THE LANCET

Supplementary appendix

This appendix formed part of the original submission and has been peer reviewed. We post it as supplied by the authors.

Supplement to: Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT–*Lancet* Commission on healthy diets from sustainable food systems. *Lancet* 2019; published online Jan 16. http://dx.doi.org/10.1016/S0140-6736(18)31788-4.

Web Appendix

Our Food in the Anthropocene: Healthy Diets from Sustainable Food systems

Supplementary Table 1. Overview of income and population changes in the shared socio-economic pathways. The pathways include a middle-of-the-road development pathway (SSP2), a more optimistic pathway with higher income and lower population growth (SSP1), and a more pessimistic pathway with lower income and greater population growth (SSP3). We used the assumptions from SSP2 in this report.

Region and parameter	Scenario (year)			
	BMK (2010)	SSP2 (2050)	SSP1 (2050)	SSP3 (2050)
East Asia and Pacific				
GDP	19,236	80,045	104,096	60,608
Population	2,184	2,261	2,173	2,351
GDP per capita	9	35	48	26
Europe				
GDP	14,628	27,780	30,571	21,342
Population	537	577	592	498
GDP per capita	27	48	52	43
Former Soviet Union (excl. Baltic States)				
GDP	2,855	8,984	10,603	7,551
Population	279	277	262	289
GDP per capita	10	32	40	26
Latin America and Caribbean				
GDP	5,834	19,164	22,838	15,894
Population	585	742	674	853
GDP per capita	10	26	34	19
Middle East and North Africa				
GDP	4,551	18,631	20,566	16,006
Population	457	715	646	808
GDP per capita	10	26	32	20
North America				
GDP	14,290	29,933	33,691	24,753
Population	344	450	460	372
GDP per capita	41	67	73	67
South Asia				
GDP	4,461	32,939	44,250	22,756
Population	1,630	2,373	2,108	2,720
GDP per capita	3	14	21	8
Sub-Saharan Africa				
GDP	1,705	13,962	19,690	9,665
Population	863	1,793	1,564	2,084
GDP per capita	2	8	13	5
World				
GDP	67,559	231,439	286,305	178,575
Population	6,879	9,187	8,479	9,975
GDP per capita	10	25	34	18

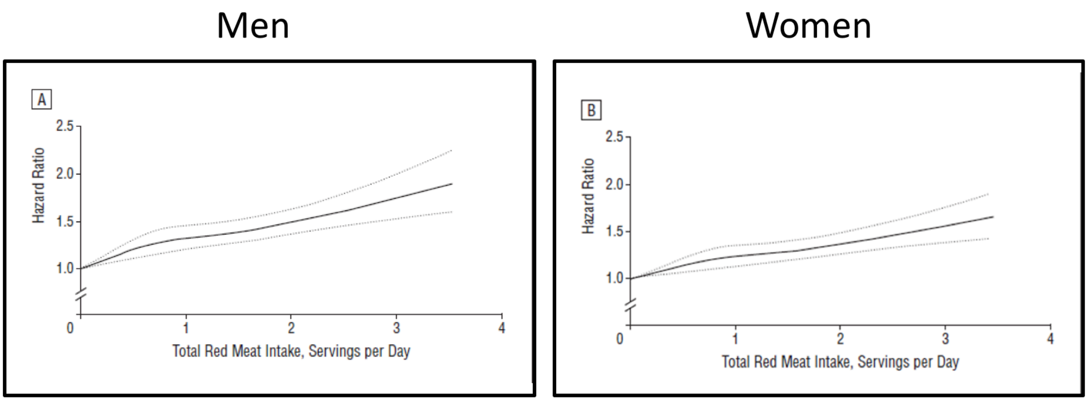
Source: Calculated from IMPACT 3.1 with population and GDP growth rates from IIASA and OECD

Note: GDP and GDP per capita are in purchasing power parity (ppp)

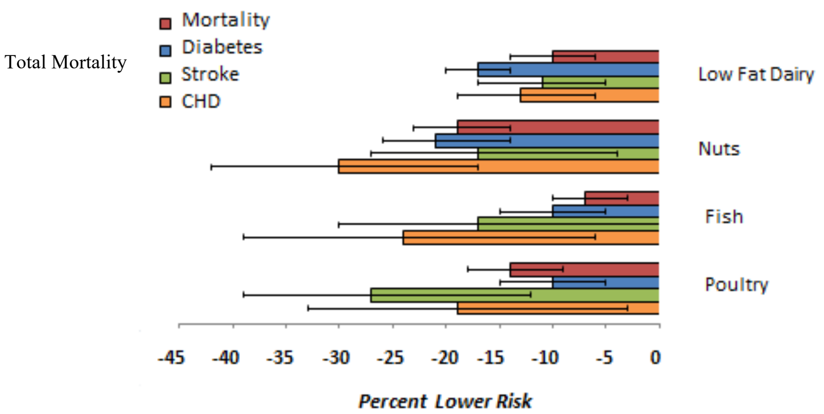
Supplementary Table 2. Global and regional population estimates for 2017, 2030, 2050, and 2100, according to the medium-variant projection.

Region	*Population (millions)*			
	2017	*2030*	*2050*	*2100*
World	7 550	8 551	9 772	11 184
Africa	1 256	1 704	2 528	4 468
Asia	4 504	4 947	5 257	4 780
Europe	742	739	716	653
Latin America and the Caribbean	646	718	780	712
Northern America	361	395	435	499
Oceania	41	48	57	72

Source: United Nations, Department of Economic and Social Affairs, Population Division (2017). *World Population Prospects: The 2017 Revision*. New York: United Nations.



Supplementary Figure 1. Multivariate relative risk of overall mortality (23,926 deaths) from red meat consumption during 2.96 million person-years of follow-up of 121,342 men and women. Relative risks are adjusted for age and major lifestyle and dietary risk factors. Source: Pan et al. 2012[1]



Supplementary Figure 2. Percent reduction in risk (95% confidence interval, CI) of major health outcomes associated with replacing red meat (one serving per day) with alternative protein sources[1-4]

