

REVIEW ARTICLE OPEN



PERSPECTIVE – The Growing Global Benefits of Limiting Salt Intake: an urgent call from the World Hypertension League for more effective policy and public health initiatives

Brent M. Egan¹, Daniel T. Lackland², Susan E. Sutherland¹, Michael K. Rakotz³, Janet Williams³, Yvonne Commodore-Mensah⁴, Daniel W. Jones⁵, Sverre-Erik Kjeldsen⁶, Norm R. C. Campbell⁷, Gianfranco Parati^{8,9}, Feng J. He¹⁰, Graham A. MacGregor¹⁰, Michael A. Weber¹¹ and Paul K. Whelton¹²

© The Author(s) 2025

Journal of Human Hypertension (2025) 39:241–245; <https://doi.org/10.1038/s41371-025-00990-1>

Excess dietary salt adversely impacts blood pressure (BP) and cardiovascular health [1–3]. Reducing salt intake is a well-established intervention for hypertension prevention and management [2–18]. Multiple converging factors magnify the health and economic toll from excess salt consumption and amplify the benefits of limiting intake to healthier levels. The aims of this paper include: (i) highlighting factors underlying the growing time-dependent benefits of limiting salt consumption on BP and cardiovascular disease (CVD) events worldwide and (ii) encouraging policy and public health initiatives to limit sodium consumption to <2000 mg daily (<5000 mg NaCl) in adults, as recommended by the World Health Organization (WHO [16]), within ten years, recognizing more time may be required. Attaining healthier levels of salt intake will be rewarded by growing health and economic dividends throughout the 21st century.

BACKGROUND

BP is a vital sign with powerful prognostic implications for cardiovascular health, healthy aging, and health equity [1–18]. Salt intake is directly related to population BP and the prevalence of hypertension. The INTERSALT Study investigators estimated a 2300 mg increase in daily sodium intake was associated with ~3.5 mmHg higher systolic BP (SBP) [1]. Subsequent reports confirmed that a 2300 mg change in daily sodium intake has a SBP effect ranging from 1–11 mmHg, which is modified by factors including age, race, potassium intake, and hypertension, with an unweighted average of ~4.4 mmHg (Fig. S1) [10, 19–24]). The mean SBP of a population is directly and strongly related to prevalent hypertension [23]. Hypertension is an important risk factor for coronary heart disease, heart failure, stroke, cognitive decline and dementia, chronic kidney disease (CKD), premature disability, and death [2–18].

Limiting salt intake is a cost-effective intervention for lowering BP, limiting age-related increases in BP [1, 25], improving hypertension control, and reducing the health (Table S2, Fig. S1) and economic toll of CVD with a 12-fold or greater return on investment [5, 26]. Effective measures are urgently needed to limit sodium intake of individuals from the current global mean of ~4300 mg to <2000 mg daily [16]. We propose a 10-year investment to achieve and sustain sodium intake at ~2000 mg daily with the promise of enduring health and economic benefits for most people.

THREE KEY POINTS HIGHLIGHT THE CRITICAL AND GROWING IMPORTANCE FOR REDUCING GLOBAL SODIUM INTAKE

Demographic changes

The pressor effects of salt (salt sensitivity) increase with age, and salt contributes to age-related increases of BP [1–7, 24, 27]. The global population is aging rapidly (Fig. 1 [28]). With short- to intermediate term salt reduction, BP falls progressively more with increasing age [10].

Furthermore, salt intake likely explains much of the age-related increase of BP [1, 25]. Estimates from the INTERSALT study suggested that a 2300 mg higher daily sodium intake was associated with ~10 mmHg rise of SBP from 25 to 55 years of age [1]. Clinical studies have indicated that the age-related increase of BP is largely confined to salt-sensitive individuals. Sixteen adults followed for 10 or more years after being defined as salt sensitive had a 1.4 mmHg/year increase in SBP or ~14 mmHg over 10 years, whereas SBP fell slightly over time in 15 salt-resistant individuals [25].

