



Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017



GBD 2017 Diet Collaborators*

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Summary

Background Suboptimal diet is an important preventable risk factor for non-communicable diseases (NCDs); however, its impact on the burden of NCDs has not been systematically evaluated. This study aimed to evaluate the consumption of major foods and nutrients across 195 countries and to quantify the impact of their suboptimal intake on NCD mortality and morbidity.

Methods By use of a comparative risk assessment approach, we estimated the proportion of disease-specific burden attributable to each dietary risk factor (also referred to as population attributable fraction) among adults aged 25 years or older. The main inputs to this analysis included the intake of each dietary factor, the effect size of the dietary factor on disease endpoint, and the level of intake associated with the lowest risk of mortality. Then, by use of disease-specific population attributable fractions, mortality, and disability-adjusted life-years (DALYs), we calculated the number of deaths and DALYs attributable to diet for each disease outcome.

Findings In 2017, 11 million (95% uncertainty interval [UI] 10–12) deaths and 255 million (234–274) DALYs were attributable to dietary risk factors. High intake of sodium (3 million [1–5] deaths and 70 million [34–118] DALYs), low intake of whole grains (3 million [2–4] deaths and 82 million [59–109] DALYs), and low intake of fruits (2 million [1–4] deaths and 65 million [41–92] DALYs) were the leading dietary risk factors for deaths and DALYs globally and in many countries. Dietary data were from mixed sources and were not available for all countries, increasing the statistical uncertainty of our estimates.

Interpretation This study provides a comprehensive picture of the potential impact of suboptimal diet on NCD mortality and morbidity, highlighting the need for improving diet across nations. Our findings will inform implementation of evidence-based dietary interventions and provide a platform for evaluation of their impact on human health annually.

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Introduction

The relationship between dietary habits and chronic non-communicable diseases (NCDs) has been extensively investigated.^{1–5} Long-term randomised trials with NCD endpoints have not been feasible for most dietary factors, but synthesis of other lines of epidemiological evidence, including long-term prospective observational studies and short-term trials of intermediate outcomes, have provided supporting evidence for potential causal relationships between specific dietary factors (eg, fruits, vegetables, processed meat, and trans fat intake) and NCDs (ischaemic heart disease, diabetes, and colorectal cancer).^{2–7} These findings have been widely used to inform national and international dietary guidelines aimed at preventing NCDs.^{8,9} However, because of the complexities of characterising dietary consumption across different nations, assessment of the health effects of suboptimal diet at the population level has not been possible.

In the past decade, efforts have been made to quantify the burden of disease attributable to specific dietary

factors.^{10–19} These efforts, although useful, had several important limitations, including insufficient geographically representative data on dietary consumption, inaccurate characterisation of population distribution of dietary intake, insufficient accounting for biases of different sources of dietary assessment, standardisation of the intake to 2000 kcal per day, and insufficient accounting for within-person variation of intake of dietary factors.

To address these limitations, as part of the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017, we systematically collected geographically representative dietary data from multiple sources, characterised the population distribution of intake for 15 foods and nutrients among adults aged 25 years or older across 195 countries, estimated the effect of each individual dietary factor on NCD mortality, and quantified the overall impact of poor dietary habits on NCD mortality. We also evaluated the relationship between diet and socioeconomic development, and assessed the trends in disease burden of diet over time. This analysis supersedes all previous results from

Research in context**Evidence before this study**

We systematically searched MEDLINE and the Global Health Data Exchange (GHDx) to identify studies providing nationally or subnationally representative estimates of consumption of 15 foods and nutrients. We included only studies reporting data collected between Jan 1, 1980, and Dec 31, 2016, in one of the 195 countries included in this analysis. Studies were excluded if done with non-random samples or among specific subpopulations. We estimated the potential health effects of each dietary risk by use of the Global Burden of Diseases, Injuries, and Risk Factors Study comparative risk assessment approach.

Added value of this study

This study provides a comprehensive picture of consumption of 15 dietary factors across nations and quantifies the potential

impact of suboptimal intake of each diet component on chronic disease mortality and morbidity among 195 countries. Additionally, this study characterises the relationship between diet and development and evaluates the trends in the burden of disease attributable to diet from 1990 to 2017. High intake of sodium, low intake of whole grains, and low intake of fruits were the leading dietary risk factors for deaths and DALYs globally and in many countries.

Implications of all the available evidence

This study highlights the need for improving diet at the global, regional, and national level. The findings inform priorities for population-level interventions to improve diet.

GBD with respect to dietary risks by comprehensively reanalysing all data from 1990 to 2017, using consistent methods and definitions.

Methods**Selection of dietary risk factors**

We selected 15 dietary risk factors (table) that met GBD selection criteria for risk factors.^{10–13} These criteria include the importance of the risk factor to either disease burden or policy; the availability of sufficient data to estimate risk factor exposure; the strength of the epidemiological evidence supporting a causal relationship between risk factor exposure and disease endpoints, and availability of data to quantify the magnitude of this relationship per unit of change in the exposure; and evidence supporting the generalisability of the effects to all populations. The process of evaluation of the strength of epidemiological evidence for the causal relationship of each diet–disease pair is described elsewhere^{10–13} and summarised in the appendix.

Dietary intake at the population level

We did a systematic review of the scientific literature to identify nationally or subnationally representative nutrition surveys providing data on consumption of each dietary factor (appendix). We also searched the Global Health Data Exchange website for nationally or subnationally representative nutrition surveys and household budget surveys. Additionally, for food groups, we used national sales data from Euromonitor and national availability data from United Nations Food and Agriculture Organization food balance sheets. For nutrients, we used data on their national availability from the Global Nutrient Database.²⁰ For sodium, we collected data on 24 h urinary sodium, where available. For trans fat, we used sales data from Euromonitor on hydrogenated vegetable oil. The list of all dietary data sources used in GBD 2017 is publicly available at the Global Health Data Exchange website. For each dietary factor, we computed a data representativeness

index as the fraction of countries for which we identified any data on the risk factor exposure (table).

Our dietary data were from multiple sources and were affected by different types of biases. We considered 24 h diet recall as the gold standard method for assessing mean intake at the population level and adjusted dietary data from other sources accordingly (appendix). Some types of dietary data (ie, availability, sales, and household data) were only available for all-age groups and both sexes. To split these data into standard age-specific and sex-specific groups, we first estimated the global age and sex patterns of intake using data from nutrition surveys and then used those patterns to split the availability, sales, and household data.

We used the spatiotemporal Gaussian process regression method to estimate the mean intake of each dietary risk factor by age, sex, country, and year (appendix). To improve our estimates in data-sparse models, we tested a wide range of covariates with plausible relationships with intake and included the covariates with best fit and coefficients in the expected direction (appendix).

See Online for appendix

Effect size of dietary risks on disease endpoints

For each diet–disease pair, we used data from published meta-analyses of prospective observational studies to estimate the relative risk of mortality and morbidity.²¹ For diet–disease pairs for which evidence was only available on morbidity, we assumed that the estimated relative risks were also applied to mortality (appendix). Considering the relationship of diet and metabolic risk factors and the well established age trend of the relative risks of metabolic risks for cardiovascular disease and type 2 diabetes, we used the age trend of the relative risks of metabolic risk factors²² to estimate the age-specific relative risk of dietary risks for cardiovascular disease and type 2 diabetes (appendix). To estimate the impact of sodium on outcomes, we first estimated the relationship between urinary sodium and change

For more on the Global Health Data Exchange see <http://ghdx.healthdata.org>

For more on Euromonitor see <https://www.euromonitor.com/>

For more on food balance sheets see <http://www.fao.org/economic/ess/fbs/en/>

For the Global Nutrient Database see <https://nutrition.healthdata.org/global-nutrient-database>

For the list of all dietary data sources see <http://ghdx.healthdata.org/gbd-2017/data-input-sources>

in systolic blood pressure, and then estimated the relationship between change in systolic blood pressure and disease outcomes.¹⁴

Optimal level of intake

We defined the optimal level of intake as the level of risk exposure that minimises the risk from all causes of death. To estimate the optimal intake for each dietary factor, we first calculated the level of intake associated with the lowest risk of mortality from each disease endpoint based on the studies included in the meta-analyses of the dietary relative risks. Then, we calculated the optimal level of intake as the weighted mean of these numbers using the global proportion of deaths from each disease as the weight. To reflect the uncertainty of optimal level of intake, we assumed a uniform uncertainty distribution of 20% above and below the mean.¹³ For sodium, the evidence supporting the selection of the optimal level of intake was uncertain.^{23,24} Therefore, we included a uniform distribution of different optimal levels of intake in the uncertainty estimation sampling.

Disease-specific deaths and disability-adjusted life-years

Data on disease-specific deaths and disability-adjusted life-years (DALYs) by age, sex, country, and year were obtained from GBD 2017. The GBD approach to estimating cause-specific mortality and DALYs has been described in detail elsewhere.^{25,26}

Disease burden of dietary risks

We used the GBD comparative risk assessment approach to estimate the population attributable fraction for each diet-disease pair by age, sex, country, and year.^{10–13} Then, we estimated the number of deaths and DALYs attributable to each dietary risk factor by multiplying the population attributable fraction by the total number of disease-specific deaths and DALYs.

To position countries on the development continuum, we used the Socio-demographic Index (SDI), which is a summary measure calculated on the basis of lag-distributed income per capita, mean educational attainment of individuals aged 15 years or older, and total fertility rate among women younger than 25 years.^{12,13}

	Exposure definition	Optimal level of intake (optimal range of intake)	Data representativeness index (%)
Diet low in fruits	Mean daily consumption of fruits (fresh, frozen, cooked, canned, or dried fruits, excluding fruit juices and salted or pickled fruits)	250 g (200–300) per day	94·9
Diet low in vegetables	Mean daily consumption of vegetables (fresh, frozen, cooked, canned, or dried vegetables, excluding legumes and salted or pickled vegetables, juices, nuts, seeds, and starchy vegetables such as potatoes or corn)	360 g (290–430) per day	94·9
Diet low in legumes	Mean daily consumption of legumes (fresh, frozen, cooked, canned, or dried legumes)	60 g (50–70) per day	94·9
Diet low in whole grains	Mean daily consumption of whole grains (bran, germ, and endosperm in their natural proportion) from breakfast cereals, bread, rice, pasta, biscuits, muffins, tortillas, pancakes, and other sources	125 g (100–150) per day	94·9
Diet low in nuts and seeds	Mean daily consumption of nut and seed foods	21 g (16–25) per day	94·9
Diet low in milk	Mean daily consumption of milk including non-fat, low-fat, and full-fat milk, excluding soy milk and other plant derivatives	435 g (350–520) per day	94·9
Diet high in red meat	Mean daily consumption of red meat (beef, pork, lamb, and goat, but excluding poultry, fish, eggs, and all processed meats)	23 g (18–27) per day	94·9
Diet high in processed meat	Mean daily consumption of meat preserved by smoking, curing, salting, or addition of chemical preservatives	2 g (0–4) per day	36·9
Diet high in sugar-sweetened beverages	Mean daily consumption of beverages with ≥50 kcal per 226·8 serving, including carbonated beverages, sodas, energy drinks, fruit drinks, but excluding 100% fruit and vegetable juices	3 g (0–5) per day	36·9
Diet low in fibre	Mean daily intake of fibre from all sources including fruits, vegetables, grains, legumes, and pulses	24 g (19–28) per day	94·9
Diet low in calcium	Mean daily intake of calcium from all sources, including milk, yogurt, and cheese	1·25 g (1·00–1·50) per day	94·9
Diet low in seafood omega-3 fatty acids	Mean daily intake of eicosapentaenoic acid and docosahexaenoic acid	250 mg (200–300) per day	94·9
Diet low in polyunsaturated fatty acids	Mean daily intake of omega-6 fatty acids from all sources, mainly liquid vegetable oils, including soybean oil, corn oil, and safflower oil	11% (9–13) of total daily energy	94·9
Diet high in trans fatty acids	Mean daily intake of trans fat from all sources, mainly partially hydrogenated vegetable oils and ruminant products	0·5% (0·0–1·0) of total daily energy	36·9
Diet high in sodium	24 h urinary sodium measured in g per day	3 g (1–5) per day*	26·2

*To reflect the uncertainty in existing evidence on optimal level of intake for sodium, 1–5 g per day was considered as the uncertainty range for the optimal level of sodium where less than 2·3 g per day is the intake level of sodium associated with the lowest level of blood pressure in randomised controlled trials and 4–5 g per day is the level of sodium intake associated with the lowest risk of cardiovascular disease in observational studies.

Table: Dietary risk factor exposure definitions, optimal level, and data representativeness index, 1990–2017

To estimate gaps in intake or excess of intake of individual components of diet, we compared the current intake of each dietary factor with the midpoint of its optimal range of intake (table). High intake of a dietary component refers to an intake level higher than the midpoint of the optimal range of intake, and low intake refers to an intake level lower than the midpoint of the optimal range of intake.

