

## Supporting information

### Analysis and valuation of the health and climate change co-benefits of dietary change

Marco Springmann<sup>a,1</sup>, H Charles J Godfray<sup>b</sup>, Mike Rayner<sup>a</sup>, Peter Scarborough<sup>a</sup>

<sup>a</sup>Oxford Martin Programme on the Future of Food, British Heart Foundation Centre on Population Approaches for Non-Communicable Disease Prevention, Nuffield Department of Population Health, University of Oxford. Old Road Campus, Headington, Oxford, OX3 7LF, UK.

<sup>b</sup>Oxford Martin Programme on the Future of Food, Department of Zoology, University of Oxford. Address: South Parks Road, Oxford OX1 3PS, UK.

<sup>1</sup>To whom correspondence should be addressed: Email: marco.springmann@dph.ox.ac.uk; Tel: +44(0)7460202512.

## Table of Contents

<b>SI.1</b>	<b>Supplementary description of dietary scenarios</b> .....	2
<b>SI.2</b>	<b>Supplementary health methods</b> .....	6
<b>SI.3</b>	<b>Supplementary environmental methods</b> .....	13
<b>SI.4</b>	<b>Supplementary economic methods</b> .....	15
<b>SI.5</b>	<b>Supplementary food system results</b> .....	18
<b>SI.6</b>	<b>Supplementary health results</b> .....	20
<b>SI.7</b>	<b>Supplementary environmental results</b> .....	25
<b>SI.8</b>	<b>Supplementary valuation results</b> .....	29
<b>SI.9</b>	<b>Emissions feedback from reductions in mortality</b> .....	31
<b>SI.10</b>	<b>Supplementary discussion of environmental results</b> .....	32
<b>SI.11</b>	<b>Supplementary discussion of valuation results</b> .....	33

## Supplementary methods

### SI.1 Supplementary description of dietary scenarios

We constructed four dietary scenarios to analyse the environmental and health benefits of different food consumption patterns (Table S1). The REF scenario is based on the food and agriculture projections of the Food and Agriculture Organization of the United Nations (FAO) (26) which cover 110 world regions and 32 food commodities for the years 2005/07, 2030, and 2050. We aggregated the FAO data to 16 commodities (Table S2) and 107 world regions (Table S3) to match the detail of data used in the environmental and health analysis; and we converted the FAO food availability data into estimates of food consumption by accounting for food waste at the consumption level (food waste at other stages of the production chain is accounted for in the FAO estimates used (26)) and food processing that separates the edible parts from non-edible ones (27).

The HGD scenario is based on global guidelines on healthy eating issued by WHO/FAO Expert Consultations on diet, nutrition (16) and human energy requirements (29) and on recommendations of the World Cancer Research Fund (28). In particular, it is recommended to consume at least five portions (400 g) of fruit and vegetables per day (16), less than 10% (~50 g) of total energy intake from free sugars (16), less than 30% of total energy intake from fats (16), less than 300 g of red meat a week (very little if any of which is processed) (29), and to not exceed recommended total energy intake. Global recommendations exist, e.g., also for other food groups, such as whole grains, pulses, and salt (16, 28), but those are either not quantified or cannot be implemented in light of current data availability at the global level. We also want to note that newer epidemiological evidence suggests that chronic disease incidence is associated more with the composition of fats in the diet rather than with total dietary fat intake per se (35). We therefore omitted the global recommendation on fat intake and concentrated on those regarding fruit and vegetables, red meat, sugar, and total energy intake.

The dietary recommendations have different health implications depending on the food group. The recommendation on sugar consumption influences the composition of diets (e.g. the proportion of staple foods), that on energy intake influences weight levels which, in turn, are associated with chronic disease mortality (18, 19, 29), and the recommendations on fruit and vegetable consumption and red meat consumption are direct, independent risk factors for chronic disease mortality (16, 41–46).

The dietary recommendations were implemented as follows. Consumption in regions which consumed less than the minimum amounts was adjusted upwards to meet the minimum requirement, whilst consumption that exceeded the maximum recommended intake was adjusted downward to meet that ceiling. We implemented the dietary recommendations as constraints on per-capita consumption in terms of grams per day (g/d) (except the total energy constraint), and calculated the calorie equivalents (kcal/d) by using conversion factors from

the FAO (26). Total energy intake was adjusted to recommended energy requirements (29) by varying the region-specific consumption of staple foods (grains, roots, and pulses), which preserved the regional character of food consumption.

The energy requirements of adults depend on physical activity levels and body weight (29). Country-specific energy requirements for maintaining a healthy body weight (as measured by having a body mass index (BMI) in the normal range (18.5 to 24.9 kg/m<sup>2</sup>) were calculated based on WHO/FAO and WCRF recommendations for maintaining moderate physical activity levels and age-specific energy requirements for the height composition of US Americans (see, e.g., Dietary Guidelines for Americans 2010 published by the US Department of Agriculture and the US Department of Health and Human Services) applied to the age composition of other regions (as an upper bound). The country-specific energy requirements (averaged across men and women) ranged from 2200 to 2300 kcal per capita per day.

The VGT and VGN scenarios are based on vegetarian and vegan dietary patterns. According to dietary food intake surveys in the EPIC-Oxford study (31) and in the Continuing Survey of Food Intake by Individuals (CSFII) (30), vegetarians eat about six to seven servings of fruits and vegetables per day (one to two more than typical meat eaters), and vegans consume about one more serving of fruits and vegetables than vegetarians. Vegetarians have a more than 4-times higher intake of legumes (94 g/d compared to 21 g/d), something that is in line with dietary advice given to vegetarians; and the consumption of sugars was within the limits set out by the global dietary recommendations considered above (see e.g. the Position of the Academy of Nutrition and Dietetics on Vegetarian Diets)

Based on those observations, we implemented the VGT and VGN scenarios as follows. In the VGT scenario, food consumption includes at least six portions of fruits and vegetables (6 x 80 g) per day, at least one serving (80 g) of legumes/pulses per day, no red meat or poultry (or fish), and otherwise meets the global dietary recommendations for sugar and total energy intake. In the VGN scenario, food consumption includes at least seven portions of fruits and vegetables per day, at least one serving (80 g) of legumes/pulses per day, no red meat, poultry, dairy, or eggs (or fish), and otherwise meets the global dietary recommendations for sugar and total energy intake.

It should be noted that vegetarian and vegan diets can differ from omnivorous diets on several other aspects. We did adjust the composition of staple foods depending on regional preferences, but we were not able to resolve nut consumption for example, an aspect with potential health implications (4) that was found to differ between vegetarians and non-vegetarians (30). It has also been found that vegetarian and vegans have, on average, lower energy intake and body weight than non-vegetarians (30, 31), an aspect which would impart further health benefits compared to non-vegetarians (36). However, because the HGD scenario based on global dietary guidelines already contains recommendations on energy intake for maintaining a healthy body weight (29), we do not adopt separate values of total

energy intake in the VGT and VGN scenarios, but instead use the same recommendations as the HGD scenario.

Table S1: Overview of diet scenarios

Scenarios	Features	Comments
Reference (REF)	Global energy intake per capita increases by 13% between 2005/07 and 2050; per-capita consumption (in grams) increases by 15% for fruits and vegetables, 15% for sugar, 35% for oils, 13% for red meat, 63% for poultry, 19-21% for eggs and dairy, 14% for roots and pulses, 20% for maize, and 32% for other grains, except wheat and rice whose consumption stays constant (wheat) or decreases by 5% (rice).	Based on projections by the Food and Agriculture Organization of the United Nations (FAO) (26), adjusted for food waste and food conversion into edible parts (27).
Global dietary guidelines (HGD)	Min five portions per day of fruits&veg, max 300 grams per week of red meat, less than 50 grams per day of sugar, total energy intake as recommended for moderately active population (2200-2300 kcal per day).	Based on global dietary recommendations by the World Cancer Research Fund (28) and WHO/FAO Expert Consultations on diet and nutrition (16) and human energy requirements (29).
Vegetarian (VGT)	Min six portions per day of fruits&veg, one portion per day of legumes, no red meat or poultry (or fish), sugar and total energy intake as recommended in HGD scenario.	Based on observed dietary patterns (30-31)
Vegan (VGN)	Min seven portions per day of fruits&veg, one portion per day of legumes, no red meat, poultry, dairy, or eggs (or fish), sugar and total energy intake as recommended in HGD scenario.	Based on observed dietary patterns (30-31).

*Notes: fish consumption is omitted as a category in FAO projections, and therefore not included in the dietary scenarios; however, kcal/d from fish consumption is included in an aggregated 'other' commodity.*

Table S2: Food commodities included in the analysis

Food commodities			
Vegetables	Beef	Wheat	Roots
Fruits	Pork	Rice	Pulses
Sugar	Poultry	Maize	Other commodities
Oils	Eggs	Other grains	
	Dairy		

*Notes: The 'Beef' category includes beef, veal and buffalo meat; and mutton, lamb and goat meat; the 'Other commodities' category includes spices, stimulants, fish and seafood; plantains are categorized as roots due to its nutritional similarity with roots (WCRF/AICR, 2007).*

Table S3: Regional aggregation

<i>Developing countries (DPG)</i>			
<i>Latin America and the Caribbean (LAC)</i>			
Argentina	Dominican Rep.	Honduras	Peru
Bolivia	Ecuador	Jamaica	Suriname
Brazil	El Salvador	Mexico	Trinidad and Tobago
Chile	Guatemala	Nicaragua	Uruguay
Colombia	Guyana	Panama	Venezuela
Costa Rica	Haiti	Paraguay	
Cuba			
<i>Sub-Saharan Africa (SSA)</i>			
Angola	Dem. Rep. of the Congo	Madagascar	Senegal
Benin	Eritrea	Malawi	Sierra Leone
Botswana	Ethiopia	Mali	Somalia
Burkina Faso	Gabon	Mauritania	Sudan
Burundi	Gambia	Mauritius	Swaziland
Cameroon	Ghana	Mozambique	Togo
Central African Rep.	Guinea	Namibia	Uganda
Chad	Kenya	Niger	United Rep. of Tanzania
Congo	Liberia	Nigeria	Zambia
Cote d'Ivoire		Rwanda	Zimbabwe
<i>Eastern Mediterranean (EMR)</i>			
Afghanistan	Iraq	Morocco	Turkey
Algeria	Jordan	Saudi Arabia	Yemen
Egypt	Lebanon	Syrian Arab Rep.	
Iran, Islamic Rep.	Libya	Tunisia	
<i>South Asia (SAS)</i>			
Bangladesh	Nepal	Sri Lanka	
India	Pakistan		
<i>East Asia (EAS)</i>			
Cambodia	Indonesia	Mongolia	Rep. of Korea
China	Lao People's Dem. Rep.	Myanmar	Thailand
Indonesia	Malaysia	Philippines	Viet Nam
<i>Developed countries (DPD)</i>			
Australia	Japan	South Africa	Other Western Europe
Canada	New Zealand	United States of America	Other Eastern Europe
Israel	Russian Federation	European Union (EU-27)	Central Asian Republics

## SI.2 Supplementary health methods

We analysed the health impacts associated with changes in food consumption by using a comparative risk assessment framework with three disease states and four diet and weight-related risk factors. The disease states included coronary heart disease (CHD), stroke, type 2 diabetes (T2DM), and cancer which is an aggregate of site-specific cancers. The four specific disease states accounted for about 60% of deaths from non-communicable diseases and for about 40% of deaths globally in 2010 (6). The weight-related risk factors included the prevalence of overweight ( $25 < \text{body mass index (BMI)} < 30$ ) and obesity ( $\text{BMI} > 30$ ), and the diet-related risk factors included fruit and vegetable consumption and red meat consumption which, together, accounted for more than half of all deaths that were attributable to diet-related risks in 2010 (4).

