In south Asia, the rise of risk factors for non-communicable diseases is shown by the substantial increase in the burden attributable to tobacco smoking including second-hand smoke, high blood pressure and other metabolic risk factors, dietary risk factors, and alcohol use. However, household air pollution from solid fuels was, despite decreases, the leading risk factor in 2010. Childhood underweight was still the fourth leading risk factor in 2010, despite its share of disease burden having more than halved from 11·9% [95% UI 10·1–14·4] of DALYs in 1990, to 4·0% [3·2–4·9] in 2010. Other risk factors for communicable disease, such as suboptimal breastfeeding and micronutrient deficiencies, fell substantially in the region as child mortality decreased.

In southeast, east, and central Asia, the epidemiological transition was already well advanced in 1990, and by 2010, high blood pressure (which is commonly associated with diets high in sodium as a prominent underlying cause[94,158]), tobacco smoking including second-hand smoke, and diets low in fruits were all among the five leading risk factors in these regions. The disease burden attributable to childhood underweight and suboptimal breastfeeding had been largely eliminated in east Asia by 2010, although they remain important in southeast Asia. In these three regions, despite decreases, household air pollution from solid fuels was still a leading risk factor in 2010, ranked third in southeast Asia, sixth in east Asia, and 12th in central Asia. Ambient particulate matter pollution accounted for a larger disease burden than did household air pollution in central and east Asia in 2010, although household solid fuels is an important source of ambient particulate matter pollution in these regions.

The North Africa and Middle East region also had a large shift from risk factors for communicable to non-communicable diseases. In 2010, risk factors for non-communicable disease almost exclusively dominated the region's causes of loss of health, with high blood pressure and high body-mass index each accounting for roughly 8% of disease burden, followed by tobacco smoking including second-hand smoke, high fasting plasma glucose, and physical inactivity or low physical activity. Ambient particulate matter pollution (seventh leading risk factor) is a notable cause of disease burden in this region, caused by a combination of polluted cities and dust from the Sahara desert.

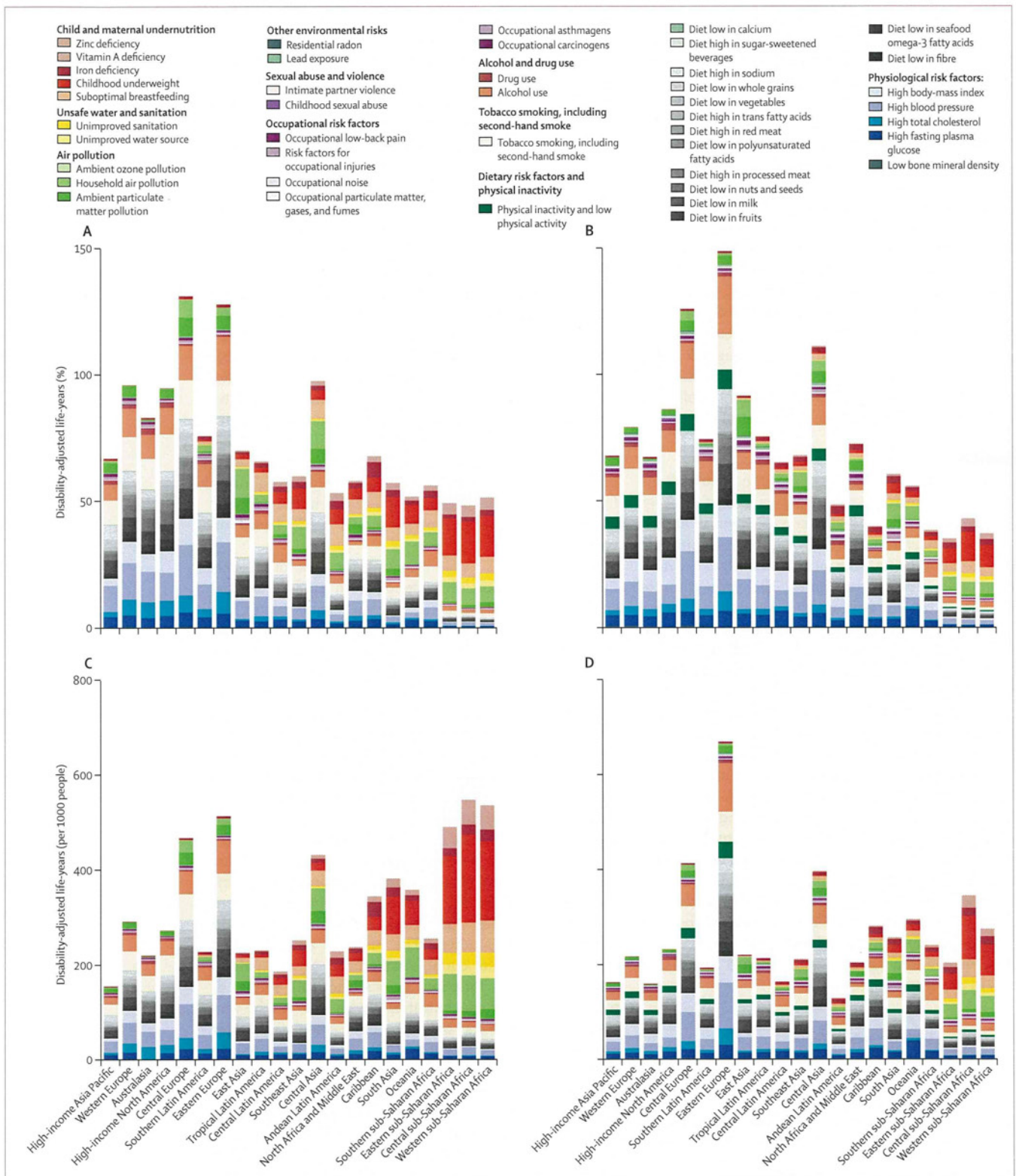
Alcohol use was an important cause of disease burden in most of Latin America. It was ranked first in central Latin America, fourth in tropical Latin America, and sixth in Andean Latin America in 1990, and first in all these regions in 2010. Risk factors for childhood communicable disease had been largely replaced by those causing non-communicable diseases in these regions by 2010, although household air pollution from solid fuels was still an important risk factor in Andean Latin America in 2010.

One of the most notable findings was the effect of alcohol use in Eastern Europe, where it accounts for almost a quarter of total disease burden. Other risk factors, such as high blood pressure, tobacco smoking including second-hand smoke, high body-mass index, and dietary risks, also feature prominently, underscoring the large underlying burden of cardiovascular disease in the region.

In North America, Australasia, southern Latin America, and western Europe, the share of disease burden attributable to tobacco smoking including second-hand smoke has fallen slightly; it has stayed almost constant in central Europe and high-income Asia Pacific. Tobacco smoking including second-hand smoke was still the leading risk factor in 2010 in North America and western Europe. Important decreases in disease burden are evident for high blood pressure and total cholesterol in North America, Australasia, and western Europe. High blood pressure is a leading risk for health in high-income Asia Pacific (accounting for 8·5% [95% UI 7·1–10·1] of disease burden) and central Europe (18·9% [16·8–20·8]); evidence from individual-level trials of salt and blood pressure and from cross-population studies indicates that this result is likely to be driven partly by high salt consumption in these regions.[94,158] Falls in disease burden attributable to tobacco smoking including second-hand smoke, high blood pressure, and high total cholesterol in high-income regions have been partly offset by the increasing burden caused by high body-mass index. In southern Latin America, high body-mass index accounted for almost 10% of overall disease burden in 2010, and is the leading risk factor in southern Latin America and Australasia.