Supplementary Panel 1 - A dietary transition in Mexico

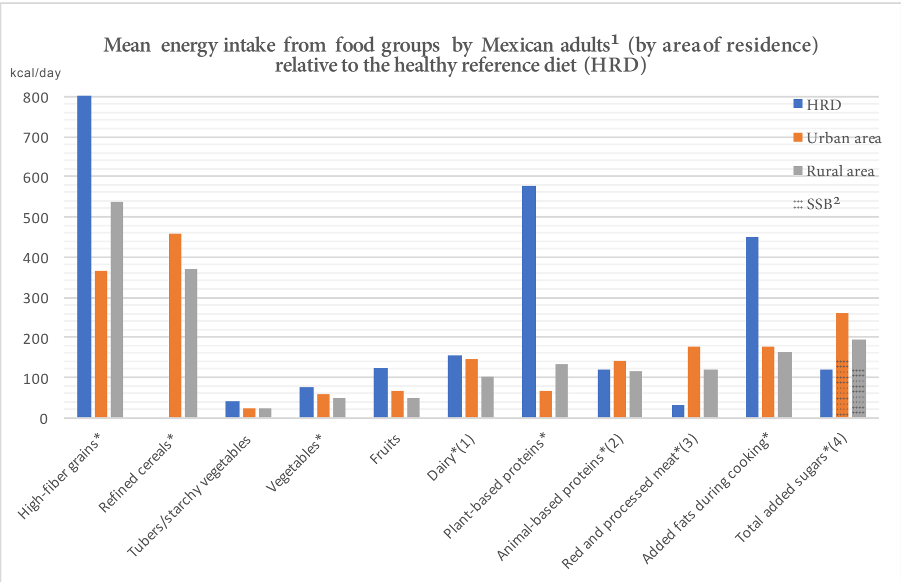
Mexico has a rich culinary tradition; a blend of the ancient pre-Columbian Mesoamerican and the Spanish gastronomies brought about a healthy and sustainable diet. Only decades ago, Mexicans consumed large amounts of plant-based proteins, particularly beans and seeds, whole grain corn tortillas, a large variety of local fruits and vegetables, small amounts of animal source protein (including insects) and added sugar. These culinary traditions were maintained for centuries, particularly by rural and indigenous populations.

However, as many other low and middle-income countries, Mexico has experienced a dietary transition. The current diet in Mexican adults is rich in sugar sweetened beverages (SSBs) and in highly processed low-nutrient food products, animal source proteins, including red and processed meat, and is poor in plant-based proteins. In a dietary survey in rural areas in Mexico in 2012, the intake of beans amounted to 36 g, a 28% reduction in six decades. Moreover, at national level, SSB and highly processed food provide on average 26% of the total energy intake and added sugars contribute 13%[5,6] while vegetables, fruits and legumes contribute only 9.5%.[7]

Relative to the reference diet (see Table 1), Mexican adults consume higher amounts of animal source proteins (excluding red meat), red meat and added sugars and lower amounts of plant-based proteins, whole grains, vegetables, and fruits. Although energy from added fats seems to be below the recommendation, these figures are probably underestimated, since fats from processed foods were not considered.

Even the more traditional rural and Indigenous populations consume around one-quarter of the intake of plant-based proteins in the reference diet and around two-thirds of whole grains. In contrast, the consumption of SSBs is 58% and 33% above recommendations for the rural and Indigenous populations, respectively. The main source of added sugars are SSBs, contributing with 63% of all added sugars in indigenous, 57% in non-indigenous, 56% in urban and 62% in rural populations.

In conclusion, Mexico, a middle-income country with traditional diets that were rich in beans and seeds, whole grain corn, fruits and vegetables is currently far away from the healthy reference diet presented by this Commission. Instead about one quarter of the total energy intake of Mexican adults are provided by SSBs and highly processed food, consumption of red meat is higher and plant-based protein and whole grains are lower than recommended. Even rural and Indigenous populations have abandoned the traditional healthy and sustainable diet and are far away from the reference diet.



[1] ≥ 20 years old
[2] Sugar-sweetened beverages
*Statistically different between urban and rural area ($p<0.05$)
(1) Butter and cream not included.
(2) Poultry, eggs and seafood (red and processed meat not included).
(3) Beef, lamb, pork and processed meat (intake of processed meat is discouraged for a healthy diet).
(4) Total added sugars include added sugars present in all food groups. Sugar-sweetened beverages contribute with 56% of the kcal from added sugars in urban population and 62% in rural population.

Supplementary Table 3. List of systematic reviews, meta-analyses and pooled analyses of primary data used as part of the evidence base for setting the scientific targets for a healthy diet.

Reference and PMID	Food Groups / Pattern	Type of Study
Appel LJ, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. Jama. 2005. PMID:16287956.[8]	Whole grains Vegetables Carbohydrate enriched High protein Chicken, fish Legumes, beans, nuts Higher fat. Olive, canola, and safflower oils, nuts and seeds.	Randomized Trial 1. carbohydrate-rich diet, similar to DASH diet. 2. High protein, with approximately half from plant sources. 3. Higher unsaturated fat, predominanty monounsaturated fat.
Orlich MJ, et al. Vegetarian dietary patterns and mortality in Adventist Health Study 2. JAMA Intern Med. 2013. PMID:23836264[9]	Vegetarian pattern	Cohort
Satija A, et al. Plant-based dietary patterns and incidence of type 2 diabetes in us men and women: results from three prospective cohort studies. PLoS Med. 2016. PMID:27299701[10]	Plant-based index as a continuous variable	Cohorts (3)
Satija A, et al. Healthful and unhealthful plant-based diets and the risk of coronary heart disease in U.S. adults. J Am Coll Cardiol. 2017. PMID:28728684.[11]	Plant-based index as a continuous variable	Cohorts (3)
Abete I, et al. Association between total, processed, red and white meat consumption and all-cause, CVD and IHD mortality: a meta-analysis of cohort studies. Br J Nutr. 2014. PMID:24932617[12]	Types of meat	Meta-analysis
Chen GC, et al. Red and processed meat consumption and risk of stroke: a meta-analysis of prospective cohort studies. Eur J Clin Nutr. 2013. PMID:23169473.[13]	Red meat, including processed and unprocessed	Meta -analysis
Feskens EJ, et al. Meat consumption, diabetes, and its complications. Curr Diab Rep. 2013. PMID:23354681.[14]	Red meat and poultry	Meta-analysis
Pan A, et al. Red meat consumption and mortality: results from 2 prospective cohort studies. Arch Intern Med. 2012. PMID:22412075[1]	Red meat, including processed and unprocessed	Cohorts (2)
Sinha R, et al. Meat intake and mortality: a prospective study of over half a million people. Arch Intern Med. 2009. PMID:19307518[4]	Red meat	cohort
Etemadi A, et al. Mortality from different causes associated with meat, heme iron, nitrates, and nitrites in the NIH-AARP Diet and Health Study: population based cohort study. BMJ. 2017. PMID:28487287.[15]	Red meat	cohort

