of these risks at the individual exposure level instead of at the PAF level as in the Independent Exposures method above. The PAF calculation for AAP using the Additive Exposures method, using the TMREL for AAP from Table 3, is:

$$PAF_{AAP} = \frac{\begin{pmatrix} Prop_{AAPonly}*RR_{AAP}+Prop_{AS,AAP}*RR_{AS+AAP}+ \\ Prop_{SHS,AAP}*RR_{SHS+AAP}+Prop_{HAP,AAP}*RR_{HAP+AAP}+ \\ Prop_{AS,HAP,AAP}*RR_{AS+HAP+AAP}+Prop_{SHS,HAP,AAP}*RR_{SHS+HAP+AAP} \end{pmatrix} - \begin{pmatrix} Prop_{AAPonly}*RR_{TMREL}+Prop_{AS,AAP}*RR_{AS+TMREL}+ \\ Prop_{SHS,AAP}*RR_{SHS+TMREL}+Prop_{HAP,AAP}*RR_{HAP+TMREL}+ \\ Prop_{AS,HAP,AAP}*RR_{AS+HAP+TMREL}+Prop_{SHS,HAP,AAP}*RR_{SHS+HAP+TMREL} \end{pmatrix}}{\begin{matrix} Prop_{AAPonly}*RR_{AAP}+Prop_{AS,AAP}*RR_{AS+AAP}+ \\ Prop_{SHS,AAP}*RR_{SHS+AAP}+Prop_{HAP,AAP}*RR_{HAP+AAP}+ \\ Prop_{AS,HAP,AAP}*RR_{AS+HAP+AAP}+Prop_{SHS,HAP,AAP}*RR_{SHS+HAP+AAP} \end{matrix}} \quad \text{(Equation 13)}$$

The other PAF equations for HAP, AS, SHS, all air pollution, all smoking, and all fine particulate matter are similar to Equation 13, with differences in the TMREL terms in the subtrahend. The PAF calculation for all fine particulate matter exposure using the Additive Exposures method is:

$$PAF_{PM2.5} = \frac{\begin{pmatrix} Prop_{AAPonly}*RR_{AAP}+Prop_{AS,AAP}*RR_{AS+AAP}+ \\ Prop_{SHS,AAP}*RR_{SHS+AAP}+Prop_{HAP,AAP}*RR_{HAP+AAP}+ \\ Prop_{AS,HAP,AAP}*RR_{AS+HAP+AAP}+Prop_{SHS,HAP,AAP}*RR_{SHS+HAP+AAP} \end{pmatrix} - (1*RR_{TMREL})}{\begin{matrix} Prop_{AAPonly}*RR_{AAP}+Prop_{AS,AAP}*RR_{AS+AAP}+ \\ Prop_{SHS,AAP}*RR_{SHS+AAP}+Prop_{HAP,AAP}*RR_{HAP+AAP}+ \\ Prop_{AS,HAP,AAP}*RR_{AS+HAP+AAP}+Prop_{SHS,HAP,AAP}*RR_{SHS+HAP+AAP} \end{matrix}} \quad \text{(Equation 14)}$$

Max Exposure

The Max Exposure method assumes that the maximum single-risk-factor exposure for a person was responsible for their $PM_{2.5}$-related health outcomes, and additional smaller $PM_{2.5}$ risks experienced by that person were negligible.

This method used the same subpopulation risk factor groupings as the Additive Exposure method above. For each subset, I extracted the relative risk from IER curves corresponding to the maximum exposure observed among the risk factors experienced in that subset. For example, an active smoker exposed to

household air pollution and ambient air pollution would be assigned the $PM_{2.5}$ exposure level for active smoking only because it exceeds the $PM_{2.5}$ levels of HAP or AAP. This is denoted in the PAF equation for AAP below by $\max(AS, HAP, AAP)$.

$$PAF_{AAP} = \frac{\begin{pmatrix} Prop_{AAPonly} * RR_{AAP} + Prop_{AS,AAP} * RR_{\max(AS,AAP)} + \\ Prop_{SHS,AAP} * RR_{\max(SHS,AAP)} + Prop_{HAP,AAP} * RR_{\max(HAP,AAP)} + \\ Prop_{AS,HAP,AAP} * RR_{\max(AS,HAP,AAP)} + Prop_{SHS,HAP,AAP} * RR_{\max(SHS,HAP,AAP)} \end{pmatrix} - \begin{pmatrix} Prop_{AAPonly} * RR_{TMREL} + Prop_{AS,AAP} * RR_{\max(AS,TMREL)} + \\ Prop_{SHS,AAP} * RR_{\max(SHS,TMREL)} + Prop_{HAP,AAP} * RR_{\max(HAP,TMREL)} + \\ Prop_{AS,HAP,AAP} * RR_{\max(AS,HAP,TMREL)} + Prop_{SHS,HAP,AAP} * RR_{\max(SHS,HAP,TMREL)} \end{pmatrix}}{\begin{matrix} Prop_{AAPonly} * RR_{AAP} + Prop_{AS,AAP} * RR_{\max(AS,AAP)} + \\ Prop_{SHS,AAP} * RR_{\max(SHS,AAP)} + Prop_{HAP,AAP} * RR_{\max(HAP,AAP)} + \\ Prop_{AS,HAP,AAP} * RR_{\max(AS,HAP,AAP)} + Prop_{SHS,HAP,AAP} * RR_{\max(SHS,HAP,AAP)} \end{matrix}} \quad \text{(Equation 15)}$$

The other PAF equations for HAP, AS, SHS, all air pollution, all smoking, and all fine particulate matter are similar to Equation 15, with differences in the TMREL terms in the subtrahend. The PAF calculation for all fine particulate matter exposure using the Max Exposure method is:

$$PAF_{PM2.5} = \frac{\begin{pmatrix} Prop_{AAPonly} * RR_{AAP} + Prop_{AS,AAP} * RR_{\max(AS,AAP)} + \\ Prop_{SHS,AAP} * RR_{\max(SHS,AAP)} + Prop_{HAP,AAP} * RR_{\max(HAP,AAP)} + \\ Prop_{AS,HAP,AAP} * RR_{\max(AS,HAP,AAP)} + Prop_{SHS,HAP,AAP} * RR_{\max(SHS,HAP,AAP)} \end{pmatrix} - (1 * RR_{TMREL})}{\begin{matrix} Prop_{AAPonly} * RR_{AAP} + Prop_{AS,AAP} * RR_{\max(AS,AAP)} + \\ Prop_{SHS,AAP} * RR_{\max(SHS,AAP)} + Prop_{HAP,AAP} * RR_{\max(HAP,AAP)} + \\ Prop_{AS,HAP,AAP} * RR_{\max(AS,HAP,AAP)} + Prop_{SHS,HAP,AAP} * RR_{\max(SHS,HAP,AAP)} \end{matrix}} \quad \text{(Equation 16)}$$

Attributable deaths

For LRI, lung cancer, ischemic heart disease, and stroke, cause-specific deaths from Aim 2 for age group 80+ were parsed out into 4 age groups (80-84, 85-89, 90-94, 95+) using the age distribution observed in all-cause deaths estimated for Indonesia 2014 by GBD.(47) PAFs were applied to these deaths for age and sex to obtain cause-specific attributable deaths for Indonesia in 2014 by age and sex with uncertainty.