Figure 6 summarises these regional patterns, in relation to the proportion of regional burden and attributable DALYs per 1000 people. Regions in figure 6 are ordered by mean age of death, a marker of the epidemiological transition. Figure 6 shows the clear transition away from risk factors for childhood communicable disease towards risk factors for non-communicable disease, with increasing mean age at death. This change is apparent from the decrease in burden of disease attributable to undernutrition and unimproved water and sanitation, with increased mean age at death, especially when the effect of risks is assessed by DALYs per 1000 people (figure 6C, D). A clear general shift occurs towards a larger proportion of overall burden arising from risk factors for non-communicable diseases, particularly metabolic risks and dietary risk factors (figure 6A, B). However, the absolute burden of risk factors for non-communicable disease does not increase with increasing mean age at death. Rather, its magnitude is lower in high-income regions than in sub-Saharan Africa and south Asia (figure 6C, D), showing the double burden of communicable and non-communicable disease in regions early in the epidemiological transition.

Some risk factors deviated from the pattern of the proportional burden (percent of region-specifc DALYs attributable to a risk factor) being closely associated with epidemiological and demographic transition (shift from


Child and maternal undernutrition
Zinc deficiency
Vitamin A deficiency
Iron deficiency
Childhood underweight
Suboptimal breastfeeding
Unsafe water and sanitation
Unimproved sanitation
Unimproved water source
Air pollution
Ambient ozone pollution
Household air pollution
Ambient particulate matter pollution
Other environmental risks
Residential radon
Lead exposure
Sexual abuse and violence
Intimate partner violence
Childhood sexual abuse
Occupational risk factors
Occupational low-back pain
Risk factors for occupational injuries
Occupational noise
Occupational particulate matter, gases, and fumes
Occupational asthmagens
Occupational carcinogens
Alcohol and drug use
Drug use
Alcohol use
Tobacco smoking, including second-hand smoke
Tobacco smoking, including second-hand smoke
Dietary risk factors and physical inactivity
Physical inactivity and low physical activity
Diet low in calcium
Diet high in sugar-sweetened beverages
Diet high in sodium
Diet low in whole grains
Diet low in vegetables
Diet high in trans fatty acids
Diet high in red meat
Diet low in polyunsaturated fatty acids
Diet high in processed meat
Diet low in nuts and seeds
Diet low in milk
Diet low in fruits
Diet low in seafood omega-3 fatty acids
Diet low in fibre
Physiological risk factors:
High body-mass index
High blood pressure
High total cholesterol
High fasting plasma glucose
Low bone mineral density
A
B
C
D
Disability-adjusted life-years (%)
Disability-adjusted life-years (per 1000 people)
0
50
100
150
0
200
400
600
800
High-income Asia Pacific
Western Europe
Australasia
High-income North America
Central Europe
Southern Latin America
Eastern Europe
East Asia
Tropical Latin America
Central Latin America
Southeast Asia
Central Asia
Andean Latin America
North Africa and Middle East
Caribbean
South Asia
Oceania
Southern sub-Saharan Africa
Eastern sub-Saharan Africa
Central sub-Saharan Africa
Western sub-Saharan Africa

communicable to non-communicable disease with increasing mean age of death). The proportion of DALYs attributable to tobacco smoking including second-hand smoke was largest in North America—where smoking among women has generally been prevalent for a long time—and central and eastern Europe. Central and eastern Europe and central Asia also had the largest proportion of disease burden attributable to risk factors with large effects on cardiovascular diseases, which are disproportionately high in these regions. Exposure to particulate matter from household and ambient sources had the most varied pattern with respect to the epidemiological transition, partly because of the heterogeneous pattern of exposure and the effects on both children and adult causes of ill health. Household air pollution from solid fuels accounted for a large proportion of disease burden in central, eastern, and western sub-Saharan Africa and it is a leading risk factor in some Asian regions and Oceania. In central and east Asia in 2010, ambient particulate matter pollution surpassed household air pollution in terms of its burden.

Discussion

The results of GBD 2010 suggest that the contributions of risk factors to regional and global burden of diseases and injuries has shifted substantially between 1990, and 2010, from risk factors that mainly cause communicable diseases in children to risk factors that mainly cause non-communicable diseases in adults. The proportion of overall disease burden attributable to childhood underweight—the leading risk factor worldwide in 1990—had more than halved by 2010, making childhood underweight the eighth risk worldwide, behind six behavioural and physiological risks, and household air pollution from solid fuels. Other risks for child mortality, such as non-exclusive and discontinued breastfeeding, micronutrient deficiencies, and unimproved water and sanitation, have also fallen. However, child and maternal undernutrition risks collectively still account for almost 7% of disease burden in 2010, with unimproved water and sanitation accounting for almost 1%. Of the non-communicable disease risks, high blood pressure, high body-mass index, high fasting plasma glucose, alcohol use, and dietary risks have increased in relative importance. This overall shift has arisen from a combination of the ageing population, substantial achievements in lowering mortality of children aged younger than 5 years, and changes in risk factor exposure.

Figure 6: **Attributable burden for each risk factor**
As percentage of disability-adjusted life-years in 1990 (A), and 2010 (B), and as disability-adjusted life-years per 1000 people in 1990 (C), and 2010 (D). Regions ordered by mean life expectancy. Burden of disease attributable to individual risk factors are shown sequentially for ease of presentation. In reality, the burden attributable to different risks overlaps because of multicausality and because the effects of some risk factors are partly mediated through other, more proximal, risks. An interactive version of this figure is available online at http://healthmetricsandevaluation.org/gbd/visualizations/regional.

These broad global patterns mask enormous regional variation in risks to health. In sub-Saharan Africa, risks such as childhood underweight, household air pollution from solid fuels, and suboptimal breastfeeding continue to cause a disproportionate amount of health burden, despite decreasing. The shift to risk factors for non-communicable disease was clear in east Asia, North Africa and Middle East, and Latin America. This regional heterogeneity underestimates even greater differences in exposure to, and health effects of, risk factors in national and subnational populations. These differences should be further elucidated in country-specific analyses using the framework and methods reported here.

Our analysis shows the large burden of disease attributable to primary and secondary tobacco smoking and to particulate matter pollution in household and ambient environments. The magnitude of disease burden from particulate matter is substantially higher than estimated in previous comparative risk assessment analyses. For example, ambient particulate matter pollution was estimated in the previous comparative risk assessment[7] to account for 0·4% of DALYs in 2000 compared with 3·1% in GBD 2010 based on interpolating our 1990 and 2005 results; for household air pollution from solid fuels the comparison is 2·7% in the previous comparative risk assessment versus 5·3% based on GBD 2010.

Several reasons could account for this difference. First, accumulation of evidence from epidemiological studies about diseases caused by particulate matter, and the use of an integrated exposure–response curve, has led to the inclusion of more outcomes than before. For example, health effects for ischaemic heart disease and stroke were not previously included for household air pollution from solid fuels, and lung cancer was included for coal smoke only. Second, the previous assessment of ambient particulate matter pollution was restricted to medium and large cities. High-resolution satellite data and chemical transport models have enabled us to quantify exposure and burden for all rural and urban populations. Third, the previous assessment of ambient particulate matter pollution did not include additional increments of risk above a concentration of 50 μg/m³ for $PM_{2·5}$, because of the narrow range of ambient particulate matter pollution levels reported in epidemiological studies. The use of an integrated exposure–response curve enabled us to estimate a continuous risk function across the full range of particulate matter concentrations, which covers the very high concentrations of ambient particulate matter exposure measured in, for example, parts of east Asia.

