difficult to isolate the reason for the differences. One reason might be the different studies used: we included 5039 site-years of vital registration and 358 of verbal autopsy data compared with 578 and 192 for CHERG. Our modelling strategy was founded on modelling each individual cause separately, using the most appropriate method for each cause, and then combining the different cause estimates into an overall assessment consistent with all-cause mortality using CoDCorrect. CHERG used separate modelling strategies for HIV/AIDS, measles, pertussis, and malaria outside of Africa, and four different models for the remainder of the child causes. Separate logistic models, each with subtly incomparable cause lists, were used for neonates and children older than 1 month, for low mortality countries excluding China, for high mortality countries excluding India, for India alone, and for China alone. This partition of the world into separate models was not justified statistically—for example, they have not shown statistically different relationships with covariates for their four sets of models. Additionally, post-estimation adjustments were applied to pneumonia, meningitis, and malaria to account for intervention effectiveness; pneumonia, sepsis, meningitis, and tetanus to account for the reliance on a combined severe infection cause in the primary model; and diarrhoea, neonatal sepsis, and sudden infant death syndrome in China to account for studies that report few causes.

We used a more empirical approach. We quantified both the root-mean squared error and validity of the UIs through cross-validation; CHERG has not to date reported any cross-validation results. Given the possibility that different relationships might exist between covariates such as access to clean water or sanitation and diarrhoeal mortality in different parts of the world, we undertook a sensitivity analysis in which we excluded vital registration data from high-income regions from the models for lower respiratory infections and diarrhoea. We detected no substantial differences for estimated global cause of death patterns in these cases. Furthermore, in CHERG, neonatal causes were assumed to not cause deaths after 1 month although high quality vital registration systems routinely report deaths from these causes that extend into the second month of life.

### Challenges and limitations

In the GBD 2013, we did not include several clinical pathways to death on the cause list, such as heart failure, sepsis, fungal infection, and acute kidney injury. These clinical entities following the underlying cause construct of the International Classification of Disease are treated as garbage codes and redistributed to the likely underlying cause. Although this approach is consistent with the idea of assigning each death uniquely to the underlying cause, it masks endpoints for clinical service delivery. For example, most fungal infections are relatively minor, but potentially millions of people contract invasive fungal diseases[121] that can be important pathways to death. Similar assessments can be made for sepsis, acute kidney injury, and heart failure. In future iterations of the GBD, we will aim to quantify mortality that occurs through these intermediate causes. Such intermediate cause estimation cannot be presented in the same causes lists as underlying causes of death but can provide supplemental and important information that would otherwise go unrecognised in global epidemiology.

Even in high-income countries with complete vital registration systems, our results differ from official statistics.[122] This difference is largely caused by the emphasis in the GBD on enhancing comparability through redistribution of deaths assigned to garbage codes. Country-specific data for cause of death show substantial national variation in coding practices. Generally, we used global or regional algorithms to redistribute deaths assigned to garbage codes. This approach is fairly coarse and does not capture local variation in certification practice or timing of implementation of coding rules. The GBD 2013 is the most detailed effort to date to try and systematically deal with garbage code redistribution. Some changes, such as the treatment of ill-defined cancers or heart failure using statistical approaches, altered the GBD 2013 results compared with the GBD 2010. We believe that the GBD results including the fraction of deaths assigned to different types of garbage codes can be useful for national statistical authorities' efforts to improve medical certification of causes of death. We also believe that through the extensive network of GBD collaborators, we can move in future research to more country-specific redistribution algorithms. To ensure comparability, however, these national variations will have to be grounded in a sound statistical approach and theory of measurement.

A study of this scope has many limitations. First is the quality of the underlying medical certification of causes of death and verbal autopsy data. Even medical certification of causes of death has limitations, which is shown by the need for garbage code redistribution.[106,123,124] Moreover, verbal autopsy data vary substantially in terms of the instrument used and the training given to physicians assigning causes of death. These shortcomings might reduce the comparability of cause of death data between countries and of our estimates based on these data.

Second, we did not incorporate uncertainty from garbage code redistribution into our estimation of UIs. Propagating such uncertainty into the CODEm models will require revision of the modelling strategy or an enormous increase in computational time. As evidenced by the change for some causes compared with the GBD 2010 as a result of changes in redistribution derived from statistical methods, this is an important area for future research.

Third, the major expansion of data for China and the associated changes in the estimates for some but not all causes, shows that UIs cannot take into account data that have not been included in the analysis.