To incorporate the uncertainty of parameters (exposure, relative risk, optimal level of intake, and mortality) as well as modelling uncertainty, we followed a Monte Carlo approach. We repeated all calculations 1000 times using one draw of each parameter at each iteration. Using these 1000 draws, we calculated the mean and 95% uncertainty interval (UI) for the final estimates.

All statistical analyses were done in Python, version 3.5.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The first author and the corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Consumption of major foods and nutrients

Globally, consumption of nearly all healthy foods and nutrients was suboptimal in 2017 (figure 1). The largest gaps between current and optimal intake were observed for nuts and seeds, milk, and whole grains, with mean consumption at 12% (95% UI 12–13; 3 g [2–3] of nuts and seeds per day), 16% (16–17; 71 g [70–72] of milk per day), and 23% (23–23; 29 g [29–29] of whole grains per day) of the optimal levels (percentages calculated on the basis of data before rounding). In parallel with suboptimal healthy food consumption, daily intake of all unhealthy foods and nutrients exceeded the optimal level globally (figure 1). The consumption of sugar-sweetened beverages (49 g per day) was far higher than the optimal intake. Similarly, global consumption of processed meat (4 g [4–4] per day, 90% greater than the optimal amount) and sodium (6 g [5–6] per day, 86% greater than the optimal amount) were far above the optimal levels. The global intake of red meat (27 g [26–28] per day) was 18% greater than the optimal intake. Men generally had a higher intake of both healthy and unhealthy foods than did women. Intake of both healthy and unhealthy foods was generally higher among middle-aged adults (50–69 years) and lowest among young adults (25–49 years) with a few exceptions. The highest intake of sugar-sweetened beverages and legumes were observed among young adults and showed a decreasing trend with age.

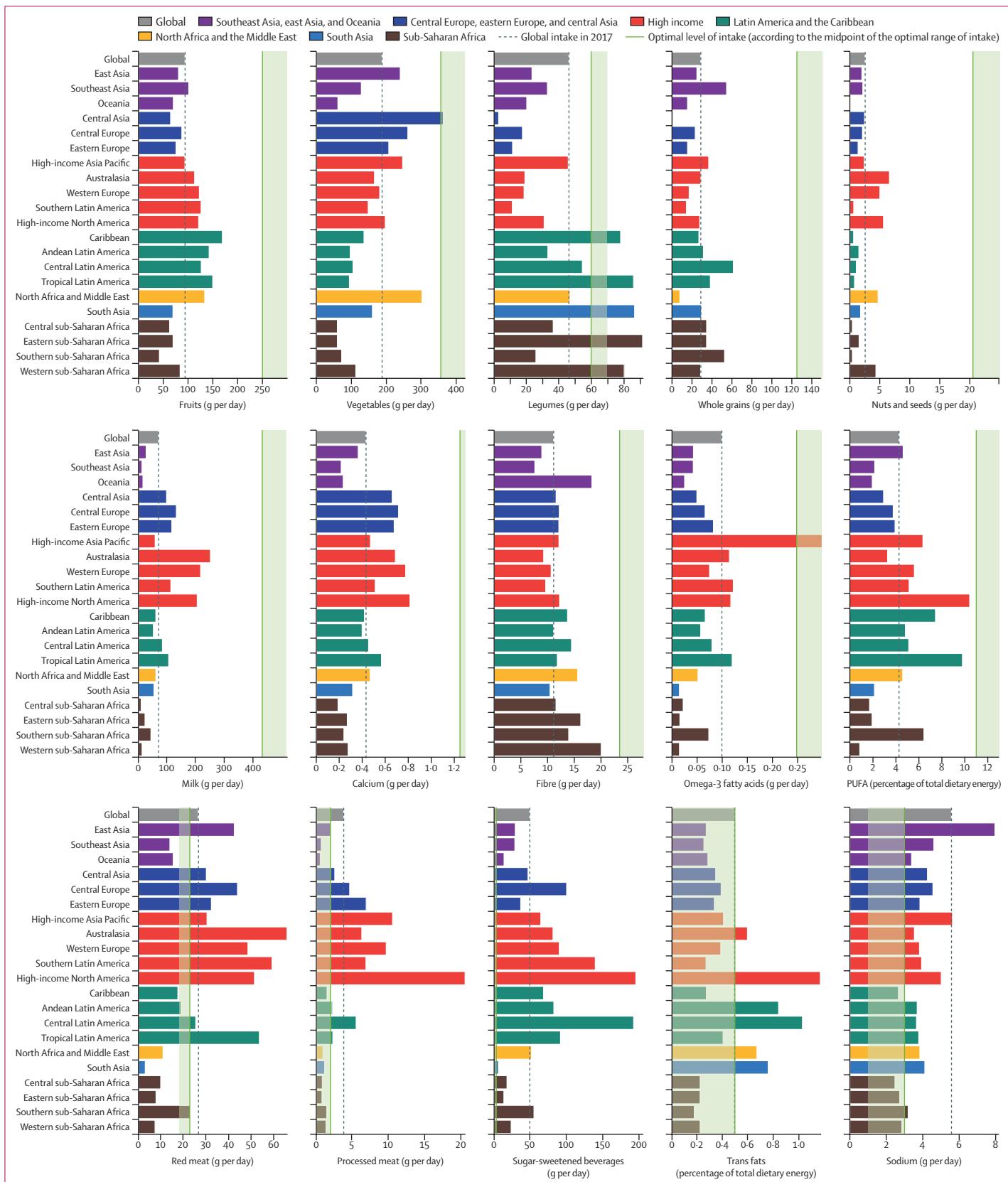
At the regional level, in 2017, the intake of all healthy foods was lower than the optimal level in all 21 GBD regions (figure 1). The only exceptions were the intake of vegetables in central Asia, seafood omega-3 fatty acids in

high-income Asia Pacific, and legumes in the Caribbean, tropical Latin America, south Asia, western sub-Saharan Africa, and eastern sub-Saharan Africa. Among unhealthy food groups, consumption of sodium and sugar-sweetened beverages were higher than the optimal level in nearly every region. Red meat consumption was highest in Australasia, southern Latin America, and tropical Latin America. High-income North America had the highest processed meat intake followed by high-income Asia Pacific and western Europe. The highest intake of trans fats was observed in high-income North America, central Latin America, and Andean Latin America.

Overall impact of diet on mortality

Globally, in 2017, dietary risks were responsible for 11 million [95% UI 10–12] deaths (22% [95% UI 21–24] of all deaths among adults) and 255 million (234–274) DALYs (15% [14–17] of all DALYs among adults; appendix). Cardiovascular disease was the leading cause of diet-related deaths (10 million [9–10] deaths) and DALYs (207 million [192–222] DALYs), followed by cancers (913 090 [743 345–1098 432] deaths and 20 million [17–24] DALYs) and type 2 diabetes (338 714 deaths [244 995–447 003] and 24 million [16–33] DALYs). More than 5 million (95% UI 5–5) diet-related deaths (45% [43–46] of total diet-related deaths) and 177 million (163–192) diet-related DALYs (70% [68–71] of total diet-related DALYs) occurred among adults aged younger than 70 years.

Across the 21 GBD regions, in 2017, the highest age-standardised rates of all diet-related deaths and DALYs among adults aged 25 years or older were observed in Oceania (678 [95% UI 616–746] deaths per 100 000 population and 17 804 [16 041–19 907] DALYs per 100 000 population; appendix). The lowest rates of all diet-related deaths among adults (aged 25 years or older) were observed in high-income Asia Pacific (97 [89–106] deaths per 100 000 population) and the lowest rates of all diet-related DALYs were observed in Australasia (2182 [1955–2444] DALYs per 100 000 population). The regions with the highest rates of diet-related cardiovascular disease deaths and DALYs were central Asia (613 [566–658] deaths per 100 000 population) and Oceania (14 755 [13 212–16 512] DALYs per 100 000 population), whereas the lowest rate of cardiovascular disease deaths and DALYs were observed in high-income Asia Pacific (68 [63–75] deaths per 100 000 population and 1443 [1329–1573] DALYs per 100 000 population). Diet-related cancer death and DALY rates were highest in east Asia (41 [34–49] deaths per 100 000 population and 878 [736–1023] DALYs per 100 000 population) and lowest in north Africa and the Middle East (nine [8–11] deaths per 100 000 population and 203 [169–243] DALYs per 100 000 population). Oceania (60 [44–78] deaths per 100 000 population and 2426 [1737–3198] DALYs per 100 000 population) had the highest age-standardised rate of diet-related diabetes



deaths and DALYs, and high-income Asia Pacific had the lowest rates (two [2–3] deaths per 100 000 population and 290 [202–395] DALYs per 100 000 population). In 2017, the highest age-standardised proportions of diet-related deaths and DALYs from cardiovascular disease were observed in Oceania (60% [95% UI 56–63] of deaths) and east Asia (64% [60–68] of DALYs), those from cancer in east Asia (15% [13–18] of deaths and 15% [12–17] of DALYs), and those from type 2 diabetes in high-income North America (41% [34–48] of deaths and 50% [42–58] of DALYs; appendix). The lowest age-standardised proportions of deaths and DALYs from these causes were in western Europe (42% [38–45] of deaths and 44% [41–47] of DALYs), western sub-Saharan Africa (5% [4–6] of deaths and 4% [4–5] DALYs), and southeast Asia (29% [20–38] of deaths and 35% [25–46] of DALYs).

In 2017, among the world's 20 most populous countries, Egypt had the highest age-standardised rate of all diet-related deaths (552 [95% UI 490–620] deaths per 100 000 population) and DALYs (11 837 [10 525–13 268] DALYs per 100 000 population) and Japan had the lowest rate of all diet-related deaths (97 [89–106] deaths per 100 000 population) and DALYs (2300 [2099–2513] DALYs per 100 000 population; figure 2). China had the highest age-standardised rates of diet-related cardiovascular disease deaths (299 [275–324] deaths per 100 000 population) and Egypt had the highest DALY rates (10 811 [9577–12 209] DALYs per 100 000 population). China had highest rates of diet-related cancer deaths and DALYs (42 [34–49] deaths per 100 000 population and 889 [744–1036] DALYs per 100 000 population), and Mexico had the highest rates of diet-related type 2 diabetes deaths and DALYs (35 [28–44] deaths per 100 000 population and 1605 [1231–2034] DALYs per 100 000 population). Japan had the lowest rate of diet-related cardiovascular disease deaths and DALYs (69 [63–75] deaths per 100 000 population and 1507 [1389–1639] DALYs per 100 000 population) and diabetes deaths and DALYs (one [1–1] death per 100 000 population and 234 [161–321] DALYs per 100 000 population). Egypt had the lowest rate of diet-related cancer deaths and DALYs (five [4–6] deaths per 100 000 population and 120 [96–146] DALYs per 100 000 population; appendix). The highest age-standardised proportion of all diet-related deaths (30% [27–33]) and DALYs (23% [21–25]) in adults aged 25 years or older were observed in Egypt, and the lowest proportion of all diet-related deaths (11% [9–12]) and DALYs (7% [6–8]) in the same age group were observed in Nigeria (appendix). The highest proportions of diet-related cardiovascular disease deaths and DALYs in 2017 were observed in Pakistan (60% [95% UI 57–64] of deaths and 66% [62–69] of DALYs), cancer deaths and DALYs in China (16% [13–18] of deaths and 15% [13–17] of DALYs), and type 2 diabetes deaths and DALYs in the USA

(41% [34–49] of deaths and 50% [43–58] of DALYs). The lowest proportions of cardiovascular disease deaths and DALYs were seen in Turkey (42% [38–47] of deaths and 44% [40–49] of DALYs), cancer deaths and DALYs in Egypt (4% [3–4] of deaths and 3% [3–4] of DALYs), and type 2 diabetes deaths and DALYs in Bangladesh (25% [17–34] of deaths and 34% [23–45] of DALYs).

Impact of individual components of diet on mortality

A small number of dietary risks had a large impact on health outcomes. In 2017, more than half of diet-related deaths and two-thirds of diet-related DALYs were attributable to high intake of sodium (3 million [95% UI 1–5] deaths and 70 million [34–118] DALYs), low intake of whole grains (3 million [2–4] deaths and 82 million [59–109] DALYs), and low intake of fruits (2 million [1–4] deaths and 65 million [41–92] DALYs; figure 3). Low intake of whole grains was the leading dietary risk factor for DALYs among men and women and the leading dietary risk factor for mortality among women. Sodium ranked first for mortality among men followed by whole grains and fruit. Low intake of whole grains was the leading risk for deaths and DALYs among young adults (aged 25–50 years) and sodium ranked first among older adults (≥ 70 years).

In 2017, across the 21 GBD regions, a diet low in whole grains was the most common leading dietary risk factor for deaths (in 16 regions) and DALYs (in 17 regions; figure 4). A diet high in sodium was the leading dietary risk factor for deaths and DALYs in east Asia and high-income Asia Pacific regions (appendix). In southern sub-Saharan Africa, a diet low in fruits and in central Latin America a diet low in nuts and seeds were the dietary risk factors responsible for the greatest proportion of deaths and DALYs in 2017.