Our integrated exposure–response curve, however, does not address how different sources of particulate matter interact in terms of effects and overlapping exposures. Studies[124,159,160] have reported broadly similar effect sizes for ambient particulate matter by smoking status (never, former, and current smokers). Other evidence[161] shows

that the effects diminish with increasing exposure for active smoking, a pattern incorporated into our exposure–response curves. We applied the effects of ambient particulate matter to both smokers and non-smokers alike to be consistent with the epidemiological evidence that emphasises independent effects of ambient particulate matter. The reasons for the independent effects of different sources of particulate matter should be further investigated. They might include different compositions of particulate matter by source, or different time patterns of exposure[162]—eg, exposure to particulate matter from active smoking is characterised by episodic, high doses whereas exposure to ambient particulate matter is more constant over time.

These limitations aside, the large attributable burden documented in our analysis represents a major shift in our understanding of disease burden arising from particulate matter and emphasises the need to design alternative fuels for household cooking and heating,[163] implement more stringent regulation of vehicle and industrial emissions,[164–166] reduce agricultural burning or land clearing by fire,[167] and curb and reverse deforestation and desertification to reduce ambient particulate matter from dust.[168–171] A large share of ambient particulate matter in Asia and sub-Saharan Africa originates from solid fuel.[172,173] Therefore the two exposures are related, and alternative cooking and heating fuels would have benefits for people who currently use solid fuels as well as those who do not, but live in the same community.[173]

Unimproved water and unimproved sanitation together accounted for 0·9% of DALYs in 2010, compared with 2·1% in 1990. These proportions are substantially smaller than the 6·8% for 1990, and 3·7% for 2000, estimated in previous GBD studies for water, sanitation, and hygiene combined.[3,7] The relatively small burden estimated for 2010 is partly related to decreases in diarrhoeal disease mortality since 1990, and partly to differences in the distributions of deaths by underlying cause of death. We have also done an updated meta-analysis of quasiexperimental and experimental studies. Historical demographic analyses suggest that the introduction of piped water into cities in the late 19th and early 20th centuries had a large beneficial effect on mortality.[174] However, our re-analysis both when restricted to experimental studies and when also including quasiexperimental studies did not detect a significantly improved effect of household water connections over improved water sources. Similarly, we did not find a significantly improved effect of water quality interventions, consistent with the findings reported by Cairncross and colleagues,[128] which showed that masked point-of-use water quality interventions did not have a significant effect on self-reported diarrhoea. As a result of this reassessment, we restricted our analysis to improved water and improved sanitation compared with unimproved sources following the MDG definitions. However, the interventions used in previous studies might not have achieved their full efficacy because of the quality of implementation. The real burden from water and sanitation could therefore be underestimated if well-implemented household connections and water quality interventions have a larger effect than improved water sources alone, and if the combination of poor water and sanitation has a larger effect than a sample interaction of individual effects. More definitive epidemiological evidence is needed to assess the effects of low quality versus high quality water, household connections versus improved water sources, and exposure based on travel time to water source.[175] Also, we could not include an assessment of personal hygiene because of the paucity of national exposure data.

Our findings on the burden of micronutrients are also substantially smaller than those in the previous comparative risk assessment for 2000 and in estimates for 2004 by Black and colleagues[10] in *The Lancet*'s Maternal and Child Undernutrition Series. For example, Black and colleagues estimated 668 000 deaths caused by vitamin A deficiency in 2004; we estimated a quarter (168 000 deaths) for 2005; for zinc deficiency, the differences are similarly large (453 000 *vs* 120 000). These differences stem from many sources. First, the estimates of Black and colleagues were based on 10·3 million child deaths worldwide, itself based on WHO estimates of global child deaths for 2004. This estimate is substantially larger than those reported by UNICEF[176] and the Institute for Health Metrics and Evaluation[177] at the time of Black and colleagues' publication.

Large differences also exist for cause-specific mortality, especially in relation to diarrhoea and lower respiratory tract infections (which can be affected by both of these risks) versus malaria (which is not).[176] The estimates also differ because of differences in the availability and interpretation of epidemiological evidence for disease outcomes and effect sizes. Maternal mortality and malaria as outcomes of vitamin A deficiency were included in the 2000 comparative risk assessment but they were not included in the present report because recent epidemiological evidence did not show a significant effect of supplementation on these outcomes. Furthermore, we excluded neonatal vitamin A deficiency since it is the subject of three ongoing randomised trials. The age at which the effects of zinc deficiency begin was increased from birth in the 2000 comparative risk assessment, to 6 months in 2004,[10] and to 12 months in the present analysis based on a reassessment of existing and new supplementation trials. Furthermore, we quantified the proportion of the population who are vitamin A or zinc deficient instead of classing whole countries as exposed or non-exposed. The evolving epidemiology of exposure to micronutrient deficiency and the subsequent health effects suggests a need to systematically reconsider most single nutrient supplementation for children in preventive strategies to lower child mortality, as suggested by the 2000 comparative risk assessment and later analyses.[10] Therapeutic zinc supplementation in health-care

settings is feasible, as is iron supplementation during pregnancy.[174–179] Our findings support the need for strengthened policy about promotion of optimal breastfeeding practices and nutritional programmes that improve child growth. The estimated number of child deaths caused by underweight has also changed substantially over successive studies: in GBD 1990 it was estimated to be 5·9 million deaths in 1990,[180] in the comparative risk assessment study for 2000 as 3·7 million deaths,[7] and 1·9 million deaths in 2004.[10] In GBD 2010 we estimated 2·3 million deaths for 1990 and 0·9 million deaths for 2010.

The evolution of estimates for deaths caused by childhood underweight is because of improvements in assessment of the population at risk. These improvements come from systematic analysis of the available data on underweight, a major modification of RRs after the change in the WHO standard in 2006, and differences in estimates of total and cause-specific mortality. We have also assessed the burden attributable to childhood wasting and childhood stunting. These analyses produce quite similar findings, for example, worldwide, childhood wasting accounted for 0·7 million deaths in 2010, and childhood stunting for 0·9 million deaths, compared with 0·9 million deaths for childhood underweight (the effects of these risks cannot be added).

The global burden of disease attributable to tobacco smoking including second-hand smoke has changed little, with decreases in high-income regions offset by increases in regions such as southeast Asia and, to a lesser extent, east and south Asia. The burden attributable to alcohol use has increased substantially in eastern Europe since 1990, mainly because of a rise in the effects of heavy drinking on cardiovascular diseases.[181] The high burden in eastern Europe was also identified in the 2000 comparative risk assessment but the data for patterns of alcohol consumption and their effects were weaker, whereas now they are supported by more surveys and epidemiological studies.[182] High blood pressure, high body-mass index, and high fasting plasma glucose are leading risk factors for disease worldwide, with blood pressure having large effects on population health in all regions, including low-income regions in sub-Saharan Africa and south Asia. This finding is consistent with previous comparative risk assessment analyses. The disease burden in south Asia and sub-Saharan Africa, caused by increased blood pressure,[70] has increased its absolute and relative importance in risk factor rankings. The large burden of high blood pressure emphasises the importance of implementing both population-wide and high-risk approaches to reduction of blood pressure.[183,184] The worldwide increase in body-mass index and blood glucose is of particular concern in view of the absence of effective interventions.[62,74] In contrast to these risks, the burden of high total cholesterol is lower than that estimated in the 2000 comparative risk assessment, because the effects on ischaemic stroke were negligible at old ages when data from the Asia-Pacific Cohort Studies Collaboration and Prospective Studies Collaboration were pooled,[68,185] and because exposure has fallen in high-income countries.[67]

A recent study estimated that 5·3 million deaths were attributable to physical inactivity in 2008.[186] This number, which has been widely quoted and equated with the number of deaths attributable to tobacco smoking,[187] used effect sizes for all-cause mortality obtained from cohorts of adults mainly from North America and Europe and applied these effects to deaths at all ages. This approach not only assumes that the cause distribution is the same in all populations, irrespective of region and age structure, but also extends the effects to people younger than those in the cohort study, including to infants and children. In other words, a proportion of deaths from maternal causes, neonatal causes, and children's infectious diseases and HIV were attributed to physical inactivity.[186] The prevalence of inactivity also included people who had sedentary patterns as well as those in the low (insufficient) activity group. By contrast, our approach—calculating attributable burden by cause and age group, and accounting for exposure in four categories—estimated substantially fewer attributable deaths: 3·2 million (2·7 million to 3·7 million) in 2010, 56% of what we attribute to tobacco smoking when second-hand smoke is excluded. This discrepancy shows the importance of comparable risk factor assessments and the importance of estimation of attributable burden taking into account differences in underlying disease and injury patterns across populations.