In Europe, the U.S., and most other countries, age-related increases of SBP continue beyond 55 years [29], especially in women [28]. Salt sensitivity increases with aging [10, 26] and is greater in women than men [30]. Given these observations, a 2300 mg higher daily sodium intake from ages 25 to 75 years and beyond may account for a 15 to 20 mmHg increase of SBP over the adult lifespan. Moreover, a

¹American Medical Association, Greenville, SC, USA. ²Medical University of South Carolina, Charleston, SC, USA. ³American Medical Association, Chicago, IL, USA. ⁴Johns Hopkins University School of Nursing, Baltimore, MD, USA. ⁵University of Mississippi Medical Center, Jackson, MS, USA. ⁶University of Oslo, Institute of Clinical Medicine, Oslo, Norway. ⁷Department of Medicine, University of Calgary, Calgary, AB, Canada. ⁸Department of Medicine and Surgery, University of Milano-Bicocca, Milan, Italy. ⁹IRCCS, Italian Auxology Institute, Dept. of Cardiology, San Luca Hospital, Milan, Italy. ¹⁰Wolfson Institute of Population Health, Barts and The London School of Medicine & Dentistry, Queen Mary University of London, London, UK. ¹¹Division of Cardiovascular Disease, State University of New York Downstate Medical Center, New York, NY, USA. ¹²Tulane University School of Public Health and Tropical Medicine, New Orleans, LA, USA. ✉email: brent.egan@ama-assn.org