For the weight-related risk assessment, we used the scenario estimates of total energy intake to estimate changes in the prevalence of overweight and obesity based on historical relationships between those weight categories and caloric availability. For that purpose, we paired FAO food availability data for the years 1980-2009 (smoothed by a three-year moving average) with WHO data on the prevalence of overweight and obesity, and estimated two linear regression models:

$$p_{\text{overweight}}(r) = 0.02462 \cdot kcal(r) - 29.67965; R^2 = 0.49; p < 0.001$$
$$p_{\text{obesity}}(r) = 0.01000 \cdot kcal(r) - 14.98936; R^2 = 0.38; p < 0.001$$

We calculated the prevalence for overweight and obesity in 2050 by first calculating percentage changes between the prevalence of overweight and obesity in 2005/07 and 2050, as estimated via the regression equation and then applying those percentage changes to FAO baseline values for 2005/07.

We estimated the mortality and disease burden attributable to dietary and weight-related risk factors by calculating population attributable fractions (PAFs). PAFs represent the proportions of disease cases that would be avoided when the risk exposure was changed from a baseline situation to a counterfactual situation. For calculating PAFs, we used the general formula (4, 17):

$$PAF = \frac{\int RR(x)P(x)dx - \int RR(x)P'(x)dx}{\int RR(x)P(x)dx} \quad (1)$$

where  $RR(x)$  is the relative risk of disease for risk factor level  $x$ ,  $P(x)$  is the number of people in the population with risk factor level  $x$  in the baseline scenario, and  $P'(x)$  is the number of people in the population with risk factor level  $x$  in the counterfactual scenario. We assumed that changes in relative risks follow a dose-response relationship (4), and that PAFs combine multiplicatively (4, 40), i.e.  $PAF_{TOT} = 1 - \prod_i (1 - PAF_i)$  where the  $i$ 's denote independent risk factors.

In addition to changes in mortality, we also calculated the years of life lost (YLS) due to a change in dietary and weight-related risk factors. For that purpose, we multiplied each age-specific death by the life expectancy expected at that age using the Global Burden of Disease standard abridged life table (40).

We used publically available data sources to parameterize the comparative risk analysis. Population and mortality data by region and 5-year age group for the year 2050 were adopted from IIASA and the United Nations Population Division, respectively. All-cause mortality rates for 2050 were decomposed into cause-specific ones for CHD, stroke, T2DM, an aggregate of cancers, and an aggregate of all other causes by using burden of disease estimates for WHO member states in 2008, projected forward to 2050 for the dietary and weight-related risk factors focussed on here. Given that dietary and weight-related risk factors are predominantly associated with chronic disease mortality, we focused on the health implications of changes in those risk factors for adults (aged 20 and older).

We restricted the selection of relative risk parameters to meta-analyses and pooled prospective cohort studies. The diet and weight-related relative risk parameters were adopted from pooled analyses of prospective cohort studies (18, 19), and from meta-analysis of prospective cohort and case-control studies (41–44, 28, 45, 46). The cancer associations have been judged as probable or convincing by the World Cancer Research Fund, and in each case a dose-response relationship was apparent and consistent evidence suggests plausible mechanisms (28). The weight-related relative risk parameters were aggregated to the BMI categories used in this study and normalized to a risk-neutral normal weight category consistent with the epidemiological evidence (18, 19). Table S4 lists the relative risk parameters adopted in this study. The selection procedure is detailed below.

We accounted for the uncertainty related to the relative risk parameters in our mortality estimates. We approximated the error distribution of relative risks by a normal distribution, which is justified for the magnitude of errors dealt with here (<50%) (see, e.g., IPCC Uncertainty Guidelines). We then used standard methods of error propagation to calculate the uncertainty intervals associated with diet and weight-related changes in mortality.

Table S4: Relative risk parameters

Risk factor	Relative risk per cause of death			
	CHD	Stroke	Cancer	T2DM
Fruit and vegetable consumption	0.96 (0.93-0.99)	0.95 (0.92-0.97)	0.93 (0.84-0.99)*	0.96 (0.92-0.99)
Red meat consumption	1.25 (1.21-1.29)	1.10 (1.05-1.15)	1.01 (1.00-1.05)*	1.15 (1.07-1.24)
Overweight	1.31 (1.24-1.39)	1.07 (0.73-1.59)	1.10 (1.04-1.17)	1.54 (1.42-1.68)
Obese	1.78 (1.64-1.92)	1.55 (1.14-2.11)	1.40 (1.30-1.50)	7.37 (5.16-10.47)

\* global average, actual relative risk is region-specific.

Sources: Dauchet et al (2005, 2006), Micha et al (2010), Chen et al (2013), WCRI/AIC (2007), Li et al (2014), Feskens et al (2013), Prospective Studies Collaboration (2009), Berrington de Gonzales et al (2010).

## **Relative risk parameters**

### *Weight-related risk parameters*

We inferred the parameters describing relative mortality risk due to weight categories from two large, pooled analyses of prospective cohort studies (18, 19). We concentrated on four causes of death: ischaemic/coronary heart disease (CHD), stroke, cancers, and type-2 diabetes mellitus (T2DM). We adopted the relative risks for CHD, stroke, and T2DM from the Prospective Studies Collaboration (19), which analysed the association between BMI and mortality among 900,000 persons in 57 prospective studies that were primarily designed to evaluate risk factors for cardiovascular disease; and we adopted the relative risks for cancer from Berrington de Gonzalez and colleagues (18) who examined the relationship between BMI and mortality in a pooled analysis of 19 prospective studies which included 1.46 million adults and which were predominantly designed to study cancer.

From each study, we adopted the relative risk rates for lifelong non-smokers to minimize confounding and reverse causality, and, to increase comparability, we normalized the relative-risk schedule to the lowest risk which, in each case corresponded to a body-mass index (BMI) of 22.5-25. We then used the number of cause-specific deaths to aggregate the BMI intervals of 2.5 that have been used in the studies to the WHO classification of BMI ranges.

### *Dietary risk parameters*

Dietary risks have been the leading risk factors for death globally in 2010 (4). The Global Burden of Disease Study included 14 different components as dietary risks, such as not eating enough fruit, nuts and seeds, vegetables, whole grains, and omega-3s and eating too much salt and processed meat (4). In this study, we focused on changes in the consumption of fruits, vegetables, and meat. Those categories constituted about two thirds of the total dietary risk in 2010 (excluding potential double counting, e.g. of fibre found in vegetables and sodium found in processed meat) (4), and country-level trends and data are available for most countries worldwide. We adopted relative-risk parameters for developing specific diseases from recent meta-analyses of existing studies.

### *Meat consumption and cardiovascular disease*

The relative risks of coronary heart diseases due to meat consumption were adopted from Micha and colleagues (41). Their comprehensive systematic review and meta-analysis of the relationship between meat consumption (processed, red, and total meat) and cardiovascular diseases (coronary heart disease (CHD), diabetes mellitus, and stroke) included 20 studies (17 prospective cohorts and 3 case-control studies) with 1,218,380 individuals from 10 countries. However, analyses of specific subcategories, e.g. total meat consumption and stroke, included significantly less studies. The results show positive associations between consumption of processed and total meat and the incidence of CHD, diabetes mellitus, and stroke. Since the publication of Micha et al (41), updated reviews of the association between meat consumption



and stroke have become available. We therefore only adopted the estimates for the association between meat consumption and coronary heart disease from the Micha et al study. We adopted the findings for total meat indicating that consumption of 100 g per day increases CHD risk by 25% (RR=1.25; 95% CI, 1.21 to 1.29). The estimate is based on data from 4 prospective cohort studies; one extremely positive finding from a case-control study was excluded in the estimate (41).

The relative risk of stroke due to meat consumption was adopted from Chen et al (42) which, for stroke, provided an updated meta-analysis of Micha et al (41) containing five large independent cohort studies (compared to two in Micha et al (41)). Chen et al (42) found that consumption of red and/or processed meat increases the risk of stroke, in particular, ischemic stroke. Their dose-response analysis of the primary studies showed that the risk of stroke increased significantly by 10% for each 100 g per day increment in total meat consumption (RR=1.10; 95% CI, 1.05–1.15), by 13% for each 100 g per day increment in red meat consumption (RR=1.13; 95% CI, 1.03–1.23) and by 11% for each 50 g per day increment in processed meat consumption (RR=1.11; 95% CI, 1.02–1.20), with low study heterogeneity. We adopted the estimate for total meat consumption.

#### *Meat consumption and diabetes*

The relative risk of T2DM due to meat consumption was adopted Feskens et al (46) who updates the meta-analysis of Micha et al (41) for T2DM. For total meat consumption, Feskens et al (46) included findings from 14 separate cohorts result, resulting in a pooled RR of 1.15 per 100 g (RR=1.15; 95% CI, 1.07-1.24), indicating that for each 100 g of total meat consumed, the risk of T2DM increases by 15 %. For red meat, the overall RR based on 14 individual studies was 1.13 per 100 g (95% CI 1.03–1.23), and for processed meat, the summary estimate of 21 separate cohorts was 1.32 per 50 g (95 % CI 1.19–1.48). We adopt the total meat estimate which includes red and processed red meat.

#### *Meat consumption and cancer*

The association between meat consumption and cancer was reviewed in the Second Expert Report "Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective" published in 2007 by the World Cancer Research Fund together with the American Institute for Cancer Research (28). The report is based on reviews and meta-analysis of over 7,000 scientific studies published on cancer prevention. It was the outcome of a 5-year project which involved a panel of 21 leading scientists and 9 research centres around the world.

With respect to meat, the report concluded that (28): red and processed meats are convincing causes of colorectal cancer; there is substantial amount of evidence, with a dose-response relationship apparent from case-control studies (red meat) and cohort studies (processed meat); there is evidence (red meat) and strong evidence (processed meat) for plausible mechanisms operating in humans. The report also noted that there is limited evidence

suggesting that red meat is a cause of cancers of the oesophagus, lung, pancreas and endometrium; and that processed meat is a cause of cancers of the oesophagus, lung, stomach and prostate.

We followed the conclusions of the expert report and its updates that highlighted a convincing causal link between meat consumption and colorectal cancer and adopted the following estimate: consumption of 100 g/day of red and processed meat increases the risk of colorectal cancer by 16% (RR=1.16, CI=1.04-1.30). We aggregated the estimate to region-specific relative risks for all cancers by weighing it by the ratio of regional deaths due to colorectal cancer to all cancer deaths in that region. Globally, this resulted in a relative risk of cancer of RR=1.01.

#### *Fruit and vegetable consumption and cardiovascular disease*

The relative risks of stroke and CHD due to fruit and vegetable consumption were adopted from Dauchet and colleagues (43, 44). Dauchet et al (44) conducted a meta-analysis for CHD and its association with fruit and vegetable consumption. The analysis included nine prospective cohort studies that consisted of 91,379 men, 129,701 women, and 5,007 CHD events. Pooled relative risks showed that CHD was decreased by 4% [RR (95% CI): 0.96 (0.93–0.99)] for each additional portion of 106 g per day of fruit and vegetable intake and by 7% [0.93 (0.89–0.96)] for fruit intake. We adopted the estimate for the aggregate of fruit and vegetable consumption, i.e. RR=0.96 (0.93-0.99).

Dauchet et al (43) undertook a similar meta-analysis for stroke. The analysis included seven cohort studies with 90,513 men, 141,536 women, and 2,955 strokes. Pooled relative risks showed that the risk of stroke was decreased by 11% (RR 95% CI: 0.89 [0.85 to 0.93]) for each additional portion of 106 g per day of fruit, by 5% (RR: 0.95 [0.92 to 0.97]) for fruit and vegetables, and by 3% (RR: 0.97 [0.92 to 1.02]) for vegetables. The study found that the association between fruit or fruit and vegetables and stroke was linear, suggesting a dose-response relationship. We adopted the estimate for the aggregate of fruit and vegetable consumption, i.e. RR=0.95 (0.92-0.97).

#### *Fruit and vegetable consumption and diabetes*

The relative risk of diabetes due to fruit and vegetable consumption was adopted from Li et al (45). In their meta-analysis, Li et al (45) included 10 prospective cohort studies. Eleven comparisons from nine studies reported an association between fruit intake and risk of T2DM, with 22 995 T2DM outcomes and 424 677 participants. Overall, fruit intake was inversely associated with risk (relative risk 0.93, 95% CI 0.88 to 0.99). Dose-response analysis indicated that a 1 serving/day (106 g/d) increment of fruit intake was associated with a 6% lower risk of T2DM (relative risk 0.94, 95% CI 0.89 to 1.00). Seven comparisons from six studies reported an association between green leafy vegetables (GLV) intake and risk of T2DM, with 19 139 T2DM outcomes and 251 235 participants. Overall, GLV intake was inversely associated with risk (relative risk 0.87, 95% CI 0.81 to 0.93). Dose-response

analysis indicated that a 0.2 serving/day (0.2 x 106 g/d) increment of GLV intake was associated with a 13% lower risk of T2D (relative risk 0.87, 95% CI 0.76 to 0.99). We weighted the GLV-related RR per 0.2 servings with a risk neutral RR per 0.8 servings of other vegetables, and with the fruit-related RR. This yielded a RR of 0.96 (0.92-0.99).