High intake of sodium was the leading dietary risk for deaths and DALYs in China, Japan, and Thailand. Low intake of whole grains was the leading dietary risk factor for deaths and DALYs in the USA, India, Brazil, Pakistan, Nigeria, Russia, Egypt, Germany, Iran, and Turkey. In Bangladesh, low intake of fruits was the leading dietary risk associated with deaths and DALYs. In Mexico, low intake of nuts and seeds ranked first for diet-related deaths and DALYs. High consumption of red meat, processed meat, trans fat, and sugar-sweetened beverages were towards the bottom in ranking of dietary risks for deaths and DALYs for most high-population countries (appendix).

Relationship between diet and SDI

Overall, in 2017, the highest age-standardised rates of all diet-related deaths and DALYs were observed in low-middle SDI countries (344 [95% UI 319–369] deaths per 100 000 population and 7797 [7265–8386] DALYs per 100 000 population) and high-middle SDI countries (347 [324–369] deaths per 100 000 population and 6998 [6534–7454] DALYs per 100 000 population; appendix).

Figure 1: Age-standardised intake of dietary factors among adults aged 25 years or older at the global and regional level in 2017

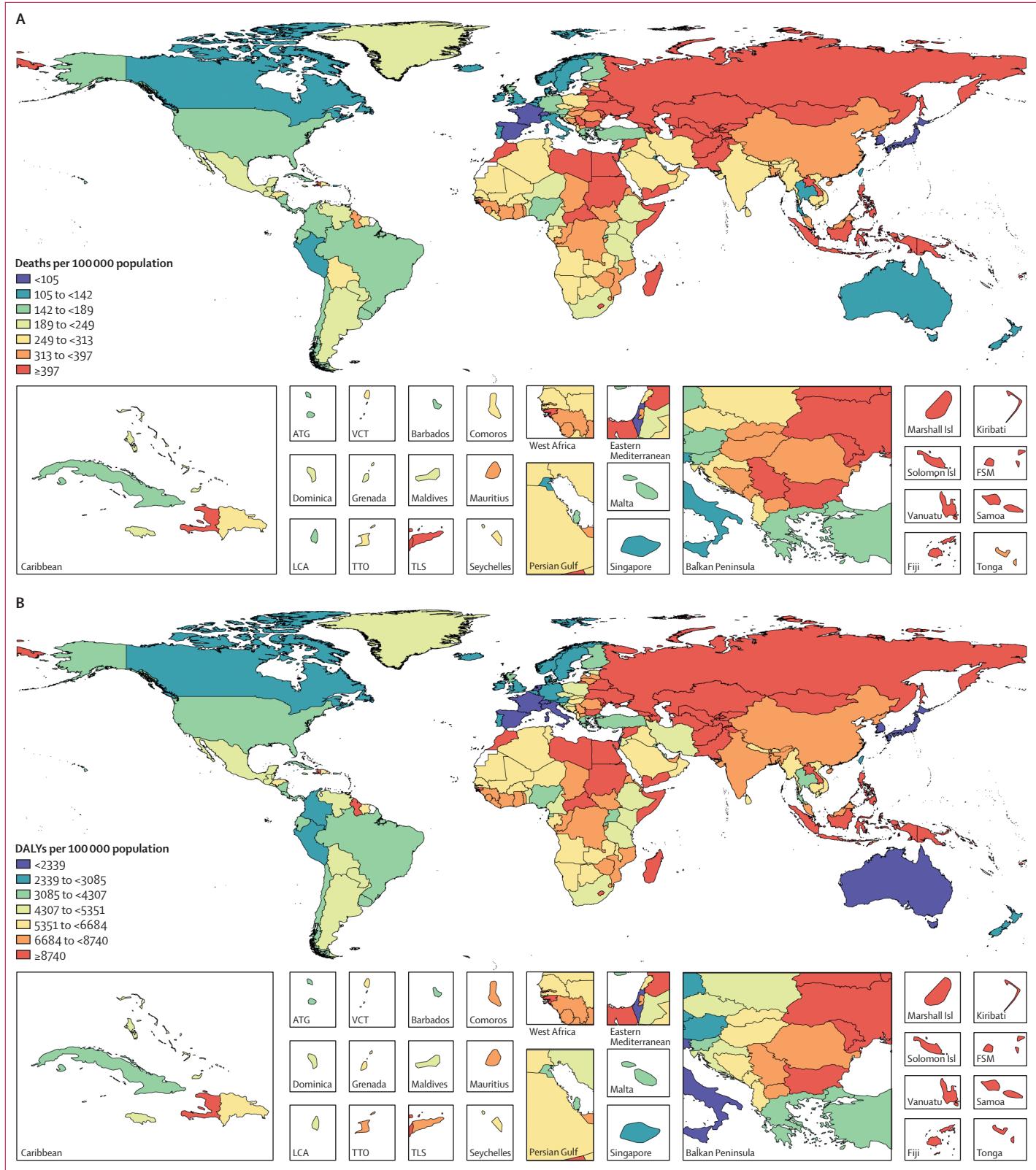


Figure 2: Age-standardised mortality rate per 100 000 population (A) and DALY rate per 100 000 population (B) attributable to diet in 2017
ATG=Antigua and Barbuda. Isl=Islands. FSM=Federated States of Micronesia. LCA=Saint Lucia. TLS=Timor-Leste. TTO=Trinidad and Tobago. VCT=Saint Vincent and the Grenadines.

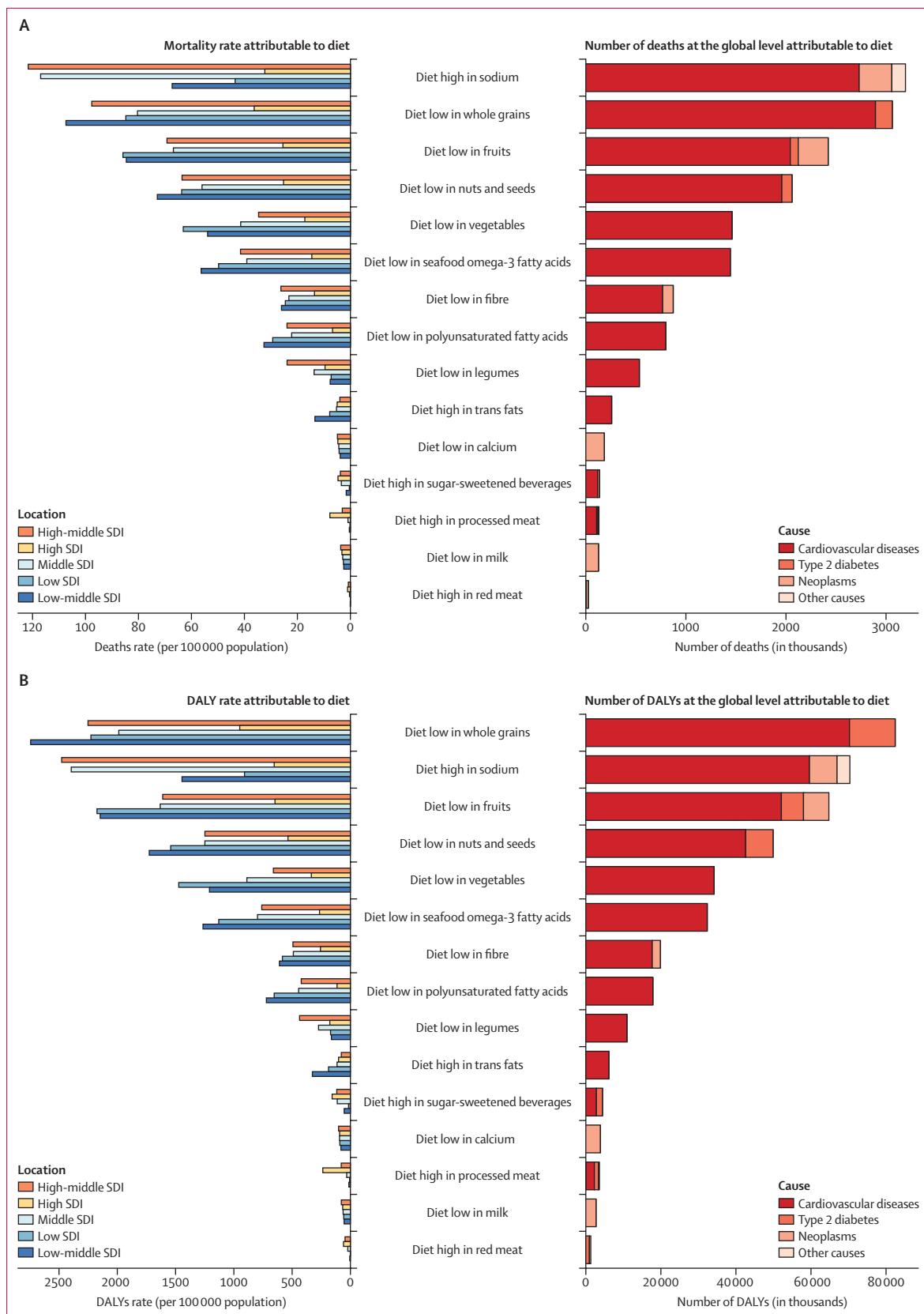


Figure 3: Number of deaths and DALYs and age-standardised mortality rate and DALY rate (per 100 000 population) attributable to individual dietary risks at the global and SDI level in 2017. DALY=disability-adjusted life-year. SDI=Socio-demographic Index.



Figure 4: Age-standardised proportions of deaths and DALYs attributable to individual dietary risks at the global and regional level in 2017
DALYs=disability-adjusted life-years.

The lowest burden of exposure to dietary risk was observed in high SDI countries (139 [129–148] deaths per 100 000 population and 3032 [2802–3265] DALYs per 100 000 population). Low-middle SDI had the highest age-standardised rates of diet-related deaths and DALYs

for cardiovascular disease (311 [288–335] deaths per 100 000 population and 6685 [6228–7161] DALYs per 100 000 population) and diabetes (14 [10–18] deaths per 100 000 population and 681 [477–914] DALYs per 100 000 population). High-middle SDI had the highest

age-standardised rates of diet-related mortality for cancer (29 [24–34] deaths per 100 000 population and 630 [529–731] DALYs per 100 000 population). The lowest age-standardised rate of diet-related deaths and DALYs for cardiovascular disease (113 [104–122] deaths per 100 000 population and 2156 [2005–2306] DALYs per 100 000 population) and diabetes (five [4–6] deaths per 100 000 population and 444 [324–587] DALYs per 100 000 population) was observed in high SDI countries and lowest mortality rate for cancer was observed in low SDI countries (15 [12–17] deaths per 100 000 population and 324 [268–376] DALYs per 100 000 population). The highest proportions of diet-related deaths and DALYs for all causes were observed in high-middle SDI countries (29% [95% UI 27–31] of deaths and 19% [17–21] of DALYs), the lowest proportion of diet-related deaths was observed in low SDI countries (16% [15–17] of deaths), and the lowest proportion of DALYs was observed in high SDI countries (10% [9–11] of DALYs; appendix). Dietary risks were responsible for 55% [51–59] of cardiovascular disease deaths and 60% [56–63] of DALYs in middle SDI countries, and 46% [42–49] of cardiovascular disease deaths and 49% [46–52] of cardiovascular disease DALYs in high SDI countries. Middle SDI countries had the highest proportion of cancer deaths (12% [10–14]) and DALYs (11% [9–13]) and high SDI countries had the lowest proportion of attributable cancer deaths (8% [7–9]) and DALYs (7% [6–9]). The highest burden of diabetes attributable to diet was observed in high SDI countries (35% [28–43] of deaths and 46% [38–55] of DALYs) and lowest attributable burden was observed in the low SDI countries (31% [22–39] of deaths and 39% [29–50] of DALYs).

High-middle and middle SDI countries were at the greatest risk of deaths and DALYs from high consumption of sodium, whereas high and low-middle SDI countries had the greatest risk caused by a diet low in whole grains (figure 3). In low SDI countries, low intake of fruit was the leading dietary risk for deaths and low intake of whole grains was the leading dietary risk for DALYs. Countries at all levels of SDI other than low SDI had the same four leading dietary risks: high sodium, low whole grains, low fruit, and low nuts and seeds. The four leading dietary risks for low SDI countries were a diet in low whole grains, low in fruit, low in nuts and seeds, and low in vegetables.