Received: 7 November 2024 Revised: 14 January 2025 Accepted: 6 February 2025

Published online: 21 March 2025

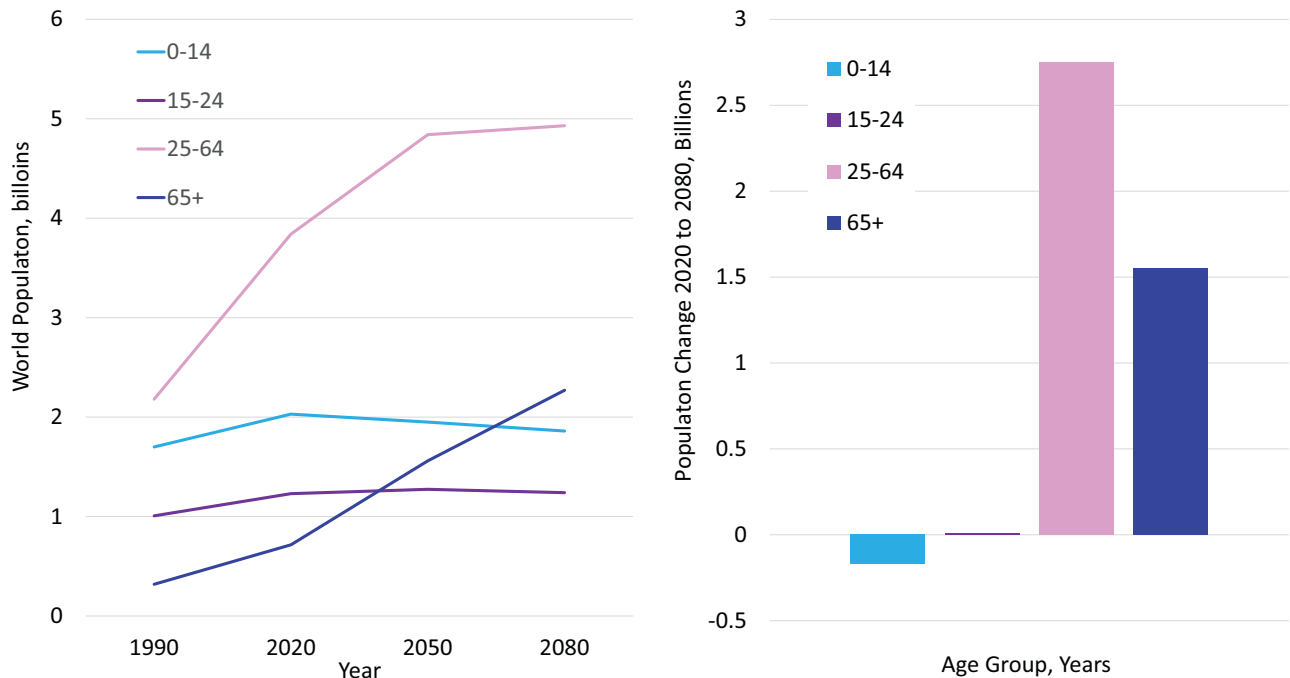


Fig. 1 World population and changes over time in the world population by age group. The world population by age group is shown from 1990 to 2080 (left side). Between 2020 and 2080, the population 25 years of age and older is projected to increase by more than 4 billion. The population <25 years old is projected to decline slightly (right side), which reflects declining birth rates. Data source: UN, World Population Prospects (2024). <https://population.un.org/wpp/Graphs/DemographicProfiles/Line/900>.

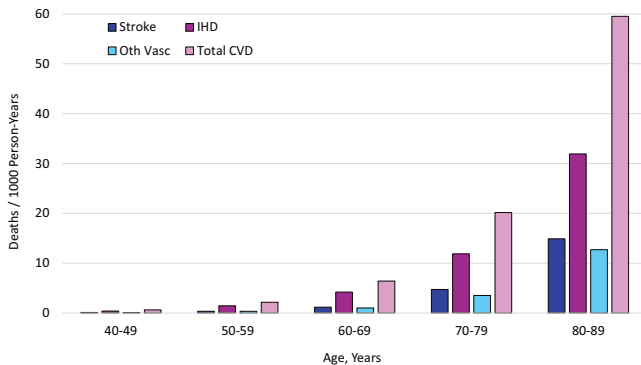


Fig. 2 Death rates from cardiovascular diseases by age group. Death rates from stroke, ischemic heart disease, other vascular diseases, and total cardiovascular disease approximately triple every ten years in adults from the fifth through ninth decades [31].

20 mmHg increase of SBP approximately doubles relative risk for CVD events [31].

Relative risk for cardiovascular events triples every 10 years at any level of SBP (Fig. 2 [31]). From the fifth to ninth decade, relative risk for CVD triples every 10 years with absolute risk increasing ~80 fold at the same SBP. If SBP also increases 20 mmHg from the fourth to ninth decades, absolute risk for CVD events would increase more than 150-fold [31]. Limiting dietary salt intake lowers BP more in older than younger individuals and likely mitigates age-related increases of BP [1–7, 10, 25]. While the relative benefit of lowering SBP on CVD events is similar in younger and older adults, absolute benefits are far greater in older adults.

Other demographic changes, which increase salt sensitivity. Populations in some Asian as well as sub-Saharan African countries and the diaspora are growing more rapidly than Euro-Caucasian

populations [32–34]. These populations are generally more sensitive to the hypertensive effects of sodium than Euro-Caucasian populations [10]. Thus, the benefits of limiting dietary salt intake are compounding as aging and other demographic changes augment salt sensitivity of the global population.

Obesity and related conditions increase salt sensitivity

The global population is becoming progressively more obese [35], a major risk factor for developing hypertension and cardiometabolic disorders including diabetes mellitus and CKD [36]. Obesity, diabetes mellitus, and CKD all amplify BP responses to salt [37]. Thus, limiting dietary salt intake to healthier levels is especially important in populations with a high and growing prevalence of obesity and cardiometabolic disorders, which combine to heighten salt sensitivity.

Low diet quality

When diet quality is low, the effect of salt on BP is magnified. For example, in the DASH-Sodium trial, the reduction of BP when limiting daily sodium consumption by 2300 mg from 3450 to 1150 mg daily was roughly twice as large on the lower quality usual diet, typical of intake in the United States, than the higher quality DASH diet [19]. Thus, limiting salt intake reduces BP more when diet quality is low, which characterizes much of the global population [38].