Reference and PMID	Food Groups / Pattern	Type of Study
Pan A, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. Am J Clin Nutr. 2011. PMID:21831992.[16]	Red meat	Cohorts (3)
Bernstein AM, et al. Major dietary protein sources and risk of coronary heart disease in women. Circulation. 2010. PMID:20713902[3]	Major protein sources	Cohort
Pan A, et al. Changes in red meat consumption and subsequent risk of type 2 diabetes mellitus: three cohorts of US men and women. JAMA Intern Med. 2013. PMID:23779232[17]	Changes in red meat	Cohorts (3)
Kromhout D, et al. Food consumption patterns in the 1960s in seven countries. Am J Clin Nutr. 1989. PMID:2718924.[18]	Overall diets	Descriptive
Chan DS, et al. Red and processed meat and colorectal cancer incidence: meta-analysis of prospective studies. PLoS One. 2011. PMID:21674008.[19]	Red meat	Meta-analysis
Bouvard V, et al. Carcinogenicity of consumption of red and processed meat. Lancet Oncol. 2015. PMID:26514947[20]	Red meat	Review
Farvid MS, et al. Adolescent meat intake and breast cancer risk. Int J Cancer. 2015. PMID:25220168[21]	Red meat and other major protein sources	Cohort
Farvid MS, et al. Dietary protein sources in early adulthood and breast cancer incidence: prospective cohort study. BMJ. 2014. PMID:24916719.[21]	Major protein sources	Cohort
Song M, et al. Association of animal and plant protein intake with all-cause and cause-specific mortality. JAMA Intern Med. 2016. PMID:27479196.[22]	Major protein sources	Cohorts (2)
Lee JE, et al. Meat intake and cause-specific mortality: a pooled analysis of Asian prospective cohort studies. Am J Clin Nutr. 2013. PMID:23902788.[23]	Meat	Meta-analysis of primary data
Talaei M, et al. Meat, dietary heme iron, and risk of type 2 diabetes mellitus: the singapore chinese health study. American Journal of Epidemiology. 2017. PMID:28535164.[24]	Meat	Cohort
Pan A, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. Am J Clin Nutr. 2011. PMID:21831992.[16]	Red meat	Cohorts (3) and Meta-analysis
Bischoff-Ferrari HA, et al. Calcium intake and hip fracture risk in men and women: a meta-analysis of prospective cohort studies and randomized controlled trials. Am J Clin Nutr. 2007. PMID:18065599.[25]	Calcium	Meta-analysis
Feskanich D, et al. Milk consumption during teenage years and risk of hip	Milk and other dairy foods	Cohort

Reference and PMID	Food Groups / Pattern	Type of Study
fractures in older adults. JAMA Pediatr. 2014. PMID:24247817.[26]		
Guo J, et al. Milk and dairy consumption and risk of cardiovascular diseases and all-cause mortality: dose–response meta-analysis of prospective cohort studies. Eur J Epidemiol. 2017.[27]	Milk and other dairy foods	Meta-analysis
Aune D, et al. Dairy products, calcium, and prostate cancer risk: a systematic review and meta-analysis of cohort studies. Am J Clin Nutr. 2015. PMID:25527754.[28]	Dairy foods and calcium	Meta-analysis
Chen M, et al. Dairy consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. BMC Med. 2014. PMID:25420418.[29]	Dairy foods	Meta-analysis
Mozaffarian D, et al. Fish intake, contaminants, and human health: evaluating the risks and the benefits. JAMA. 2006.[30]	Fish	Review
Virtanen JK, et al. Fish consumption and risk of major chronic disease in men. Am J Clin Nutr. 2008. PMID:19064523.[31]	Fish	Cohort
Oken E, et al. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. Am J Epidemiol. 2008. PMID:18353804[32]	Fish	Cohort
Del Gobbo LC, et al. Omega-3 polyunsaturated fatty acid biomarkers and coronary heart disease: pooling project of 19 cohort studies. JAMA Intern Med. 2016. PMID:27357102.[33]	N-3 fatty acids	Meta-analysis of primary data from cohort studides
Rong Y, et al. Egg consumption and risk of coronary heart disease and stroke: dose-response meta-analysis of prospective cohort studies. BMJ. 2013. PMID:23295181[34]	egg	Meta-analysis
Iannotti LL, et al. Eggs in Early Complementary Feeding and Child Growth: A Randomized Controlled Trial. Pediatrics. 2017. PMID:28588101.[35]	egg	Randomized clinical trial
Kris-Etherton PM, et al. The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. J Nutr. 2008. PMID:18716180.[36]	nuts	Review
Sabate J, et al. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. Arch Intern Med. 2010. PMID:20458092.[37]	nuts	Meta-analysis

Reference and PMID	Food Groups / Pattern	Type of Study
Grosso G, et al. Nut consumption and age-related disease. Maturitas. 2016. PMID:26586104.[38]	nuts	Review
Mayhew AJ, et al. A systematic review and meta-analysis of nut consumption and incident risk of CVD and all-cause mortality. Br J Nutr. 2016. PMID:26548503.[39]	nuts	Meta-analysis
Luo C, et al. Nut consumption and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a systematic review and meta-analysis. Am J Clin Nutr. 2014. PMID:24847854.[40]	Nuts	Meta-analysis
Bao Y, et al. Association of nut consumption with total and cause-specific mortality. N Engl J Med. 2013. PMID:24256379.[41]	Nuts	Cohorts
Estruch R, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med. 2018. PMID: 29897866.[42]	Nuts as part of Mediterranean diet	Randomized clinical trial
Kushi LH, et al. Cereals, legumes, and chronic disease risk reduction: evidence from epidemiologic studies. Am J Clin Nutr. 1999. PMID:10479217.[43]	Cereals, legumes	Review
Afshin A, et al. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. Am J Clin Nutr. 2014. PMID:24898241.[44]	Nuts and legumes	Meta-analysis
Lee SA, et al. Adolescent and adult soy food intake and breast cancer risk: results from the Shanghai Women's Health Study. Am J Clin Nutr. 2009. PMID:19403632.[45]	Soy	cohort
Zong G, et al. Whole grain intake and mortality from all causes, cardiovascular disease, and cancer: a meta-analysis of prospective cohort studies. Circulation. 2016. PMID:27297341.[46]	Whole grains	Meta-analysis
Dehghan M, et al. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. Lancet. 2017. PMID:28864332.[47]	Fats and carbohydrate	Multiple cohorts
Mensink RP, et al. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr. 2003. PMID:12716665.[48]	Dietary fats	Meta-analysis of controlled feeding studies with risk factor outcomes

Reference and PMID	Food Groups / Pattern	Type of Study
Jeppesen J, et al. Effects of low-fat, high-carbohydrate diets on risk factors for ischemic heart disease in postmenopausal women. Am J Clin Nutr. 1997. PMID:9094889[49]	Fat vs carbohyrate	Controlled feeding study with risk factor outcomes
Liu S, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. Am J Clin Nutr. 2000. PMID:10837285.[50]	Carbohydrate	cohort
Muraki I, et al. Potato consumption and risk of type 2 diabetes: results from three prospective cohort studies. Diabetes Care. 2016. PMID:26681722.[51]	Potatoes	Cohorts (3)
Borgi L, et al. Potato intake and incidence of hypertension: results from three prospective US cohort studies. BMJ. 2016. PMID:27189229.[52]	Potatoes	Cohorts (3)
Bertoia ML, et al. Changes in intake of fruits and vegetables and weight change in united states men and women followed for up to 24 years: Analysis from three prospective cohort studies. PLoS Med. 2015. PMID:26394033.[53]	Specific fruits and vegetables	Cohorts (3)
Wang X, 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. BMJ. 2014. PMID:25073782[54]	Fruits and Vegetables	Meta-analysis
Aune D, et al. Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality-a systematic review and dose-response meta-analysis of prospective studies. Int J Epidemiol. 2016. PMID:28338764.[55]	Fruits and Vegetables	Meta-analysis
Muraki I, et al. Fruit consumption and risk of type 2 diabetes: results from three prospective longitudinal cohort studies. BMJ. 2013. PMID:23990623.[56]	Fruits	Cohorts (3)
Hung HC, et al. Fruit and vegetable intake and the risk of major chronic disease. J Natl Cancer Inst. 2004. PMID:15523086.[57]	Fruits and vegetables	Cohorts (2)
Boffetta P, et al. Fruit and vegetable intake and overall cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC). J Natl Cancer Inst. 2010. PMID:20371762.[58]	Fruits and vegetables	Cohort
Wang DD, et al. Association of specific dietary fats with total and cause-specific mortality. JAMA Intern Med. 2016. PMID:27379574.[59]	Dietary fats	Cohorts (2)