### *Fruit and vegetable consumption and cancer*

The relative risks of various cancers due to fruit and vegetable consumption were adopted from the 2007 expert report of the World Cancer Research Fund and the American Institute for Cancer Research (28). The expert report concluded that:

“[N]on-starchy vegetables and also fruits probably protect against cancers of the mouth, larynx, pharynx, oesophagus, and stomach, and that fruits also probably protect against lung cancer. The case that vegetables, fruits, and pulses (legumes) may be protective against cancers of some sites is supported by evidence on foods containing micronutrients found in these and other plant foods. Foods containing carotenoids probably protect against cancers of the mouth, pharynx, larynx, and lung; foods containing beta-carotene and also vitamin C probably protect against oesophageal cancer; foods containing selenium and also lycopene probably protect against prostate cancer; and foods containing folate probably protect against pancreatic cancer. [...] [It was also found that] foods containing dietary fibre, found in plant foods (particularly when in whole or relatively unprocessed forms), probably protect against colorectal cancer.”

We adopted the following relative risk parameters for which the expert report indicated a substantial amount of consistent evidence for plausible mechanisms and a dose-response relationship:

Cancers of the mouth, pharynx, and larynx:

- Consumption of non-starchy vegetables reduces the risk of cancers of the mouth, pharynx, and larynx by 28% per 50-g serving per day (RR=0.72; 95% CI: 0.63-0.82).
- Consumption of fruits reduces the risk of cancers of the mouth, pharynx, and larynx by 28% per 100-g serving per day (95% CI: 0.59-0.87).
- We adopted the simple average of vegetable and fruit consumption for the relative risk of cancers of the mouth, pharynx, and larynx, i.e. RR=0.72 (0.61-0.85).

Oesophageal cancer:

- Consumption of raw vegetables reduces the risk of oesophageal cancer by 31% per 50-g serving per day (RR=0.69; 95% CI: 0.58-0.83).
- Consumption of fruits reduces the risk of oesophageal cancer by 44% per 100-g serving per day (RR=0.56; 95% CI: 0.42-0.74).
- We adopted the simple average of vegetable and fruit consumption for the relative risk of oesophageal cancer, i.e. RR=0.63 (0.50-0.79).

Stomach cancer:

- Consumption of non-starchy vegetables reduces the risk of stomach cancer by 30% per 100-g serving per day (RR=0.70; 95% CI: 0.62-0.79).

[Estimates of green-yellow vegetables yield  $RR=0.59$  (0.46-0.75) per 100 g/d; green, leafy vegetables yield  $RR=0.43$  (0.24-0.77) per 50 g/d; and raw vegetables yield  $RR=0.5$  (0.38-0.65) per 100 g/d]

- Consumption of fruits reduces the risk of stomach cancer by 33% per 100-g serving per day ( $RR=0.67$ ; 95% CI: 0.59-0.76).
- We adopted the simple average of vegetable and fruit consumption for the relative risk of stomach cancer, i.e.  $RR=0.69$ .

Lung cancer:

- Consumption of fruits reduces the risk of lung cancer by 6% per 80-g serving per day ( $RR=0.94$ ; 95% CI: 0.90-0.97).
- We adopted the simple average of vegetable and fruit consumption for the relative risk of lung cancer (assuming no effect of vegetable consumption,  $RR=1$ ), i.e.  $RR=0.97$ .

Overall relative risk:

- We aggregated the cause-specific relative-risk estimates to region-specific all-cancer estimates by weighing each risk by the ratio of regional deaths due to the specific cancer divided by all cancer deaths in that region. Globally, this yielded an aggregate all-cancer risk of  $RR=0.93$ .

### SI.3 Supplementary environmental methods

We calculated the environmental impacts associated with the different dietary scenarios by using commodity-specific GHG emissions factors. The emissions factors are adopted from a recent meta-analysis of life-cycle analyses (LCAs) which estimated the ‘cradle to farm gate’ emissions of different food items (12) and adjusted to account for productivity improvements that would reduce GHG intensity over time based on output (3).

The meta-analysis included 120 publications with 555 LCAs of GHG emissions from 82 different food items (12). The system boundaries of those LCAs included emissions from pre-farm activities, such as fertilizer and feed production, as well as infrastructure construction, but excluded emissions from land-use change and post-farm-gate activities, such as processing, packaging, and transportation to the household. We adopted the emissions factors for 15 food commodities corresponding to the commodity detail chosen in this study (Table S5). We did not account for GHG emissions related to the consumption of fish and seafood, because those food groups are not resolved in the projections of food demand used in this study (26).

To obtain estimates of the total GHG emissions associated with each diet scenario, we multiplied commodity-specific gross consumption estimates (i.e. without deducting food waste) (measured in kcal/d/cap) by the corresponding emissions factors (measured in g/kcal) and population numbers, and converted the result to GHG emissions per region per year. We used the standard deviation reported in the meta-analysis (12) to calculate error bounds for our estimates.

Significant improvements in the GHG intensity of cereals and livestock products occurred over the period 1961-2010 due to a move from less to more intensive production systems (3). To account for future productivity improvements, we used the historical changes in GHG emissions intensity per change in output between 1961-2010 for beef, pork, poultry, eggs, dairy, and rice (3) (excluding small absolute increases in the emissions intensity for wheat and maize) and applied those changes to the changes in production between the base year of 2005/07 and 2050. The resulting reductions in GHG emissions intensity were 14.9% for beef, 5.8% for pork, 9.8% for poultry, 10.0% for eggs, 22.5% for dairy, and 6.2% for rice (Table S6). It is possible that part of those efficiency improvements could be offset by increased emissions from land-use change that may be associated with increased production. However, our system boundary, given by the baseline emissions intensities (12), does not include emissions from land-use change. We therefore did not attempt to quantify such offsetting effects, but we discuss potential emissions feedbacks in the discussion section of the main text.

For contextualization in terms of climate change, we calculated the ratio of food-related GHG emissions to GHG emissions from all sources. We used historical data for the base year 2005/07, and we adopted emissions estimates from the International Panel on Climate Change (IPCC) for an emissions pathway consistent with a likely (>66%) chance of limiting global

temperature increase to below 2°C (32), an aim that was agreed to by the international community in 2010 as part of the Cancun Agreements to the United Nations Framework Convention on Climate Change.

Table S5: GHG emissions factors by commodity (adopted from Tilman and Clark, 2014).

Food item	Carbon-eq emissions (mean and standard deviation (std))			
	g/kcal	g/kcal_std	g/serving	g/serving_std
Sugar	0.02	0.00004	0.9	0.002
Roots	0.03	0.02	0.84	0.04
Pulses	0.02	0.002	1.9	0.22
Maize	0.03	0.004	3	0.36
Wheat	0.06	0.009	5.2	0.86
Other grains	0.05	0.005	5.4	0.46
Fruits*	0.12	0.03	7.75	1.85
Rice	0.14	0.02	14	2.1
Vegetables	0.68	0.25	14	3.5
Oils	0.16	0.04	20	5.4
Eggs	0.59	0.03	24	1
Poultry	1.3	0.05	52	2.1
Pork	1.6	0.1	61	3.6
Dairy	0.52	0.04	74	2.5
Beef	5.6	0.41	330	18
Other commodities**	1.2	0.325	27	7.25

\* The GHG coefficient for fruits is taken as average between temperate and tropical fruits (0.10 g/kcal and 0.14 g/kcal, respectively).

\*\* includes stimulants, spices, and seafood; we don't account for changes in that category, and therefore also omit this item in the main scenarios' GHG accounting.

Table S6: Improvements in GHG emissions intensity by commodity. Future changes in emissions intensity were calculated based on historical changes in emissions intensity per change in output. Past changes in emissions intensity and production were adopted from Tubiello et al. (2014).

Food item	Change in production (%)		Change in emissions intensity (%)	
	1961-2010	2005/07-2050	1961-2010	2005/07-2050
Beef	132	73	-27	-15
Pork	335	43	-45	-6
Poultry	1,011	123	-80	-10
Eggs	359	63	-57	-10
Dairy	110	65	-38	-22
Rice	230	29	-49	-6

## SI.4 Supplementary economic methods

We estimated the economic benefits of changes in diets by monetizing the changes in GHG emissions and health. For monetizing the GHG emissions, we used estimates of the social cost of carbon (SCC) which represents the monetized damages associated with an incremental increase in carbon emissions. The values adopted are based on a comprehensive integrated-assessment modelling exercise facilitated by technical experts from several US agencies (20). The interagency group selected four SCC values for use in regulatory analyses. Three values are based on the average SCC across three integrated-assessment models and socio-economic and emissions scenarios at discount rates of 2.5, 3, and 5 percent. The fourth value is the SCC value for the 95th percentile at a 3 percent discount rate, which is meant to represent the higher-than-expected economic impacts from climate change further out in the tails of the SCC distribution. For the year 2050, the SCC estimates are 27, 71, 98, and 221 USD/tCO<sub>2</sub> for discount rates of 5, 3, 2.5 percent, and the 95th percentile at a 3 percent discount rate.

For monetizing the health impacts, we relied on two complementary costing methods, the value of statistical life approach and the cost-of-illness approach. The value of statistical life (VSL) is a measure for the willingness to pay for a mortality risk reduction defined as the marginal rate of substitution between money and mortality risk in a defined time period (22). The VSL does not represent the value of life itself, but rather the value of small risks to life which can be estimated either from market decisions that reveal the implicit values reflected in behaviour (revealed preference studies), or by using surveys which elicit respondents' willingness to pay for small reductions in mortality risks directly (stated preference studies).

We based our valuation on a comprehensive global meta-analysis of stated preference surveys of mortality risk valuation undertaken for the Organisation for Economic Co-operation and Development (OECD) (21). Following OECD recommendations, we adopted a VSL base value for the EU-27 of USD 3.5 million (1.75-5.25 million) and used the benefit-transfer method to calculate VSLs in other regions (22). In the benefit-transfer method, the VSL base value is adjusted by income ( $Y$ ) subject to an elasticity of substitution ( $\beta$ ):

$$VSL_r = VSL_{base} \left( \frac{Y_r}{Y_{base}} \right)^\beta$$

Following OECD recommendations, we used GDP per capita adjusted for purchasing power parity (PPP) as a proxy for income, and we adopted an elasticity of 0.8 for benefit transfers to high-income countries and an elasticity of 1.0 for benefit transfers to low and middle-income countries (22). Baseline data on GDP per capita were sourced from the World Bank Development Indicator database, and GDP projections for 2050 were based on projections by the OECD for a "Middle of the Road" socio-economic development pathway (SSP Database, available at: <https://tntcat.iiasa.ac.at/SspDb>). In line with World Bank methodology, we adjusted the income classification of countries depending on their GDP per capita (adjusted for purchasing power parity) in each period.

The error in applying the benefit-transfer method is expressed by the low and high range of the VSL base value. We estimated the uncertainty in the monetized health impacts by using standard methods of error propagation.

The current OECD guideline (22) recommends against differentiating the VSL for different age groups. Our main valuation therefore consists of applying the region-specific VSL estimates directly to changes in mortality. However, since mostly older people are affected by changes in diets through changes in chronic-disease mortality, we also monetized the health impact in terms of years of life lost by using the value of statistical life year (VSLY). We calculated the VSLY for each region by expressing the VSL as the discounted net present value of the VSLY throughout a lifetime:

$$VSL_r = \sum_{age} \frac{VSLY_r}{(1 + dr)^{age}}$$

where we used a discount rate ( $dr$ ) of 3% and a maximum age of 86 adopted from the Global Burden of Disease standard lifetable. We used non-linear programming (GAMS, NLP solver) to numerically solve the equation above for the VSLYs per region.