DESIGNING AND IMPLEMENTING NATIONAL PLANS FOR GLOBAL ACTION TO REDUCE SALT INTAKE AND IMPROVE CARDIOVASCULAR HEALTH

Given the three previously mentioned factors, the health and economic benefits of reducing global sodium consumption [5, 26] and the costs of inaction are both substantial and progressively growing. Two broad strategies to reduce sodium intake, including public policy and public health mass media campaigns, separately and combined are supported by current evidence, previous recommendations, and experience [2, 3, 11, 14, 39, 40]. Non-

discretionary intake predominates as the source of excessive sodium consumption in many high-income countries. Conversely, discretionary salt intake is a greater challenge in many low- and middle-countries (LMIC), especially in rural areas [41].

While this Perspective aims at reinvigorating national efforts to reduce sodium consumption from ~4300 mg to <2000 mg daily within ten years, it seems prudent to dedicate the first two years to building consensus and laying the foundation for sustainable action. Following a focused two-year coalition building and planning period, the next 8 years would focus on implementation, ongoing data monitoring, and periodic plan refinement as required to remain on target. In fact, reducing sodium intake could take longer given historical challenges and recognizing that sources of excessive sodium intake can change with time.

Reducing non-discretionary salt consumption

For nations where the predominant source of sodium is non-discretionary, policies to reduce sodium added during food processing and in meals prepared outside the home by 10% can be implemented and repeated at yearly intervals without detection by most consumers [5, 16, 20]. Assuming the global average of 4300 mg daily sodium intake, when non-discretionary intake is high, approximately 3100 of daily sodium intake is added in processing and meals prepared outside the home. These foods naturally contain approximately 400 mg of sodium.

A 10% annual reduction of non-discretionary sodium intake for eight years would reduce non-discretionary intake from 3100 to 1334 mg, lowering total sodium intake from 4300 to 2534 mg daily. If discretionary intake of ~800 mg daily also fell 7% annually for 8 years, then sodium consumption would fall to ~2180 mg daily, approaching the WHO target of <2000 mg (Fig. 3).

Reducing discretionary salt consumption

When discretionary intake is high, as it is in many low- and middle-income countries [41, 42], substituting a salt that contains 75% NaCl and 25% KCl instead of the usual 100% NaCl combination has been acceptable and effective in reducing BP, major CVD events, and death [42, 43]. Where discretionary intake is high, one can generally assume that approximately 400 mg of salt is naturally present in foods, 800 mg is added during food processing, and 3100 mg is added by the consumer. If consumers replace 20% of their usual NaCl intake with a 75% NaCl: 25% KCl, then sodium consumption would decline 5% annually from 3100 mg at baseline to 2057 mg after 8 years (Fig. 4). Phasing in KCl containing salt substitutes over time provides an opportunity for

consumer marketing to increase the use of salt substitutes as well as for monitoring of safety concerns due to hyperkalemia. The ~1040 mg reduction (45 mmol) in daily sodium consumption over eight years would parallel a rise of 1800 mg (45 mmol) in daily potassium intake. If non-discretionary sodium intake could also be reduced 10% annually, then daily sodium consumption would fall to ~2800 mg/d. While sodium intake remains above the <2000 mg/d target, potassium intake rises. The combined benefits of reducing sodium and increasing potassium intake include lowering SBP ~4–6 mmHg and CVD mortality approximately 10% [41, 42].

Safety of potassium-enriched salt. The 75% NaCl: 25% KCl approach to reducing sodium intake is especially relevant to LMIC where daily potassium intake is 1000–1800 mg below the WHO recommended level of 3500 mg [42]. In the example above, daily potassium intake would increase by ~1800 mg. In the Salt Substitution and Stroke Study (SSaSS), 24-urine potassium increased 800 mg daily to ~2200 mg/ daily, which remained below the WHO recommended level of 3500 mg, recalling that ~90% of potassium ingested is excreted in the urine [43]. Hyperkalemia and sudden death did not increase in SSaSS [42], and fewer arrhythmias were observed in participants assigned to potassium-substituted salt [44]. Individuals with known CKD and those taking potassium supplements and potassium-sparing diuretics were excluded, although patients with diabetes mellitus and those taking renin-angiotensin system blockers were included. Evidence suggests that potassium-enriched salt is safe for most adults in LMIC, where baseline potassium intake is low.