Results

The Independent Exposures method resulted in higher PAF estimates for all risk-outcome pairs (including and especially risk factor aggregates), followed by the Proportional PAF method (Table 6, full results for all ages and by age group in Supplementary Table 1 appended). For male ischemic heart disease deaths, all $PM_{2.5}$ risk factors combined are attributable for 52.2% (95% UI 49.4%-55.3%) of deaths using the Independent Exposures method, 50.2% (95% UI 47.6%-53.0%) using the Proportional PAF method, 39.4% (95% UI 37.7%-41.1%) using the Additive Exposures method, and 38.9% (95% UI 37.2%-40.6%) using the Max Exposures method (Table 6). For PAFs due to all $PM_{2.5}$, the Independent

Exposures method estimates were 29-36% higher than the Additive Exposures method for IHD, stroke, and LRI deaths among males and 11% higher for lung cancer deaths among males. For females, the discrepancy is higher, with the Independent Exposures method estimates 30-58% higher than the Additive Exposures method for all four causes of death among females. Estimates from the Proportional PAF method were only slightly closer to those from the Additive Exposures method, ranging from 27-29% higher for male IHD, stroke, and LRI deaths and 10% higher for male lung cancer deaths, and 27-41% higher for female deaths due to all four causes (Table 6).

Table 6. Estimated sex-specific PAFs with 95% uncertainty by risk/outcome pair for Indonesia in 2014, for all fine particulate matter combined and separately for AAP, HAP, AS, and SHS.

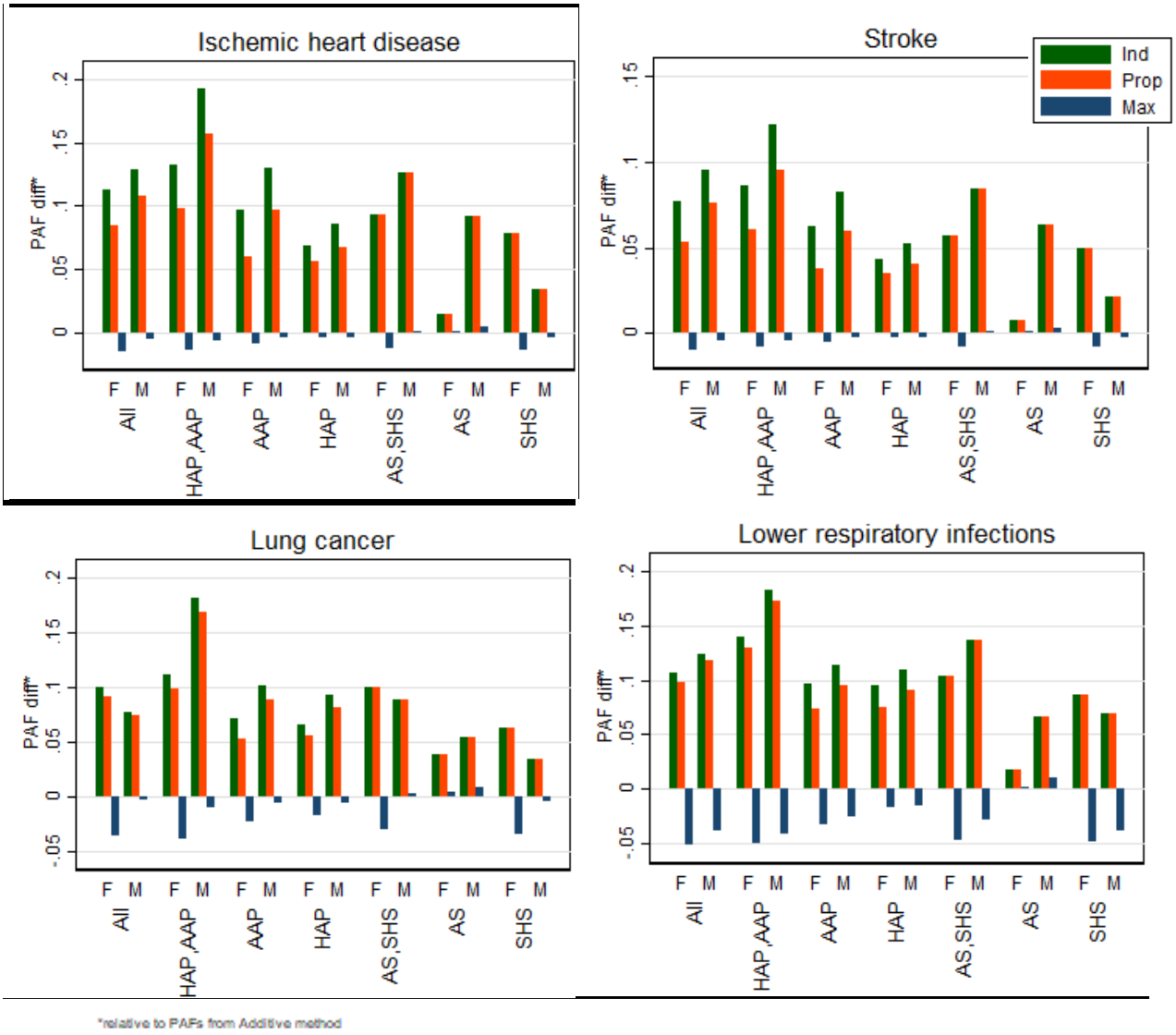
	PAF (95% UI)							
	Independent		Proportional PAF		Additive		Max	
MALES								
Ischemic heart disease								
All PM2.5	0.522	(0.494, 0.553)	0.502	(0.476, 0.530)	0.394	(0.377, 0.411)	0.389	(0.372, 0.406)
AAP	0.144	(0.121, 0.170)	0.111	(0.091, 0.134)	0.015	(0.011, 0.018)	0.011	(0.008, 0.014)
HAP	0.095	(0.077, 0.115)	0.076	(0.059, 0.096)	0.008	(0.006, 0.012)	0.005	(0.002, 0.009)
AS	0.356	(0.340, 0.373)	0.356	(0.340, 0.373)	0.264	(0.244, 0.283)	0.269	(0.249, 0.288)
SHS	0.041	(0.035, 0.048)	0.041	(0.035, 0.048)	0.007	(0.006, 0.009)	0.003	(0.002, 0.005)
Stroke								
All PM2.5	0.359	(0.320, 0.398)	0.340	(0.305, 0.373)	0.264	(0.244, 0.281)	0.260	(0.240, 0.277)
AAP	0.093	(0.068, 0.119)	0.071	(0.052, 0.092)	0.011	(0.008, 0.015)	0.009	(0.006, 0.012)
HAP	0.058	(0.043, 0.075)	0.047	(0.034, 0.062)	0.006	(0.004, 0.008)	0.003	(0.002, 0.006)
AS	0.231	(0.214, 0.246)	0.231	(0.214, 0.246)	0.168	(0.152, 0.183)	0.171	(0.155, 0.186)
SHS	0.026	(0.020, 0.033)	0.026	(0.020, 0.033)	0.005	(0.004, 0.006)	0.002	(0.001, 0.003)
Lower respiratory infections								
All PM2.5	0.550	(0.464, 0.632)	0.543	(0.461, 0.620)	0.426	(0.364, 0.481)	0.387	(0.333, 0.441)
AAP	0.156	(0.084, 0.236)	0.137	(0.086, 0.196)	0.043	(0.029, 0.062)	0.017	(0.008, 0.028)
HAP	0.178	(0.124, 0.235)	0.158	(0.110, 0.213)	0.068	(0.034, 0.103)	0.052	(0.022, 0.086)
AS	0.214	(0.179, 0.250)	0.214	(0.179, 0.250)	0.147	(0.112, 0.188)	0.157	(0.120, 0.198)
SHS	0.136	(0.101, 0.175)	0.136	(0.101, 0.175)	0.066	(0.038, 0.091)	0.028	(0.015, 0.042)
Lung cancer								
All PM2.5	0.817	(0.767, 0.862)	0.814	(0.764, 0.859)	0.739	(0.704, 0.771)	0.736	(0.699, 0.767)
AAP	0.111	(0.063, 0.167)	0.097	(0.058, 0.144)	0.009	(0.006, 0.013)	0.004	(0.002, 0.006)
HAP	0.104	(0.063, 0.152)	0.093	(0.058, 0.136)	0.011	(0.007, 0.016)	0.005	(0.002, 0.009)
AS	0.729	(0.692, 0.761)	0.729	(0.692, 0.761)	0.675	(0.635, 0.715)	0.683	(0.643, 0.722)
SHS	0.042	(0.027, 0.059)	0.042	(0.027, 0.059)	0.008	(0.006, 0.010)	0.003	(0.002, 0.004)
FEMALES								
Ischemic heart disease								
All PM2.5	0.317	(0.284, 0.355)	0.289	(0.259, 0.321)	0.205	(0.186, 0.226)	0.190	(0.171, 0.211)
AAP	0.130	(0.110, 0.153)	0.094	(0.077, 0.112)	0.033	(0.026, 0.041)	0.024	(0.020, 0.030)
HAP	0.100	(0.083, 0.118)	0.087	(0.070, 0.105)	0.031	(0.024, 0.040)	0.027	(0.018, 0.037)
AS	0.034	(0.027, 0.042)	0.034	(0.027, 0.042)	0.020	(0.015, 0.025)	0.020	(0.016, 0.025)
SHS	0.104	(0.092, 0.118)	0.104	(0.092, 0.118)	0.026	(0.022, 0.029)	0.012	(0.007, 0.017)
Stroke								
All PM2.5	0.210	(0.170, 0.253)	0.187	(0.151, 0.223)	0.133	(0.110, 0.155)	0.123	(0.101, 0.146)
AAP	0.085	(0.064, 0.109)	0.061	(0.045, 0.078)	0.022	(0.016, 0.030)	0.017	(0.012, 0.023)
HAP	0.062	(0.048, 0.078)	0.054	(0.041, 0.068)	0.019	(0.014, 0.025)	0.017	(0.011, 0.023)
AS	0.019	(0.015, 0.024)	0.019	(0.015, 0.024)	0.012	(0.009, 0.015)	0.012	(0.009, 0.015)
SHS	0.065	(0.051, 0.080)	0.065	(0.051, 0.080)	0.015	(0.013, 0.018)	0.007	(0.004, 0.010)
Lower respiratory infections								
All PM2.5	0.468	(0.373, 0.560)	0.459	(0.372, 0.547)	0.361	(0.292, 0.428)	0.310	(0.249, 0.374)
AAP	0.156	(0.084, 0.236)	0.132	(0.084, 0.189)	0.059	(0.040, 0.086)	0.026	(0.013, 0.043)