Our second costing method relied on the cost-of-illness approach which captures the direct and indirect costs associated with treating a specific disease, including medical and health-care costs (direct), and costs of informal care and from lost working days (indirect) (see e.g. ref (23)). Because global cost-of-illness studies of cardiovascular disease and cancer do not exist at present, we again used a cost transfer method, which is similar to the benefit transfer method used in the value of statistical life approach and which has been used in other global assessments of the global economic burden of cardiovascular disease and cancer (34).

We based our cost-of-illness estimation on a comparative assessment of the economic burden of cardiovascular diseases (23, 24) and cancer (25) across the European Union. We adopted the total cost estimate associated with CHD, stroke, and cancer for the EU in 2009, which included direct costs (healthcare expenditure, health service utilization, expenditure on medication) and indirect costs (opportunity costs of informal care, productivity costs due to mortality and morbidity). We calculated the costs per death due to CHD, stroke, and cancer using EU-wide death-by-disease statistics for the same year (24). The costs per death by disease ( $d$ =CHD, stroke, cancer) in the EU and other regions ( $r$ ) in the year 2050 were then estimated by scaling the EU base values for direct and indirect costs by the ratio of health expenditure per capita for direct costs, and by the ratio of GDP per capita (adjusted for purchasing power parity) for indirect costs:

$$COI_{r,d}^{direct} = COI_{EU,d}^{direct} \left( \frac{hexp_r}{hexp_{EU}} \right)$$

$$COI_{r,d}^{indirect} = COI_{EU,d}^{indirect} \left( \frac{GDP_r}{GDP_{EU}} \right)$$



Productivity losses due to morbidity and mortality, which are a part of the indirect costs, were only included for deaths occurring among those of working age (< 65 years old).

Baseline data on GDP per capita and health expenditure per capita were sourced from the World Bank Development Indicator database, and GDP and population projections for 2050 were based on projections by the OECD and IIASA for a “Middle of the Road” socio-economic development pathway (SSP Database, available at: <https://tntcat.iiasa.ac.at/SspDb>).

Projections for health expenditure per capita in 2050 are based on own projections. For those, we linearly regressed past health expenditure per capita on past GDP per capita for the period 1995-2013, and then used the relationships to project future health expenditure per capita based on future GDP trajectories. Most regions exhibited a good fit to the linear regression model (p-values smaller than 0.01 (99% significance level) for n=141 out of 174 regions; 96% with p-values smaller than 0.001). For regions that did not exhibit a good statistical fit (p-values larger than 0.01; n=33 out of 174 regions), we used WHO estimates of health expenditure as percentage of GDP in 2010, and calculated future health expenditure by using future GDP values, holding the percentage of health expenditure to GDP constant. (This is likely to have yielded a conservative estimate as global health expenditure as a percentage of GDP increased by approximately 7% in each of the last three five-year periods.)

We added a transfer error (uncertainty) of 30% to the cost-of-illness estimates based on sub-sample comparison. A comparison of the costs per disease death for individual EU countries in 2009 between estimates based on the cost transfer method and the original estimates indicated a population-weighted deviation of 8-14% in total costs, and of 8-31% in direct costs.

For diabetes, globally comparable health expenditure estimates were available (Zhang et al., 2010) which we adopted directly and adjusted for potential double-counting of cost components by using incremental cost estimates (Köster et al., 2011; ADA, 2013). Diabetes is associated with a high risk of developing complications and co-morbidities, such as CHD, stroke, blindness, renal failure, and amputation, which makes cost coding difficult and diabetes-related healthcare spending difficult to determine precisely. We adopted estimates of diabetes-related deaths and diabetes-related healthcare expenditure by country from the Diabetes Atlas (6<sup>th</sup> Edition), and used those to calculate diabetes-related healthcare expenditure per death by region. To avoid double counting of cost components, we adjusted the estimates of diabetes-related health expenditure produced for the Diabetes Atlas by estimates of the incremental cost components that are specifically attributable to diabetes. We adopted an incremental-cost ratio of 50% which is the average of available incremental cost estimates (46-57%) (Köster et al., 2011; ADA, 2013). Finally, we projected the healthcare expenditure attributable to diabetes death forward to 2050 by multiplying the 2013 values by the region-specific increase in healthcare spending per capita between 2013 and 2050. Based on earlier version of the Diabetes Atlas (Zhang et al., 2010), we assumed an uncertainty interval of 50% around the mean estimates.

# Supplementary results

## SI.5 Supplementary food system results

According to our analysis, the adherence to global dietary guidelines is weak in many regions and projected to remain weak in the future (Figure S1). In the base year of 2005/07, the majority of regions did not meet dietary recommendations for the consumption of fruits and vegetables (90 out of 107 regions covered in the analysis) and sugar (65), and more than a third of all regions did not meet dietary recommendations for red meat (41), and exceeded recommendations for total energy intake (47). In the reference scenario in 2050, more regions were projected to meet recommended minimum intakes of fruits and vegetables (with 74 regions not meeting recommendation), but less regions were projected to meet the recommended maximum intakes of red meat (65), sugar (74), and total energy intake (91). As a consequence, large changes in the food system would be necessary to achieve the dietary patterns considered here (Table S6).

Figure S1: Number of regions (out of a total of 107) that do not meet global dietary guidelines in the years 2005/07 and 2050. The dietary recommendations include min 5 portions of fruits and vegetables per day (16), less than 50 g of sugar per day (16), max 300 g of red meat per week (28), and 2200-2300 kcal per day depending on age and sex composition of the population (29).

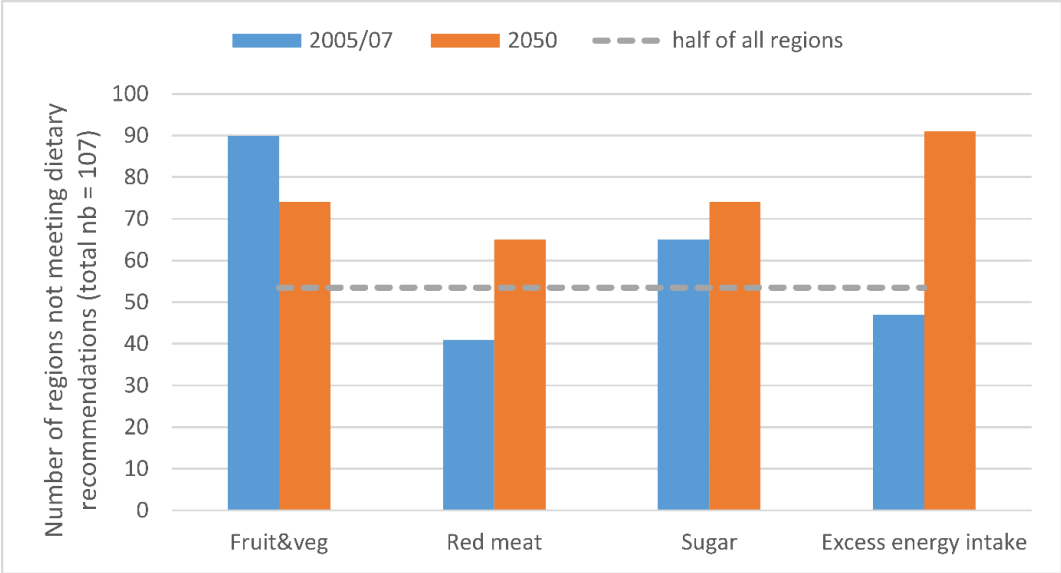


Table S7: Food consumption in the REF scenario (lower columns) by region, and in the alternative diet scenario in aggregate (upper columns). Food consumption by food groups is given in grams per day, except for total energy intake which is given in kilo-calories per day. Red shading indicates that dietary recommendations (listed under HGD) are not met in aggregate.

Region	Total kcal	Red Meat	Fruit & Veg	Sugar	Oils	Poultry	Eggs	Dairy	Pulses	Staples
<i>2005/07(WLD)</i>	2,318	66	341	59	32	31	22	215	17	453
<i>HGD(WLD)</i>	2,243	33	492	45	43	50	26	260	16	391
<i>VGT(WLD)</i>	2,243	0	545	45	43	0	26	260	80	352
<i>VGN(WLD)</i>	2,243	0	606	45	43	0	0	0	80	436
WLD	2,624	75	393	68	43	50	26	260	19	481
DPD	2,737	135	465	87	54	89	35	549	8	381
DPG	2,603	64	380	64	41	43	24	205	21	500
HIC	2,773	145	482	85	59	95	35	570	9	354
MIC	2,625	104	412	91	39	68	35	483	6	464
LAC	2,782	116	355	110	44	100	33	366	28	417
SSA	2,387	31	170	41	37	12	7	101	37	719
EMR	2,599	39	520	79	39	58	20	257	17	461
SAS	2,570	18	245	76	45	30	15	316	25	441
EAS	2,744	126	638	50	40	54	45	103	4	432

## SI.6 Supplementary health results

Figure S2: Total number of avoided deaths (left axis) and years of life saved (right axis) in 2050 compared to 2005/07 by risk factor ( $\Delta$ weight: change in prevalence of overweight and obesity combined,  $\Delta$ C(fruit&veg): change in fruit and vegetable consumption;  $\Delta$ C(red meat): change in red meat consumption; Total: all risk factors combined; Total YLS: all risk factors combined for the measure of years of life saved).

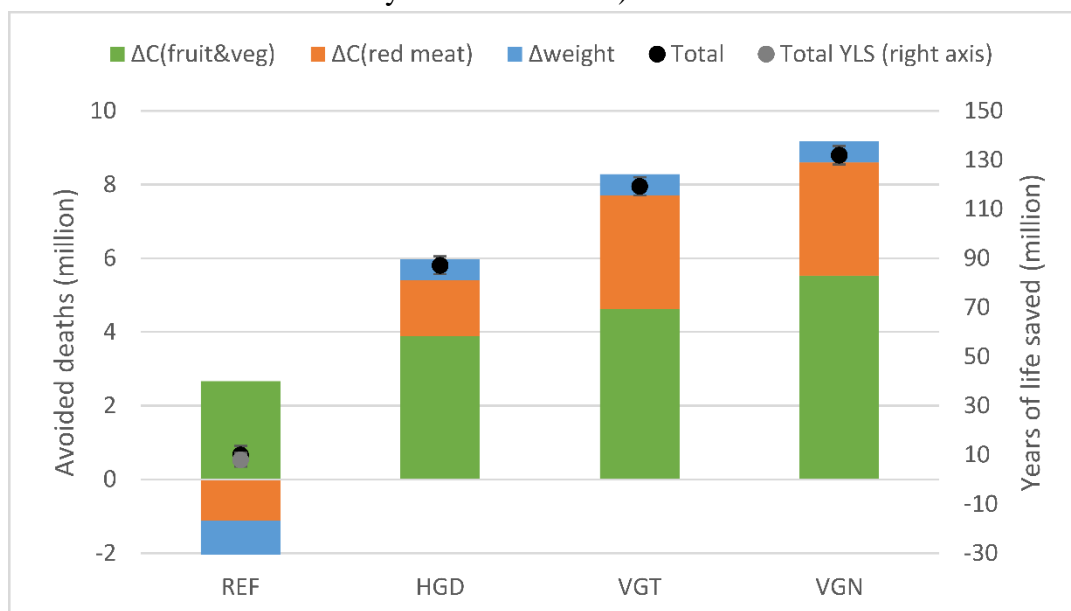


Figure S3: Total number of avoided deaths (left axis) and years of life saved (right axis) in 2050 compared to 2005/07 by cause of death: coronary heart disease (CHD), stroke, cancer, type 2 diabetes (T2DM), and all causes combined (“Total” for avoided deaths, and “Total YLS” for years of life saved).