Reference and PMID	Food Groups / Pattern	Type of Study
Prentice RL, et al. Low-fat dietary pattern and risk of invasive breast cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. Jama. 2006. PMID:16467232.[60]	Dietary fat	Randomized Clinical Trial
Jakobsen MU, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. Am J Clin Nutr. 2009. PMID:2676998.[61]	Dietary fats	Meta-analysis of primary data from 11 cohorts
Farvid MS, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. Circulation. 2014. PMID:25161045.[62]	Dietary fat, linoleic acid	Meta-analysis of cohorts
Chowdhury R, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. Ann Intern Med. 2014. PMID:24723079.[63]	Dietary fats	Meta-analysis
Sun Y, et al. Palm oil consumption increases LDL cholesterol compared with vegetable oils low in saturated fat in a meta-analysis of clinical trials. J Nutr. 2015. PMID:25995283.[64]	Dietary fats, palm oil	Meta-analysis
Kabagambe EK, et al. The type of oil used for cooking is associated with the risk of nonfatal acute myocardial infarction in costa rica. J Nutr. 2005. PMID:16251629.[65]	Dietary fats	Case-control study
de Lorgeril M, et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease -Erratum in: Lancet 1995;345:738.-Lancet. 1994. PMID: 7911176.[66]	Dietary fat, ALA	Randomized clinical trial
Chen M, et al. Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. Am J Clin Nutr. 2016. PMID:27557656.[67]	Dairy fat	Cohorts (3)
Knopp RH, et al. Long-term cholesterol-lowering effects of 4 fat-restricted diets in hypercholesterolemic and combined hyperlipidemic men. The Dietary Alternatives Study. JAMA. 1997. PMID:9363971.[68]	Dietary fat	Controlled feeding study with risk factor outcomes
Tobias DK, et al. Effect of low-fat diet interventions versus other diet interventions on long-term weight change in adults: a systematic review and meta-analysis. Lancet Diabetes Endocrinol. 2015. PMID:26527511.[69]	Dietary fat	Meta-analysis

Reference and PMID	Food Groups / Pattern	Type of Study
Chiavaroli L, et al. Effect of fructose on established lipid targets: a systematic review and meta-analysis of controlled feeding trials. J Am Heart Assoc. 2015. PMID:26358358.[70]	Fructose	Meta-analysis of controlled feeding studies with risk factor outcomes
Barclay AW, et al. Glycemic index, glycemic load, and chronic disease risk--a meta-analysis of observational studies. Am J Clin Nutr. 2008. PMID:18326601.[71]	Carbohydrates	Meta-analysis of cohort studies
Te Morenga L, et al. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2012. PMID:23321486.[72]	Sugar	Meta-analysis of randomized trials for weight control
Malik VS, et al. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 2010. PMID:20693348.[73]	Sugar	Meta-analysis of cohort studies
Yang Q, et al. Added sugar intake and cardiovascular diseases mortality among US adults. JAMA Intern Med. 2014. PMID:24493081.[74]	Sugar	Cohort
Maslova E, et al. Maternal protein intake during pregnancy and offspring overweight 20 y later. Am J Clin Nutr. 2014. PMID:25099541.[75]	Protein	Cohort
Brantsaeter AL, et al. Does milk and dairy consumption during pregnancy influence fetal growth and infant birthweight? Food and Nutrition Research. 2012. PMID:23185146.[76]	Milk and other dairy foods	Review
Sanchez HP, et al. Adherence to the Mediterranean diet and quality of life in the SUN Project. Eur J Clin Nutr. 2012. PMID:21847137.[77]	Mediterranean dietary pattern	Cohort
Bhushan A, et al. Adherence to Mediterranean diet and subjective cognitive function in men. Eur J Epidemiol. 2018. PMID:29147948.[78]	Mediterranean dietary pattern	Cohort
Morris MC, et al. MIND diet associated with reduced incidence of Alzheimer's disease. Alzheimers Dement. 2015. PMID:25681666.[79]	Dietary patterns and combinations	Senior cohort
Appel LJ, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997. PMID:9099655.[80]	Dietary pattern	Randomized trial with blood pressure outcome

Reference and PMID	Food Groups / Pattern	Type of Study
Sacks FM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med. 2001. PMID:11136953.[81]	Dietary pattern	Randomized trial with blood pressure outcome
Chiuve SE, et al. The association between a nutritional quality index and risk of chronic disease. Am J Prev Med. 2011. PMID:21496749.[82]	Dietary pattern	Cohorts (2)
Wang DD, et al. Improvements In US Diet Helped Reduce Disease Burden And Lower Premature Deaths, 1999-2012; Overall Diet Remains Poor. Health Aff. 2015. PMID:26526250.[83]	Dietary patterns	Cohort
Schwingshackl L, et al. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. J Acad Nutr Diet. 2015. PMID:25680825.[84]	Dietary patterns	Meta-analysis
Onvani S, et al. Adherence to the Healthy Eating Index and Alternative Healthy Eating Index dietary patterns and mortality from all causes, cardiovascular disease and cancer: a meta-analysis of observational studies. J Hum Nutr Diet. 2017. PMID:27620213.[85]	Dietary patterns	Meta-analysis
Cespedes EM, et al. Multiple healthful dietary patterns and type 2 diabetes in the women's health initiative. Am J Epidemiol. 2016. PMID:26940115.[86]	Dietary patterns	Cohort
Mehta RS, et al. Dietary patterns and risk of colorectal cancer: analysis by tumor location and molecular subtypes. Gastroenterology. 2017. PMID:28249812.[87]	Dietary patterns	Cohorts (2)
Hou L, et al. Association between dietary patterns and coronary heart disease: a meta-analysis of prospective cohort studies. Int J Clin Exp Med. 2015. PMID:25785058.[88]	Dietary patterns	Meta-analysis
Trichopoulou A, et al. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003. PMID:12826634.[89]	Dietary pattern	Cohort
Samieri C, et al. The association between dietary patterns at midlife and health in aging: an observational study. Ann Intern Med. 2013. PMID:24189593.[90]	Dietary patterns	Cohort

Reference and PMID	Food Groups / Pattern	Type of Study
Sotos-Prieto M, et al. Association of changes in diet quality with total and cause-specific mortality. N Engl J Med. 2017. PMID:28700845.[91]	Dietary patterns	Cohorts (2)
Chan DS, et al. Red and processed meat and colorectal cancer incidence: meta-analysis of prospective studies. PLoS One. 2011. PMID:21674008.[19]	Red meat	Meta-analysis
Feskens EJ, et al. Meat consumption, diabetes, and its complications. Curr Diab Rep. 2013. PMID:23354681.[14]	Meat	ohort
Micha R, et al. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes--an updated review of the evidence. Curr Atheroscler Rep. 2012. PMID:23001745.[92]	meat	Meta-analysis
Zheng J, et al. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. Public Health Nutr. 2012. PMID:21914258.[93]	Fish	Meta-analysis
Aune D, et al. Nut consumption and risk of cardiovascular disease, total cancer, all-cause and cause-specific mortality: a systematic review and dose-response meta-analysis of prospective studies. BMC Med. 2016. PMID:27916000.[94]	Nuts	Meta-analysis

Supplementary Table 4. Assessment of nutrient adequacy of the Healthy Reference Diet by analysing the nutrient composition of this diet using data primarily from US sources.