PROJECTED IMPACT OF ATTAINING WHO TARGETS FOR SALT INTAKE ON BP, CVD EVENTS, AND MORTALITY

Preventing 1.89 million premature deaths annually is a compelling reason for more effective policy initiatives and public health programs to limit salt consumption [16], yet even more non-fatal events are averted. Data from Norfolk, United Kingdom, documented that the ratio of total to fatal CVD was 4.6 in men and 6.8 in women and was greater in adults <60 years ($\geq 10:1$) than ≥ 60 years ($\geq 4:1$) [45]. Thus, limiting sodium intake to <2000 mg daily may prevent ~1.9 million fatal CVD and at least 7.5 million non-fatal CVD events annually. CVD prevention with salt reduction may be underestimated as benefits have been attributed to BP reduction, although BP-independent effects may contribute, especially for stroke [46].

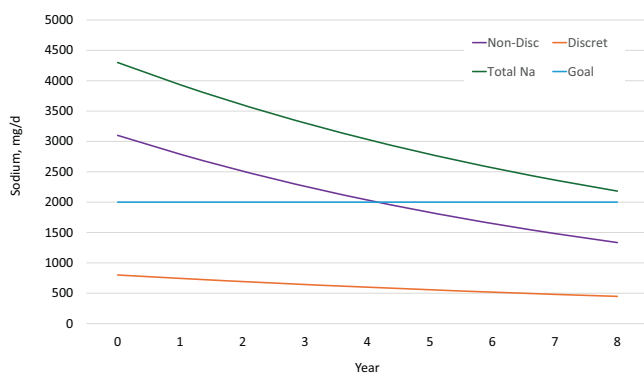


Fig. 3 Projections for reducing sodium consumption when non-discretionary intake is high. Assuming roughly 3100 mg salt is added to food processed and prepared outside the home each day, a 10% annual reduction for 8 years would lower non-discretionary intake from 3100 to ~1334 mg and total sodium intake from 4300 to 2534 mg daily. If discretionary sodium intake were also reduced 7% per year (orange line), then total sodium intake would decline to ~2180 mg daily.

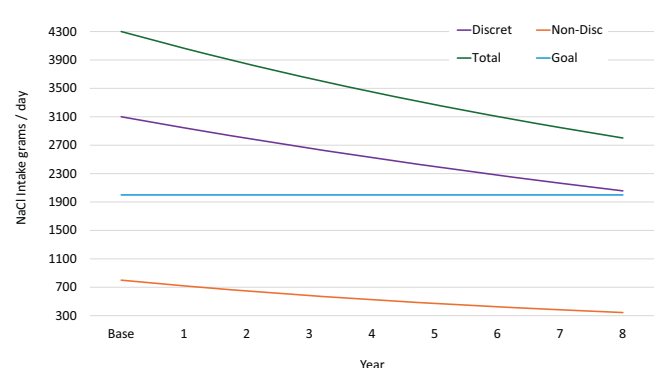


Fig. 4 Projections for reducing sodium consumption when discretionary intake is high. The estimate assumes 3100 mg sodium/d is added in food preparation and seasoning at home. Discretionary would fall 5% annually by replacing 20% of added salt with 75% NaCl: 25% KCl each year for 8 years. If non-discretionary sodium also declines 10% annually, then total sodium intake would fall from 4300 to 2800 mg daily. Potassium intake would rise ~1800 mg/d.