HAP	0.206	(0.145, 0.269)	0.186	(0.131, 0.247)	0.111	(0.057, 0.167)	0.094	(0.044, 0.150)
AS	0.034	(0.023, 0.049)	0.034	(0.023, 0.049)	0.016	(0.010, 0.026)	0.018	(0.011, 0.028)
SHS	0.174	(0.129, 0.220)	0.174	(0.129, 0.220)	0.088	(0.051, 0.117)	0.038	(0.020, 0.057)
Lung cancer								
All PM2.5	0.437	(0.334, 0.537)	0.427	(0.329, 0.523)	0.336	(0.271, 0.397)	0.300	(0.234, 0.359)
AAP	0.111	(0.063, 0.167)	0.092	(0.055, 0.135)	0.039	(0.025, 0.057)	0.016	(0.009, 0.024)
HAP	0.144	(0.095, 0.199)	0.133	(0.087, 0.183)	0.078	(0.054, 0.106)	0.061	(0.036, 0.093)
AS	0.141	(0.111, 0.178)	0.141	(0.111, 0.178)	0.103	(0.080, 0.131)	0.108	(0.084, 0.137)
SHS	0.120	(0.079, 0.166)	0.120	(0.079, 0.166)	0.058	(0.045, 0.071)	0.023	(0.013, 0.034)

For individual risk factors, the Independent Exposures method estimates for male PAFs are over double those from the Additive Exposures method for all causes related to SHS, AAP, and HAP, and from 6.4 to 12.3 times the male PAFs for ischemic heart disease, stroke, and lung cancer attributable to AAP and/or HAP, which correspond to absolute differences in PAFs of 5-19% (Figure 3). The effect is smaller among females because smoking rates are much lower and do not dominate the $PM_{2.5}$ exposures experienced. For females, the most consistent PAF estimates across method occurs for deaths from any cause attributable to AS, and deaths attributable to SHS for males (Figure 3).

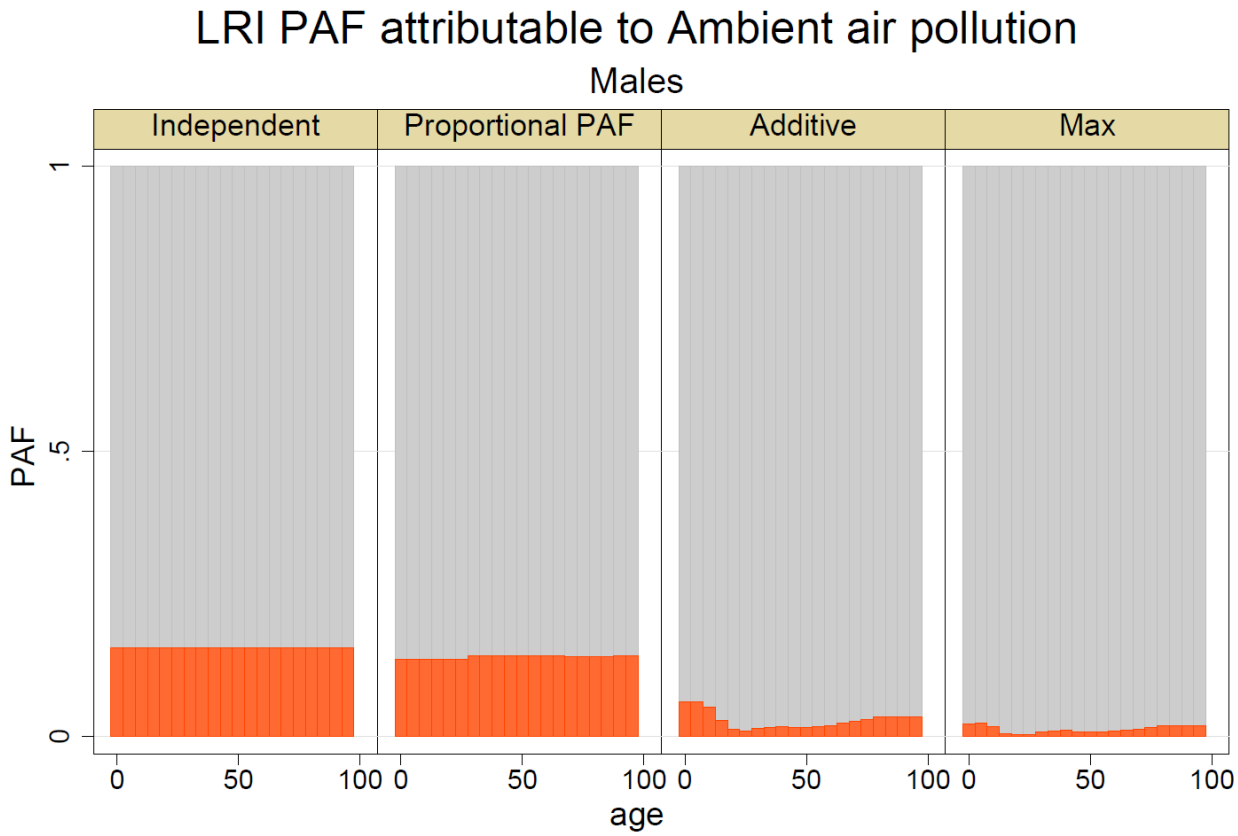
For all causes related to AAP and HAP for both males and females, the differences between Proportional PAF method estimates and the Additive Exposures method estimates are just slightly lower than that for the Independent Exposure method. Estimates are identical for PAFs attributable to AS and SHS because their exposures and PAFs have the same exposure definitions and calculations (Figure 3). The Max Exposures method resulted in PAFs more similar to those from the Additive Exposures method, with differences from -0.05 LRI deaths attributable to all air pollution among females, to 0.01 for LRI attributable to AS among males.