See pages 35-36 for details

Methods for analyses of total diets: nutrient adequacy and mortality

Comparative Risk Model

Diet scenarios

We estimated baseline food intake for 158 countries by adapting food demand projections based on a harmonised dataset of country-specific food availability data, and adjusting those for food waste at the household level.[1,2] For estimating the prevalence of underweight (BMI<18), overweight (BMI>25) and obesity (BMI>30) in each country, we fitted log-normal distributions to WHO estimates of mean BMI and the prevalence of overweight and obesity using a cross-entropy method that jointly minimised the deviation of the prevalence data,[3] and we projected weight changes by using correlations between changes in mean BMI and changes in food availability.[3]

We assessed dietary changes towards balanced dietary patterns, including the reference diet and more specialised dietary patterns, including pescatarian, vegetarian, and vegan dietary patterns. The reference diet contains no processed meat, low amounts of red meat (including beef, lamb, pork) and sugar, moderate amounts of poultry, dairy and fish, and generous amounts of fruits, vegetables, legumes, and nuts. The other three dietary patterns replace either meat (pescatarian, vegetarian) or all animal source foods (vegan) to one third by fruits and vegetables and to two thirds by either fish and seafood (pescatarian diets) or legumes (vegetarian and vegan diets). We regionalised the dietary patterns for each country by preserving the current national preferences for types of grains, fruits, red meat and fish.

Nutrient analysis

We analysed the nutrient adequacy of the diet scenarios by calculating their nutrient content and comparing it to international recommendations. For calculating the nutrient content, we paired the consumption of each food group with its nutrient density as reported in the Global Expanded Nutrient Supply (GENuS) dataset, a global dataset of nutrient supply of 23 nutrients across 225 food categories for over 150 countries.[4] For our analysis, we aggregated the nutrient dataset to the commodity and regional detail of our consumption data, and we normalised calorie densities to those of the Food and Agriculture Organization for consistency with our diet scenarios. We then compared the calculated nutrient content of the diet scenarios to recommendations of the World Health Organization (WHO) [5,6]. Because the recommendations differ by age and sex, we calculated population-level average values for each nutrient by using the age and sex structure for the year of analysis based on data by the Global Burden of Disease project and forward projections by the Population Division of the United Nations.[7,8] Our estimates of recommended energy intake take into account the age and sex-specific energy needs for a moderately active population of US height as an upper bound,[9,10] and include the energy costs of pregnancy and lactation.[9] Our estimates of calcium intake take into account the average calcium content of drinking water, in line with previous assessments.[11] Because the WHO did not set guidelines for phosphorus and copper, we adopted their recommended intakes from the US Institute of Medicine.

Mortality analysis

To analyse the implications of dietary change for chronic disease mortality, we constructed a comparative risk assessment framework with nine risk factors and five disease endpoints.[12] The risk factors included high consumption of red meat, low consumption of fruits, vegetables, nuts and seeds, fish, and legumes, as well as being underweight (BMI<18.5), overweight (25<BMI<30), and obese (BMI>30). The disease endpoints included coronary heart disease (CHD), stroke, type-2 diabetes mellitus (T2DM), cancer (in aggregate and as site-specific ones, such as colon and rectum cancers), and an aggregate of other causes that are associated with changes in weight. The disease endpoints accounted for about half of all deaths in 2015,[7] and the risk factors were responsible for two thirds of deaths attributable to dietary risk factors in 2015, and for a third of all attributable deaths in that year.[13]

We estimated the mortality and disease burden attributable to dietary risk factors by calculating population impact fractions (PIFs) and applying those to age and country-specific mortality rates.[7,12,13] PIFs represent the proportions of disease cases that would be avoided when the risk exposure was changed from a baseline situation (the benchmark diet) to a counterfactual situation (the dietary scenarios). Relative risk estimates that relate the risk factors to the disease endpoints were adopted from meta-analyses of prospective cohort studies for dietary risks,[14–21] and a pooled cohort study for weight-related risks.[22] In line with the meta-analyses, we included non-linear dose-response relationships for fruits and vegetables,[16] nuts and seeds,[15] and fish,[21] and assumed linear dose-response relationships for the remaining risk factors.[14,17–20] As our analysis was primarily focused on mortality from chronic diseases, we focused on adults aged 20 year or older, and we adjusted the relative-risk estimates for attenuation with age based on a pooled analysis of cohort studies focussed on metabolic risk factors,[23] in line with other assessments.[13,24] In addition to changes in total mortality, we also calculated years of life lost using the standard abridged lifetable from the Global Burden of Disease project.

Uncertainty analysis

In our uncertainty analysis, we accounted for the major uncertainties in each analysis. In the comparative risk analysis, we calculated uncertainty intervals associated with changes in mortality using error propagation and the confidence intervals of the relative risk parameters. In the nutritional analysis, we explicitly calculated low and high supply values of each nutrient based on the reported confidence intervals.

Data availability

The country-level results generated during the current study will be uploaded to the Oxford University Research Archive (ORA) upon acceptance.

Empirical Disease Risk

We applied the Alternate Healthy Eating Index (AHEI) to assess the dietary quality in different countries around the world. The AHEI is based on a combination of food and nutrient variables that have established relationships with the incidence of major chronic disease and mortality.[95-97] For this study, we included 10 out of 11 components of the original AHEI (excluding alcohol intake). The 10-dimensional AHEI ranged from 0 (non-adherence) to 100 (perfect-adherence); each of the components was scored from 0 to 10 (Supplementary Table 6). For fruits, vegetables, whole grains, nuts and legumes, long-chain (n-3) fatty acids (mainly from seafood), and polyunsaturated fats, a higher score indicated higher intake. For *trans* fat, sugar-sweetened beverages and fruit juices, red/processed meat, and sodium, a higher score indicated lower intake. We used data from the Global Dietary Database (GBD) as input in the calculation of AHEI in 187 countries.[98] The GBD compiled data by sex, age and year for 10 foods and 10 nutrients in adults aged 20 years or older based on 325 dietary surveys (including 233 nationally representative surveys) and the UN FAO food balance sheets.[99,100] The estimated sodium intake also incorporated data of urinary sodium from 142 surveys with 24-hour urine collections.[101] We first estimated sex-specific national and global mean intakes by weighting the intake level in each age and sex stratum by the population distribution of each country. We then applied the AHEI scoring criteria to the national and global means to calculate the sex-specific mean AHEI in each country and globally. We also scored the Reference Diet (Supplementary Table 5) based on the AHEI criteria.