OVERCOMING THE BARRIERS AND CHALLENGES TO ATTAINING GLOBAL SODIUM INTAKE OF <2000 MG DAILY

This Perspective concurs with several prior reports cited above that document the adverse effects of sodium consumption on population health. Our Perspective highlights the impact of increasing age, diabetes, obesity, CKD, and greater numerical growth of non-Caucasian than Caucasian populations. These factors converge to increase salt sensitivity and magnify the benefits of lowering population sodium intake. These initiatives must align with the sources of dietary sodium. While most estimates on the benefits of healthier levels of sodium intake have focused on CVD mortality, non-fatal events occur more often and contribute to a large and costly burden of disability adjusted life years. The time for effective and sustained multilateral action is now as time lost is costly and lives are being lost [47]. The adverse effects of limiting sodium consumption to <2000 mg daily [5, 6] are minimal relative to the documented health and economic benefits. The time has come for a rationale balance of benefits, risks, and competing interests to coalesce around a multi-national initiative to attain and sustain sodium consumption at the WHO recommended target of <2000 mg daily.

REFERENCES

- Intersalt Cooperative Research Group. Intersalt: an International Study of Electrolyte Excretion And Blood Pressure. Results for 24 h urinary sodium and potassium excretion. *BMJ*. 1988;297:319–28.
- Dickinson BD, Havas S. Reducing the population burden of cardiovascular disease by reducing sodium intake. *Arch Intern Med*. 2007;167:1460–8.
- He FJ, MacGregor GA. A comprehensive review on salt and health and current experience of worldwide salt reduction programmes. *J Hum Hypertens*. 2009;23:368–84.
- Bibbins-Domingo K, Chertow GM, Coxson PG, Moran A, Lightwood JM, Pletcher MJ, et al. Projected effect of dietary salt reductions on future cardiovascular disease. *NEJM*. 2010;362:590–9.
- He FJ, Li J, MacGregor GA. Effect of longer-term modest salt reduction on blood pressure. *Cochrane systematic review and meta-analysis of randomised trials*. *BMJ*. 2013;346:f1325.
- Aburto NJ, Ziolkovska A, Hooper L, Elliott P, Cappuccio FP, Meerpohl JJ. Effect of lower sodium intake on health: systematic review and meta-analysis. *BMJ*. 2013;346:f1326.
- Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, et al. Global sodium consumption and death from cardiovascular causes. *NEJM*. 2014;371:624–34.
- Wong MMY, Arcand J, Leung AA, Thout SR, Campbell NRC, Webster J. The science of salt: a regularly updated systematic review of salt and health outcomes (December 2015–March 2016) from the World Hypertension League. *J Clin Hypertens*. 2017;19:322–32.
- Wang Y-J, Tzu-Lin Y, Shih M-C, Tu Y-K, Chien K-L. Dietary sodium intake and risk of cardiovascular disease: a systematic review and dose-response meta-analysis. *Nutrients*. 2020;12:2934.
- Huang L, Trieu K, Yoshimura S, Neal B, Woodward M, Campbell NRC, et al. Effect of dose and duration of reduction in dietary sodium on blood pressure levels: systematic review and meta-analysis of randomised trials. *BMJ*. 2020;368:m315.
- He FJ, Tan M, Ma Y, MacGregor GA. Salt reduction to prevent hypertension and cardiovascular disease. *JACC*. 2020;75:632–47.
- Chen X, Du J, Wu X, Cao W, Sun S. Global burden attributable to high sodium intake from 1990 to 2019. *Nutr Metab Cardio Dis*. 2021;31:3314–21.
- Ma Y, He FJ, Sun Q, Yuan C, Kleneker LM, Curhan GC, et al. 24-h urinary sodium and potassium excretion and cardiovascular risk. *NEJM*. 2022;386:252–63.
- Campbell NRC, Whelton PK, Orias M, Wainford RD, Cappuccio FP, Ide N. 2022 World Hypertension League, Resolve to Save Lives, International Society of Hypertension dietary sodium (salt) global call to action. *J Hum Hypertens*. 2023;37:428–37.
- Wang K, Jin Y, Wang M, Liu J, Bu X, Mui J, et al. Global cardiovascular disease burden attributable to high sodium intake from 1990 to 2019. *J Clin Hypertens*. 2023;25:868–79.
- WHO. WHO global report on sodium intake reduction. Geneva: World Health Organization; 2023.
- Song J, Tan M, Wang C, Brown MK, Pombo-Rodrigues S, MacGregor GA, et al. Salt intake, blood pressure and cardiovascular disease mortality in England, 2003–2018. *J Hypertens*. 2023;41:1713–20.
- Moreno SV, Grimes C, Bolton KA, Uddin R, Siopis G, Maddison R, et al. The burden of cardiovascular disease attributable to high dietary sodium intake in Australia between 1990 and 2019. *J Hypertens*. 2024;42:1163–72.
- Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *NEJM*. 2001;344:3–10.
- He FJ, MacGregor GA. Effect of modest salt reduction on blood pressure: a meta-analysis of randomized trials. Implications for public health. *J Hum Hypertens*. 2002;16:761–70.
- Pimenta E, Gaddam KK, Oparil S, Aban I, Husain S, Dell'Italia LJ, et al. Effects of dietary sodium reduction on blood pressure in subjects with resistant hypertension: results from a randomized trial. *Hypertension*. 2009;54:475–81.
- Graudal NA, Hubeck-Graudal T, Jürgens G. Effects of low-sodium diet vs. high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). *Am J Hypertens*. 2012;25:1–15.
- Rose G, Day S. The population mean predicts the number of deviant individuals. *BMJ*. 1990;310:1031–4.
- Little R, Ellison DH. Modifying dietary sodium and potassium intake: an end to the 'Salt Wars'? *Hypertension*. 2024;81:415–25.
- Weinberger MH, Fineberg NS. Sodium and volume sensitivity of blood pressure: age and pressure change over time. *Hypertension*. 1991;18:67–71.
- Wang G, Labarthe D. The cost-effectiveness of interventions designed to reduce sodium intake. *J Hypertension*. 2011;29:1693–99.
- United Nations, DESA, Population Division. World population prospects 2024. <http://population.un.org/wpp/>.
- Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, Hense H-W, Joffres M, et al. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *JAMA*. 2003;289:2363–9.
- Chapman N, Ching SM, Konradi AO, Nuyt AM, Khan T, Twumasi-Ankrah B, et al. Arterial hypertension in women: state of the art and knowledge gaps. *Hypertension*. 2023;80:1140–49.
- Barris CT, Faulkner SL, Belin de Chantemèle EJ. Salt sensitivity of blood pressure in women. *Hypertension*. 2023;80:268–78.
- Lewington S, Clarke R, Qizilbash N, Peto R, Collins R, Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*. 2002;360:1903–13.
- World Population Review. Caucasian countries. 2024. <https://worldpopulationreview.com/countryrankings/caucasian-countries>. Accessed 14 Feb 2025.
- Newsweek.com. Map reveals world's 10 fastest growing populations. 2024. <https://www.newsweek.com/map-world-population-growing-fastest-1937121>. Accessed 14 Feb 2025.
- Richardson SJ, Freedman BI, Ellison DH, Rodriguez CJ. Salt sensitivity: a review with a focus on non-hispanic blacks and hispanics. *J Am Soc Hypertens*. 2013;7:170–9.
- GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet*. 2020;396:1223–49.
- Nawaz S, Chinnadurai R, Al-Chalabi S, Evans P, Kalra PA, Syed AA, et al. Obesity and chronic kidney disease: a current review. *Obes Sci Prac*. 2023;9:61–74.
- Bailey MA, Dhaun N. Salt sensitivity: causes, consequences, and recent advances—review. *Hypertension*. 2024;81:476–89.
- Miller, Webb V, Cudheaf P, Shi P, Zhang J, Reedy J, et al. Global dietary quality in 185 countries from 1990 to 2018 show wide differences by nation, age, education, and urbanicity. *Nature Food*. 2022;3:694–702.
- Campbell N, Lackland D, Chockalingam A, Lisheng L, Schiffrin EL, Harrap S, et al. The World Hypertension League and International Society of Hypertension call on governments, nongovernmental organizations, and the food industry to work to reduce dietary sodium. *J Clin Hypertension*. 2014;16:99–100.
- Campbell NR, Lackland DT, Niebylski ML, Orias M, Redburn KA, Nilsson PM, et al. 2016 dietary salt fact sheet and call to action: The World Hypertension League, International Society of Hypertension, and the International Council of Cardiovascular Prevention and Rehabilitation. *J Clin Hypertension*. 2016;18:1082–5.
- Xu X, Zeng L, Jha V, Cobb LK, Shibuya K, Appel LJ, et al. Potassium-enriched salt substitutes: a review of recommendations in clinical management guidelines. *Hypertension*. 2024;81:400–14.
- Neal B, Wu Y, Feng X, Zhang R, Shi J, Zhang J, et al. Effect of salt substitution on cardiovascular events and death. *NEJM*. 2021;385:1067–77.
- Nohara-Shitama U, Adachi H, Enomoto M, Fukami A, Kumagai E, Nakamura S, et al. Twenty-four-hour urinary potassium excretion, but not sodium excretion, is associated with all-cause mortality in a general population. *JAHA*. 2018;7:e007369.
- Yu J, Arnott C, Li Q, Di Tanna GL, Tian M, Huang L, et al. Secondary analysis of the salt substitute and stroke study (SSaSS): effects of potassium-enriched salt on cardiac outcomes. *Hypertension*. 2024;81:1031–40.
- Jorstad HT, Colekesen EB, Boekholdt SM, Tijssen JG, Wareham NJ, Khaw KT, et al. Estimated 10-year cardiovascular mortality seriously underestimates overall cardiovascular risk. *Heart*. 2016;102:63–8.

