Global Burden of Raised Blood Pressure
Coming Into Focus

Mark D. Huffman, MD, MPH; Donald M. Lloyd-Jones, MD, ScM

In this issue of JAMA, Forouzanfar and colleagues report updated estimates of the global burden of raised systolic blood pressure (SBP) from 1980 to 2015 using data from 844 reports of nationally representative samples comprising 8.7 million individuals from the Global Burden of Diseases, Injuries, and Risk Factors Study 2015 (GBD 2015). Using complex, state-of-the-art methods including spatiotemporal Gaussian process regression, the authors compared estimates for 1990 with 2015 and reported that approximately 3.5 billion people aged 25 years or older had SBP of at least 110 mm Hg (an 11% unadjusted increase), and 0.9 billion had SBP of 140 mm Hg or higher (an 18% unadjusted increase). The authors further reported that these prevalence estimates correspond with a 49% increase in the underestimated number of annual deaths due to SBP of 110 mm Hg or higher (10.7 million [95% uncertainty interval (UI), 9.6-11.8 million]) and a 51% increase due to an SBP of 140 mm Hg or higher (7.8 million [95% UI, 7.0-8.7 million]) in 2015. Since population growth and aging account for the majority (55%) of increases in numbers of deaths related to cardiovascular disease from 1990 to 2013, the downward trends in age-adjusted death rates due to raised SBP for men (0.92% per year [95% UI, 0.80%-1.03%]) and women (1.37% [95% UI, 1.26%-1.50%]) are important for comparing estimates over time.

Additionally, Forouzanfar et al describe variations in the age-adjusted burden of raised SBP across regions as estimated by the disability-adjusted life-years (DALYs) and deaths attributed to raised SBP (>5-fold increase for SBP ≥10 mm Hg and >10-fold increase for SBP ≥140 mm Hg). This heterogeneity is a key highlight, as in previous GBD reports, and large low- and middle-income countries face the greatest absolute burden of disease based on population growth, aging population, SBP trends, and changing competing risks because of declines in the rates of death caused by maternal and childhood illnesses and some infectious diseases. Strategies to reduce the burden of disease due to raised SBP need to be contextualized to each setting based on capacity, priorities, ongoing activities, and political will, particularly for the upstream drivers of raised SBP that may be different in different countries.

The authors provide country-level estimates for 195 countries based on imputation because only 154 countries had data across the entire study period. Could this method lead to model overfitting because of data gaps? Would it be better if no data were reported from such countries? Uncertainty intervals derived from the authors’ models are reported but do not include uncertainties associated with intermediate predictive steps, which may or may not be important in these estimates or those of other researchers. Some epidemiologists and other researchers may argue about the potential for overcounting based on the inclusion of deaths and disability related to endocarditis, rheumatic heart disease, and chronic kidney disease. Although raised blood pressure may be associated with worse prognosis when associated with these conditions, these relationships may be partially confounded or perhaps even susceptible to reverse causation, particularly in the case of chronic kidney disease.

The theoretical minimal risk exposure level of SBP greater than or equal to 110 mm Hg (rather than ≥140 mm Hg or even ≥115 mm Hg) may also overestimate burden and raise questions about what may be attributable risk (compared with what may be modifiable risk) based on SBP levels. Changing population-level exposures (such as blood pressure) to individual minimal-risk exposures may also have different consequences than changing external risk exposures, such as tobacco, for which lower (or no) exposure is always better. This is likely not the case for blood pressure. Conversely, the authors do not report data about raised diastolic blood pressure, which may lead to an underestimate of the prevalence of raised blood pressure, especially among younger individuals.

As in the current report, articles from the GBD provide an extensive collection of tables and figures showing data on major trends in cardiovascular health. Mean SBP, proportional changes, and cause-specific mortality trends adjusted for population growth and aging are presented with a surprising degree of precision given the uncertainties in the underlying data, but they likely approximate the best estimates, even acknowledging gaps in data. The estimates of DALYs remain useful, particularly given the increasing global burden of chronic diseases, but if nearly two-thirds of the world’s population has low-quality vital statistics data based on estimates from the World Health Organization, then the global DALY estimates should be interpreted with caution.