We have expanded the set of components of diet included from a combined category of fruits and vegetables in the 2000 comparative risk assessment to 15 components in GBD 2010; together these dietary risk factors account for a tenth of global disease burden. Of the dietary risk factors, the aetiological effect sizes for sodium, polyunsaturated fatty acids replacing saturated fatty acids, and seafood omega-3 fatty acids were informed fully (for sodium) or partly by randomised controlled trials. Disease burden attributable to diet high in sodium was a third of that for high blood pressure. The theoretical-minimum-risk exposure distribution was selected on the basis of values reported in randomised trials; studies of populations with low prevalence of cardiovascular disease suggest that benefits are likely to continue to lower levels.[158]

The large attributable burden for dietary risk factors such as diets low in fruits, vegetables, whole grains, nuts and seeds, and seafood omega-3 fatty acids might surprise some readers. The large burden is caused by both high exposure—eg, low intake of fruits in many regions—and large effect sizes. We did supplementary analyses using information from studies of dietary patterns and randomised controlled feeding studies to examine the robustness of the effect sizes used in GBD 2010. The findings of these supplementary analyses were consistent with those from the meta-analyses of single risk factors.

However, we stress that these results should still be interpreted with caution, particularly because of the debate surrounding the effects of seafood omega-3 fatty acids.[143,188] Empirical assessments show that the pooled effect of risks and interventions trends towards a null result over time[189,190] and this pattern could apply to seafood omega-3 fatty acids since the earlier, primarily observational effect sizes tended to show a larger effect than did the more recent randomised controlled trials. Because the difference between results of observational studies and randomised controlled trials is not statistically significant we have quantified the attributable burden by use of the combined effect size. However, the validity of this approach could change as new evidence accumulates. Also, evidence from randomised controlled trials does not exist for several of the dietary components with a large attributable burden—fruits, vegetables, and nuts and seeds—although, as previously noted, evidence from randomised controlled trials does exist for intermediate outcomes. Further work is needed to confirm the effect size of dietary components and to establish to what degree the benefits continue, preferably through intervention studies of fatal and non-fatal events.

The extended analysis of components of diet does not include saturated fat beyond its replacement by polyunsaturated fats. Ecological studies suggest that saturated fat intake is a significant risk factor for mortality from ischaemic heart disease.[191] However, observational studies indicate that there might be no benefits if saturated fat reduction is associated with an increase in carbohydrates,[91] which is also supported by the absence of benefits from a low fat diet in the Women's Health Initiative.[192] Together with data for seafood omega-3 fatty acids, these findings show the complexity of the relation between dietary fat and health and suggest that the traditional health education message focused on lowering saturated fat alone needs to be expanded greatly to encompass several other key components of diet, including increased consumption of healthy foods that are presently missing from most diets.

The strengths of our study include a more comprehensive set of risk factors than any previous global or national analysis, consistent analyses in 1990, and 2010, which enables assessment of changes in risk factor burden, the incorporation of substantially more data for risk-factor exposure, improved methods to deal with missing and incomparable data, strong emphasis on comparability of methods related to exposure, disease outcomes, and effect sizes, and use of theoretical-minimum-risk exposure distribution as the consistent alternative exposure distribution with which current exposures are compared.

Like all population-based analysis, our study also has some limitations. First, despite the massive improvement in the availability of exposure data and methods, exposure estimates for many risk factors are affected by data limitations, especially for 2010, since fewer data could be included. This limitation will become even more salient in applications of our methods to individual countries and shows the importance of surveillance of national risk factors as a crucial component of national health information systems. More importantly, for some risk factors we have less direct measures of exposure than for others. For example, for household air pollution from solid fuels we measured exposure on the basis of household fuel use rather than personal exposure to particulate matter; for other risks, such as blood pressure, we have direct biological measurements of exposure.

Second, the presence of residual confounding in the estimates of effect sizes cannot be definitively ruled out, particularly for those without evidence from intervention studies, either because they have not yet been done or the risk is not amenable to intervention. For example, no large-scale trials have been done of interventions for high body-mass index that measured cause-specific deaths although effects on disease incidence have been investigated in trials.[193] Observational studies of the effect sizes for body-mass index have controlled for some potential confounders.[75–77] As noted, the pooled effect of risks and interventions trends towards the null result over time;[189,190] the implication being that risks for which only a few studies have been done might have their effect overestimated compared with risks for which a large body of evidence exists.

Third, with the exception of risk factors for which much evidence has been accumulated across diverse populations and age groups, such as the metabolic risks, uncertainty remains as to the extent to which effect sizes are generalisable to different populations. Similarly, the large body of epidemiological evidence for cardiovascular risk factors shows a relation between age and the effect size of risk factors for cardiovascular disease. Such age-related changes might be present for other outcomes. Fourth, we have combined epidemiological evidence for effect sizes using studies across different periods, which could mask underlying temporal changes in risk; no data presently exist to enable an examination of the extent to which effect sizes might change over time.

Fifth, we have excluded risks for which insufficient information exists to enable estimation of exposure, or for which the evidence of effect sizes is scarce. This approach excludes several risk–outcome pairs that have been previously included in global and regional assessments of risk factor attributable burden, such as unsafe sex and global climate change. Unsafe sexual practices were included in the 2000 comparative risk assessment but we excluded it because of the absence of robust estimates of exposure or available approaches to determine the proportion of HIV infection that is attributable to unsafe sexual practices by country over time. If quantifiable, unsafe sexual practices would probably account for a large fraction of global health burden; the direct burden of HIV is 3·3% of DALYs in 2010; other sexually transmitted infections account for 0·4% of DALYs. Similarly, we have been unable to

control for confounding in observational studies of late initiation of breastfeeding, which is associated with an increased risk of neonatal mortality. Infants who might too ill or weak to breastfeed are more likely to die. In our analysis, we could not assess low birthweight as an outcome for maternal iron deficiency, despite evidence from randomised trials. Similarly, we could not assess low birthweight as an outcome for maternal alcohol use. Low birthweight was not a disease outcome in GBD 2010 but is associated with an increased risk of neonatal mortality. We excluded several other risk–outcome pairs that had insufficient evidence to estimate effect sizes or that had substantial potential of residual confounding—eg, the effect of addictive drugs (cannabis, amphetamines, and opioids) on unintentional and intentional injuries; or the effects of intimate partner violence, on HIV or other sexually transmitted infections.

Sixth, we included few risks that affected three of the leading communicable diseases—HIV/AIDs, tuberculosis, and malaria (beyond deaths in childhood). Overall, we have not included risks for 126 of the 241 most detailed causes included in the GBD, which account for 26·3% of global disease health burden. This shortcoming emphasises the need for a more deliberate research focus to identify and quantify risk factors for the outcomes for which there are presently no risks or few large risks.

Seventh, we have quantified the attributable burden of risk factors, holding all other independent factors constant. For clusters of risk factors we have approximated the joint effects, assuming that risk factors within each cluster are independent. A more accurate quantification of the joint effects of multiple risk factors is an important area for future research. Finally, it is important to stress that the size of the attributable risk factor burden does not equal priority for action since prioritisation also depends on availability, cost, and effectiveness of intervention strategies to reduce exposures to these risks.