Impact of nutrition transition on exposure to dietary risks

Since 1990, the number of deaths (8 million [95% UI 7–8] deaths) and DALYs (184 [172–197] DALYs) attributable to dietary risks significantly increased to 11 million (10–12) deaths and 255 million (234–274) DALYs in 2017 (appendix). The main contributors to this increase were population growth and population ageing. After removing the effect of population growth and population ageing, the age-standardised attributable death and DALY rates showed a significant decrease between 1990

and 2017; from 406 (381–430) deaths per 100 000 population to 275 (258–292) deaths per 100 000 population, and from 8536 (8063–9013) DALYs per 100 000 population to 6080 (5685–6472) DALYs per 100 000 population. This decrease seemed to be driven mostly by decreases in the background mortality rate because, during the same period, the proportion of deaths and DALYs related to dietary risk remained relatively stable.

Discussion

Our systematic evaluation of dietary consumption patterns across 195 countries provides a comprehensive picture of the health effects of poor dietary habits at the population level. We found that improvement of diet could potentially prevent one in every five deaths globally. Our findings show that, unlike many other risk factors, dietary risks affected people regardless of age, sex, and sociodemographic development of their place of residence. Although the impact of individual dietary factors varied across countries, non-optimal intake of three dietary factors (whole grains, fruits, and sodium) accounted for more than 50% of deaths and 66% of DALYs attributable to diet.

Our findings show that suboptimal diet is responsible for more deaths than any other risks globally, including tobacco smoking,^{11,12} highlighting the urgent need for improving human diet across nations. Although sodium, sugar, and fat have been the main focus of diet policy debate in the past two decades,^{27,28} our assessment shows that the leading dietary risk factors for mortality are diets high in sodium, low in whole grains, low in fruit, low in nuts and seeds, low in vegetables, and low in omega-3 fatty acids; each accounting for more than 2% of global deaths. This finding suggests that dietary policies focusing on promoting the intake of components of diet for which current intake is less than the optimal level might have a greater effect than policies only targeting sugar and fat, highlighting the need for a comprehensive food system interventions to promote the production, distribution, and consumption of these foods across nations.

Over the past decade, the effectiveness of a range of population-level dietary interventions has been systematically evaluated and several promising interventions have been identified.^{29–31} These include mass media campaigns, food and menu labeling, food pricing strategies (subsidies and taxation), school procurement policies, and worksite wellness programmes. Cost-effectiveness analyses of these interventions have shown that targeting specific dietary factors (eg, sodium) might not only be cost-effective but cost-saving.^{32–35} However, improvement of diet through population-level interventions faces several major challenges. First, the observed effects for most of these dietary interventions are far below the level required to achieve optimal diet globally.^{29,30} Second, there is almost no evidence on the effectiveness of these interventions on several important

dietary factors (ie, nuts, whole grains, seafood, red meat, and processed meat). Third, cost-effectiveness analyses of dietary interventions are generally based on a range of simplifying assumptions and do not take into account the reactions of consumers (eg, substitution effect), the food industry (eg, food reformulations and pricing strategies), and other stakeholders in the real world.^{32–35} Fourth, despite the growing public and political will for the implementation of some of these policies (eg, trans fat bans), few countries have successfully adopted and implemented them.^{36,37} Fifth, many of these policies only target consumers but not the wide range of interconnected factors, such as food production, processing, and distribution, that exist throughout the food system. Indeed, these factors might affect dietary consumption, and it is important to include them to improve diet.^{38,39} Therefore, in view of the magnitude of the disease burden attributable to diet and the limitations of the existing interventions, development of novel food system interventions is urgently needed.

Our results show a need for extensive changes in various sectors of the food system at the global, regional, and national levels to improve diet. Changes in agricultural practices, if not done properly, might raise concerns over potential environmental effects on climate change, biodiversity loss, degradation of land and soil, and freshwater depletion.^{40–43} A growing body of evidence has emerged in the past decade showing that shifting diet from unhealthy animal-based foods (eg, red meat and processed meat) to healthy plant-based foods (eg, fruits, vegetables, and whole grains) might be associated with lower emission of greenhouse gases and thus might be more environmentally sustainable.^{40–43} The few studies evaluating other environmental effects of the shift from animal-based to plant-based diet have also demonstrated that this shift might be associated with lower land use and water footprint.⁴¹ However, because of the variations in the methods and research questions across these studies and scarcity of reliable estimates on dietary consumption patterns across nations, a comprehensive assessment of environmental effects of achieving optimal diet globally has not been possible to date. GBD estimates the dietary consumption of key foods and nutrients across 195 nations annually. These data provide a unique opportunity to quantify the environmental burden of current dietary consumption patterns at global, regional, and national levels in a consistent and comparable way. Additionally, these data could potentially be used to evaluate the effect of various food system interventions on human health and environment.⁴²

Our study also demonstrates the gaps in nationally representative individual-level data on intake of key foods and nutrients in different regions of the world, highlighting the importance of establishing national surveillance and monitoring systems for key dietary risk factors.^{17,18} For example, although many countries collect

data on fruit and vegetable intake, data on intake of specific nutrients such as sodium are scarce. The FAO/WHO Global Individual Food consumption data Tool⁴⁴ aims to address this problem, but several important gaps will remain. In the absence of reliable biomarkers or more accurate methods of dietary assessment, the 24 h diet recall or diet record remains the gold standard method of dietary assessment. However, evidence from validation studies suggests that it is not highly reliable for assessment of foods and nutrients due to recall bias or potential social desirability.^{45,46} This evidence highlights the need for development and validation of innovative dietary assessment methods. In the past decade, new methods have been developed; however, they have not been widely used and their validity has not been systematically evaluated.⁴⁷ Furthermore, accurate estimation of nutrients (eg, fibre, calcium, and polyunsaturated fatty acids), remains a major challenge. Many countries do not have local food composition tables and rely on data from food composition tables from other countries (eg, US Department of Agriculture food composition tables). Additionally, the recipes of mixed dishes as well as formulation of the food products, particularly their content of fat, sugar, and sodium, varies across countries and over time, which makes estimation of the true intake of nutrient more challenging.

Our systematic evaluation of epidemiological evidence shows several important limitations in existing dietary relative risks. The effect sizes of the dietary risk factors on disease endpoints were mostly obtained from meta-analyses of prospective observational studies. Although many of these dietary relative risks have been adjusted for the major confounders (eg, age, sex, smoking, and physical activity), the possibility of residual confounding cannot be excluded. To remove the effect of energy intake as a potential confounder and address measurement error in dietary assessment tools, most cohorts have adjusted for total energy intake in their statistical models. This energy adjustment means that diet components are defined as risks in terms of the share of diet and not as absolute levels of exposure. In other words, an increase in intake of foods and macronutrients should be compensated by a decrease in intake of other dietary factors to hold total energy intake constant. Thus, the relative risk of change in each component of diet depends on the other components for which it is substituted. However, the relative risks estimated from meta-analyses of cohort studies do not generally specify the type of substitution. The definition of dietary factors (eg, whole grains) also varies across studies. Additionally, given the intake of healthy dietary factors are generally positively correlated with each other and inversely correlated with harmful dietary factors, the effect size of the individual dietary factors might be overestimated. Many of the observational studies used for estimation of the relative risks have not corrected risk estimates for dietary measurement error, and some have adjusted for

factors along the causal pathways. Although many cohort studies have collected dietary data, only a few of them have published results of their assessment, which increases the possibility of publication bias. These limitations highlight the need for a collaborative effort to collect and harmonise all available dietary data from cohort studies and to do a pooled analysis for each diet–disease pair and quantify the effect size after adjusting for the same set of confounders.

Other potential limitations should also be considered in interpreting and using the findings of our study. We did not evaluate the effect of other forms of malnutrition (ie, undernutrition and obesity). The epidemiological evidence supporting a causal relationship between dietary risks and disease endpoints were mostly from observational studies, and the strength of evidence was generally weaker than the strength of evidence supporting a causal relationship between other established risks factors (eg, tobacco use and high systolic blood pressure) and chronic diseases. Additionally, the strength of evidence varied across foods and nutrients. Dietary data were from mixed sources and were not available for all countries. These factors increase the statistical uncertainty of our estimates for exposure to dietary risks. For sodium, we did not include data from spot urine sample, which resulted in a lower data representativeness index for sodium than that of other dietary risks. In estimation of the NCD burden of diet, we assumed that the distribution of dietary factors is independent within each unit of analysis (ie, country, age, and sex group), which might have resulted in underestimation or overestimation of the combined effect of dietary factors. To quantify the effect of correlation of dietary factors, we used individual-level data from the US National Health and Nutrition Examination Survey and estimated the overall burden of dietary risks (ie, joint population attributable fractions) with and without taking into account their correlation. The absolute difference in the joint population attributable fractions, on average, was less than 2%. Additionally, deaths due to some dietary risk factors might not be mutually exclusive, which could result in overestimation of the burden of disease attributable to diet.

In summary, we found that poor dietary habits are associated with a range of chronic diseases and can potentially be a major contributor to NCD mortality in all countries worldwide. This finding highlights the urgent need for coordinated global efforts to improve the quality of human diet. Given the complexity of dietary behaviours and the wide range of influences on diet, improving diet requires active collaboration of a variety of actors throughout the food system, along with policies targeting multiple sectors of the food system.

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AAF prepared the first draft. KAF and KMC constructed the figures and tables. AAF and PJS developed models for dietary risks. CJLM provided overall guidance. JSS managed the project. AAF and ECM finalised the manuscript on the basis of comments from other authors and reviewer feedback. All other authors provided data, reviewed results, or reviewed and contributed to the paper.

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JMG reports grants from Unilever. LJ reports personal fees from Mills Scientific Council. SL reports personal fees from Amgen, Berlin-Chemie, Merck Sharp & Dohme (MSD), Novo Nordisk, Sanofi-Aventis, Synlab, Unilever, and Upfield, and non-financial support from Preventicus. SL is also a member of the Scientific Board of the German Nutrition Society and a coauthor of the evidence-based guideline Fat Intake and Prevention of Nutrition-Related Diseases of the German Nutrition Society. WMä reports grants and personal fees from Siemens Diagnostics, Aegerion Pharmaceuticals, Amgen, AstraZeneca, Danone Research, Pfizer, BASF, Numares AG, and Berlin-Chemie; personal fees from Hoffmann LaRoche, MSD, Sanofi, and Synageva; grants from Abbott Diagnostics; and employment with Synlab Holding Deutschland GmbH. WMe is currently a program analyst for Population and Development at the Peru Country Office of the United Nations Population Fund-UNFPA, an institution that does not necessarily endorse this study. RMi reports grants from the US National Institutes of Health, Bill & Melinda Gates Foundation, and Unilever; and personal fees from World Bank and Bunge. DM reports research funding from the US National Institutes of Health and the, Bill & Melinda Gates Foundation; personal fees from GOED, DSM, Nutrition Impact, Pollock Communications, Bunge, Indigo Agriculture, Amarim, Acasti Pharma, and America's Test Kitchen; scientific advisory board roles with Elysium Health (with stock options), Omada Health, and DayTwo; and chapter royalties from UpToDate. In addition, DM is listed as a co-inventor on patents US8889739 and US9987243 issued to Tufts University (Somerville, MA, USA; unlicensed) for use of trans-palmitoleic acid to prevent and treat insulin resistance, type 2 diabetes, and related conditions, as well as reduce metabolic risk factors. CDR reports personal fees from Dairy Management Institute. AES reports personal fees from IEM, Novartis, Servier, and Abbott. AGT reports grants from National Health and Medical Research Council, Australia. All other authors declare no competing interests.

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Health Effects of Dietary Risks in 195 Countries: Findings from the Global Burden of Diseases Study 2017

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Overview

The process of estimating the burden of disease attributable to each dietary risk factor has been shown in Supplemental Figure 1. Briefly, using the World Cancer Research Fund evidence grading criteria, we evaluated the strength of epidemiologic evidence supporting causal relationship between dietary factors and chronic diseases and selected the dietary factors for which we found sufficient evidence on their causal relationship with chronic disease endpoints. We then systematically collected dietary data from multiple sources and harmonized them. Then, we used spatio-temporal Gaussian process regression to estimate the mean exposure level for each dietary factor; used linear regression to estimate the standard deviation of intake based on the mean intake for each dietary factor; and used ensemble approach to characterize the distribution of intake for each dietary factor at the population level. We obtained the relative risk of each disease endpoint per serving of the dietary components from recent dose-response meta-analyses of prospective observational studies and estimated the optimal level of intake based on the level of intake associated with the lowest risk of mortality in prospective cohort studies. Using the exposure level, relative risk, and optimal level of intake, we estimated the population attributable fraction (PAF) for each diet-disease pair. Finally, using disease-specific PAFs and mortality and Disability-Adjusted Life Year (DALY), we calculated the total number of attributable deaths and DALYs across all relevant outcomes.

Selection of dietary risks

We used the World Cancer Research Fund evidence grading criteria to separately assess the strength of the epidemiologic evidence on the causal relationship between each dietary risk factor and disease endpoint, and only included dietary risk factors for which we found convincing or probable evidence on their relationship with chronic diseases. Supplemental Tables 1 and 2 summarize epidemiologic evidence supporting the causal relationship for the risk-outcome pairs included in our analysis.