46. Tomonari T, Fukuda M, Miura T, Mizuno M, Wakamatsu TY, Ichikawa T, et al. Is salt intake an independent risk factor of stroke mortality? Demographic analysis by regions in Japan. *J Am Soc Hypertens*. 2011;5:456–62.
47. Song J, Brown MK, Cobb LK, Jacobson MF, Ide N, MacGregor GA, et al. Delayed finalization of sodium targets in the United States may cost over 250 000 lives by 2031. *Hypertension*. 2022;79:798–808.

ACKNOWLEDGEMENTS

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the American Medical Association.

AUTHOR CONTRIBUTIONS

Regarding the Perspective, BME, DTL, SES, MAW, and PKW participated in design, drafting and revision; MKR, JW, YC-M, DWJ, S-EK, NRCC, GP, FJH, and GAM participated in interpreting and revising the draft. All authors approved the final decision to submit the Perspective to the *Journal of Human Hypertension* and each author accepts accountability for the content.

FUNDING

No financial report was received in support of this Perspective.

COMPETING INTERESTS

BME, none; DTL, none; SES, none; MKR, none; JW, none; YC-M, none; DWJ, none; SEK reports lecture honoraria from Emcure, Getz, Glenmark, J.B. Pharma, Merck Healthcare KGaA, and Vector-Intas in the past 3 years; NRCC, none; GP, none; FJH is an unpaid member of Action on Salt and World Action on Salt, Sugar and Health (WASSH); GAM is the unpaid Chair of Action on Salt, Action on Sugar, World Action on Salt, Sugar and Health (WASSH), and Blood Pressure UK; MAW, none; PKW, none.

ADDITIONAL INFORMATION

Supplementary information The online version contains supplementary material available at <https://doi.org/10.1038/s41371-025-00990-1>.

Correspondence and requests for materials should be addressed to Brent M. Egan.

Reprints and permission information is available at <http://www.nature.com/reprints>

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Open Access This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

© The Author(s) 2025