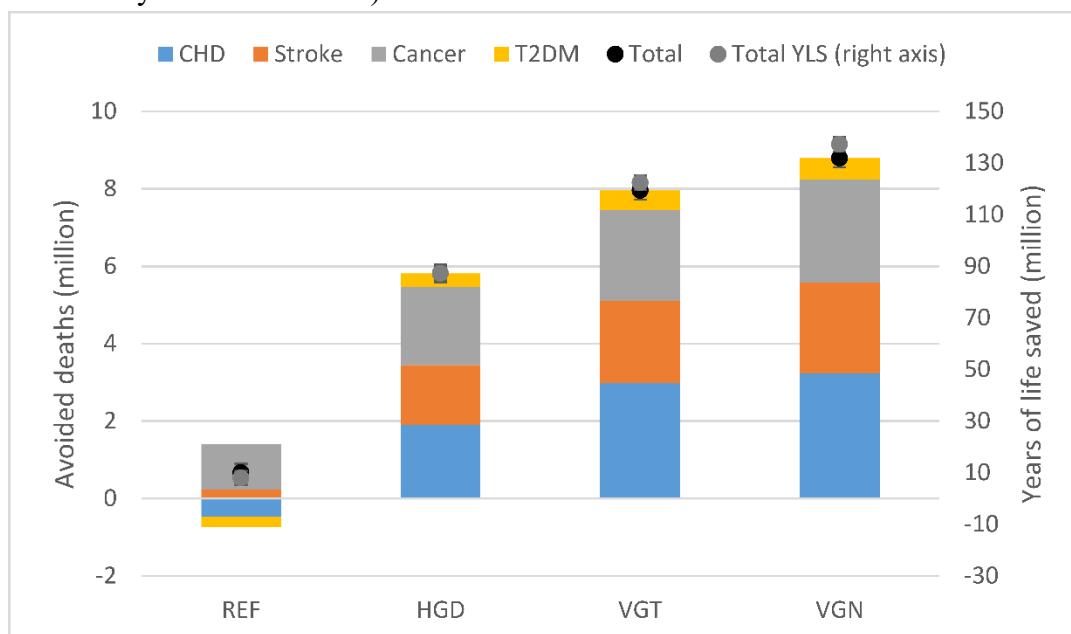


Table S8: Total deaths in 2050 among population aged 20 and older by cause of death (coronary heart disease (CHD), stroke, cancer, type 2 diabetes (T2DM), other causes of deaths (other), and all causes combined) and region (developed countries (DPD), developing countries (DPG); Western high-income countries (HIC), Western middle-income countries (MIC), Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), East Asia (EAS)).

Region	CHD	Stroke	Cancer	T2DM	Other	All causes
WLD	14,062	12,904	12,846	2,615	43,024	85,451
DPD	3,648	2,051	3,984	409	7,292	17,384
DPG	10,415	10,853	8,863	2,205	35,732	68,068
LAC	907	629	1,108	443	3,096	6,183
SSA	771	839	583	322	5,791	8,306
EMR	1,261	691	637	165	2,600	5,354
SAS	3,739	2,529	1,646	517	12,257	20,687
EAS	3,685	6,134	4,843	742	11,840	27,243

Figure S4: Total number of avoided deaths in 2050 compared to 2005/07 by age group.

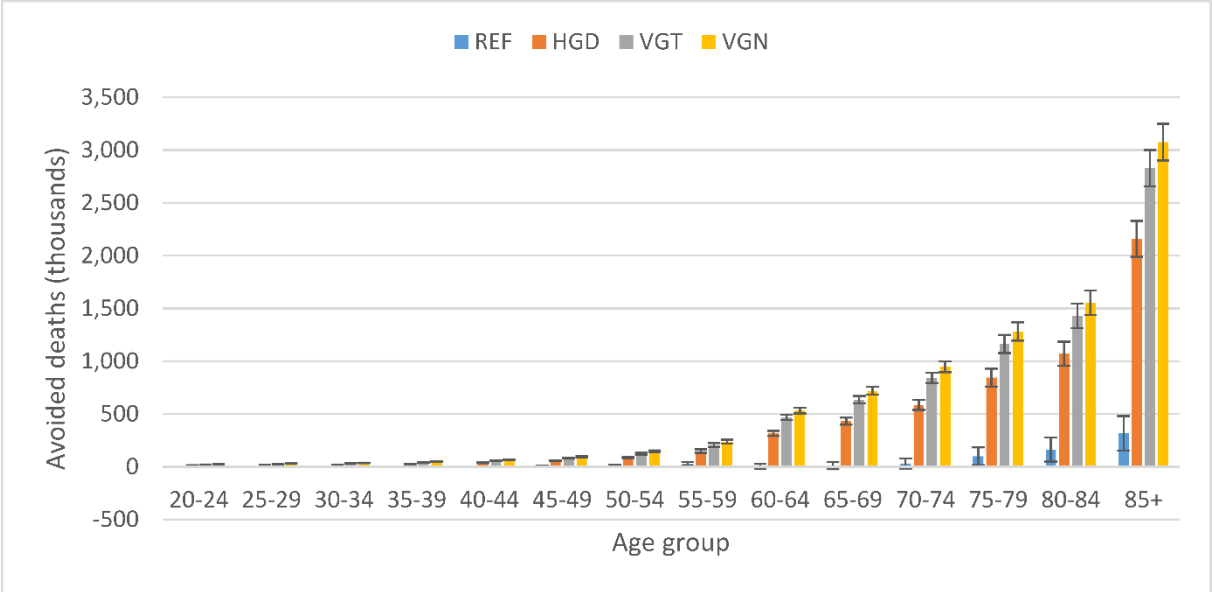


Figure S5: Health analysis of dietary change for the year 2050 relative to 2005/07 by risk factor and region. Risk factors include change in fruit and vegetable consumption ( $\Delta C(\text{fruit\&veg})$ ), change in red meat consumption ( $\Delta C(\text{red meat})$ ), change in prevalence of overweight and obesity combined ( $\Delta \text{weight}$ ), and all risk factors combined (Total). Regions include an aggregate of all world regions (WLD); developed countries (DPD), which include Western high-income countries (HIC) and Western middle-income countries (MIC); and developing countries (DPG), which include Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), and East Asia (EAS). (*Upper panel*) Number of avoided deaths (in millions) in the dietary scenarios in 2050 relative to 2005/07 by risk factor and region. (*Lower panel*) Number of avoided deaths per capita (in deaths per thousand people) in the dietary scenarios in 2050 relative to 2005/07 by risk factor and region.

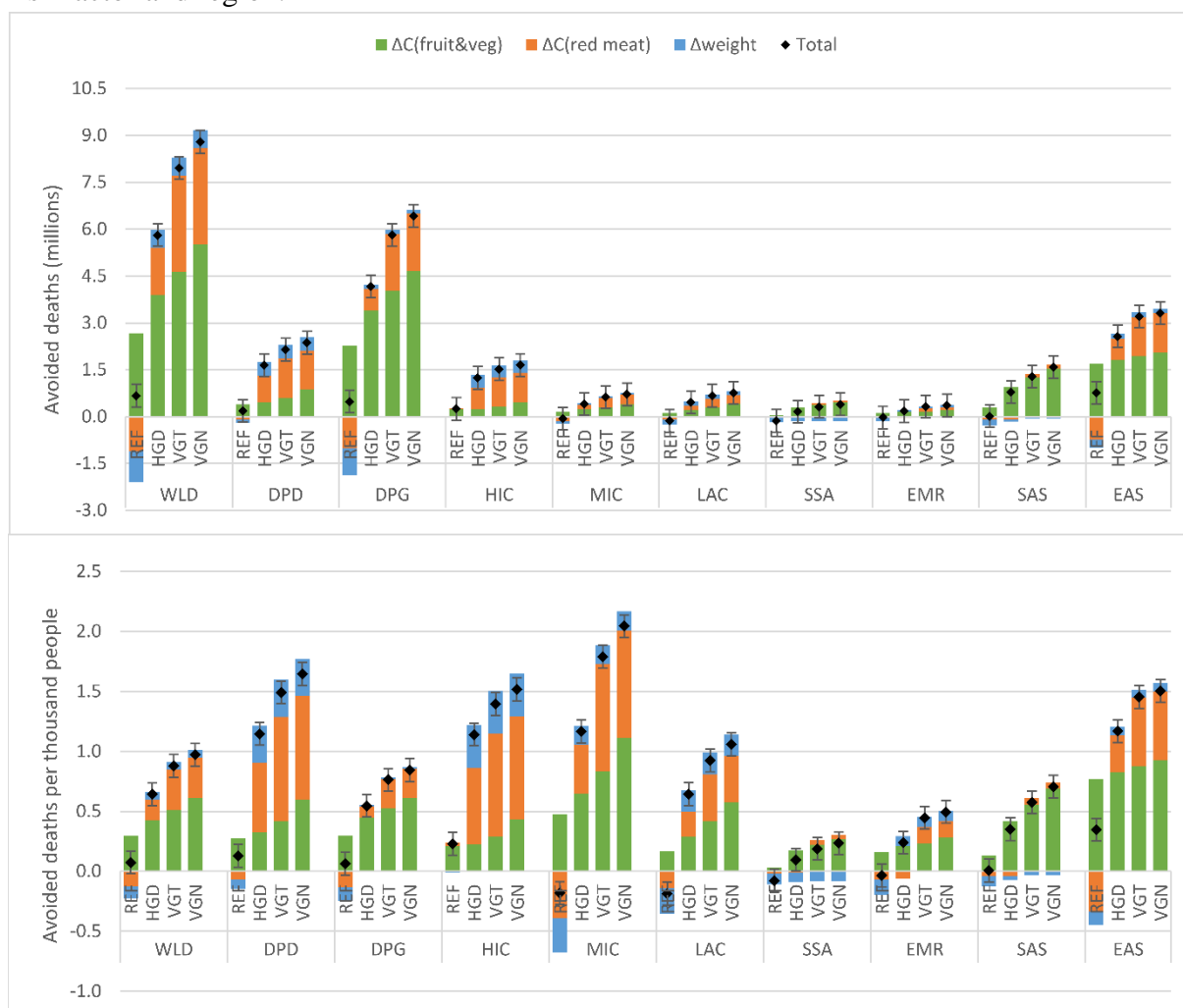


Figure S6: Health analysis of dietary change for the year 2050 relative to the reference scenario by risk factor and region. Risk factors include change in fruit and vegetable consumption ( $\Delta C(\text{fruit\&veg})$ ), change in red meat consumption ( $\Delta C(\text{red meat})$ ), change in prevalence of overweight and obesity combined ( $\Delta \text{weight}$ ), and all risk factors combined (Total). Regions include an aggregate of all world regions (WLD); developed countries (DPD), which include Western high-income countries (HIC) and Western middle-income countries (MIC); and developing countries (DPG), which include Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), and East Asia (EAS). (*Upper panel*) Number of avoided deaths (in millions) in the dietary scenarios in 2050 relative to the reference scenario by risk factor and region. (*Lower panel*) Number of avoided deaths per capita (in deaths per thousand people) in the dietary scenarios in 2050 relative to the reference scenario by risk factor and region.