Estimating the dietary intake

Data Sources

We systematically searched Medline to identify studies providing nationally or subnationally representative estimates of consumption of dietary factors. Additionally, we searched the Global Health Data Exchange (GHDx) database for individual-level data from nutrition surveys or household budget surveys providing dietary data. We included all nationally or subnationally representative studies providing data on mean intake of each dietary factor by age and sex. We only included studies reporting data collected between 1 January 1980 and 31 December 2016 in one of the 195 countries included in this analysis. Studies were excluded if using non-random samples (e.g., case-control studies or convenience samples); conducted among specific subpopulations (e.g., pregnant women, racial or ethnic minorities, immigrants, or individuals with specific diseases); having sample sizes of less than 20 per 5-year age-sex group; providing inadequate information on any of the inclusion criteria. We excluded non-English articles. For eight food groups (fruits, vegetables, legumes, nuts and seeds, red meat, processed meat, milk, SSBs), we used sales data from Euromonitor to capture recent trends in the intake. For six food groups (fruits, vegetables, legumes, nuts and seeds, milk, and red meat), we

used data from United Nations Food and Agriculture Organization (FAO) food balance sheets (FBS). Additionally, we estimated the national availability of nutrients (i.e., fiber, calcium, omega-3 fatty acids, polyunsaturated fatty acids availability, and saturated fatty acids) using data from FAO's Supply Utilization Accounts (SUA) and the United States Department of Agriculture's National Nutrition Database for Standard Reference. For whole grains, we used FBS and SUAs data to estimate the availability of refined grains and total grains at the country level and then calculated the availability of whole grains by difference. Types of data sources (other than 24-hour dietary recall) used in modeling of each dietary factor is summarized in Table A.

Table A. Types of data sources (other than 24-hour dietary recall) and covariates used in modeling of each dietary factor.

	Data Sources				Country level covariate
	Sales	FFQ ¹	HBS ²	FAO	
Diet low in fruits	●	●	●	●	Lag-distributed income, total available kilocalories per person per day
Diet low in vegetables	●	●	●	●	Lag-distributed income, total available kilocalories per person per day
Diet low in legumes	●	-	●	●	Lag-distributed income, total available kilocalories per person per day
Diet low in whole grains	-	●	-	●	Lag-distributed income
Diet low in nuts and seeds	●	-	●	●	Lag-distributed income, total available kilocalories per person per day
Diet low in milk	●	●	●	●	Lag-distributed income, total available kilocalories per person per day
Diet high in red meat	●	●	●	●	Lag-distributed income, total available kilocalories per person per day
Diet high in processed meat	●	●	●	-	National availability of red meat (grams/person/day), National availability of pig meat (% of energy/person/day), Lag-distributed income
Diet high in sugar-sweetened beverages	●	●	●	-	National availability of sugar (grams/person/day), Lag-distributed income, total available kilocalories per person per day
Diet low in fiber	-	●	-	●	Lag-distributed income, total available kilocalories per person per day
Diet suboptimal in calcium	-	●	-	●	Lag-distributed income, total available kilocalories per person per day
Diet low in seafood omega-3 fatty acids	-	-	-	●	Landlocked nation (Yes/No), Lag-distributed income
Diet low in polyunsaturated fatty acids	-	●	-	●	Lag-distributed income, total available kilocalories per person per day
Diet high in trans fatty acids	●	●	-	-	-
Diet high in sodium ³	-	-	-	-	-

¹Food Frequency Questionnaire

²Household Budget Survey

³For sodium, we used data from the 24-hour urinary sodium and 24-hour dietary recall.

Crosswalks

We used dietary data from multiple sources and each type of dietary data was affected by specific types of biases. To adjust for biases of each dietary assessment method and to make our dietary data more comparable, we considered 24-hour diet recall as the gold standard method for assessing the mean intake at the population level and adjusted dietary data from other sources accordingly in a subset of countries with data from both methods of assessment. For sodium, 24-hour urinary sodium was

considered as the gold standard and we converted dietary sodium to urinary equivalent using a multiplier estimated from the surveys reporting both dietary and urinary sodium. Given that some of our data sources (i.e., availability, sales, and household data) were only providing data for all-age groups and both sexes, we first split these data into standard age-sex groups. Then, we matched dietary data from each non-gold-standard source data to the dietary data from the gold-standard source (i.e., 24-hour diet recall) by age, sex, country, and year and performed a linear regression analysis to estimate the consumption data from data on availability, sales, and household availability by age (a) and sex (s) using the following equation:

$$\text{Dietary Intake}_{a,s} = \beta * \text{Availability}_{a,s} + \text{age} + \text{sex}$$

Age and sex splitting

Data from UN FAO, Euromonitor, and household budget surveys were for all-age groups and both sexes. To split these data into standard age-sex groups, we first characterized the global age and sex patterns of intake for each dietary factor using data from 24-hour dietary recalls. Then, we applied the identified age patterns to split the availability, sales, and household data into standard age-sex groups.

Estimating the mean exposure level

We used a spatio-temporal Gaussian process regression (ST-GPR) to estimate the full time series of national intake of each diet component (flowchart). This approach is a stochastic modelling technique that is designed to detect signals amidst noisy data. It also serves as a powerful tool for interpolating non-linear trends.^{1,2} Unlike classical linear models that assume that the trend underlying data follows a definitive functional form, GPR assumes that the specific trend of interest follows a Gaussian Process, which is defined by a mean function $m(\cdot)$ and a covariance function $\text{Cov}(\cdot)$. For example, let $p_{c,a,s,t}$ be the exposure, in normal, log, or logit space, observed in country c , for age group a , and sex s at time t :

$$(p_{c,a,s,t}) = g_{c,a,s}(t) + \epsilon_{c,a,s,t}$$

where

$$\begin{aligned}\epsilon_{c,a,s,t} &\sim \text{Normal}(0, \sigma_p^2), \\ g_{c,a,s}(t) &\sim GP\left(m_{c,a,s}(t), \text{Cov}\left(g_{c,a,s}(t)\right)\right).\end{aligned}$$

The derivation of the mean and covariance functions, $m_{c,a,s}(t)$ and $\text{Cov}\left(g_{c,a,s}(t)\right)$, along with a more detailed description of the error variance (σ_p^2), is described below.

Estimating mean functions

We estimated mean functions using a two-step approach. To be more specific, $m_{c,a,s}(t)$ can be expressed, depending on the exposure transformation, as:

$$\log(p_{c,a,s}(t)) = X_{c,a,s}\beta + h(r_{c,a,s,t})$$

$$\text{logit}(p_{c,a,s}(t)) = X_{c,a,s}\beta + h(r_{c,a,s,t})$$

$$p_{c,a,s}(t) = X_{c,a,s}\beta + h(r_{c,a,s,t})$$

where $X\beta$ is the summation of the components of a hierarchical mixed-effects linear regression, including the intercept and the product of covariates with their corresponding fixed effect coefficients. The second part of the equation, $h(r_{c,a,s,t})$, is a smoothing function for the residuals, $r_{c,a,s,t}$, derived from the linear model. Table A provides a description of covariates used in linear model for each dietary factor.

While the linear component captures the general trend in exposures over time, much of the data variability may still not be adequately accounted for. To address this, we fit a locally weighted polynomial regression (LOESS) function $h(r_{c,a,s,t})$ to systematically estimate this residual variability by borrowing strength across time, age, and space patterns (the spatiotemporal component of ST-GPR). The time adjustment parameter, defined by λ , aims to borrow strength from neighbouring time points (ie, the exposure in this year is highly correlated with exposure in the previous year but less so further back in time). The age-adjustment parameter, defined by ω , borrows strength from data in neighbouring age groups. The space-adjustment parameter, defined by ξ , aims to borrow strength across the hierarchy of geographical locations. We further combined the spatial and temporal weights into a single space-time weight, to allow the amount of spatial weight given to a particular point $r_{c,a,s,t}$ to fluctuate given the data availability at each time t and location-level l in the location hierarchy.

Estimating error variance

σ_p^2 represents the error variance in normal or transformed space including sampling variance of the estimates and prediction error from the crosswalks performed. First, variance was systematically imputed if the data extraction did not include any measure of uncertainty. When some sample sizes for data were available, missing sample sizes were imputed as the 5th percentile of available sample sizes. Missing variances were then predicted from the mean using a regression. When sample sizes were entirely missing and could not be imputed, the 95th percentile of available variances at the most granular geographic level (ie, first country, then region, etc.) were used to impute missing variances. Finally, prior to GPR, an approximation of non-sampling variance was added to the error variance. Calculations of non-sampling variance were performed on normal-space variances. Non-sampling variance was calculated as the variance of inverse-variance weighted residuals from the space-time estimate at a given location level hierarchy. If there were fewer than ten data points at a given level of the location hierarchy, the non-sampling variance was replaced with that of the next highest geography level with more than ten data points.

Estimating the covariance function

The final input into GPR is the covariance function, which defines the shape and distribution of the trends. Here, we have chosen the Matern-Euclidian covariance function, which offers the flexibility to model a wide spectrum of trends with varying degrees of smoothness. The function is defined as follows:

$$M(t, t') = \sigma^2 \frac{2^{1-\nu}}{\Gamma(\nu)} \left(\frac{d(t, t')\sqrt{2\nu}}{l} \right)^\nu K_\nu \left(\frac{d(t, t')\sqrt{2\nu}}{l} \right)$$

where $d(\cdot)$ is a distance function; σ^2 , v , l , and K_v are hyperparameters of the covariance function – specifically σ^2 is the marginal variance, v is the smoothness parameter that defines the differentiability of the function, l is the length scale, which roughly defines the distance between which two points become uncorrelated, and K_v is the Bessel function. We approximated σ^2 by taking the normalised median absolute deviation $MADN(r'_c)$ of the difference, which is the normalised absolute deviation of the difference of the first-stage linear regression estimate from the second-stage spatiotemporal smoothing step for each country. We then took the mean of these country-level MADN estimates for all countries with 10+ country-years of data, to ensure that differences between first- and second-stage estimates had sufficient data to truly convey meaningful information on model uncertainty. We used the parameter specifications $v = 2$ for all models.

Prediction using GPR

We integrated over $g_{c,t}(t_*)$ to predict a full time series for country c , age a , sex s , and the prediction time t_* :

$$p_{c,a,s}(t_*) \sim N\left(m_{c,a,s,t}(t_*), \sigma_p^2 I + Cov(g_{c,a,s,t}(t_*))\right)$$

Random draws of 1,000 samples were obtained from the distributions above for every country for a given indicator. The final estimated mean for each country was the mean of the draws. In addition, 95% uncertainty intervals were calculated by taking the 2.5th and 97.5th percentile of the sample distribution. The linear modelling process was implemented using the lmer4 package in R, and the ST-GPR analysis was implemented through the PyMC2 package in Python.

Characterizing the distribution of intake

To characterize the population distribution of intake for each dietary factor by age, sex, country, and year, in addition to mean intake, we estimated the standard deviation of intake and the shape of the distribution for each dietary factor. To generate standard deviation for each dietary factor, we first modeled the relationship between standard deviation and mean among adults aged 25 years and older in nationally representative nutrition surveys using 24-hour diet recalls:

$$\ln(\text{Standard deviation}_i) = \beta_0 + \beta_1 \times \ln(\text{Mean}_i)$$

These coefficients were then applied to draws of mean intake from ST-GPR to generate draws of standard deviation for each dietary factor. Using 24-hour recalls with multiple measurements, we also quantified the within-person variation in consumption of each dietary component and adjusted the standard deviations accordingly.

To model the distribution of each dietary risk factor, we used an ensemble technique in which a model selection algorithm is used to choose the best model for each risk factor. We drew the initial set of candidate models from commonly used probability density functions (PDFs) families. These included: beta, exponential, gamma, gumbel, inverse gamma, inverse Weibull, log-logistic, lognormal, mirrored gamma, mirrored gumbel, normal, and Weibull. We fitted each PDFs candidate family to each dataset using the method of moments (MoM), and used the Kolmogorov-Smirnov test as the measure of goodness of fit (GoF). Preliminary analysis showed that the GoF ranking of PDF families varied across

datasets for any particular risk factor and that combining the predictions of differently fitted PDF families could dramatically improve the GoF for each dataset. Therefore, we developed a new model for prediction using the ensemble of candidate models, which is a weighted linear combination of all candidate models, $\{f\}$, where a set of weights $\{w\}$ is chosen such that it is the sum of the weights equals to one and the values of the weights were determined by a second GoF criterion with its own validation process. Because of basic differences among risk factors, their distributions, and the risk attribution process, the model selection process was often slightly different for each risk factor. The details can be summarised by (1) the summary statistics for each dataset; (2) a table showing the Kolmogorov-Smirnov statistic for each candidate model and URD; (3) the criterion used for determining the overall GoF; (4) summary results of the validation process; and (5) the weights defining the final ensemble model for each dataset.