Figure 3. Absolute difference between estimated PAFs from the each method and those from the Additive Exposures method by risk/outcome pair and sex (M=Male, F=Female) for all ages. (Ind=Independent Exposures, Prop=Proportional PAF, Max=Max Exposures)



In terms of age patterns, the Independent Exposures and Proportional PAFs methods for AAP results in a constant PAF over age for related outcomes without age-specific IER curves and over sex for all outcomes because a single exposure level for a given location-year is applied to the entire population (Figure 4). The other two methods exhibit fluctuations over both age and sex for AAP attributable burden due to the influence of smoking behavior and household air pollution by age and sex. Figure 4 shows an example of these differences among the methods for male lower respiratory infections attributable to AAP, and Supplementary Figure 1 (appended) contains these figures of PAFs for all risk/outcome pairs by sex.

Figure 4. Lower respiratory infections attributable to ambient air pollution for males in Indonesia, by PAF estimation method. The proportions highlighted in orange represent the estimated PAFs by age.



Supplementary Figure 2 (appended) displays the age-specific attributable number of deaths by risk/outcome pair for males and females. The age pattern among attributable deaths is driven by both the PAF age pattern and the age pattern observed in the cause of death estimates from Aim 2. These graphs show the impact of the large differences in PAF estimates between the Independent Exposures method and the other three PAF methods on the calculated number of attributable deaths to each risk factor.

Discussion

Overall, a large fraction of fatal IHD, stroke, lung cancer, and LRI burden in Indonesia in 2014 is attributable to active smoking and secondhand smoke in all four methods of calculating attributable burden, especially for males (Supplementary Figure 1, Supplementary Table 1). This is due to the very high prevalence of active male smokers in Indonesia—65% of males compared to 4% of females over age 15—and the average consumption of over 10 cigarettes per day for males and 7 for females.(21) This prevalence disparity results in higher PAFs due to active smoking for males than females, and higher PAFs due to SHS for females than males.

The commonly utilized Independent Exposures method assumes that the health effects of fine particulate matter from each source of $PM_{2.5}$ occur independently of the other $PM_{2.5}$ sources. This assumption allows for separate estimation of attributable burden to each risk factor, with any relevant exposure definition for which corresponding relative risks can be obtained. In this method, the counterfactual TMREL scenario for each risk has a RR of 1, rather than taking into account the remaining risk associated with other sources of $PM_{2.5}$ in the population. Some advantages of this method are its simplicity to compute, flexible exposure definitions, and that it is currently used by the WHO. Unfortunately, the high-impact simplifying assumptions of this method lead to overestimation of attributable burden to all PM2.5 risk factors (individually and as aggregates) as demonstrated by the comparison with the other three methods in this analysis.

The Proportional PAF method recognizes that AAP and HAP exposures and health effects are related by assuming that AAP and HAP exposures are additive to extract the RR for all $PM_{2.5}$ air pollution from IER curves (like the Additive Exposures method). However, the counterfactual TMREL scenario for all air pollution, SHS, and AS still assume a RR of 1 rather than accounting for remaining sources of PM2.5. This method also assumes the AAP and HAP PAFs are additive and mutually exclusive to break down the aggregate PAF into PAFs for HAP and AAP separately (proportionally according to exposure level), which essentially assumes a linear relationship with RR rather than curved as observed in the IER curves. Similar to the Independent Exposures method, this method assumes that the health effects of fine particulate matter from all air pollution and smoking occur independently of one another. The benefits of this method include its simplicity to compute, flexible AS and SHS exposure definitions, that it accounts for some of the exposure relationship between AAP and HAP, and that it is currently in use by GBD. This method still contains high-impact simplifying assumptions of independence and improper TMREL counterfactuals in the PAF calculations, and it also ignores the curved relationship between exposure and RR by proportionally splitting the all-air-pollution PAF into AAP and HAP attributable burdens. The Proportional PAF method also overestimates attributable burden to fine particulate matter when compared to the Additive Exposures and Max Exposure methods.

The Additive Exposures method recognizes that all PM2.5 exposures and health effects are related and that if one is eliminated, exposure to the other risks remains. This method results in much lower attributable burden to AAP and HAP in particular because the TMREL for these individual risk factors would still include exposure to smoking, so the reduction in RR would occur on the plateau portion of the IER curves and would therefore be much smaller than if RR were assumed to reduce to 1. A distinct advantage of this method is the more precise use of IER curves and counterfactual RRs to account for additive PM2.5 exposure across all combinations of these risk factors experienced in a population. Also, very little additional data would be needed to implement this approach as proposed: only the conversion of active smoking to location-specific associated PM2.5 concentrations would need to be conducted using cigarette consumption data (number of cigarettes consumed daily and average amount of fine particulate matter consumed per type of cigarette consumed in the location). However, this method does require that the exposures be measured in terms of PM2.5 concentration and currently cannot incorporate lags or cumulative exposure measures over time.

The Maximum Exposure method also recognizes that all PM2.5 exposures and health effects are related, but it also assumes that the cause of the largest portion of PM2.5 is the primary cause of the health effects. In the counterfactuals, if one risk is eliminated, there is still exposure to the others, and this approach utilizes the same data as the Additive Exposures method. An advantage of this method is the more accurate counterfactuals than the Independent Exposures or Proportional PAF methods, but these counterfactuals are not as precise as in the Additive Exposures method. The biggest disadvantage is the simplifying assumption that the risk with the highest level of observed PM2.5 is responsible for the PM2.5-related health effects. This assumption does not simplify the actual calculation process but was conducted in order to provide an interesting comparative approach to the Additive Exposures method.

Sometimes (for AS), the PAF from the Max Exposure method is greater than from the Additive Exposures method, which may seem counterintuitive at first because one might think that assigning the exposure from the single highest risk experienced by a subpopulation would always result in lower PAF estimates than the other methods. However, this seeming paradox results from the influence of SHS and HAP on AS PAF estimates; these two risk factors cause higher theoretical minimum relative risks for active smoking in the Additive Exposures method when summed, than the Max Exposure method when only the one with the higher $PM_{2.5}$ concentration informs the relative risk. So in those cases, the Max Exposure method subtracts a slightly smaller value in the numerator of the PAF equation relative to the first term when compared with the Additive Exposures method, resulting in a slightly larger overall fraction. This is not observed for the PAFs for the other risk factors because their theoretical minimum relative risk—the term being subtracted in the numerator—is dominated by active smoking.

Limitations

There are several limitations in this analysis for the exposure definitions, relative risk relationships, and burden attribution methods.