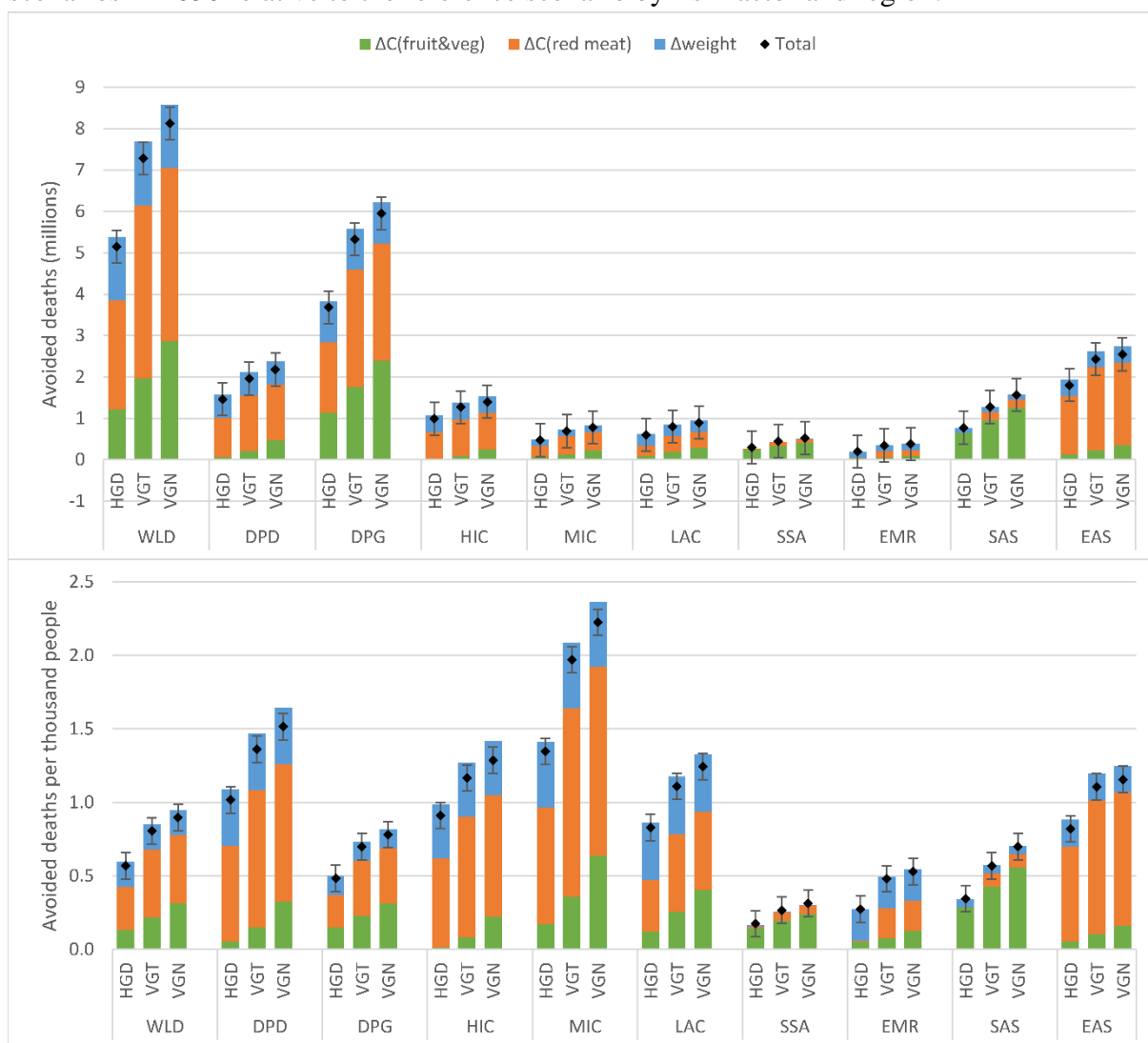
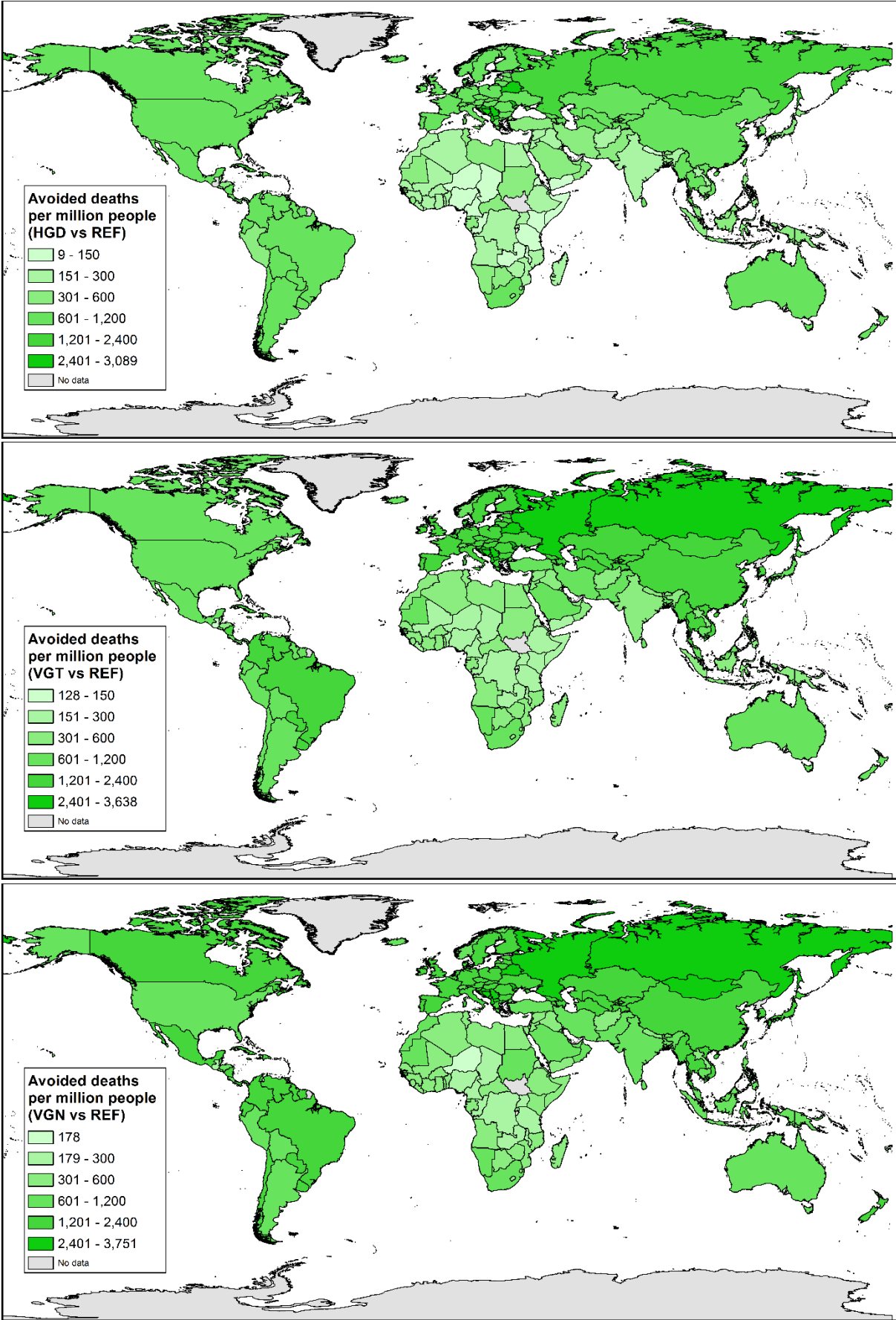


Figure S7: Change in avoided deaths per capita relative to the reference scenario in 2050.





## SI.7 Supplementary environmental results

Figure S8: Food-related GHG emissions (GtCO<sub>2</sub>-eq) in 2005/07 and projected to 2050 for each dietary scenarios by commodity. The percentages within the bars refer to the percentage of food-related GHG emissions to total GHG emissions, using historical emissions data for 2005/07 and an emissions pathway that would limit global temperature increase to below 2°C (32).

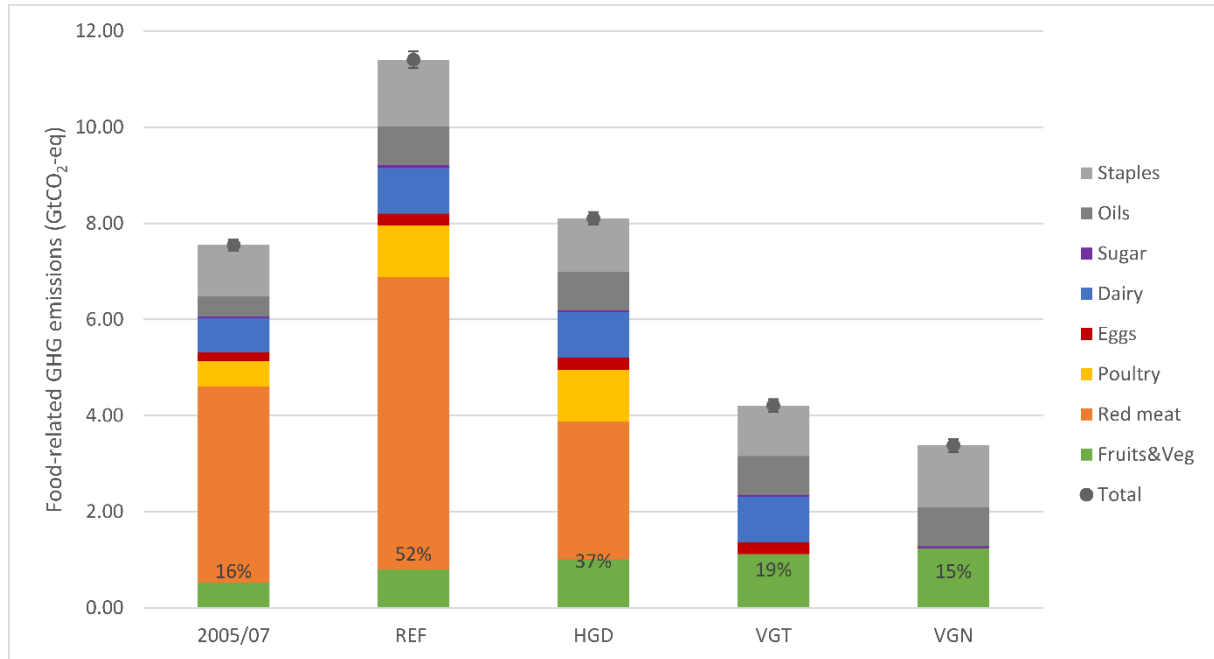


Fig. S9: Environmental analysis of dietary change for the year 2050 by food group and region. Regions include an aggregate of all world regions (WLD); developed countries (DPD), which include Western high-income countries (HIC) and Western middle-income countries (MIC); and developing countries (DPG), which include Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), and East Asia (EAS). (*Upper panel*) Food-related greenhouse gas (GHG) emissions in the dietary scenarios in 2050 by food group and region. (*Lower panel*) Food-related GHG emissions per capita in the dietary scenarios in 2050 by food group and region.