Relative risk

We obtained the relative risk of each disease endpoint per serving of the dietary components from recent dose-response meta-analyses of prospective observational studies. Considering the well-established age trend of the relative risks of metabolic risk factors for cardiovascular disease and diabetes,⁵ we conducted a literature review to identify the most important metabolic mediators for each dietary factor and used the age trend of the relative risk of that mediator(s) and the disease endpoint to estimate the age-specific relative risk for each dietary factors (Supplemental Table 3). We took the following steps to estimate the age-specific relative risk for each dietary factor:

- For each dietary factor, we conducted a systematic review of literature and identified the metabolic risks that could potentially mediate the effect of diet on the disease endpoint (Table B).
- We obtained the age-specific relative risk for each mediator and disease endpoint from a meta-analysis of pooled cohort studies.⁵
- For each dietary risk and disease endpoint, we estimated the median age at event from the meta-analysis used to estimate the relative risks of the diet-disease pair.
- We assigned the relative risk reported in the meta-analysis to age group that included median age at event.
- We estimated the percent change in the relative risks between age at event and each group for all relevant metabolic mediators and took the average of them.
- We applied the average percent change in the relative risk of metabolic mediators to the relative risk of the dietary factor at age at event and estimated the age-specific relative risk of the dietary factor.

Table B. Metabolic mediators used to determine the age trend of the effect of dietary factors on cardiometabolic outcomes.

	Body Mass Index	Total Serum Cholesterol	Fasting Plasma Glucose	Systolic Blood Pressure
Diet low in fruits	●	●	●	●
Diet low in vegetables	●	●	●	●
Diet low in legumes	●	●	●	●
Diet low in whole grains	●	●	●	-
Diet low in nuts and seeds	●	●	●	●
Diet high in red meats	●	-	●	-
Diet high in processed meats	●	-	●	●

Diet low in fiber	-	●	-	-
Diet low in seafood omega-3 fatty acids	●	-	-	●
Diet low in polyunsaturated fatty acids	-	●	●	-
Diet high in trans fatty acids	●	●	-	-

Theoretical minimum risk exposure level

To estimate the TMREL for each dietary factor, we first calculated the level of intake associated with the lowest risk of mortality from each disease endpoint based on the studies included in the meta-analyses of the dietary relative risks.⁶ Then, we calculated the TMREL as the weighted average of these numbers using the global number of deaths from each of outcome as the weight. To reflect the uncertainty of TMREL, we have assumed a uniform uncertainty distribution of 20% above and below the mean. For sodium, the evidence supporting the selection of the TMREL is uncertain. Therefore, we included in the uncertainty estimation sampling a uniform distribution of different TMRELS. The manuscript Table provides the range and distribution of the uncertainty in the TMREL.

Population attributable fraction

For each dietary risk factor, the attributable burden was estimated by comparing observed health outcomes to those that would have been observed if the past exposure level had been sustained at an optimal level, here referred to as theoretical minimum risk exposure level (TMREL). The main inputs to this analysis included the exposure level (where l is the lowest level of intake and u is highest level of intake) for each risk factor (p); the effect size of the risk factor on each disease endpoint (RR); the risk factor level associated with the lowest risk (TMREL); and the total number of deaths from each disease endpoint. Using the first three inputs, we estimated the population attributable fraction (PAF) for each risk-disease pair by age (a), sex (s), country (c), and year (t). Then, we used disease-specific (o) PAFs and mortality to calculate the total number of attributable deaths across all relevant outcomes (w):

$$PAF_{asct} = \frac{\int_l^u RR_{as}(x)P_{asct}(x)dx - RR_{as}(TMREL)}{\int_l^u RR_{as}(x)P_{asct}(x)dx}$$

$$Total\ attributable\ deaths_{asct} = \sum_{o=1}^w Death_{oasct} PAF_{oasct}$$

Assuming no correlation between dietary factors, we estimated the PAFs and number of deaths for the overall effect of all dietary factors relevant to each outcome based on the following equations:

$$PAF_{oasct} = 1 - \prod_i^n (1 - PAF_{ioasct})$$

$$Total\ attributable\ deaths_{asct} = \sum_{o=1}^w Death_{oasct} PAF_{oasct}$$

To account for potential overlaps between the effect of foods and nutrients that are associated with the same outcome (e.g., milk and calcium with colorectal cancer), we developed a mediation matrix and only accounted for the effect of either the food item or the nutrient.

Uncertainty of PAF estimates was calculated from 1,000 draws resulting from PAF calculations using the 1,000 draws of exposure estimates, 1,000 draws of relative risk estimates, and 1,000 draws of the theoretical minimum risk exposure level. The 1,000 draws of PAF estimates were multiplied by 1,000 draws of deaths and DALYs to produce 1,000 draws of attributable burden. All components of the PAF calculation analysis were assumed to be independent of each other.

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Supplemental Table 1. Epidemiological evidence supporting causality between dietary risk factors and disease endpoints.

Risk	Outcome	RCTs (n)*	RCTs with significant effect in the opposite direction (%)	RCTs with null findings (%)	Prospective observational studies (n)*	Prospective observational studies with significant association in the Case-control studies assessing the risk-outcome pair relationship (n)†‡	Case-control studies that show significant association in the opposite direction (%)	Lower limit of RR > 1.5†	Dose-response relationship	Biological plausibility	Analogy
Dietary risks											
Diet low in fruits	Lip and oral cavity cancer	0	-	-	2	0	15	0	●	●	●
Diet low in fruits	Nasopharynx cancer	0	-	-	2	0	15	0	●	●	●
Diet low in fruits	Other pharynx cancer	0	-	-	2	0	15	0	●	●	●
Diet low in fruits	Oesophageal cancer	0	-	-	5	0	-	-	●	●	●
Diet low in fruits	Larynx cancer	0	-	-	2	0	15	0	●	●	●
Diet low in fruits	Tracheal, bronchus, and lung cancer	0	-	-	22	0	-	-	●	●	●
Diet low in fruits	Ischaemic heart disease	0	-	-	9	0	-	-	●	●	●
Diet low in fruits	Ischaemic stroke	0	-	-	9	0	-	-	●	●	●
Diet low in fruits	Diabetes mellitus	0	-	-	9	0	-	-	●	●	●
Diet low in vegetables	Oesophageal cancer	0	-	-	5	0	-	-	●	●	●
Diet low in vegetables	Ischaemic heart disease	0	-	-	9	0	-	-	●	●	●
Diet low in vegetables	Ischaemic stroke	0	-	-	8	0	-	-	●	●	●
Diet low in vegetables	Haemorrhagic stroke	0	-	-	5	0	-	-	●	●	●
Diet low in legumes	Ischaemic heart disease	0	-	-	5	0	-	-	●	●	●
Diet low in whole grains	Ischaemic heart disease	0	-	-	7	0	-	-	●	●	●
Diet low in whole grains	Ischaemic stroke	0	-	-	6	0	-	-	●	●	●
Diet low in whole grains	Haemorrhagic stroke	0	-	-	6	0	-	-	●	●	●
Diet low in whole grains	Diabetes mellitus	0	-	-	10	0	-	-	●	●	●
Diet low in nuts and seeds	Ischaemic heart disease	1	0	100	6	0	-	-	●	●	●
Diet low in nuts and seeds	Diabetes mellitus	1	0	100	5	0	-	-	●	●	●
Diet low in milk	Colon and rectum cancer	0	-	-	7	0	-	-	●	●	●
Diet high in red meat	Colon and rectum cancer	0	-	-	8	0	-	-	●	●	●
Diet high in red meat	Diabetes mellitus	0	-	-	9	11	-	-	●	●	●
Diet high in processed meat	Colon and rectum cancer	0	-	-	9	11	-	-	●	●	●
Diet high in processed meat	Ischaemic heart disease	0	-	-	5	0	-	-	●	●	●
Diet high in processed meat	Diabetes mellitus	0	-	-	8	0	-	-	●	●	●
Diet high in sugar-sweetened beverages	Diabetes mellitus type 2	0	-	-	17	0	-	-	●	●	●
Diet high in sugar-sweetened beverages	Ischemic heart disease	0	-	-	4	0	-	-	●	●	●
Diet low in fibre	Colon and rectum cancer	0	-	-	15	0	-	-	●	●	●
Diet low in fibre	Ischaemic heart disease	0	-	-	12	0	-	-	●	●	●
Diet low in calcium	Colon and rectum cancer	0	-	-	13	0	-	-	●	●	●
Diet low in seafood omega-3 fatty acids	Ischaemic heart disease	17	0	94	16	0	-	-	●	●	●
Diet low in polyunsaturated fatty acids	Ischaemic heart disease	8	0	75	11	0	-	-	●	●	●
Diet high in trans fatty acids	Ischaemic heart disease	0	-	-	13	0	-	-	●	●	●
Diet high in sodium	Stomach cancer	0	-	-	10	0	-	-	●	●	●
Diet high in sodium‡	Systolic blood pressure	45	0	73	0	-	-	-	●	●	●

*The numbers in the table represent the independent RCTs and prospective observational studies evaluated the relationship between each risk-outcome pairs. If there were multiple reports from one study, they were counted as one study

†To evaluate the magnitude of the effect size for continuous risks, we evaluated the RR comparing the 75th percentile to the 25th percentile of the exposure distribution at the global level

‡The health effects of sodium on cardiovascular outcomes and chronic kidney disease were assessed through systolic blood pressure. Evidence on the direct effect of sodium on cardiovascular disease mainly comes from prospective cohort studies. Considering that, in GBD, we have only evaluated the effect of sodium mediated through systolic blood pressure, we did not present epidemiologic evidence on the direct effect of sodium on cardiovascular disease in this table. Evidence on the effect of sodium on systolic blood pressure mostly comes from randomized controlled trials. While some cohort studies evaluated the relationship between sodium and systolic blood pressure, we did not identify a systematic evaluation of these studies.

Supplemental Table 2. Citation of the epidemiological studies used to evaluate the causal relationship between dietary risk-outcome pairs in Supplemental Table 1.