Supplementary Table 5. The Alternate Healthy Eating Index scoring method and the Scientific Targets for Healthy Diets.

Component	Criteria for minimum score (0)	Criteria for maximum score (10)	Scientific Targets for Healthy Diets	
			Intake	AHEI
Vegetables,[1] *servings/d*	0	≥5	300 g/d	9.23
Fruit,[2] *servings/d*	0	≥4	200 g/d	7.69
Whole grains,[3] *servings/d*	0		232 g/d	10
Women		5		
Men		6		
Sugar-sweetened beverages and fruit juice,[4] *servings/d*	≥1	0	0 serving/d	10
Nuts and legumes,[5] *servings/d*	0	≥1	125 g/d	10
Red/processed meat,[6] *servings/d*	≥1.5	0	14 g/d	9.07
trans Fat, *% of energy*	≥4	≤0.5	0% of energy	10
Long-chain (n-3) fats (EPA + DHA), *mg/d*	0	250	250 mg/d	10
PUFA, % *of energy*	≤2	≥10	10% of	10
Sodium, *mg/d*	Highest decile	Lowest decile	2300 mg/d	8
Total	0	100		94

We estimated the biological effects of dietary quality, i.e., multivariable-adjusted sex-specific hazard ratios (HRs) per one unit of AHEI on cause-specific mortality (including cancer, cardiovascular disease, respiratory disease, neurodegenerative disease, kidney disease, diabetes, digestive system disease, and other causes except injury and infection), from two ongoing prospective cohorts, the Nurses' Health Study (NHS) [102] of 121,700 women and Health Professionals Follow-Up Study (HPFS) [103] of 51,529 men. The AHEI in the NHS and the HPFS was calculated from dietary information collected using validated semi-quantitative food frequency questionnaires every 2 or 4 years over the follow-up of the two cohorts. We applied Cox proportional hazard models that included AHEI as an exposure variable and cause-specific mortality as outcomes, and simultaneously adjusted for potential confounding variables (including age, total energy intake, ethnicity, marital status, physical activity level, smoking status, alcohol consumption, multivitamin use, current aspirin use, family histories of myocardial infarction, diabetes and cancer, baseline histories of hypertension and hypercholesterolemia, and menopausal status and hormone use in women) to calculate the HRs. These biological effects of dietary quality are likely to represent the best evidence to date on the relationship between long-term dietary intake and health outcomes because of the unique features of the NHS and the HPFS, including repeated and detailed measurements of diet and covariates, extended follow-up, and large sample size. We calculated the population-attributable fraction (PAF) due to a hypothetical improvement in dietary quality from current intake level to the Reference Diet using the comparative risk assessment framework. [104] The calculation of PAF incorporated sex-specific distribution of AHEI in each country and the biological effects of AHEI. The numbers of preventable cause-specific deaths attributed to the hypothetical improvement in dietary quality were calculated by multiplying the cause-specific PAF by the number of deaths due to that cause in each country. The cause-specific deaths in each country were derived from the World Health Organization (WHO) Mortality Database.[105] Because the GBD and WHO Mortality databases only have 118 countries in common, the calculation of preventable deaths was conducted in the 118 countries. To calculate the numbers of preventable total deaths (excluding injury and infection), we summed up the preventable deaths across different causes in each country. The preventable total and cause-specific deaths at global level were calculated by summing up the deaths across different countries. In addition to calculating disease burden associated with the improvement in total AHEI, we also estimate the PAFs and preventable deaths attributable to improvement in each component of AHEI from current intake level to recommended level in the Scientific Targets for Healthy Diets. All the analyses were conducted with SAS version 9.4 (SAS Institute, Cary, NC).

Supplementary Table 6. Examples of the development and applications of the planetary boundaries framework and list of primary data used as part of the evidence base for using the planetary boundaries framework as a guide in this report.

National and regional studies (* = policy-oriented applications)	
Canada	Fanning et al 2016[106]
China	Dearing et al 2014[107]
Finland	*SITRA/Expert Panel on Sustainable Development
Germany	*National Sustainability Strategy and Integrated Environment Programme 2016
South Africa	Cole et al. 2014[108]
Spain	Fanning et al 2016[106]
Sweden	*Swedish Environmental Protection Agency; Nykvist et al 2013
Switzerland	*National Sustainable Development Strategy 2017 Frischnknecht et al 2016[109]; Dao et al[110]
Europe	*EU 7th Environmental Action Plan ("*living well, within limits of the planet*") ESDN/Pisano and Berger 2013 (8 countries)[111] EEA/Häyhä et al 2018[112]
Land, water, climate boundaries for 28 countries	Fang et al 2015[113]
Land, water, climate and biogeochemical flows for 151 countries (also social well-being)	O'Neill et al 2018[114]
Sector studies	
Urban	Hoornweg et al. 2016[115]
Conservation	WWF/IUCN Living Planet Reports 2014, 2016[116,117]
Business and private sector	Whiteman et al. 2013[118] Butz et al. 2018[119] Action2020/World Business Council For Sustainable Development
Food and agricultural systems	Kahiluoto et al. 2015 Jägermeyr, J., et al. 2017[120] Campbell et al., 2017[121]

Textiles sector	Houdini Sportswear (https://www.houdinisportswear.com/en/sustainability/planetary-boundaries)
Links to SDGs	Heck et al. 2018[122]
Processes and interactions studies	
Nexus links and trade-offs - climate/land/water/ecosystems	Hoff 2011[123] Heck et al. 2018[122,124] Erb et al. 2015[124]
N&P	Hoff H. et al. 2015[123] Carpenter and Bennett 2011[125] De Vries et al. 2013[126] Kahiluoto et al. 2014[127]
Water	Gerten D. et al. 2013[128] Bogardi et al. 2013[129]
Biodiversity	Mace et al. 2014[130] Barnosky et al 2012[131] Rothman 2017[132]
Chemical pollution/novel entities	Diamond et al 2015[133] Persson et al 2013[134] Sala and Goralczyk 2013[135] Handoh and Kawai 2011[136]
Earth system	Waters et al 2016[137] Barnosky 2015[131,138]

Countries studied in Fang et al: Australia, Austria, Belgium, Brazil, Canada, China, Denmark, Finland, France, Germany, India, Indonesia, Ireland, Italy, Japan, Mexico, Netherlands, Norway, Poland, Russia, South Africa, South Korea, Spain, Sweden, Switzerland, Turkey, UK, and USA.