Exposures

First, in terms of exposure definitions, secondhand smoke does not take into account consumption patterns (type and quantity of cigarettes) and behaviors (where smoking occurs) of smokers, the number of smokers in the nonsmoker's household, nor smoke exposure outside the household experienced by the nonsmoker. In the future, consumption patterns could potentially be used to adjust the literature-based conversion of secondhand smoke exposure to $PM_{2.5}$ concentration to more accurately reflect the Indonesian smoking environment. For sources of secondhand smoke outside the household, in the absence of individual-level data tracking these various sources and levels of secondhand smoke on a daily basis, this proxy of within-household smoking status must suffice until more thorough data are available. Related to this, active smokers can also be exposed to the secondhand smoke of other smokers, which is not taken into account in the exposure definition for SHS. Household exposure to SHS for active smokers could be extracted from microdata in a similar manner as for nonsmokers, though the same exposure definition limitations mentioned above would still apply.

Second, the exposure to AAP is defined as the population-weighted mean PM2.5 for the country with uncertainty. A more accurate exposure definition for AAP would be the full distribution of PM2.5

experienced by the population. Currently, the satellite-based method for AAP analysis gives a grid resolution of approximately 11 km by 11 km overlaid with local population numbers to enable a weighted average of AAP up to the subnational or national level. These concentrations could simply retain their geographic detail, rather than being averaged, to inform burden attribution.

Third, for both the Additive Exposures and Max Exposure methods, the subpopulations were generated using risk factor prevalence rates as independent probabilities to calculate the prevalence of every combination of risk factors in the population. A more robust method to achieve this would be to determine joint exposure of HAP, AS (both smoking status and number of cigarettes smoked per day), and SHS using census and survey microdata. These joint exposures could be mapped to the more precise values of AAP based on granular locations, as suggested in the second point above. This analysis assumes independent probabilities as a simplifying assumption, and future work could improve the calculations of these subpopulation exposures.

Fourth, neither secondhand smoke nor household air pollution directly incorporate amount of time exposed to either in the current exposure definition. An opportunity for improvement would include collecting any published or survey data of proportion of the day spent inside the home, preferably stratified by asleep or awake (such as that collected by Semple et al 2015(34)), modeling those proportions by age, sex, year, and location (national or by province, for Indonesia), and applying those proportions to the $PM_{2.5}$ concentrations for secondhand smoke and household air pollution. Even better would be individualized $PM_{2.5}$ measurements over a series of 24 hours to create an average daily exposure distribution.

Fifth, the concentration levels of PM2.5 exposure related to active smoking, secondhand smoke, and household air pollution are all point values for this analysis and in GBD. Each of these values could be estimated with uncertainty intervals to incorporate distributions around these values in the comparative risk analysis computations.

Sixth, this analysis ignores any lagged or cumulative effects of $PM_{2.5}$ exposure, which would underestimate or overestimate attributable burden to each risk factor if the exposure is decreasing or increasing within cohorts of the population, respectively, over time relative to disease onset and progression.

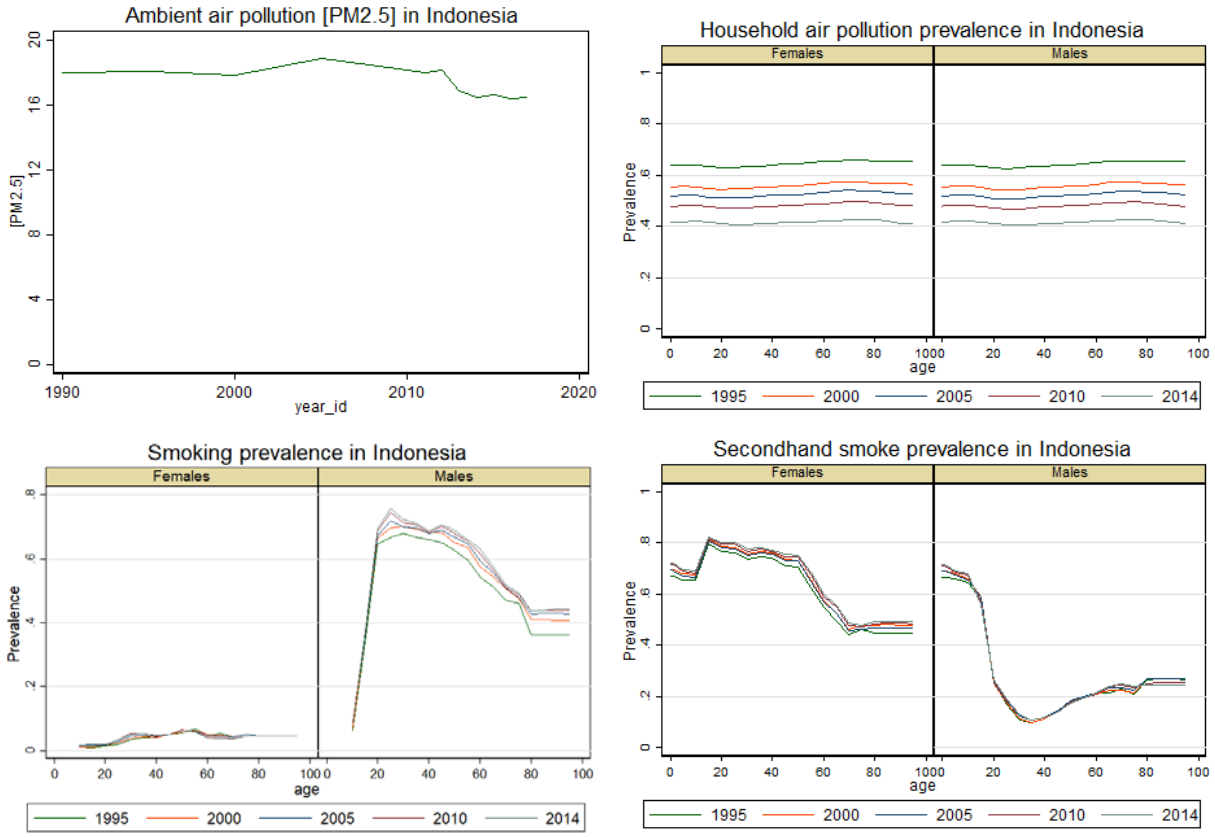
Cumulative effects refer to the dose-effect relationship between risk of health outcome and the total cumulative exposure levels experienced by a person or population over their lifetime(s), such as total pack-years smoked as used in GBD 2017 for many active-smoking-related health outcomes.(21) For example, a 20-year-old smoker who has smoked 10 cigarettes a day for one year should have a different risk of disease than a smoker who is 80 and has smoked 10 cigarettes a day for 60 years because their cumulative exposures are quite different. The IER curves are age-specific for IHD and stroke which may somewhat account for this difference, but those are only stratified by age and not cumulative exposure. The IER curves for lung cancer and LRI are not age-specific, so the same RR would apply to both of these smokers for these outcomes. If using the Additive Exposures or Max Exposure method, this problem can be mitigated by stratifying the lung cancer and LRI IER curves by age, or by creating new IER curves stratified by binned cumulative $PM_{2.5}$ exposure rather than current $PM_{2.5}$ exposure if such data were

available. For the Independent Exposures and Proportional PAF methods, exposure definitions are more flexible and can utilize cumulative exposure measures like pack-years, as in GBD, instead of converting them to PM2.5 concentrations.