Public policy to improve the health of populations will be more effective if it addresses the major causes of disease burden. Even small reductions of population exposure to large risks will yield substantial health gains.[194] The principal advantage of doing a comprehensive and comparable scientific assessment of disease burden caused by different risk factors is that it provides the evidence base for informing discussion about policy. Coupled with evidence of their present burden, most of the leading risk factors, except high body-mass index and high fasting plasma glucose, have decreased in at least some regions and countries, showing that substantial reduction of their effect through targeted prevention strategies is feasible. If predictions about huge increases in disease burden worldwide are to be proved wrong, then countries, with appropriate global public health leadership, must urgently implement measures to control exposure to leading hazards, particularly risks for non-communicable diseases.

Contributors

CJLM, SSL, and ME wrote the first draft. SSL, TV, AF, GD, KS, ADL, CJLM, and ME revised the report. ME, CJLM, and ADL designed the study and provided overall guidance. SSL, EC, GF, CA, ESa, KA, REE, and LCR did comparative analyses of risk factors. All other authors developed the estimates of risk-specific exposure, theoretical-minimum-risk exposure distribution, and RR inputs, and checked and interpreted results.

Conflicts of interest

A Davis is employed by the NHS on works for the UK Dept of Health as lead adviser on audiology. E R Dorsey has been a consultant for Medtronic and Lundbeck and has received grant support from Lundbeck and Prana Biotechnology. M Ezzati chaired a session and gave a talk at the World Cardiology Congress (WCC), with travel cost reimbursed by the World Heart Federation. At the WCC, he also gave a talk at a session organised by Pepsico with no financial remuneration. G A Mensah is a former employee of PepsiCo. D Mozaffarian has received: ad hoc travel reimbursement and/or honoraria for one-time specific presentations on diet and cardiometabolic diseases from Nutrition Impact (9/10), the International Life Sciences Institute (12/10), Bunge (11/11), Pollock Institute (3/12), and Quaker Oats (4/12; modest); and Unilever's North America Scientific Advisory Board (modest). B Neal is the Chair of the Australian Division of World Action on Salt and Health. He has consulted to Roche and Takeda. He has received lecture fees, travel fees, or reimbursements from Abbott, Amgen, AstraZeneca, George Clinical, GlaxoSmithKline, Novartis, PepsiCo, Pfizer, Pharmacy Guild of Australia, Roche, Sanofi-Aventis, Seervier, and Tanabe. He holds research support from the Australian Food and Grocery Council, Bupa Australia, Johnson and Johnson, Merck Schering-Plough, Roche, Servier, and United Healthcare Group. He is not employed by a commercial entity and has no equity ownership or stock options, patents or royalties, or any other financial or non-financial support that might be viewed as a conflict of interest. L Rushton received honorarium for board membership of the European Centre for Ecotoxicology and Toxicology of Chemicals and research grants to Imperial College London (as PI) from the European Chemical Industry Council and CONCAWE.

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Data for better health—and to help end poverty

See Comment pages 2053, 2054, 2058, 2060, 2062, and 2063
See Special Report page 2067
See Articles pages 2071, 2095, 2129, 2144, 2163, 2197, and 2224

The World Bank Group welcomes the publication of the new Global Burden of Disease Study (GBD). The Bank commissioned the first GBD in 1990, and continues to make extensive use of this signal contribution to global health. Like its predecessors, the new, methodologically updated GBD 2010 marks a milestone in global health knowledge and our capacity for evidence-based action. It will once again set the terms of health policy, planning, and funding discussions for years to come.

The GBD gives us a data-rich framework for comparing the importance of different diseases, injuries, and risk factors in causing premature death and disability within and across populations. Its value lies not only in the data but the critical discussions it makes possible. Specifically, the GBD has sharpened thinking on issues as diverse as the measurement of comorbidities; the role of culture in mediating the experience of disease; the meaning of disability; and the impact of poverty on health. The GBD challenges us to be rigorous and clear in our arguments about the criteria that should guide programming and investment decisions at country, regional, and global levels.

GBD 2010 shows the remarkable health achievements of the past two decades, as well as the continuing, and emerging, challenges that require action. Life expectancy is rising, and the prevalence of many communicable diseases, including HIV/AIDS, is dropping. Yet in some parts of the world, preventable illnesses, such as diarrhoea, remain stubborn causes of death in childhood. We must confront the growing burden of non-communicable diseases, and the fundamental shift from premature death towards increasing years lived with chronic illnesses and debilitating conditions.

To respond effectively to these challenges, national and local health systems must be strengthened, even transformed, and policy and funding decisions across the development spectrum must be reassessed—from safety nets to urban planning. GBD 2010 is an indispensable resource for public health and development leaders to ensure that their investments yield the greatest possible health benefits, and to help end poverty and boost prosperity. The remarkable body of evidence and analysis in GBD 2010 will help us foster the conversations that are needed across the whole of government, not just in ministries of health, to fulfil this responsibility.

Jim Yong Kim
The World Bank, Washington, DC 20433, USA
president@worldbank.org

I am President of the World Bank Group. I declare that I have no conflicts of interest.

GBD 2010: a multi-investigator collaboration for global comparative descriptive epidemiology

See Comment pages 2053, 2054, 2058, 2060, 2062, and 2063
See Special Report page 2067
See Articles pages 2071, 2095, 2129, 2144, 2163, 2197, and 2224

The data, methods, and findings of the Global Burden of Disease Study 2010 (GBD 2010) are described in detail in *The Lancet*. This large collaboration is an evolution of a body of work that began with GBD 1990.[1] The number of diseases, injuries, and risk factors evaluated and the geographical units of analysis have greatly expanded in the past 20 years, and change over time has been assessed. Nevertheless, GBD 2010 follows the basic principles of GBD 1990: trying to use all the relevant published and unpublished evidence; capturing fatal and non-fatal health outcomes with comparable metrics; and separating epidemiological assessment from advocacy concerns or entanglement of agendas.[2] At the time of GBD 1990, the sum of cause-specific deaths presented by different disease groups substantially exceeded the number of deaths in the world, thereby highlighting the importance of firewalling epidemiological assessment from programmatic advocacy and of overcoming the differences in epidemiological traditions for individual diseases and risk factors.

In a 5-year study, the goal of GBD 2010 was to provide the strongest evidence-based assessment of people's health problems around the world. We sought to achieve this by incorporating expert knowledge through the engagement of the global health scientific community, collating the world's data on health outcomes,


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substantially strengthening analytical methods, and ensuring comparability across diseases, injuries, and risk factors. The study was organised around four broad components with different functions: expert groups on diseases, injuries, and risk factors; new data collection for disability weights and methods development; a strong analytical core; and the governance of the study.

The first component, expert groups, developed from an open call for participation in *The Lancet* in 2007;[3] experts were selected from the individuals who expressed interest and also through additional recruitment of leading experts. Most of the experts contributed their time and good will with minimal resources. Experts wrote lay descriptions for health states, led or contributed substantially to the review and selection of relevant data sources, provided input on epidemiological models for diseases, injuries, or risk factors, reviewed and reacted iteratively to the estimates of burden by diseases, injuries, and risk factors, and helped guide the interpretation of results in light of broader epidemiological evidence.

Rising expectations for statistical rigour and the need to estimate all quantities of interest with uncertainty led to an ambitious programme of new methods development. Innovations include new methods for analysing mortality data on child and adult survival,[4-6] new model life tables,[7] new methods for data synthesis,[8] new and more detailed methods for analysing garbage coding in causes of death,[9] the Cause of Death Ensemble model (CODEm),[10] the Codcorrect algorithm,[11] the development of a dedicated Bayesian meta-regression framework for disease and risk factor prevalence (DisMod-MR),[12] new methods for collecting and analysing data on assessments of disability, the comorbidity microsimulation environment,[12] new methods for estimating risk factor trends,[13,14] and the extensive computational machinery required to propagate uncertainty from all sources into the final estimates. Field data collection in five countries and an internet survey was also part of this component to provide a strong empirical basis for the new disability weights.