Fourth, for some causes, CODEm produces larger UIs in high-income countries than might be expected. This difference is largely the result of heterogeneity across high-income countries for a cause that cannot be explained by the models. This effect is more notable for causes such as diabetes, for which there are reasons to believe that large variation in certification practice remains in high-income countries.[125] Because diabetes or increased fasting plasma glucose is a risk factor for macrovascular outcomes, differences in how physicians interpret the meaning of underlying cause could explain such national variation in practice. In the GBD, the full consequences of high fasting plasma glucose are captured in the risk factor assessment;[8] deaths caused by diabetes in our analysis were only those that were recorded on the death certificate to be the underlying cause.

Fifth, although we tried to improve the comparability of cause of death data over time through mapping variants of the International Classification of Diseases and garbage-code redistribution, some time trends might be affected by changes in diagnostic technology. Some causes, such as cancers, might have been less likely to have been diagnosed in the 1980s and 1990s, when imaging and other diagnostic techniques were not widespread.

Sixth, for chronic kidney disease, the breakdown into deaths from diabetes, hypertension, acute glomerulonephritis, and other depends on both detailed cause of death data and renal registry data. In clinical practice, assigning chronic kidney disease to a particular cause might be difficult for patients with both hypertension and diabetes.

Seventh, in some unusual cases such as chronic respiratory diseases in India, the sum of modelled estimates for CoDCorrect level 2 causes are much smaller than the level 1 modelled estimate leading to very large corrections for the CoDCorrect step. Very large corrections for CoDCorrect suggests that the component models for these causes can be improved in the future with better data or methods.

Eighth, for natural history models, most notably for HIV/AIDS, changes in parameter assumptions such as the death rate on or off antiretroviral therapy, can have a large effect on estimated mortality. We believe that progressive revision of these models improves the estimates but nevertheless, validation of natural history models is difficult. For CODEm, we were able to quantify with the cross-validation strategy model performance but this is not possible with the natural history models.

Ninth, a strength of the GBD approach is that all estimates of cause-specific mortality must sum to all-cause mortality in a country-age-sex-year group. However, this means that estimates for a specific cause are affected by the estimates for all other causes. Causes of death such as malaria, that have very wide UIs are particularly affected by the estimates of other causes.

Tenth, models used to generate estimates of all-cause mortality and cause-specific mortality make use of a long list of covariates. Uncertainty in these covariates, such as GDP per head, was not routinely quantified but nevertheless might be substantial. We were not able to propagate uncertainty in the independent variables used in the modelling stages into the final results. 95% UIs might therefore be under-estimated. However, when we have tested in a few cases the effect of propagating uncertainty in the independent variables in the case of the HIV crude death rate, the changes to UIs, were minor (data not shown).

Eleventh, we made extraordinary efforts to propagate uncertainty throughout our all-cause mortality estimation process, which is not yet common practice in modern demographic research. However, uncertainty in covariates used in the first stage model of child and adult mortality rate was not included because of the complexity of added computation and the fact that these covariates have little effect on our final estimates, as indicated by our preliminary testing.

Lastly, empirical age patterns of mortality, which are vital for the estimation of mortality for many low-income and middle-income countries, mostly come from high-income countries with great vital registration systems and some low-income and middle-income countries in the most recent period. Countries in the sub-Saharan African region are least represented in our empirical database of age pattern of mortality (appendix pp 81–89). Propagating uncertainty from both under-5 and adult mortality rates (two key entry parameters for our new model life-table system), and from the standard life-table generation process has given our death estimates in sub-Saharan African countries substantial uncertainty; accurate documentation of age pattern of mortality in these countries are key for producing best all-cause mortality estimates in the future.

## Conclusion

Global public policy to reduce premature death needs a detailed, up-to-date, and accurate understanding of progress (or lack thereof) of disease and injury control strategies. This understanding applies not just to diseases that have been the focus of global public health efforts for the past few decades, but increasingly, as we have shown, for newly recognised contributors to global health trends. Through the process of providing yearly updates, the GBD is transforming into a collective approach to global health surveillance. Ideally, it will aggregate data from all available sources and provide a coherent view of health levels and trends that is timely, valid, and local. To fully achieve a collective process of global health surveillance, the time lag will need to be shortened between data collection, reporting, and inclusion in the GBD. Public policy in countries will be much better informed if more frequent assessments are accompanied by less uncertainty around the estimates. Uncertainty will decrease not so much as a result of further methodological advances in disease

modelling and data synthesis, but much more as a result of greater investment and awareness among countries and donors alike of the need to strengthen vital registration systems.