Lagged effects of $PM_{2.5}$ exposure refer to the time-to-health outcome-onset or progression. For example, GBD uses 5-year lagged daily smoking prevalence as the exposure measure for 10 smoking-related health outcomes.(21) This is a helpful measure when prevalence rates change quickly over time and some current burden is due to that lagged exposure, such as a disease usually takes 5 years to develop after exposure, regardless of the cumulative exposure. If former smokers are analyzed separately, however, then a lagged smoking prevalence measure for current smokers may contain some former smokers and result in muddled results. Both lagged and cumulative effects of smoking have been more studied than those of AAP, HAP, or SHS. The lagged and cumulative effects of $PM_{2.5}$ exposure in general represent a research area that needs further study.

According to GBD estimates (21), average national AAP $PM_{2.5}$ concentration in Indonesia has remained steady from 1990 until approximately 2010, after which there is a slight decline (Figure 5). Proportion exposure to HAP has the greatest changes from 1995 to 2014, declining from over 60% to just over 40% of all males and females exposure to HAP. Smoking rates have declined within cohorts of the population from 1995 to 2014, while the age-specific rates remain fairly constant over time. For example, within the age group 50-55 in 2010, their cumulative exposure would include their smoking habits as ages 45-49 in 2005, 40-44 in 2000, and so on, and smoking prevalence was higher in those age groups than older age groups for all years. Exposure to SHS has also changed within cohorts, as expected given the smoking prevalence changes over age.

Figure 5. Exposures to AAP (as $PM_{2.5}$ concentration), HAP (as proportion exposed to HAP), AS (as proportion active smokers), and SHS (as proportion exposed to SHS) over time in Indonesia.(21)



However, the shapes of the IER curves themselves were estimated using current $PM_{2.5}$ exposures to these risk factors by aggregating all available studies with both a derivable current exposure concentration and associated relative risk. Pope et al and GBD publications have set this precedence of combining relative risks and current exposures to these four risk factors in IER curves, making this limitation less relevant, and the benefits of using the Additive Exposures method to estimate attributable burden outweigh the current alternative of ignoring the interactions of these risk factors, even at the cost of discounting past exposure. Part of that past exposure can be accounted for separately in calculations of burden attributable to former smoking, which is not included in this analysis.

One concession to fully converting to the Additive Exposures method may be possible with active smoking, because of the existence of data estimating the risk of health outcomes for lagged and cumulative exposure to smoking; similar data for lagged and cumulative effects of SHS, AAP, and HAP are not yet available. It could be worth exploring the use of current active smoking exposures as presented in this analysis to inform burden attributable to AAP, HAP, and SHS and all aggregates, but to also separately estimate the burden attributable to AS alone using current GBD methods that incorporate lags and cumulative exposure as pack-years. This would create an inconsistent exposure

definition within the comparative risk analysis (current smoker versus cumulative smoking exposure), but it would be an improvement on current methods by taking full advantage of available data on lagged and cumulative exposure to smoking and also the additive relationship of risk factors measured by fine particulate matter to prevent overestimation of burden attributable to $PM_{2.5}$. In fact, GBD already contains an inconsistent exposure definition for active smoking by using current smoking as the exposure when generating the IER curves, and cumulative exposure when calculating PAFs, so this approach may end up being a useful compromise.

Relative risks

Regarding relative risks, all of these approaches assume that the relative risk is only a function of $PM_{2.5}$ concentration level of fine particulate matter, regardless of the source or content of the exposure. It treats the airborne byproducts of cigarette smoke the same as from burning coal in the stove, which likely overlooks the nuanced health effects influenced by the constituents of different particulate matter sources.(48,49)

Conclusion

Comparative risk assessment can be reductive because it often depends on simplifying assumptions based on the availability and quality of supporting data—i.e. treating AAP, HAP, AS, and SHS as unrelated risk factors—which can mislead a person attempting to interpret the results and apply them to a population. In order for the estimates to be more reliable and useful, CRA methods must be updated when possible to reduce implausible simplifying assumptions, and the results assessed by competent stakeholders within the context of their target population. The findings of this analysis are of direct relevance to researchers working with similar comparative risk assessment data where relative risks are obtained from IER curves and exposures are measured by (or can be converted to) the same definition.

Most of the limitations of this analysis are related to the detailed exposure definitions rather than the comparison of burden attribution methods and do not affect the method selection process—with the exception of the desire to include lagged and cumulative effects of exposure. These exposure definitions could be improved in future research as suggested above to lead to more accurate final estimates.

The main disadvantage of the Independent Exposures and Proportional PAF methods used by the WHO and GBD is that they do not account for a proper counterfactual for these 4 risks, where removal of one would still leave the other three. By ignoring their joint effects, these methods overestimate the attributable burden to all $PM_{2.5}$-related risk factors, in particular AAP, HAP, and SHS in a location like Indonesia where smoking rates are high among men.

The Additive Exposures method does incorporate appropriate counterfactuals to more accurately estimate attributable burden, and it demonstrates that the multiplicative function for aggregation performs poorly. When compared to the Additive Exposures method, the commonly utilized Independent Exposures method overestimates burden attributable to $PM_{2.5}$-related risk factors and

should be abandoned in favor of the Additive Exposure method for outcomes that have IER curves. The current GBD method of Proportional PAFs corrects some of this overestimation, but it falls short of addressing the full impact of the coexistence of these $PM_{2.5}$ risk factors and does not contain proper counterfactuals. The Additive Exposures method is advantageous over all three of the other methods because it accounts for total current $PM_{2.5}$ exposure, the associated risks, and correct counterfactuals. The Maximum Exposure method provides an interesting comparison method but its simplifying assumption in the exposure definitions does not simplify the actual PAF calculation and therefore has no advantages over the Additive Exposures method.

The methods used by researchers for comparative risk assessment should be assessed periodically and updated as improved methods emerge. This analysis demonstrates disadvantages of current methods widely used for attributing burden to risk factors related to fine particulate matter and explores a method that addresses these disadvantages with few concessions which could improve the accuracy and utility of CRA estimates for decision making. Burden of disease researchers should strongly consider utilizing the Additive Exposures method—or the adapted Additive Exposures method described in the sixth exposures limitation above—for estimating burden attributable to $PM_{2.5}$ risk factors.

References

1. Scorecard: The pollution information site. What is Comparative Risk Analysis? [Internet]. 2011. Available from: http://scorecard.goodguide.com/comp-risk/def/comprisk_explanation.html

2. McBride G, Ross T, Dufour A. Chapter 10: Comparative risk analysis. In: Animal waste, water quality and human health. World Health Organization; 2012. p. 361–403.

3. Ezzati M. Annex 4.1: Comparative Risk Assessment in the Global Burden of Disease Study and the Environmental Health Risks. In: Methods for quantifying environmental health impacts [Internet]. p. 31–49. Available from: http://www.who.int/quantifying_ehimpacts/methods/en/wsh0007an4.pdf

4. WHO | The world health report 2002 - Reducing Risks, Promoting Healthy Life [Internet]. WHO. [cited 2015 Sep 14]. Available from: http://www.who.int/whr/2002/en/

5. WHO | The global burden of disease: 2004 update [Internet]. WHO. [cited 2015 Sep 14]. Available from: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/

6. GBD 2016 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017 Sep 16;390(10100):1345–422.

7. WHO | Data and statistics [Internet]. WHO. [cited 2018 Dec 14]. Available from: http://www.who.int/quantifying_ehimpacts/en/

8. World Cancer Research Fund. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. American Institute for Cancer Research [Internet]. 2007 [cited 2018 Sep 4]; Available from: https://wiki.cancer.org.au/policy/Citation:World_Cancer_Research_Fund,_American_Institute_for_Cancer_Research_2007_2

9. Pope CA, Burnett RT, Turner MC, Cohen A, Krewski D, Jerrett M, et al. Lung Cancer and Cardiovascular Disease Mortality Associated with Ambient Air Pollution and Cigarette Smoke: Shape of the Exposure–Response Relationships. Environ Health Perspect. 2011 Nov;119(11):1616–21.