Supplementary Panel 2 - Negative emissions

In the Paris Agreement, all countries pledged to keep total global temperature "well below" 2°C and to "pursue efforts to limit the temperature increase even further to 1.5°C". However, all options investigated by the Intergovernmental Panel on Climate Change (IPCC) for keeping the global temperature rise to well below 2°C require using "negative emissions" to remove massive amount of CO_2 from the atmosphere and store it on land, underground, or in the oceans.[139] This was supported by a recent study that showed 1.5°C is achievable, but only by using negative emissions.[140]

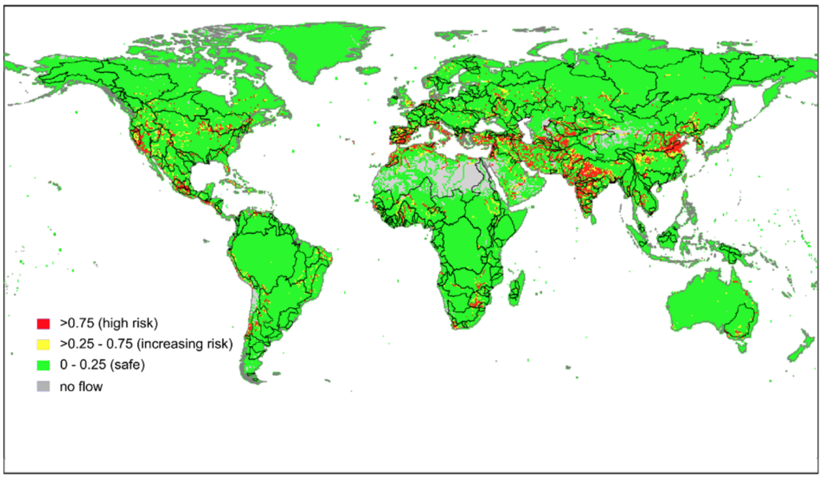
All models for keeping emissions well below 2°C require that global emissions peak by 2020 and decline sharply thereafter, with net CO_2 emissions to zero by 2050 and increasingly negative in the second half of the century. Even with rapid reductions, however, all scenarios considered an overshoot of 1.5°C warming in the 2040s, followed by a decline thereafter as more CO_2 is taken out of the atmosphere using negative emission technologies.[140]

One of the most commonly proposed technologies for removing CO_2 from the atmosphere is bioenergy combined with carbon capture and storage (BECCS). BECCS realises negative emissions by combining cultivation of plant biomass to pull CO_2 from the atmosphere, burning the biomass for energy in power plants and then capturing the CO_2 released during combustion using carbon capture and storage (CCS) technologies. The captured CO_2 is then stored in underground reservoirs.

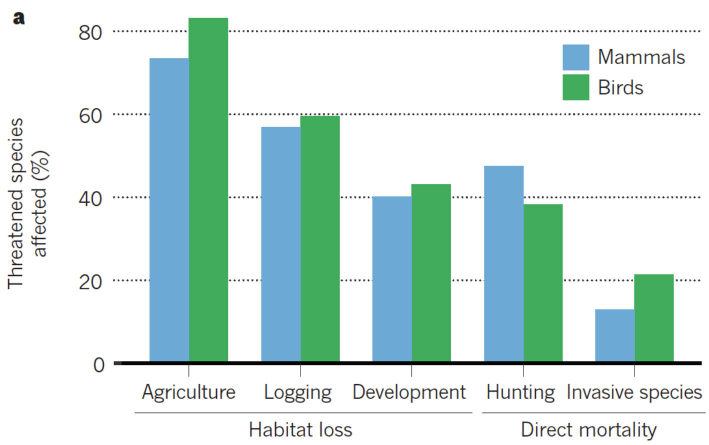
However, full scale implementation of BECCS has major implications for land use and food production. In most integrated assessment models, land use for BECCS is minor in 2050, but by 2080 will rise to 430-580 million ha globally, a third of arable land.[141] Land use for large scale implementation of BECCS would compete for both land and water needed for food production, as well as land needed for biodiversity.[142]

Agriculture offers promising potential for removing CO_2 from the atmosphere. With nearly 40% of the terrestrial land surface occupied by crop and pasture lands, agricultural land remains the largest land surface available for sequestering carbon. Removal of CO_2 from the atmosphere and its storage in both soils and above-ground biomass in croplands, pasture and agroforestry systems could become an important carbon sink. The global potential for carbon removals into soils under cropland is estimated at 3.1 to 6.8 Gt CO_2-eq yr^{-1} [143], while above-ground biomass in trees on farms provides further global potential of 1 Gt CO_2-eq yr^{-1} carbon sequestration (95% CI: 0.5–1.4)[144].

Forestry has an even higher potential than agriculture for storage of existing carbon and removals of atmospheric CO_2. Halting food production's role in forest clearance and land degradation can leverage large-scale carbon stocks in natural systems. For example, reforestation and afforestation on degraded rangelands offer a large mitigation potential of 10 Gt CO_2-eq yr^{-1} (95% CI: 2–17), while halting forest conversion adds a further 3.6 Gt (95% CI: 3.0–4.2).[144] In several regions, land expansion has markedly reversed and natural restoration of forests has occurred, such as the hardwood forests of the eastern United States.[145] Carbon storage in agriculture and forestry is limited by saturation, however, because carbon uptake rates slow down as vegetation matures and soil organic carbon reaches a maximum.



Supplementary Figure 3. Transgression of the allowed monthly water withdrawals as % of mean monthly river flow (fraction of maximum allowed level) during months that show such an exceedance. For example, green (within planetary boundary for water use) means that average exceedance in the respective months is still below the uncertainty range. Source: Steffen et al.[146]



Supplementary Figure 4. Relative impact of agriculture and other activities on mammals and bird species threatened with extinction based on IUCN extinction risks. Source: Tilman et al.[147]

Descriptions of global biomes assessed in Figure 3

Brief descriptions of each biome taken and abbreviated from the WWF biome descriptions (https://www.worldwildlife.org/biomes/):

Tropical and subtropical moist broadleaf forests are found in large, discontinuous patches centered on the equatorial belt and between the Tropics of Cancer and Capricorn, Tropical and Subtropical Moist Forests (TSMF) are characterized by low variability in annual temperature and high levels of rainfall (>200 centimeter annually). Forest composition is dominated by semi-evergreen and evergreen deciduous tree species.

Tropical and subtropical dry broadleaf forests occur in climates that are warm year-round, and may receive several hundred centimeters or rain per year, they deal with long dry seasons which last several months and vary with geographic location. Deciduous trees predominate these forests, and during the drought a leafless period occurs, which varies with species type.

Tropical and Subtropical Coniferous forests are found predominantly in North and Central America, these tropical regions experience low levels of precipitation and moderate variability in temperature. Tropical and Subtropical Coniferous Forests are characterized by diverse species of conifers, whose needles are adapted to deal with the variable climatic conditions.

Temperate Broadleaf and Mixed forests experience a wide range of variability in temperature and precipitation. In regions where rainfall is broadly distributed throughout the year, deciduous trees mix with species of evergreens. Structurally, these forests are characterized by 4 layers: a canopy composed of mature full-sized dominant species and a slightly lower layer of mature trees, a shrub layer, and understory layer of grasses and other herbaceous plants.

Temperate Conifer Forests are found predominantly in areas with warm summers and cool winters, and vary enormously in their kinds of plant life. In some, needleleaf trees dominate, while others are home primarily to broadleaf evergreen trees or a mix of both tree types.

Boreal Forests/Taiga are characterized by low annual temperatures of northerly latitudes; precipitation ranges from 40-100 centimeters per year and may fall mainly as snow. This combination, along with nutrient poor soils favors the preponderance of conifer species although species of deciduous trees are also rather common. Ground cover in Boreal Forests and Taiga is dominated by mosses and lichens.