Risk	Outcome	Citation/Note
Diet low in fruits	Lip and oral cavity cancer	Key TJ. Fruit and vegetables and cancer risk. <i>British Journal of Cancer</i> 2011; 104: 6–11.
Diet low in fruits	Lip and oral cavity cancer	Jin, Jian, Zhiguo Ouyang, and Zhaoyan Wang. 2014. “Association of Fruit and Vegetables with the Risk of Nasopharyngeal Cancer: Evidence from a Meta-Analysis.” <i>Scientific Reports</i> 4 (July): srep05229. doi:10.1038/srep05229.
Diet low in fruits	Lip and oral cavity cancer	Pavia, Maria, Claudia Pileggi, Carmelo GA Nobile, and Italo F. Angelillo. 2006. “Association between Fruit and Vegetable Consumption and Oral Cancer: A Meta-Analysis of Observational Studies.” <i>The American Journal of Clinical Nutrition</i> 83 (5): 1126–34.
Diet low in fruits	Nasopharynx cancer	Key TJ. Fruit and vegetables and cancer risk. <i>British Journal of Cancer</i> 2011; 104: 6–11.
Diet low in fruits	Nasopharynx cancer	American Institute for Cancer Research, and World Cancer Research Fund, eds. 2007. <i>Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective: A Project of World Cancer Research Fund International</i> . Washington, D.C: American Institute for Cancer Research.
Diet low in fruits	Nasopharynx cancer	Pavia, Maria, Claudia Pileggi, Carmelo GA Nobile, and Italo F. Angelillo. 2006. “Association between Fruit and Vegetable Consumption and Oral Cancer: A Meta-Analysis of Observational Studies.” <i>The American Journal of Clinical Nutrition</i> 83 (5): 1126–34.
Diet low in fruits	Other pharynx cancer	Key TJ. Fruit and vegetables and cancer risk. <i>British Journal of Cancer</i> 2011; 104: 6–11.
Diet low in fruits	Other pharynx cancer	American Institute for Cancer Research, and World Cancer Research Fund, eds. 2007. <i>Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective: A Project of World Cancer Research Fund International</i> . Washington, D.C: American Institute for Cancer Research.
Diet low in fruits	Other pharynx cancer	Pavia, Maria, Claudia Pileggi, Carmelo GA Nobile, and Italo F. Angelillo. 2006. “Association between Fruit and Vegetable Consumption and Oral Cancer: A Meta-Analysis of Observational Studies.” <i>The American Journal of Clinical Nutrition</i> 83 (5): 1126–34.
Diet low in fruits	Larynx cancer	Key TJ. Fruit and vegetables and cancer risk. <i>British Journal of Cancer</i> 2011; 104: 6–11.
Diet low in fruits	Larynx cancer	American Institute for Cancer Research, and World Cancer Research Fund, eds. 2007. <i>Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective: A Project of World Cancer Research Fund International</i> . Washington, D.C: American Institute for Cancer Research.
Diet low in fruits	Larynx cancer	Pavia, Maria, Claudia Pileggi, Carmelo GA Nobile, and Italo F. Angelillo. 2006. “Association between Fruit and Vegetable Consumption and Oral Cancer: A Meta-Analysis of Observational Studies.” <i>The American Journal of Clinical Nutrition</i> 83 (5): 1126–34.
Diet low in fruits	Oesophageal cancer	Liu J, Wang J, Leng Y, Lv C. Intake of fruit and vegetables and risk of esophageal squamous cell carcinoma: a meta-analysis of observational studies. <i>Int J Cancer</i> 2013; 133: 473–85.
Diet low in fruits	Tracheal, bronchus and lung cancer	Vieira AR, Abar L, Vingeliene S, et al. Fruits, vegetables and lung cancer risk: a systematic review and meta-analysis. <i>Ann Oncol</i> 2016; 27: 81–96.
Diet low in fruits	Ischaemic heart disease	Wang X, Ouyang Y, Liu J, et al. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. <i>BMJ</i> 2014; 349: g4490.
Diet low in fruits	Ischaemic stroke	Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke: a meta-analysis of prospective cohort studies. <i>Stroke</i> 2014; 45: 1613–9.
Diet low in fruits	Hemorrhagic stroke	Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke: a meta-analysis of prospective cohort studies. <i>Stroke</i> 2014; 45: 1613–9.
Diet low in fruits	Diabetes mellitus	Li M, Fan Y, Zhang X, Hou W, Tang Z. Fruit and vegetable intake and risk of type 2 diabetes mellitus: meta-analysis of prospective cohort studies. <i>BMJ open</i> 2014; 4(11): e005497.
Diet low in vegetables	Oesophageal cancer	Liu J, Wang J, Leng Y, Lv C. Intake of fruit and vegetables and risk of esophageal squamous cell carcinoma: a meta-analysis of observational studies. <i>Int J Cancer</i> 2013; 133: 473–85.
Diet low in vegetables	Ischaemic heart disease	Wang X, Ouyang Y, Liu J, et al. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. <i>BMJ</i> 2014; 349: g4490.
Diet low in vegetables	Ischaemic stroke	Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke: a meta-analysis of prospective cohort studies. <i>Stroke</i> 2014; 45: 1613–9.
Diet low in vegetables	Hemorrhagic stroke	Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke: a meta-analysis of prospective cohort studies. <i>Stroke</i> 2014; 45: 1613–9.
Diet low in legumes	Ischaemic heart disease	Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. <i>Am J Clin Nutr</i> 2014; 100: 278–88.
Diet low in whole grains	Diabetes mellitus	Aune D, Norat T, Romundstad P, Vatten LJ. Whole grain and refined grain consumption and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of cohort studies. <i>Eur J Epidemiol</i> 2013; 28: 845–58.
Diet low in whole grains	Ischaemic heart disease	Aune D, Keum N, Giovannucci E, et al. Whole grain consumption and risk of cardiovascular disease, cancer, and all cause and cause specific mortality: systematic review and dose-response meta-analysis of prospective studies. <i>BMJ</i> 2016; 353: i2716.
Diet low in nuts and seeds	Ischaemic heart disease	Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. <i>Am J Clin Nutr</i> 2014; 100: 278–88.
Diet low in nuts and seeds	Diabetes mellitus	Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. <i>Am J Clin Nutr</i> 2014; 100: 278–88.
Diet low in milk	Colon and rectum cancer	World Cancer Research Fund, American Institute for Cancer Research, Imperial College London. WCRF/AICR Systematic Literature Review Continuous Update Project Report: The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer. Oct 2010.
Diet high in red meat	Colon and rectum cancer	World Cancer Research Fund, American Institute for Cancer Research, Imperial College London. WCRF/AICR Systematic Literature Review Continuous Update Project Report: The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer. Oct 2010.
Diet high in red meat	Diabetes mellitus	Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. <i>Am J Clin Nutr</i> 2011; 94: 1088–96.

Supplemental Table 2. Citation of the epidemiological studies used to evaluate the causal relationship between dietary risk-outcome pairs in Supplemental Table 1.

Risk	Outcome	Citation/Note
Diet high in processed meat	Colon and rectum cancer	World Cancer Research Fund, American Institute for Cancer Research, Imperial College London. WCRF/AICR Systematic Literature Review Continuous Update Project Report: The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer. Oct 2010.
Diet high in processed meat	Ischaemic heart disease	Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. <i>Circulation</i> 2010; 121: 2271–83.
Diet high in processed meat	Diabetes mellitus	Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. <i>Am J Clin Nutr</i> 2011; 94: 1088–96.
Diet high in sugar-sweetened beverages	Diabetes mellitus	Imamura F, O'Connor L, Ye Z, et al. Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction. <i>BMJ</i> 2015; 351: h3576.
Diet high in sugar-sweetened beverages	Ischaemic heart disease	Xi B, Huang Y, Reilly KH, et al. Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. <i>Br J Nutr</i> 2015; 113: 709–17.
Diet low fibre	Colon and rectum cancer	World Cancer Research Fund, American Institute for Cancer Research, Imperial College London. WCRF/AICR Systematic Literature Review Continuous Update Project Report: The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer. Oct 2010.
Diet low fibre	Ischaemic heart disease	Threapleton DE, Greenwood DC, Evans CE, et al. Dietary fibre intake and risk of cardiovascular disease: systematic review and meta-analysis. <i>BMJ</i> (Clinical research ed) 2013; 347: f6879.
Diet low in calcium	Colon and rectum cancer	World Cancer Research Fund, American Institute for Cancer Research, Imperial College London. WCRF/AICR Systematic Literature Review Continuous Update Project Report: The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer. Oct 2010.
Diet low in seafood omega-3 fats	Ischaemic heart disease	Chowdhury R, Stevens S, Gorman D, et al. Association between fish consumption, long chain omega 3 fatty acids, and risk of cerebrovascular disease: systematic review and meta-analysis. <i>BMJ</i> (Clinical research ed) 2012; 345: e6698.
Diet low in polyunsaturated fats	Ischaemic heart disease	Farvid MS, Ding M, Pan A, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. <i>Circulation</i> 2014; 130: 1568–78.
Diet low in polyunsaturated fats	Ischaemic heart disease	Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. <i>PLoS Med</i> 2010; 7: e1000252.
Diet high in trans fats	Ischaemic heart disease	Mozaffarian D, Clarke R. Quantitative effects on cardiovascular risk factors and coronary heart disease risk of replacing partially hydrogenated vegetable oils with other fats and oils. <i>Eur J Clin Nutr</i> 2009; 63(Suppl 2): S22-33.
Diet high in trans fats	Ischaemic heart disease	http://www.bmjjournals.org/content/bmjj-suppl/2015/08/11/bmj.h3978.DC1/sour025275.ww2_default.pdf ; pg. 44
Diet high in sodium and high systolic blood pressure	n/a	Aburto NJ, Ziolkowska A, Hooper L, Elliott P, Cappuccio FP, Meerpol JJ. Effect of lower sodium intake on health: systematic review and meta-analyses. <i>BMJ</i> 2013; 346: f1326.
Diet high in sodium	Stomach cancer	World Cancer Research Fund, American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: AICR, 2007.
Diet high in sodium	Stomach cancer	D'Elia, Lanfranco, Giovanni Rossi, Renato Ippolito, Francesco P. Cappuccio, and Pasquale Strazzullo. 2012. "Habitual Salt Intake and Risk of Gastric Cancer: A Meta-Analysis of Prospective Studies." <i>Clinical Nutrition</i> 31 (4): 489–98. doi:10.1016/j.clnu.2012.01.003.
Diet low in nuts and seeds	Ischaemic heart disease and diabetes mellitus	Experimental evidence on the relationship of nuts with ischaemic heart disease and diabetes mellitus come from the PREDIMED trial; a randomized trial consisting of three arms: a Mediterranean diet with extra-virgin olive oil, a Mediterranean diet with nuts, and a control diet. Given that the intake of dietary factors other than nuts changed in the intervention arms of this trial, the observed effect might be fully attributable to nuts.
Diet high in sodium	Cardiovascular diseases	Evidence on the direct effect of sodium on cardiovascular disease mainly comes from prospective cohort studies. Considering that, in GBD, we have only evaluated the effect of sodium mediated through systolic blood pressure, we did not present epidemiologic evidence on the direct effect of sodium on cardiovascular disease in this table. Evidence on the effect of sodium on systolic blood pressure mostly comes from randomized controlled trials. While some cohort studies evaluated the relationship between sodium and systolic blood pressure, we did not identify a systematic evaluation of these studies.

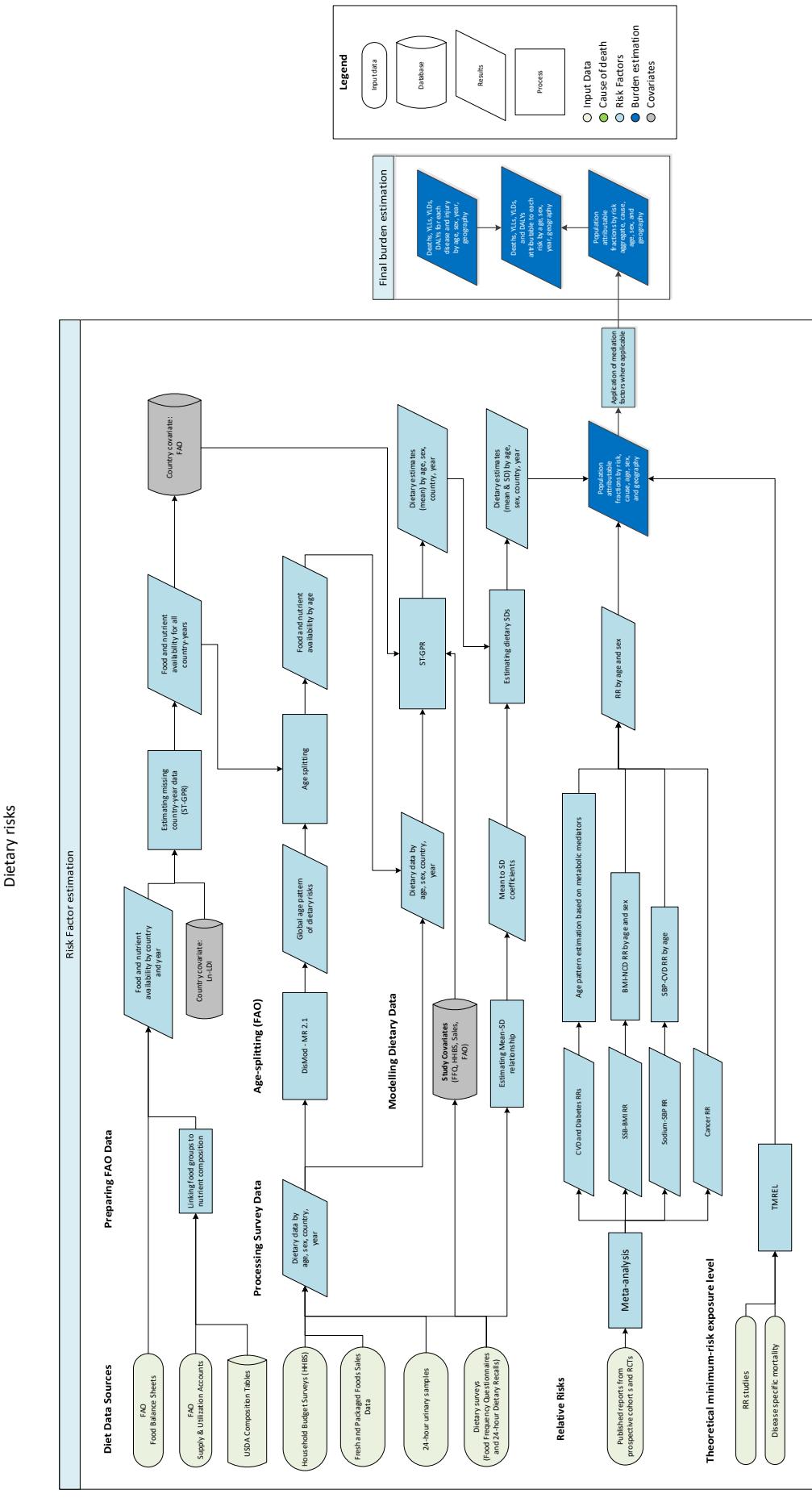
supplemental Table 3. Relative risks used by age and sex and for each outcome for all diet risk factors.

Supplemental Table 3. Relative risks used by age and sex and for each outcome for all diet risk factors.