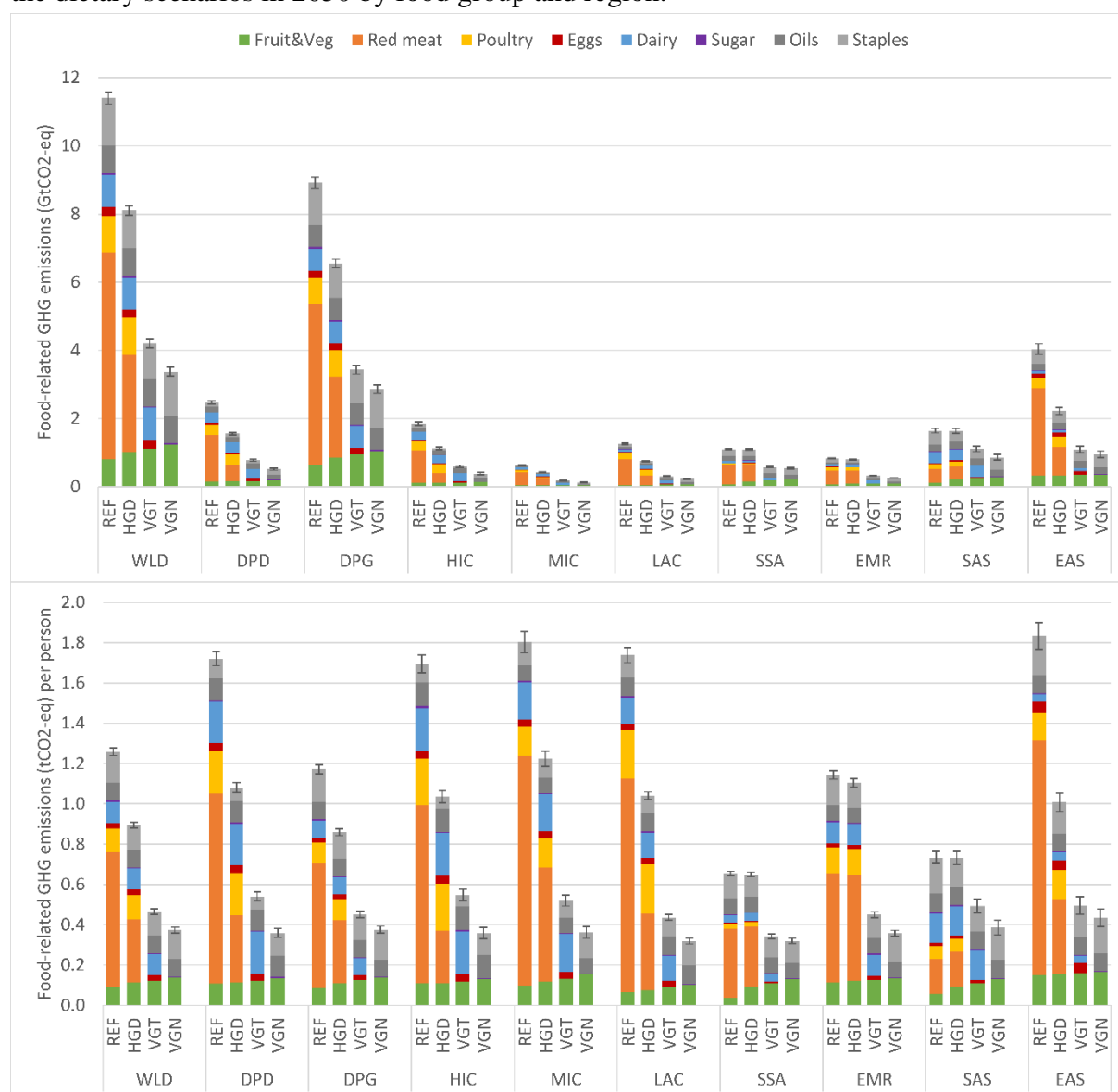


Fig. S10: Environmental analysis of dietary change for the year 2050 relative to the reference scenario by food group and region. Regions include an aggregate of all world regions (WLD); developed countries (DPD), which include Western high-income countries (HIC) and Western middle-income countries (MIC); and developing countries (DPG), which include Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), and East Asia (EAS). (Upper panel) Changes in food-related greenhouse gas (GHG) emissions in the dietary scenarios relative to the reference scenario in 2050 by food group and region. (Lower panel) Changes in food-related GHG emissions per capita in the dietary scenarios relative to the reference scenario in 2050 by food group and region.

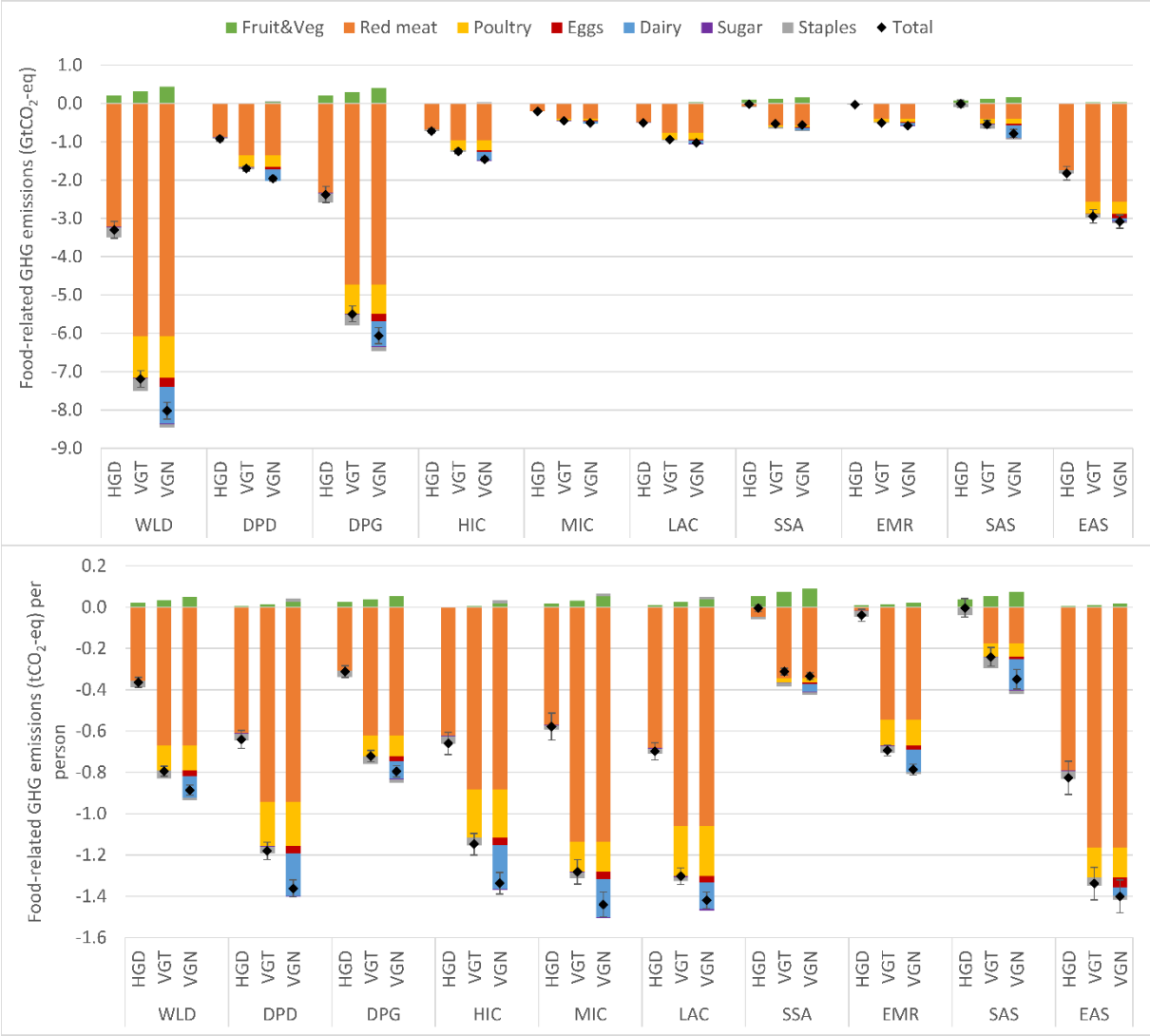
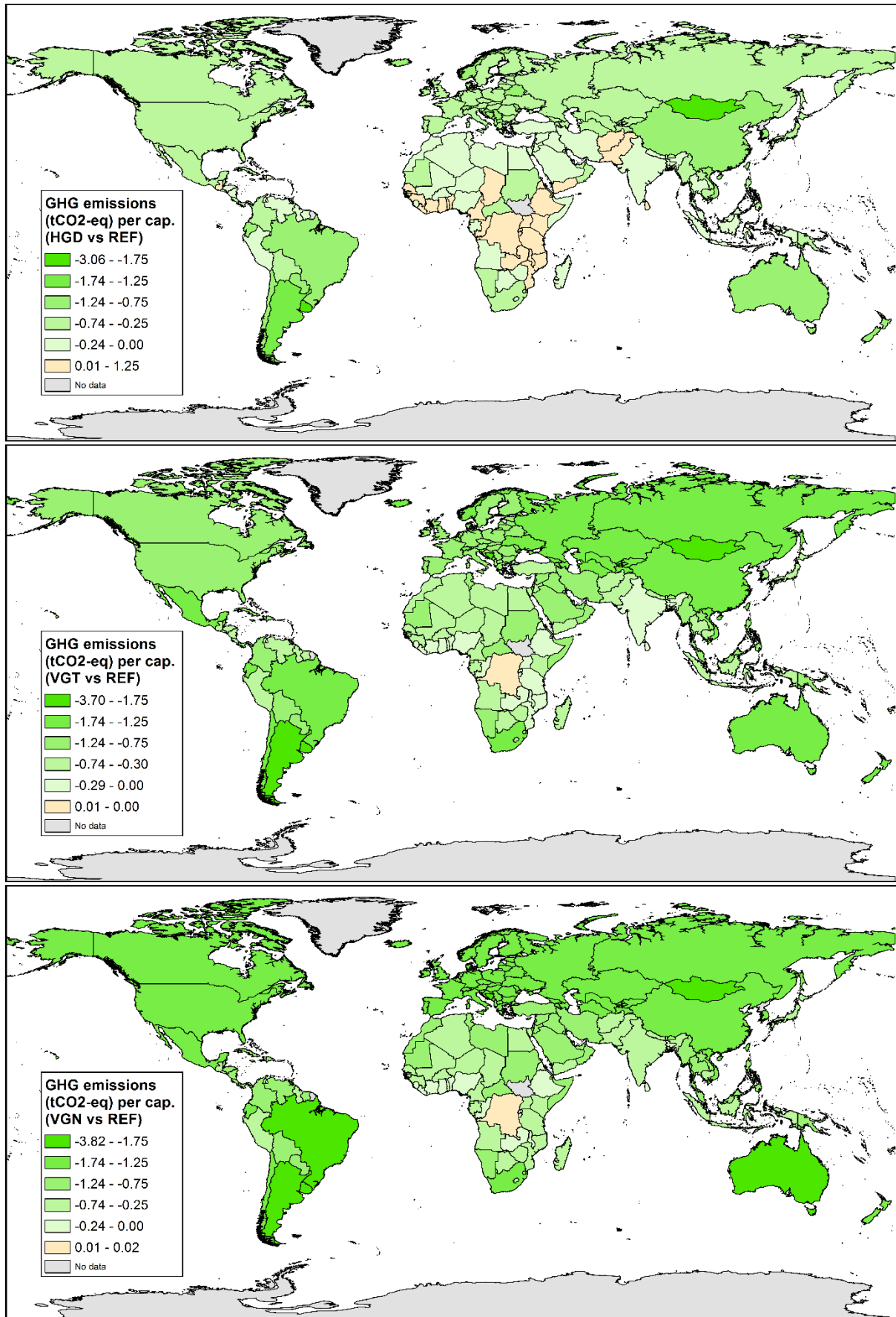


Figure S11: Change in GHG emissions per capita relative to reference scenario in 2050.



**SI.8 Supplementary valuation results**

Figure S12: Healthcare-related benefits (USD billion) by region and cost component of total healthcare benefits, including saved direct healthcare costs (direct) and indirect costs of informal care (indirect(care)) and lost labour productivity (indirect(productivity)).

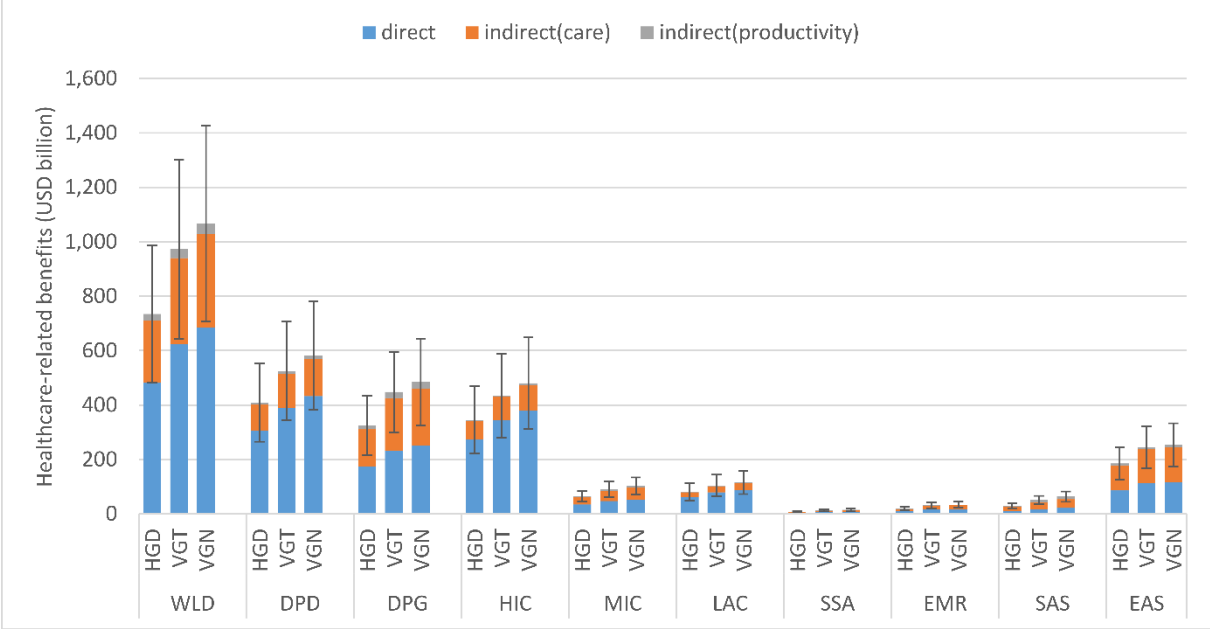


Figure S13: Health benefits estimated by value-of-statistical-life (VSL) approach by region: developed countries (DPD), developing countries (DPG); Western high-income countries (HIC), Western middle-income countries (MIC), Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), East Asia (EAS).