10. Pope CA, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, et al. Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure-response relationship. Circulation. 2009 Sep 15;120(11):941–8.

11. Glantz SA. Air pollution as a cause of heart disease. Time for action. J Am Coll Cardiol. 2002 Mar 20;39(6):943–5.

12. Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 2010 Jun 1;121(21):2331–78.

13. Brauer M, Amann M, Burnett RT, Cohen A, Dentener F, Ezzati M, et al. Exposure assessment for estimation of the global burden of disease attributable to outdoor air pollution. Environ Sci Technol. 2012 Jan 17;46(2):652–60.

14. Evans J, van Donkelaar A, Martin RV, Burnett R, Rainham DG, Birkett NJ, et al. Estimates of global mortality attributable to particulate air pollution using satellite imagery. Environ Res. 2013 Jan;120:33–42.

15. Anenberg SC, Horowitz LW, Tong DQ, West JJ. An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. Environ Health Perspect. 2010 Sep;118(9):1189–95.

16. Burnett RT, Pope CA, Ezzati M, Olives C, Lim SS, Mehta S, et al. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. Environ Health Perspect. 2014 Apr;122(4):397–403.

17. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, 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 Dec 15;380(9859):2224–60.

18. The FTC Cigarette Test Method for Determining Tar, Nicotine, and Carbon Monoxide Yields of U.S. Cigarettes: Smoking and Tobacco Control Monograph No. 7: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute: 9781499642179: Amazon.com: Books [Internet]. [cited 2018 Sep 18]. Available from: https://www.amazon.com/Cigarette-Determining-Nicotine-Monoxide-Cigarettes/dp/1499642172

19. WHO | Ambient and household air pollution and health [Internet]. WHO. [cited 2015 Sep 23]. Available from: http://www.who.int/phe/health_topics/outdoorair/databases/en/

20. WHO | WHO report on the global tobacco epidemic 2017 [Internet]. WHO. [cited 2018 Oct 4]. Available from: http://www.who.int/tobacco/global_report/2017/en/

21. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1923–94.

22. Bonjour S, Adair-Rohani H, Wolf J, Bruce NG, Mehta S, Prüss-Ustün A, et al. Solid fuel use for household cooking: country and regional estimates for 1980-2010. Environ Health Perspect. 2013 Jul;121(7):784–90.

23. GBD 2013 Risk Factors Collaborators, Forouzanfar MH, Alexander L, Anderson HR, Bachman VF, Biryukov S, et al. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015 Sep 10;

24. Balakrishnan K, Sambandam S, Ramaswamy P, Mehta S, Smith KR. Exposure assessment for respirable particulates associated with household fuel use in rural districts of Andhra Pradesh, India. J Expo Anal Environ Epidemiol. 2004;14 Suppl 1:S14-25.

25. Dionisio KL, Howie S, Fornace KM, Chimah O, Adegbola RA, Ezzati M. Measuring the exposure of infants and children to indoor air pollution from biomass fuels in The Gambia. Indoor Air. 2008 Aug;18(4):317–27.

26. Gao X, Yu Q, Gu Q, Chen Y, Ding K, Zhu J, et al. Indoor air pollution from solid biomass fuels combustion in rural agricultural area of Tibet, China. Indoor Air. 2009 Jun;19(3):198–205.

27. Cynthia AA, Edwards RD, Johnson M, Zuk M, Rojas L, Jiménez RD, et al. Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico. Indoor Air. 2008 Apr;18(2):93–105.

28. Dasgupta S, Huq M, Khaliquzzaman M, Pandey K, Wheeler D. Who suffers from indoor air pollution? Evidence from Bangladesh. Health Policy Plan. 2006 Nov;21(6):444–58.

29. Gurley ES, Salje H, Homaira N, Ram PK, Haque R, Petri WA, et al. Seasonal concentrations and determinants of indoor particulate matter in a low-income community in Dhaka, Bangladesh. Environ Res. 2013 Feb;121:11–6.

30. Devakumar D, Semple S, Osrin D, Yadav SK, Kurmi OP, Saville NM, et al. Biomass fuel use and the exposure of children to particulate air pollution in southern Nepal. Environ Int. 2014 May;66:79–87.

31. Piadé J-J, Roemer E, Dempsey R, Hornig G, Deger Evans A, Völkel H, et al. Toxicological assessment of kretek cigarettes: Part 2: kretek and American-blended cigarettes, smoke chemistry and in vitro toxicity. Regul Toxicol Pharmacol. 2014 Dec;70 Suppl 1:S15-25.

32. Melikian AA, Djordjevic MV, Hosey J, Zhang J, Chen S, Zang E, et al. Gender differences relative to smoking behavior and emissions of toxins from mainstream cigarette smoke. Nicotine Tob Res. 2007 Mar;9(3):377–87.

33. Cigarette & Tobacco Industry Indonesia: Rising Pressures in 2018? | Indonesia Investments [Internet]. [cited 2018 Sep 17]. Available from: https://www.indonesia-investments.com/news/todays-headlines/cigarette-tobacco-industry-indonesia-rising-pressures-in-2018/item8471?

34. Semple S, Apsley A, Azmina Ibrahim T, Turner SW, Cherrie JW. Fine particulate matter concentrations in smoking households: just how much secondhand smoke do you breathe in if you live with a smoker who smokes indoors? Tob Control. 2015 Oct;24(e3):e205-211.

35. Jedrychowski WA, Perera FP, Pac A, Jacek R, Whyatt RM, Spengler JD, et al. Variability of total exposure to PM2.5 related to indoor and outdoor pollution sources Krakow study in pregnant women. Sci Total Environ. 2006 Jul 31;366(1):47–54.

36. Van Deusen A, Hyland A, Travers MJ, Wang C, Higbee C, King BA, et al. Secondhand smoke and particulate matter exposure in the home. Nicotine Tob Res. 2009 Jun;11(6):635–41.

37. Klepeis NE, Hughes SC, Edwards RD, Allen T, Johnson M, Chowdhury Z, et al. Promoting smoke-free homes: a novel behavioral intervention using real-time audio-visual feedback on airborne particle levels. PLoS ONE. 2013;8(8):e73251.

38. Ratschen E, Thorley R, Jones L, Opazo Breton M, Cook J, McNeill A, et al. A randomised controlled trial of a complex intervention to reduce children's exposure to secondhand smoke in the home. Tob Control. 2018 Mar;27(2):155–62.

39. Rice JL, Brigham E, Dineen R, Muqueeth S, O'Keefe G, Regenold S, et al. The feasibility of an air purifier and secondhand smoke education intervention in homes of inner city pregnant women and infants living with a smoker. Environ Res. 2018;160:524–30.

40. Semple S, Turner S, O'Donnell R, Adams L, Henderson T, Mitchell S, et al. Using air-quality feedback to encourage disadvantaged parents to create a smoke-free home: Results from a randomised controlled trial. Environ Int. 2018 Aug 1;120:104–10.

41. Malson JL, Lee EM, Murty R, Moolchan ET, Pickworth WB. Clove cigarette smoking: biochemical, physiological, and subjective effects. Pharmacol Biochem Behav. 2003 Feb;74(3):739–45.

42. Barrett KE, Barman SM, Boitano S, Brooks HL. Ganong's Review of Medical Physiology, Twenty-Fifth Edition. 25 edition. New York; Blacklick: McGraw-Hill Education / Medical; 2015. 768 p.