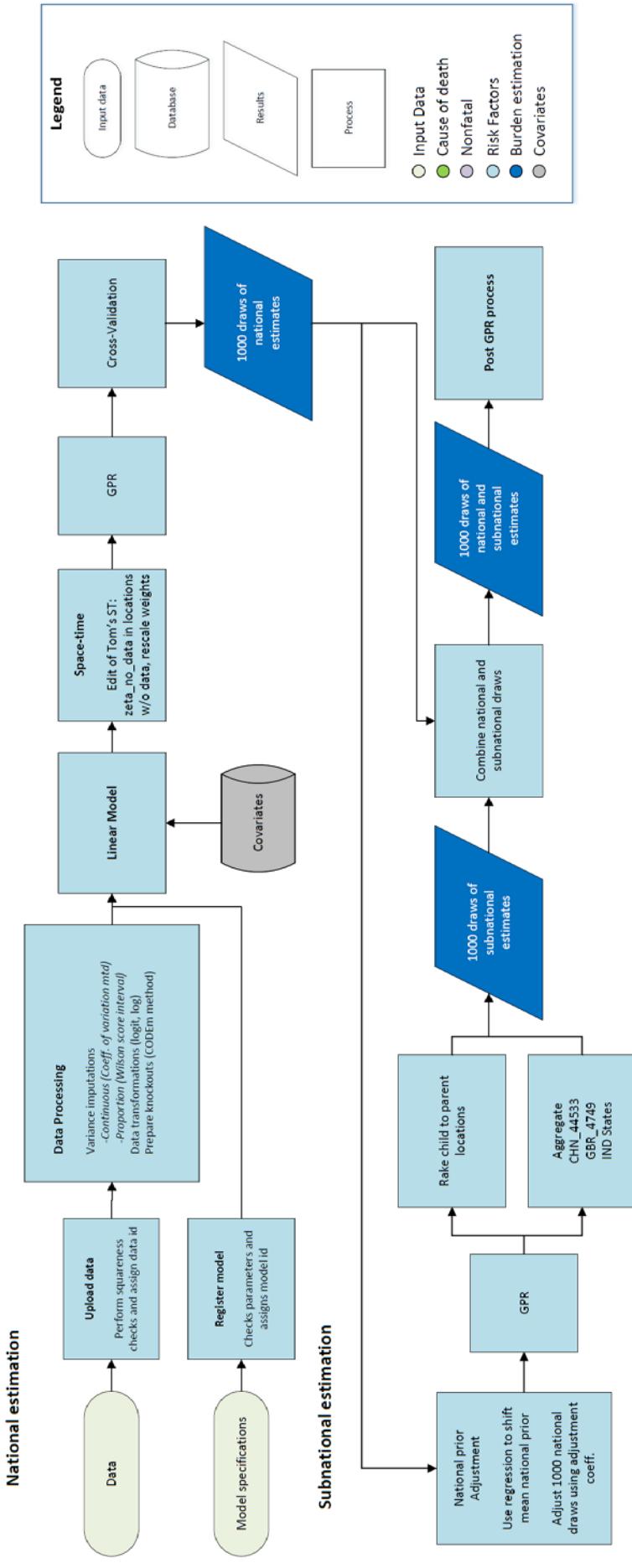
Risk - Outcome	Category / Units	Morbidity / Mortality	Sex	25-29 years	30-34 years	35-39 years	40-44 years	45-49 years	50-54 years	55-59 years	60-64 years	65-69 years	70-74 years	75-79 years	80-84 years	85-89 years	90-94 years	95+ years
Diet high in sugar-sweetened beverages																		
Ischemic heart disease																		
Diabetes mellitus type 2																		
Diet low in fibre																		
Colon and rectum cancer																		
Ischemic heart disease																		
Diet low in calcium																		
Colon and rectum cancer																		
Diet low in seafood omega-3 fatty acids																		
Ischemic heart disease																		
100 mg/day																		
Ischemic heart disease																		
Diet low in polyunsaturated fatty acids																		
Ischemic heart disease																		
Diet high in trans fatty acids																		
Ischemic heart disease																		
Diet high in sodium *																		
Non-Black, Non-Hypertensive																		
Non-Black, Hypertensive																		
Black, Non-Hypertensive																		
Black, Hypertensive																		
Stomach cancer																		

*Shifts are reported for diet high in sodium as the estimation is based on mediation through high systolic blood pressure.

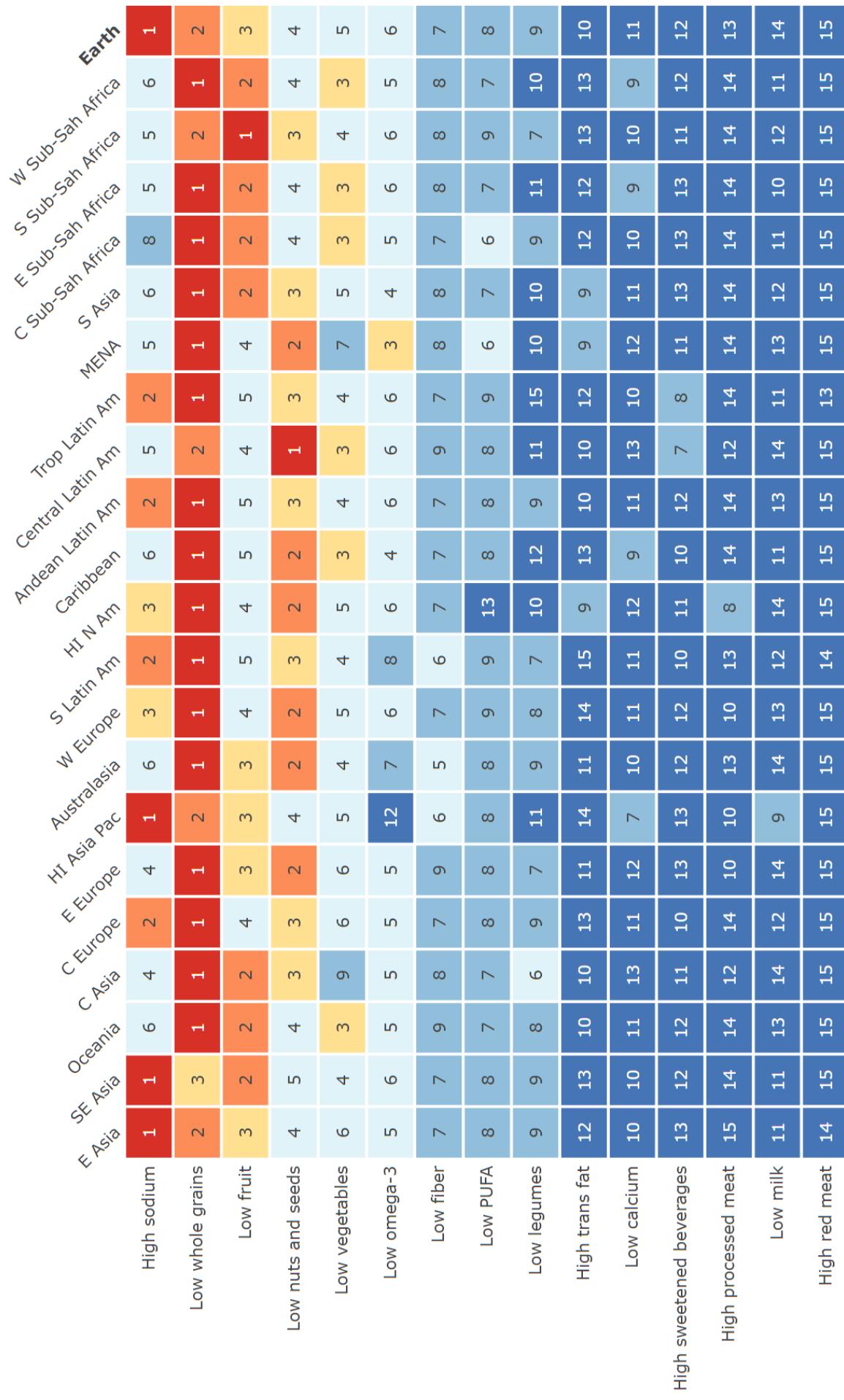
Supplemental Figure 1. Flowchart of the estimation process of dietary risk factors.



Supplemental Figure 2. Flowchart of the analytic process for Spatiotemporal Gaussian Process Regression.



Supplemental Figure 3. Ranking of dietary risk factors according to their attributable deaths at the global and regional level.



Supplemental Figure 4. Ranking of dietary risk factors according to their attributable DALYs at the global and regional level.

	Earth									
	W Sub-Saharan Africa					S Sub-Saharan Africa				
	E Sub-Saharan Africa		C Sub-Saharan Africa			S Asia		MENA		
	1	2	3	4	5	6	7	8	9	10
Low whole grains	2	1	1	1	1	1	1	1	1	1
High sodium	1	3	6	4	2	4	1	6	4	5
Low fruit	3	2	2	3	2	3	4	5	3	2
Low nuts and seeds	4	4	3	3	4	3	2	4	2	2
Low vegetables	5	5	4	9	6	6	5	5	3	4
Low omega-3	6	6	5	5	5	13	7	6	7	6
Low fiber	7	7	9	8	7	9	6	7	8	8
Low PUFA	9	8	7	7	9	8	10	15	8	8
Low legumes	8	9	8	6	8	7	12	9	12	15
High trans fat	14	13	10	11	14	11	14	15	10	13
High sweetened beverages	13	11	11	10	10	12	11	12	9	9
Low calcium	10	10	12	13	11	13	10	12	13	10
High processed meat	15	14	12	13	10	9	13	10	12	11
Low milk	11	12	13	14	12	14	8	15	13	14
High red meat	12	15	15	15	15	11	14	13	15	15

Supplemental Figure 5. Ranking of dietary risk factors according to their attributable deaths among the world's 20 most populous countries.

	China	India	USA	Indonesia	Pakistan	Brazil	Nigeria	Japan	Mexico	Philippines	Egypt	Vietnam	Germany	Iran	Congo DR	Turkey	Thailand	
High sodium	1	6	3	1	5	2	7	3	4	1	8	1	6	7	1	3	4	1
Low whole grains	2	1	1	3	1	1	4	1	2	3	2	2	1	3	1	1	2	1
Low fruit	3	2	4	2	2	5	2	1	3	3	4	5	1	5	2	1	6	5
Low nuts and seeds	4	3	2	5	4	3	5	5	2	4	1	3	4	2	4	2	4	4
Low omega-3	5	4	6	6	6	4	6	6	6	14	6	6	5	3	5	6	3	6
Low vegetables	6	5	5	4	3	4	3	2	5	5	2	4	3	11	7	5	10	3
Low fiber	7	8	8	7	8	7	9	9	6	10	7	8	8	6	7	7	7	7
Low PUFA	8	7	14	8	7	9	6	8	8	9	8	7	6	8	9	8	6	5
Low legumes	9	12	10	9	9	15	14	9	7	11	12	9	13	9	10	8	9	12
Low calcium	10	10	12	10	11	10	9	11	12	7	13	11	9	12	9	13	12	10
Low milk	11	11	13	11	12	11	10	12	14	9	14	12	10	13	11	14	13	11
High trans fat	12	9	9	12	10	12	12	10	11	13	7	13	11	4	12	12	6	12
High sweetened beverages	13	13	11	13	14	8	11	14	13	12	5	10	12	10	13	11	13	8
High red meat	14	15	15	15	13	15	15	15	15	15	15	15	15	14	15	15	15	15
High processed meat	15	14	7	14	13	14	13	13	10	10	11	14	14	15	10	14	14	14

Supplemental Figure 6. Ranking of dietary risk factors according to their attributable DALYs among the world's 20 most populous countries.

	China	India	USA	Indonesia	Pakistan	Brazil	Nigeria	Russian Federation	Japan	Philippines	Ethiopia	Vietnam	Egypt	Germany	Iran	Congo DR	Turkey	Thailand
High sodium	1	6	2	3	5	4	7	4	4	1	8	2	6	7	2	4	4	1
Low whole grains	2	1	1	2	1	1	1	3	1	2	4	1	1	1	1	1	1	2
Low fruit	3	2	3	1	2	5	2	1	2	3	3	5	2	5	1	2	5	4
Low nuts and seeds	4	3	4	5	3	2	4	5	3	4	1	3	4	2	4	3	2	5
Low vegetables	5	5	6	4	4	3	3	2	5	5	5	4	3	11	7	5	10	3
Low omega-3	6	4	7	6	6	5	6	6	15	6	6	5	3	5	6	3	5	7
Low fiber	7	8	8	7	7	8	7	8	7	11	7	8	8	6	7	7	7	6
Low legumes	8	12	11	9	9	15	14	9	7	12	14	9	14	10	10	8	9	8
Low PUFA	9	7	15	8	8	9	6	8	9	10	10	8	7	6	8	10	8	8
Low calcium	10	10	13	10	11	11	9	11	13	6	12	11	9	12	9	12	10	9
Low milk	11	11	14	12	12	13	10	12	14	9	15	12	10	13	11	14	13	11
High red meat	12	15	12	15	15	10	15	15	14	13	15	15	15	12	15	15	15	15
High sweetened beverages	13	13	9	13	14	8	11	14	11	11	2	10	12	9	13	11	11	13
High trans fat	14	9	10	11	10	12	12	10	12	13	7	13	11	4	14	13	6	12
High processed meat	15	14	5	14	13	14	13	13	10	8	9	14	13	14	15	9	14	14