To ensure comparability, a strong analytical core of researchers were used to estimate causes of death, disease incidence and prevalence, risk factor exposure and attributable-burden, and healthy life expectancy.

GBD 2010 was governed by a core team whose charge included guiding the overall study, making decisions when consensus could not be achieved with the relevant expert groups, and approving all final estimates. This role for the core team was defined in the original study protocol given the likelihood that in such a large, complex scientific undertaking there would be topics on which consensus could not be reached.

No results were final until the very end of the study, because of the interconnections between components, such as all-cause mortality, cause-specific mortality, and disease or injury models. Although these interconnections made the work of expert groups and the analytical core more complex and iterative, they are also an important strength for the GBD approach. Evidence on a particular disease or injury is cross-validated against evidence on all-cause mortality with many safeguards built into the estimation process. In GBD 2010, these efforts at cross-validation have been extended to include a range of disabilities, such as vision loss, hearing loss, or anaemia.

For the comparative risk assessment, there are fewer internal validity checks since each risk factor or cluster of risk factors is evaluated on its own; multicausality means the same outcome can be related to multiple risks. To promote comparability and rigour, clear inclusion criteria were developed and applied by the core team, in consultation with epidemiological experts, to proposals on which risk-outcome pairs should be included in the study. The absence of public health or medical interventions such as vaccination or contraception was not considered a risk factor, although these should be included in intervention modelling studies. Other risk factors, such as total caloric intake, vitamin D and folate deficiencies, unsafe sexual behaviours, and personal hygiene could not be assessed because of the extreme lack of data on exposure.

In some cases, there was vigorous debate between the GBD core team and an expert group, and even within the GBD core team or within expert groups themselves, on inclusion or estimation: the potential for residual confounding of dietary risks, air pollution effects in smokers versus non-smokers, the effects of ambient air pollution on birth outcomes, maternal vitamin A deficiency on neonatal mortality, alcohol on tuberculosis, or intimate partner violence on HIV incidence. In each case, after lengthy and vigorous exchanges with the relevant experts, and when possible external experts, the core team—following the GBD protocol—convened and decided on whether the standard of evidence set

for the study had been met. Other groups of scientists might have used a lower bar for evidence or might have made different interpretations and choices on the basis of their knowledge of specific studies or even disciplinary background; nevertheless, a consistent approach was applied across risks in GBD 2010.

The innovations that were essential to modernise GBD methods and provide uncertainty intervals for all quantities of interest also created managerial challenges for the completion of the study. Experts involved in the collaboration were understanding about delays in key components, such as mortality or causes of death or of processing large amounts of disease and risk factor specific data that at times arrived simultaneously. To ensure a standardised approach to expert group consultation, from January, 2012 to June, 2012, every expert group was sent a detailed written report and set of global and regional tables on the results of the analysis for a disease, injury, or risk factor. These expert group reports were the basis of a final round of discussions and iterative corrections, as much as possible within the realm of a study with finite, although extended, time. At the end of GBD 2010, the final papers collectively have 486 authors from 302 institutions in 50 countries who have reviewed the final articles. In some cases, experts chose not be authors, possibly because their scientific interpretation of the evidence differed from the judgment of the GBD core team. This is reasonable and to be expected. Irrespective of the disagreements, these experts' inputs and views contributed to the GBD study and strengthened its findings. When evidence is strong, consensus is usually easy to obtain. When data are limited and there are only one or two studies available on a topic, reasonable scientists will disagree. Inclusion of uncertainty intervals in GBD 2010 conveys to users the limitations of the analysis. However, some choices are not reflected in the uncertainty intervals, such as which disease sequelae or risk-outcome pairs are included in the study or the absence of studies that measure the hazards of a risk factor, such as dietary salt intake or unimproved water, through the full exposure range. To the extent possible, these limitations are qualitatively discussed in the accompanying articles.

Future studies and data will strengthen the evidence, help overcome these limitations, reduce uncertainties, and confirm some of our results and revise others. The key principle is to synthesise and reflect the current state of the evidence using a set of clearly defined criteria and analytical methods; this is what the GBD 2010 collaboration has taken a major step towards. We do not expect that our processes, or the scientific basis that motivates them, will be universally acceptable: vast uncertainty, as we have quantified for some parameters and outcomes, ought to foster legitimate scientific discourse and debate. We welcome this response, which can only strengthen the evidence base and methodological armamentarium for future efforts to measure disease burden. Meanwhile, we believe that our rigorous adherence to established scientific principles and criteria will encourage greater confidence in the comparability of the results of GBD 2010, and thereby greater use of them.

*Christopher J L Murray, Majid Ezzati, Abraham D Flaxman, Stephen Lim, Rafael Lozano, Catherine Michaud, Mohsen Naghavi, Joshua A Salomon, Kenji Shibuya, Theo Vos, Alan D Lopez

Institute for Health Metrics and Evaluation, University of Washington, Seattle, WA 98121, USA (CJLM, ADF, SL, RL, MN); MRC-HPA Centre for Environment and Health, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, UK (ME); China Medical Board, Boston, MA, USA (CM); School of Public Health, Harvard University, Boston, MA, USA (JAS); School of Population Health, University of Queensland, Brisbane, Australia (TV, ADL); and Department of Global Health Policy, University of Tokyo, Tokyo, Japan (KS)

cjlm@uw.edu

We are all members of the GBD 2010 core team. ME chaired a session and gave a talk at the World Cardiology Congress (WCC) with travel cost reimbursed by the World Heart Federation. At the WCC, he also gave a talk at a session organised by PepsiCo with no financial or other remuneration. The other authors declare that they have no conflicts of interest.

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AIDS is not over

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See **Comment** pages 2053, 2054, 2055, 2060, 2062, and 2063

See **Special Report** page 2067

See **Articles** pages 2071, 2095, 2129, 2144, 2163, 2197, and 2224

Optimism and momentum has been building around the real possibility that an AIDS-free generation is imminent. Public enthusiasm is fuelled by news about the rapid scale-up of antiretroviral therapy, evidence that HIV treatment can prevent new infections, and expanded coverage of programmes to prevent mother-to-child transmission of HIV. Yet, the most recent estimates of HIV prevalence and incidence and of AIDS-related mortality released by UNAIDS[1], together with data from the Global Burden of Disease Study 2010 in *The Lancet*,[2,3] make it clear that AIDS is not over.

The estimates from the Global Burden of Disease Study 2010 confirm that HIV/AIDS remained a leading cause of disease burden and death in 2010.[3] It was ranked 33rd in 1990, but its burden had moved up to fifth by 2004[4] and remained there in 2010, despite major declines in AIDS-related mortality as a result of fewer new infections and the increased availability of antiretroviral therapy, care, and support. Looking at the most common causes of death globally, HIV/AIDS ranked sixth in 2004[4] and held the same position in 2010.[2] The Global Burden of Disease Study 2010 estimates 1·5 million AIDS-related deaths in 2010,[2] whereas UNAIDS data show 1·8 (range 1·6–2·0) million AIDS-related deaths.[1] Both estimates highlight a persistent, significant, and egregious burden of avoidable death.