43. Sureda X, Fernández E, López MJ, Nebot M. Secondhand tobacco smoke exposure in open and semi-open settings: a systematic review. Environ Health Perspect. 2013 Jul;121(7):766–73.

44. Carnahan E, Lim SS, Nelson EC, Gillespie CW, Mokdad AH, Murray CJ, et al. Validation of a new predictive risk model: measuring the impact of major modifiable risks of death for patients and populations. The Lancet. 2013 Jun;381:S26.

45. WHO | Household air pollution [Internet]. WHO. [cited 2018 Oct 4]. Available from: http://www.who.int/gho/phe/indoor_air_pollution/en/

46. WHO | Methodology for assessment of environmental burden of disease [Internet]. WHO. [cited 2018 Dec 14]. Available from: https://www.who.int/quantifying_ehimpacts/publications/methodoly/en/

47. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016 Oct 8;388(10053):1459–544.

48. Indoor Air Quality [Internet]. American Lung Association. [cited 2015 Sep 24]. Available from: http://www.lung.org/associations/charters/mid-atlantic/air-quality/indoor-air-quality.html

49. Dai L, Bind M-A, Koutrakis P, Coull BA, Sparrow D, Vokonas PS, et al. Fine particles, genetic pathways, and markers of inflammation and endothelial dysfunction: analysis on particulate species and sources. J Expo Sci Environ Epidemiol. 2016 Jun;26(4):415–21.

Tables

Supplementary Tables

Figures

Supplementary Figures

Conclusion

Before the work of this dissertation began, there were no published comprehensive burden of disease statistics at the province level in Indonesia. I set out to fill some of this knowledge gap in terms of mortality, by generating subnational all-cause mortality estimates, breaking these deaths down by cause using small area estimation methods, and assessing four methods of attributing these cause-specific deaths to fine particulate matter exposure. With the recently decentralized government in Indonesia, having accurate burden of disease estimates at the province level enables provincial policy discussions to include a better understanding of the current local disease landscape.

Several analyses from Aim 1 directly contributed to the first set of province-level burden of disease estimates for Indonesia created by GBD 2017: mapping province boundaries over time to enable consistent boundaries for annual estimates, annual estimates of population numbers by province, and a subnational time series of lag-distributed income. Additional data extraction of birth histories and household deaths also contributed to the GBD computations. In addition, Aim 1 also included empirical life tables from Indonesian provinces to inform the age-specific mortality rates, and these life tables should also be included in future GBD mortality analyses to more precisely inform mortality in Indonesia.

In Aim 2, two key components will be useful for researchers doing burden of disease analysis when using verbal autopsy data and/or small sample sizes. First, verbal autopsy data has limitations of the cause of death accuracy, as determined by the Population Health Metrics Research Consortium (PHMRC). The PHMRC conducted validation studies across four countries of physician-certified VA, which is used in Aim 2; VA using the Tariff Method; VA using InterVA; VA using Random forests; VA using Simplified Symptom Pattern Method; and VA using the King and Lu method (1). I utilized the validation study of physician-certified VA to create an analytical cause list that maximizes the accuracy of PCVA by cause group and the relevance of the cause groups included in the list. My method could be used to assess or create a cause list to analyze VA data using any of the cause of death assignment methods validated by the PHMRC study. Until now, published literature using PCVA has coerced the data into an existing cause list, regardless of PCVA performance in terms of accuracy for each cause group (2).

Second, the small area estimation methods I used in Aim 2 from http://www.r-inla.org/ are free, fast, flexible, easy to learn, incorporate spatial autocorrelation not captured in nested hierarchical models, and are resilient to data with small sample sizes. For researchers without the cluster capabilities the GBD uses, these methods can be easily applied to obtain subnational estimates within country, of cause fractions or any health indicator of interest. Hopefully this Aim 2 work can empower local research groups in countries or institutes with fewer resources to take ownership of their own estimates and priorities.

Aim 3 challenges how burden of disease is currently attributed to risk factors defined by fine particulate matter exposure by GBD, the WHO, and any work that follows their methodology. A person's exposure to secondhand smoke is not independent of their exposure to household air pollution, for example, but

rather additive. In Aim 3, I compared four methods to calculate attributable burden to ambient air pollution, household air pollution, active smoking, and secondhand smoke. There is precedence of these four exposures being combined on the same integrated exposure-risk curves to estimate a continuum of risk according to exposure to fine particulate matter, and it logically follows that exposures experienced by subsets of a population should also be additive along these risk curves. GBD began to incorporate this partially in their 2017 estimates by adding ambient and household air pollution exposure (3), but their Proportional PAF method falls short of calculating the counterfactuals appropriately or including smoking exposures.

There are several major unresolved issues within these three aims that would benefit from future development. In Aim 1, the modeling approaches for 5q0, 45q15, and the education covariate could better incorporate the influence of neighboring provinces on one another, perhaps using INLA like Aim 2. In such a large, diverse country as Indonesia, these spatial effects could have an influence on final estimates and should be taken into account.

In Aim 2, a major limitation is the lack of a time trend. As more cause of death data become available, incorporating time into the models would allow the generation of a time series of cause-specific mortality. This would be much more useful for local stakeholders in planning for and prioritizing disease burden prevention and intervention. In addition, a limited list of causes of death can be identified in physician-certified verbal autopsy data. Development of a sample vital registration system in Indonesia would provide more detailed cause of death assignments which would enable more detailed cause of death estimation.

Aim 3 calculated exposure distributions among the population using probabilities of exposure to each risk factor by age and sex. However, a more precise method of calculating subpopulation exposure combinations would be extracting the exposures simultaneously from microdata. This would result in a precise exposures dataset containing the observed number of current smokers who are also exposed to household air pollution, for example, rather than multiplying independent probabilities of each to obtain the overlap. These microdata extractions of household air pollution, active smoking, and secondhand smoke could then be mapped geographically to the ambient air pollution exposure levels from satellite data to obtain total fine particulate matter exposure by person.

Burden of disease estimation methods contain many areas for improvement, and these three aims contribute to the development of these methods. All-cause mortality estimates form the basis of most subsequent burden of disease estimates and should be generated with care, utilizing all possible data at the national and subnational levels. Cause-specific mortality estimation can be cumbersome, especially for smaller research groups, but this difficulty can be avoided by using computationally efficient methods more flexible for subnational analysis.

Also, Aim 3 shows that the current methods used by GBD and the WHO overestimate burden attributable to all sources of fine particulate matter, exemplified using deaths caused by ischemic heart disease, stroke, lung cancer, and lower respiratory infections. This is especially true for the burdens attributable to household and ambient air pollution, which do not take smoking exposure into account in their calculations. By incorporating the cumulative exposure to smoking and air pollution sources of

fine particulate matter, my proposed Additive method of calculating population attributable fractions results in more accurate estimates of attributable burden to these risk factors.

That the proposed method estimates lower attributable burden to these risk factors does not negate the importance of addressing air pollution and secondhand smoke exposure in Indonesia or elsewhere. Rather, the measurement of their impact needs to be improved by GBD, the WHO, and other burden of disease research groups in order to increase the accuracy and utility of that impact in public health policy discussions.

Burden of disease methodology is not and should not be stagnant, but rather it should evolve as more data (both quantity and type) become available and as methods develop. This work contributes to that development while recognizing that many areas for improvement remain.

References

1. Population Health Metrics Research Consortium (PHMRC) [Internet]. Institute for Health Metrics and Evaluation. 2014 [cited 2018 Nov 7]. Available from: http://www.healthdata.org/population-health-metrics-research-consortium-phmrc

2. GBD 2017 Causes of Death Collaborators. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1736–88.

3. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018 Nov 10;392(10159):1923–94.