Global collective action to reduce mortality from major communicable diseases such as diarrhoea, measles, tetanus, tuberculosis, and, more recently, HIV/AIDS and malaria, is working, but will require continued intervention efforts and resources and will probably be even more responsive if periodic assessments such as that reported here are available and used. While progress is being made to control several major non-communicable diseases of global concern, others have been largely neglected but are rising in importance, particularly drug use disorders, cirrhosis, diabetes, and chronic kidney disease. Greater prominence to reducing disease burden from these diseases, as well as continuing priority for injury control, is strongly suggested by our analysis. The findings on global, regional, and national trends in mortality from diseases should provide an important baseline for discussions about the next generation of health goals and targets after the Millennium Development Goals.

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ADL and CJLM conceived the study and provided overall guidance. CJLM, ADL, MN, and HW prepared the first draft. All other authors provided data, developed models, reviewed results, initiated modelling infrastructure, or reviewed and contributed to the report.

### Declaration of interests

BDG works for AMP, which receives grant support for vaccine and immunisation related work from Crucell, GlaxoSmithKline, Merck, Novartis, Pfizer, and Sanofi Pasteur; however, none of this support is for work related to the present report. KJ reports has consulted for GlaxoSmithKline on projects outside the submitted work. WM is program analyst at the UNFPA country office in Peru, which does not necessarily endorse the study. JAS has received research grants from Takeda and Savient and consultant fees from Savient, Takeda, Regeneron, and Allergan. JAS is a member of the executive of OMERACT, which receives funding from 36 companies; a member of the American College of Rheumatology's Guidelines Subcommittee of the Quality of Care Committee; and a member of the Veterans Affairs Rheumatology Field Advisory Committee. RFG is associate editor of *Annals of Epidemiology* for which he receives a stipend. CK receives research grants from Brazilian public funding agencies Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), and Fundação de Amparo à Pesquisa do Estado do Rio Grande do Sul (FAPERGS). He has also received authorship royalties from publishers Artmed and Manole. GR has consultancy agreements with Alexion Pharmaceuticals, Reata Pharmaceuticals, Bayer Healthcare, and Novartis Pharma, and is a member of the Abbvie Atrasentan Steering Committee; GR does not accept personal remuneration, compensations are paid to his institution for research and educational activities. MDH has received research support from the National Heart, Lung, and Blood Institute and World Heart Federation for its Emerging Leaders program, which is supported by unrestricted educational grants from AstraZeneca and Boehringer Ingelheim. FP-R has received investigation grants from Ministerio de Sanidad, Gobierno de España, Asociación de Reumatólogos del Hospital de Cruces, Fundación Española de Reumatología; has been a consultant (with or without payment) for Astra-Zeneca, Menarini, Metabolex, Ardea Biosciences, SOBI, Novartis, and Pfizer; and has been a speaker for AstraZeneca and Menarini. KBG received the NHMRC-Gustav Nossai scholarship sponsored by CSL Behring in 2013. MGS has previously served as consultant for Ethicon on global surgery. PJ is supported by a career development fellowship from the Wellcome Trust, Public Health Foundation of India, and a consortium of UK universities. DAQ was supported by The Eunice Kennedy Shriver National Institute of Child Health and Human Development of the National Institutes of Health (number 5T32HD057822). AK has received institutional support (intramural funding) from the Oklahoma State University Center for Health Sciences. RAL receives funding through the Farr Institute of Health Informatics Research. The Farr Institute is supported by Arthritis Research UK, British Heart Foundation, Cancer Research UK, Economic and Social Research Council, Engineering and Physical Sciences Research Council, Medical Research Council, National Institute of Health Research, National Institute for Social Care and Health Research (Welsh Government), and the Chief Scientist Office (Scottish Government Health Directorates), (MRC grant MR/K006525/1). DM reports ad hoc honoraria from Bunge, Pollock Institute, and Quaker Oats; ad hoc consulting for Foodminds, Nutrition Impact, Amarin, AstraZeneca, Winston and Strawn LLP, and Life Sciences Research Organization; membership of Unilever North America Scientific Advisory Board; and chapter royalties from UpToDate. RD and LB are employed by the US Department of Veterans Affairs. VC is on the speaker bureau for Boehringer Ingelheim Baker. MS is an employee of Novartis Pharma. All

other authors declare no competing interests. The authors alone are responsible for the views expressed in this Article and they do not necessarily represent the views, decisions, or policies of the institutions with which they are affiliated.

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