Worldwide AIDS-related deaths increased dramatically during the late 1980s and peaked in 2005–06, followed by a steep decline to 2010–11. Yet, despite substantial reductions in AIDS mortality rates in many countries, AIDS remains the leading cause of death in southern and eastern Africa, and ranks number three in eastern Europe.[2] Furthermore, AIDS continues to affect young people disproportionately. In 2010, AIDS was the leading cause of death in women aged 15–49 years (14·4%) and the second most common cause of death for men aged 15–49 years (10·7%).[2]

UNAIDS estimated that 34 (range 31·4–35·9) million people lived with HIV in 2011,[1] with substantial geographical variations. Adult prevalence remains highest in sub-Saharan Africa at 4·9% (range 4·6–5·1%).[1] The good news is that since 2001, annual HIV incidence has fallen in 38 countries, most of them in sub-Saharan Africa. However, new infections are on the rise in some countries in eastern Europe, central Asia, the Middle East, and north Africa. It is a cause for concern that 2·5 (range 2·2–2·8) million people were newly infected with HIV in 2011.[1]

One of the great global health achievements of the past decade has been the scale-up of HIV treatment. In 2011, more than 8 million people living with HIV in low-income and middle-income countries received antiretroviral treatment.[1] Largely because of this unprecedented scale-up, supplemented by expanded HIV prevention services, the numbers of AIDS-related deaths and incidence rates worldwide have steadily decreased.[1]

To consolidate and intensify the accomplishments of the past decade, and to save millions of lives now in jeopardy, we must confront four realities. First, it will be impossible to sustain current efforts to tackle HIV and AIDS with current levels of funding. In 2015, when resource needs are expected to peak, an estimated US$22–24 billion per year will be needed,[5] but international AIDS funding has been stagnant since 2009 at about $8·2 billion per year. Many countries have increased their domestic funding for HIV, notably Benin, China, and South Africa, and they are to be supported and further encouraged. However, global solidarity remains essential to sustain HIV efforts in many of the poorest and most affected African countries. Moreover, international resources are critical to support programmes for marginalised populations in many countries. As treatment is scaled up, disability-adjusted

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特集／「社会保障と税の一体改革」とはいったい何だったのか

～消費税増税で社会保障は充実するのか～

視点　我が国の医療の進むべき道：グローバルヘルスの観点から

●第37回診療報酬請求事務能力認定試験（医科）：問題と解答

医学通信社

我が国の医療の進むべき道：グローバルヘルスの観点から

東京大学医学系研究科国際保健政策学教室教授　渋谷健司

1 保健医療は投資という発想

私は国内外の保健医療政策の研究を専門にしているが，この道に足を踏み入れたのは千葉県の田舎の病院で救急当直の合間にたまたま読んだ一冊のレポート，世界銀行の「世界開発報告1993年度版：健康への投資」であった[1]。当時はラリー・サマーズが主任エコノミストであり，世界銀行が従来のインフラ整備から人間開発へとシフトを始めた時期であった。また，世界保健機関（WHO）のリーダーシップ欠如に対する批判が世界中で巻き起こり，世界の保健政策の中心がジュネーブからワシントンへ移ろうとしている時期でもあった。

そのレポートには，発展途上国においても急速に高齢化と疾病構造の変化が進展していること，費用効果分析によると予防のみならず治療にも対費用効果の高い介入があること，そして，何よりも**健康は投資であり必ずしもコストではないこと**，が実証的に示されていた。それまで，WHOを中心とした，途上国といえば感染症と母子保健対策，そして基本的サービスへのアクセスを軸とした政策議論に慣れていた私には目から鱗の落ちる思いであり，筆頭著者を調べ，彼に会いにボストンまで行ったのが，保健医療政策との付き合いの始まりであった。

時は巡り，ちょうど20年後の2013年，世界の保健政策は再度，ジュネーブからワシントン，そしてシアトルへと移り，国内では社会保障が大きな政治アジェンダになった。しかし，世界的には欧州を中心とした経済危機の影響が世界を蝕み，国内的には惰性と既得権益のために医療を含む社会保障に関しては時代遅れの制度が継続し，その結果，真の弱者への保護は手薄く，また若い世代への負担が増大している。

現行の税と社会保障の一体改革は，増税という既存の制度の維持に必要な財源の調達に関する議論に終始している。しかし，**今こそ「健康への投資」というメッセージを再度検討すべき時期に来ているのではないだろうか。**そして，それは，必ずしも健康な生産労働人口を増やすというエコノミスト的ロジックのみでなく，斜陽化する製造業に代わる産業としての保健医療の構築という意味合いも含まれる。事実，保健医療の海外展開は世界の潮流であり，本稿では，グローバルな文脈から我が国の医療制度，そして我が国が今後国内外において採るべき戦略に関して私見を述べたい。

2 グローバル化する保健医療

保健医療制度は元来，各国の歴史や文化，社会経済状態，法制度に密接に関わるローカルなものである。しかし，グローバル化の流れのなかで，保健医療もそれと無関係ではいられなくなってきた。

「**グローバルヘルス**」とは，主に国内の人口を対象とする公衆衛生，植民地熱帯病を対象とする熱帯医学，先進国から途上国への技術移転を目的とする国際保健，それらがグローバル化の流れのなかで結びついた分野のことである。日本語では「**国境を越える保健医療課題**」と訳されるが，それは，先進国と発展途上国間での双方向の連携，そして経験と知識の共有が必要であり，きわめて学際的かつイノベーションを重視し，社会医学に限らず，ワクチン開発等の基礎研究や臨床も含まれる[2]。

このグローバルヘルス興隆の始まりは2000年に遡る。当時の国連事務総長コフィ・アナンが提唱し，国連加盟189カ国が合意したミレニアム開発目標（MDGs）である。MDGsは2015年までに国連加盟各国が達成すべき開発目標であるが，8つの目標のうち実に3つが保健医療関連目標であり，このMDGsによって保健医療は世界の開発のアジェンダとなった。

このような流れを受け，アメリカでは2005年頃から「グローバルヘルス」という言葉が使われ出したが，近年，この言葉は瞬く間に世界中に広まった。今や世界の主な大学にはグローバルヘルスを標榜する教室が存在し，さ

渋谷　健司（しぶや　けんじ）
1991年，東京大学医学部医学科卒．同年，帝京大学付属市原病院麻酔科医員（研修医）として勤務。93年，東京大学医学部付属病院医師（産婦人科）を経て，米国ハーバード大学リサーチ・フェロー。99年に同大学より公衆衛生学博士号取得。同年，帝京大学医学部産婦人科助手。2000年，衛生学公衆衛生学講師。01年に世界保健機関（WHO）シニア・サイエンティスト（保健政策のエビデンスのための世界プログラム）就任。04年にWHOコーディネーター（評価・保健情報システム/保健統計・エビデンス）を経て，現職。

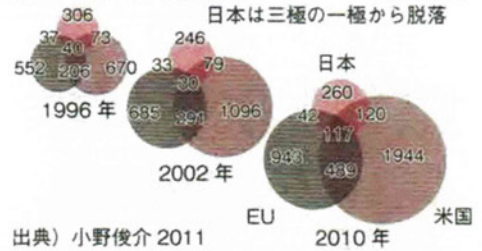
らには，米戦略国際問題研究所（CSIS）や英王立国際問題研究所（チャタムハウス）といった著名な外交政策シンクタンクにおいてもグローバルヘルスに関する部門が設けられている。

このように，保健医療のグローバル化は世界の潮流となっている。アジア諸国においても，タイやシンガポール，インドはメディカルツーリズムを推進しており，患者も医師も国境を越えて移動している。また，韓国は医療を国家戦略と定め，済州島での医療特区構想（各国の医師免許を容認，医師の所得税撤廃）を提唱し，韓流ブーム戦略さながらの大胆な施策を打ち出している。さらに，世界各地で「財源不足，医師不足，低収入の環境で，どのように良い医療を提供するか」という課題に対する様々な革新的取組みがなされており（例：営利型慈善病院，バウチャー制度，タスクシフティング，ICT活用等），我が国がこのような事例から学ぶべきものは多い。