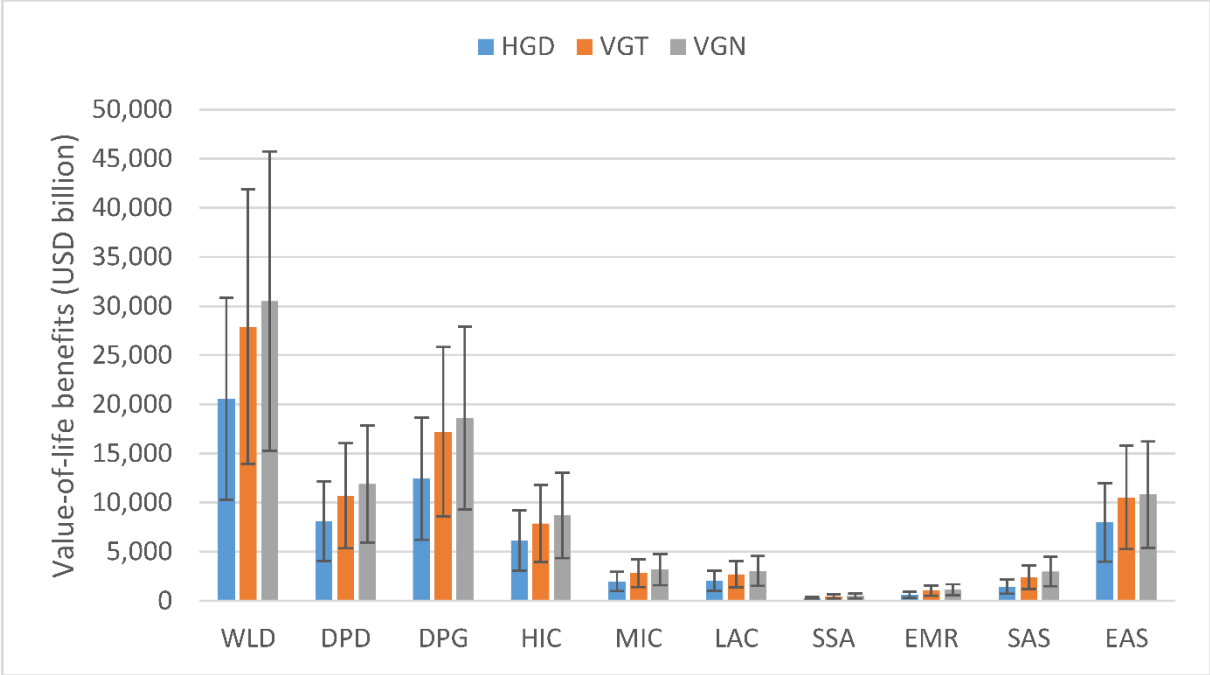
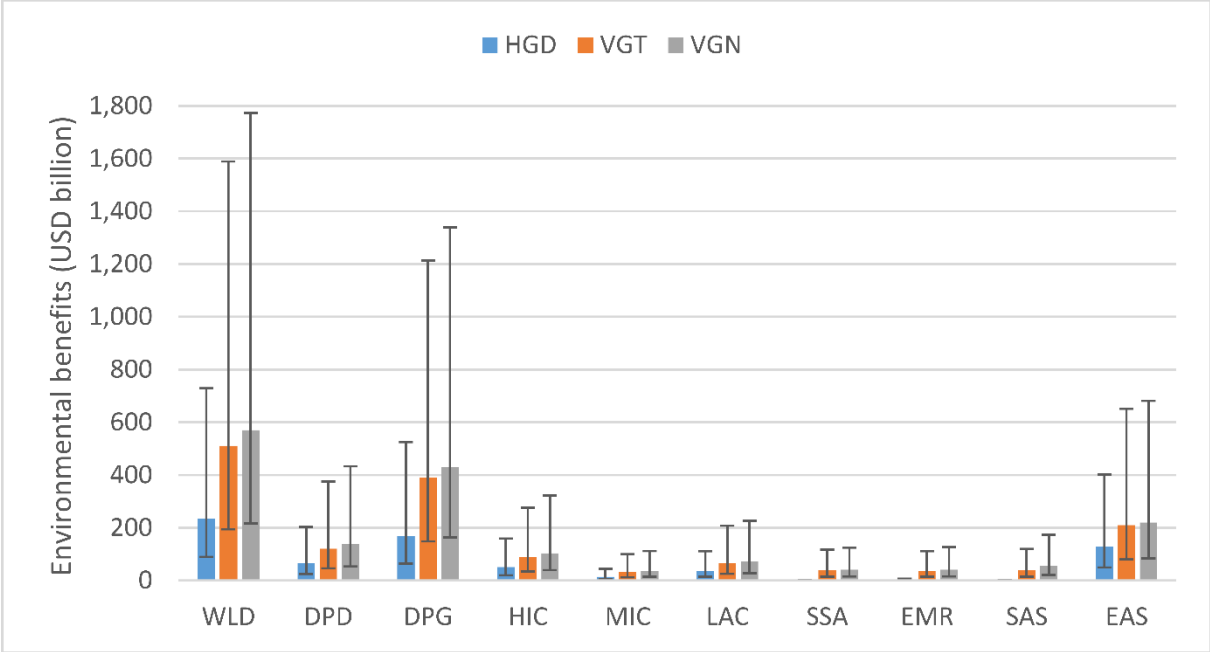


Figure S14: Value of environmental benefits by region: developed countries (DPD), developing countries (DPG); Western high-income countries (HIC), Western middle-income countries (MIC), Latin America and the Caribbean (LAC), Sub-Saharan Africa (SSA), Eastern Mediterranean (EMR), South Asia (SAS), East Asia (EAS). The error bars denote environmental benefits obtained by using different social costs of carbon which are associated with different discount rates (main: 3%, low: 5%, high: 95<sup>th</sup> percentile of 3%).



## Supplementary discussion

### SI.9 Emissions feedback from reductions in mortality

In our emissions analysis, we accounted for the impacts that dietary changes have on food-related GHG emissions. However, changes in mortality could generate feedbacks on emissions which might counteract the diet-related impacts. For example, one would expect that if people lived longer due to diet-reductions in mortality, they would also contribute a greater amount of GHG over their life cycle. Here we analyse such feedbacks and find that the additional emissions due to diet-related reductions in mortality could compensate about 4-6% of the total reductions in food-related GHG emissions that are associated with dietary changes (Table S9). One way of contextualizing these emissions feedbacks is by noting that the number of avoided deaths (5.1-8.1 million, depending on the scenario) represent less than a tenth of a percent of the projected population in 2050 (> 9 billion). Thus, changes in the dietary habits of the whole population have a much greater impact on food-related GHG emissions than the feedback on those emissions from the relatively small number of avoided deaths.

Table S9: Sensitivity analysis of emissions feedback related to changes in mortality.

Row	Parameter	Scenario			
		HGD	VGT	VGN	
1	Years of life saved (YLS) (millions)	79	114	129	
2	Food-related emissions per capita (tCO <sub>2</sub> -eq per person per year)	0.89	0.46	0.37	
3	Food-related emissions reductions (GtCO <sub>2</sub> -eq)	3.3	7.2	8	
4	Food-related emissions due to YLS (MtCO <sub>2</sub> -eq)	Absolute emissions (MtCO <sub>2</sub> -eq)	71	53	48
5		Percent of food-related emissions reduction (%)	2.2	0.7	0.6
6	Ratio of non-food-related GHG emissions under a 2°C emissions trajectory to food-related emissions (see Fig. S8)	2.7	5.3	6.7	
7	Total YLS-related emissions (MtCO <sub>2</sub> -eq)	Absolute emissions (MtCO <sub>2</sub> -eq)	191	280	322
8		Percent of food-related emissions reductions (%)	5.8	3.9	4

Notes: Rows 1-3 and 6 are main results; row 4 is calculated by multiplying rows 1 and 2; row 5 is calculated by dividing row 4 by row 3; row 7 is calculated by multiplying rows 4 and 6; row 8 is calculated by dividing row 7 by row 3.

## SI.10 Supplementary discussion of environmental results

In our environmental analysis, we projected food-related GHG emissions to increase from 7.6 GtCO<sub>2</sub>-eq in 2005/07 to 11.4 GtCO<sub>2</sub>-eq in the reference scenario in 2050; and we projected dietary changes in the HGD, VGT, and VGN scenarios to decrease the reference emissions in 2050 by 3.3-8.0 GtCO<sub>2</sub>-eq (29-70%). In aggregate, our environmental results are in line with the environmental literature on dietary change. Hedenus et al (13) projected food-related GHG emissions to increase from 8 GtCO<sub>2</sub>-eq in 2000 to 12 GtCO<sub>2</sub>-eq in 2050, and that dietary changes that ranged from replacing 75% of ruminant meat and dairy by other meat to replacing 75% of all animal foods by pulses and cereals on kcal basis would reduce food-related GHG emissions by 3.4-5.2 GtCO<sub>2</sub>-eq (net of technical mitigation in the agricultural sector and increased productivity which would add another 2 and 1.7 GtCO<sub>2</sub>-eq to the estimate). Tilman and Clark (12) projected food-related GHG emissions to increase from 8.43 GtCO<sub>2</sub>-eq in 2009 to 15 GtCO<sub>2</sub>-eq in 2050; and that adopting Mediterranean, pescatarian, and vegetarian diets would reduce emissions by 4.22-8.44 GtCO<sub>2</sub>-eq (30-60%). Although we adopted the same baseline GHG emissions factors as Tilman and Clark, our estimates are slightly lower than theirs, because we also accounted for output-based productivity improvements in agriculture which also improve (i.e., lower) emissions intensities, and we did not account for the GHG emissions associated with the consumption of fish and seafood. Another difference between our study and Tilman and Clark's is that we use consumption projections produced by FAO, whereas Tilman and Clark generate income-dependent dietary projections. Our final environmental comparison is to Bajzelj et al (7) who projected that adopting a dietary pattern based on dietary recommendations would decrease food-related GHG emissions by 5.8-6.4 GtCO<sub>2</sub>-eq depending on the underlying reference scenario. In contrast to our study, Bajzelj et al included land-use emissions (which is why reference emissions cannot be compared) and their dietary scenario is largely based on advice from US institutions and associations (Harvard Medical School, American Heart Association), which makes it more stringent than our HGD scenario that is based on global recommendations.



## SI.11 Supplementary discussion of valuation results

We are not aware of other studies that contrasted the value of environmental and health benefits. However, some separate valuation studies exist. In our economic analysis, we estimated the value of environmental benefits from avoided damages to range from 89-1,773 billion in 2050 (0.04-0.77% of global GDP) depending on the dietary scenario and the discount rate used. Using an integrated assessment model, Stehfest et al (11) estimated mitigation cost savings of 0.56% of GDP if dietary recommendations on healthy eating would be adopted – an estimate that is within the range obtained here. With regards to health-related costs, Bloom et al (34) estimated the economic burden of cancer and cardiovascular diseases in 2030 to be USD 1.46 trillion using a cost-of-illness approach and USD 43.4 trillion using a value-of-statistical-life approach. Re-doing our analysis for the year 2030 yielded reductions in the number of deaths from stroke, CHD, and cancer of 12-20% and monetized health benefits of USD 314 billion (219-408 billion), USD 445 billion (311-578 billion), and USD 500 billion (350-650 billion) in HGD, VGT, and VGN scenario, respectively, when using the cost-of-illness approach, and of USD 10 trillion (5-15 trillion), USD 14 trillion (7-22 trillion), and USD 16 trillion (8-24 trillion) in the HGD, VGT, and VGN scenario, respectively, when using the value-of-statistical-life approach. Applying the percentage mortality reduction to the economic estimates of Bloom et al yields values that fall within those ranges (USD 175-292 billion for the cost-of-illness estimate and USD 5.2-8.7 trillion for the value-of-statistical-life estimate).