These data supersede previous estimates from the GBD investigators, but readers and stakeholders (such as ministries of health, public health experts, and researchers) may wonder what differences exist between these data and other recent reports, including those from the NCD (noncommunicable diseases) Risk Factor Collaboration, which reported blood pressure estimates from 1479 studies that included 19.1 million adults in 2016. How much do the differences in data sources and methods influence the results? These reports provide slightly different yet overlapping estimates.
and may represent an informal check and balance system. Trust in data collection, synthesis, reporting, and sharing has improved in global burden estimates and will remain important regardless of the source.

Estimating the burden of single risk factors has helped define high-burden regions, although country, regional, and global estimates of overall cardiovascular health may be more useful for stakeholders. An integrated approach to estimating cardiovascular health trends, using the approach developed by the American Heart Association, may influence policy and programmatic prioritization to achieve greater prevalence of ideal cardiovascular health through a comprehensive and cohesive approach that encompasses strategies across the entire spectrum of prevention throughout the life course. For example, while age-adjusted trends in global tobacco use are encouraging, the increasing prevalence of obesity and diabetes threaten to reverse major gains in age-adjusted death rates due to cardiovascular disease. Even blood pressure levels, which have fluctuated modestly for at least the past 25 years, may start to worsen significantly because of the worldwide epidemic of obesity.

Several key takeaways emerge as implications of these latest GBD data. First, both broad population-level and high-risk clinical strategies are needed to reduce the burden of cardiovascular and other diseases related to elevated SBP. Whereas in many settings it is normative for SBP levels to increase steadily through adulthood, this is not an inevitable consequence of aging. For example, individuals who maintain stable weight from young adulthood to middle age do not experience age-related increases in SBP. Second, exposure to increasing blood pressure levels, even within the prehypertensive range, results in accumulating myocardial and vascular damage over time. Preventing the onset of clinical hypertension (ie, primary prevention) and blunting or abolishing the rise in SBP from 110 mm Hg through prehypertensive levels to 140 mm Hg or higher is imperative to prevent premature death and disability from cardiovascular diseases. Clinical trials, such as the Trial of Preventing Hypertension (TROPHY), while controversial, suggest that drug therapy for prehypertension may be feasible for prevention of hypertension if maintained. At the other end of the prevention spectrum, natural experiments and policy-driven programs suggest that sodium reduction in the food supply, particularly in high-income countries, appears useful for the majority of the population at risk for developing hypertension or with hypertension. Translating these lessons to low- and middle-income countries requires ongoing work.

These data cannot inform clinical practice guidelines regarding appropriate levels for initiation of blood pressure-lowering therapy or goal levels for treatment. However, these data strengthen the case to lower the risk for cardiovascular diseases in those with SBP of 140 mm Hg or higher by all effective means available, including improving uptake of healthy diets, minimizing weight gain or promoting weight loss in overweight and obese individuals, and promoting uptake and adherence to effective blood pressure-lowering drugs as well as management of related cardiovascular risk.

The GBD project continues to evolve and engage the medical community with its impressive scope of data. It is difficult to imagine not having a group performing global-level synthesis to provide the best estimates of disease burden. The project remains relatively new by historical standards, yet has made remarkable progress, including raising the awareness of the importance of data systems to inform and improve health. The project works to be transparent about its complex methods, which remain challenging for most readers, but it also works to encourage global participation, which has increased since the project began. Even if the extensive amounts of data are fuzzy and imperfect, they provide valuable estimates of current global disease burden.

ARTICLE INFORMATION

Author Affiliations: Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, Illinois (Huffman, Lloyd-Jones); Division of Cardiology, Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, Illinois (Huffman, Lloyd-Jones).

Corresponding Author: Donald M. Lloyd-Jones, MD, ScM, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, 680 N Lake Shore Dr, Ste 1400, Chicago, IL 60611 (djll@northwestern.edu).

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REFERENCES