他方，我が国では，こうした世界の潮流に逆行している。不活化ポリオワクチン輸入と国内生産の例をとっても明らかなように，**数十年前の金融行政の護送船団を思わせる旧態然とした仕組みは，我が国の保健医療のグローバル化と発展を大きく妨げている**。例えば臨床開発の分野においては，**図表1**に示すように，欧米では特に共同開発数が急激に増加しているが，我が国のみが過去15年間ほとんど変わらない[3]。

また，我が国の保健医療のグローバル化の遅れは，保健関連ODAにも如実に示されている。2000年にMDGsが宣言されて以降，世界的には保健関連ODA予算は急増したのに対し，OECD加盟国のうち我が国のみが縮小している。また，日本の保健医療分野に対するODAは，ODA全体のわずか2%であり，これはOECD諸国平均の15%と比べてきわめて低い[4]。未だに「健康への投資」という戦略的発想がないのが日本なのである。

図表1　日米欧・臨床開発プロジェクト数の推移



出典）小野俊介 2011

3 皆保険制度がグローバルヘルスのアジェンダに

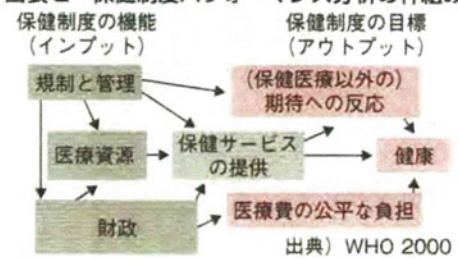
現在のグローバルヘルスの特徴は2つある。まず，その関係者が多様であること。WHOの財政的・政策的求心力の低下に伴い，官民連携型の国際機関やビル・ゲイツがマイクロソフトを引退後に設立したビル・アンド・メリンダ・ゲイツ財団（ゲイツ財団）などの民間財団，そして近年では民間企業の存在感が増している。また，活動の中心が小規模な個別のプロジェクトから多国間・官民連携を軸とする大規模なプログラム，そしてアジェンダ設定やルール作りへと変化していることが挙げられる。

次に，世界的な高齢化と疾病構造の変化により，優先課題が感染症から生活習慣病対策，そして皆保険制度構築へと変化している。2005年の第58回世界保健機関総会では，財政的に持続可能な皆保険制度の構築に向け努力することを加盟国に求める決議が採択された。実際，過去10年間でガーナやルワンダといった低所得国においても，低コストで国民皆保険を実現するための保険制度が導入されはじめている。皆保険制度構築は今最もホットなグローバルヘルスのアジェンダなのである[5]。

4 なぜランセットが日本の保健医療制度の特集をしたか？

2011年は，我が国が皆保険制度を達成してから50年目にあたる年であった。その節目に，イギリスのランセット誌と共同で，日本の保健医療制度を特集する機会を得た[6]。ご存知のように，ランセット誌は世界で数百万人の読者をもつ世界で最も権威のある医学雑誌の一つである。しかし，ランセット誌がニュー・イングランド・ジャーナル・オブ・メディシンやJAMAなどのライバル誌と異なるユニークな点は，現編集長のリチャード・ホートンの編集方針によるところが大きい。もちろん最大の読者である一般臨床家対象の論文が中心であるが，世界の医療制度，人権，健康と社会的公正，戦争等のテーマも定期的に取り上げる，きわめて社会派的な雑誌なのである。それもそのはず，1823年の創刊時の編集長

図表2　保健制度パフォーマンス分析の枠組み



出典）WHO 2000

トーマス・ウェイクリーのモットーは，「読者に情報を伝え，楽しませ，そして，社会を変革すること」であり，その伝統が今も連綿と生きている。

なぜそのランセットが日本の医療制度の特集を企画したかといえば，それは，リチャード・ホートン本人の言葉がすべてを物語るであろう。「**日本の医療制度は日本国民のみならず，世界の人々の健康のバロメーターであるという点でも，きわめて重要である。…日本は大変なソフトパワーをもっている。世界における確固たる地位を確保する努力と国内での政策を改善する力を発揮しようとしている**」[7]。閉塞感に覆われた国内状況だが，世界の我が国に対する信頼と期待はいまだに高いのである。

特に，我が国の医療制度は2つの点で世界的にも注目を集めている。まず，**低コストで良好な健康指標を実現し，公平性を徐々に高めてきた皆保険制度は，今まさにグローバルヘルスの主要課題となっており**，特に，高度経済成長を迎えようとする発展途上国のモデルとなりうること。次に，**高度経済成長期に作られた現行制度が少子高齢化の進む現在の日本では持続不可能**になっており，今後どのような制度を構築していくのか，我が国の将来ビジョンが試されている点である。

5 保健医療制度パフォーマンス分析の枠組み

ランセット日本特集号では，編集部から3つの要望があった。まず，過去と現在のみならず将来を見据えること。次に，日本の特殊事情のみならずグローバルな教訓も示すこと。そして，エビデンスに基づく議論をすることであった。分析の枠組みは，筆者もその枠組み作りに関わった「世界保健報告2000年度版：保健制度パフォーマンスの改善」の枠組みを用いた（**図表2**）[8]。

保健制度パフォーマンス分析は，元々は次の5つの重要な比較分析を行うことを目的としたものであった。①健康アウトカムのばらつきはどのくらい保健医療制度の相違によって説明できるのか，②保健医療制度パフォーマンスの改善によって健康アウトカムはどのくらい改善できるか，③どの保健医療制度が健康アウトカムを改善するのによいか，④どの保健医療制度が対費用効果が高いか，⑤保健医療制度のパフォーマンスの決定要因は何か。

この枠組みは，保健医療制度をその機能（インプット）と目標（アウトプット）に分けたシンプルなものであるが，ともするとインプット（財源や医療従事者数など）の議論に終始する医療制度改革の議論において，何が本質であるかを忘れないためにはきわめて有用である。保健医療制度の主な目標は，健康アウトカムの増進であり，それに加えて，保健サービス以外の期待への対応や医療費の公平な負担を達成することが重要であるとしている[8]。

6 我が国の保健医療制度の現状と課題：グローバルヘルスの観点から

Savedoffらの研究によると，皆保険が成り立つ条件としては，経済成長，人口構成が若いこと，そして，政治的後押しがあることの3つがあるという[5]。我が国が皆保険を達成した1961年前後の政治，社会経済状況を鑑みれば，日本はまさにその3条件を満たしていた。つまり，我が国の皆保険制度は，加入者の負担による社会保険制度をもとに，まだ若く経済成長のまっただなかにできた，いわば発展途上国モデルである。50年後の今，この条件が満たされつつあるのが，現在のアジアやアフリカの多くの新興国である。第2次大戦後，発展途上国型の皆保険制度を完璧に作り上げた我が国のこれまでの経験と教訓こそが，これから世界で生かされるのである。

北原茂実氏（医療法人社団KNI理事長）は，こうした点を鑑み，我が国の保健医療の産業化と制度のパッケージ輸出を提言している[9]。実際，経済成長が急速に起こる場合，保健医療供給体制のキャッチアップは通常遅れるために，確実に保険制度が導入されるのであれば，初期投資は十分に回収できる。この際，大切なことは，保険制度に関する研修や単独の病院建設といった従来のODAプロジェクトや企業のCSRではなく，**現地で持続可能なビジネスモデルを開発することや付加価値のある戦略形成支援**である。例えば，日本型の医療を中心とし，保健医療システムにITを導入し，同時に日本式教育での現地の人材育成，さらには公務員共済や企業共済を組み合わせて日本の病院と提携し，企業の福利厚生を充実させることで日本式システムをパッケージとして導入することが可能であり，経済的リターンとともに外交的に