Tropical and Subtropical grasslands and savannas are large expanses of land in the tropics that do not receive enough rainfall to support extensive tree cover. The Tropical and Subtropical Grasslands, Savannas, and Shrublands are characterized by rainfall levels between 90-150 centimeters per year. Grasses dominate the species composition of these ecoregions, although scattered trees may be common.

Montane grasslands and shrublands includes high elevation (montane and alpine) grasslands and shrublands. They are tropical, subtropical, and temperate. The plants and animals of tropical montane paramos display adaptations to cool, wet conditions and intense sunlight.

Tundra is a treeless polar desert found in the high latitudes in the polar regions. The region's long, dry winters feature months of total darkness and extremely frigid temperatures. Structurally, the Tundra is a treeless expanse that supports communities of sedges and heaths as well as dwarf shrubs. Most precipitation falls in the form of snow during the winter while soils tend to be acidic and saturated with water where not frozen.

Mediterranean Forests, Woodlands, and Scrub are characterized by hot and dry summers, while winters tend to be cool and moist. Only 5 regions in the world experience these conditions: the Mediterranean, south-central and southwestern Australia, the fynbos of southern Africa, the Chilean matorral, and the Mediterranean ecoregions of California. Most plants are fire adapted, and dependent on this disturbance for their persistence.

Deserts and Xeric Shrublands vary greatly in the amount of annual rainfall they receive; generally, however, evaporation exceeds rainfall in these biomes, usually less than 10 inches annually. Temperature variability is also extremely diverse.

Mangroves occur in the waterlogged, salty soils of sheltered tropical and subtropical shores. They are subject to the twice-daily ebb and flow of tides, fortnightly spring and neap tides, and seasonal weather fluctuations. They stretch from the intertidal zone up to the high-tide mark.

Methods for Chapter 4

Food systems model and scenarios
The analysis contained in chapter 4 extends the analysis by Springmann and colleagues with additional scenarios and sensitivity analyses.[148] Below we detail the methods used.

Food systems model
For our analysis, we constructed a food systems model that connects food consumption and production across regions. The model is based on the database and model equations of the International Model for Policy Analysis of Agricultural Commodities and Trade (IMPACT).[1] The IMPACT model projects food production and demand until 2050 for 62 agricultural commodities and 159 countries. The projections are based on statistical association with changes in income and population, and are in line with other projections.[2]

Because we were interested in analysing the environmental impacts associated with specific dietary scenarios, we reformulated the IMPACT model such that food demand is an input parameter and food production is an output. For that purpose, we distinguished several steps along the food chain, starting from trade in processed commodities and animals, feed demand for animals, demand of primary commodities to process oils and refined sugar, trade in primary commodities, and primary production, including non-food uses, e.g. in industry. Below we summarise the main model equations. A full description of the IMPACT-related parameters is provided elsewhere.[1]

Starting from final consumption demand ($QD_{c,r}^{cns}$) for commodity *c* in region *r*, we first add demands other than food demand, in particular stock variation, seed demand, and demand for industrial use ($QD_{c,r}^{oth}$), as well as demand for biofuels ($QBF_{c,r}$):

$$QD_{c,r}^{cns+oth} = QD_{c,r}^{cns} + QD_{c,r}^{oth} + QBF_{c,r}$$

Then we calculate the feed demand that supports the consumption of animal-based foods in the specific dietary scenarios. Because feed requirements differ by region, we first estimate where livestock is produced by accounting for trade flows ($QL_{c,r}^{trd} = QL_{c,r} - QL_{c,r}^{imp} + QL_{c,r}^{exp}$). For that purpose, we use import-to-demand fractions ($FI_{c,r} = \frac{QI_{c,r}}{QD_{c,r}^{cns+oth}}$) to calculate the percentage of livestock that is imported ($QL_{c,r}^{imp}$), and balance imports with exports ($QL_{c,r}^{\exp}$) in line with projected imports and exports ($QI_{c,r}$, $QE_{c,r}$) by using the ratio of regional exports to all exports ($FE_{c,r} = \frac{QE_{c,r}}{\sum_r QE_{c,r}}$), a method that implicitly assumes that in each dietary scenarios, current exporters stay exports, and current importers stay importers. Feed demand ($QF_{c,r}$) is then calculated in relation to regional feed requirements ($FR_{c,r}$):

$$QL_{c,r}^{imp} = FI_{c,r} \cdot QL_{c,r}$$
$$QL_{c,r}^{\exp} = FE_{c,r} \cdot \sum_r QL_{c,r}^{imp}$$
$$QF_{c,r} = FR_{c,r} \cdot QL_{c,r}^{trd}$$

Next we calculate the intermediate demand for primary commodities that supports the consumption of processed goods (vegetable oils, oil meals, refined sugar) in the dietary scenarios. For that purpose, we first adjust the mix of intermediate processed commodities for trade ($P_{c,r}^{trd} = QP_{c,r} - QP_{c,r}^{imp} + QP_{c,r}^{exp}$), and then use region-specific processing factors for oils and sugar ($PF_{c,r}$) to calculate the demand for primary commodities (oil crops, sugar crops):

$$QInt_{c,r} = PF_{c,r} \cdot QP_{c,r}^{trd}$$

Finally, we account for trade in those primary commodities that satisfy the demand for processing ($QInt_{c,r}^{trd} = QInt_{c,r} - QInt_{c,r}^{imp} + QInt_{c,r}^{exp}$), in feed that consists of primary commodities ($QF_{c,r}^{trd} = QF_{c,r} - QF_{c,r}^{imp} + QF_{c,r}^{exp}$), and in the primary commodities that are demanded in unprocessed form ($QD_{c,r}^{cns+oth,trd} = QD_{c,r}^{cns+oth} - QD_{c,r}^{cns+oth,imp} + QD_{c,r}^{cns+oth,exp}$). The production of primary commodities is then given by the sum of:

$$QS_{c,r} = QD_{c,r}^{cns+oth,trd} + QF_{c,r}^{trd} + QInt_{c,r}^{trd} - QL_{c,r} - QP_{c,r}$$

Environmental accounts

To assess the environmental impacts of the food system, we paired the food system model with a set of country-specific environmental footprints related to GHG emissions, cropland use, bluewater use, and nitrogen and phosphorus application. In line with projections of the allowable agricultural emissions budget,[3] and our separate treatment of land use, we focused on the non-CO_2 emissions of agriculture, in particular methane and nitrous oxide. Data on GHG emissions were adopted from country-specific analyses of GHG emissions from crops,[4] and livestock.[5] Non-CO_2 emissions of fish and seafood were calculated based on feed requirements and feed-related emissions of aquaculture,[6] and on projections of the ratio between wild-caught and farmed fish production.[7,8] Our baseline emissions estimate agrees well with existing ones that follow the same methodology.[9,10]

Data on cropland and consumptive bluewater use were adopted from the IMPACT model.[1] To derive commodity-specific footprints, we divided use data by data on primary production, and we calculated the footprints of processed goods (vegetable oils, refined sugar) by using country-specific conversion ratios,[1] and splitting coproducts (oils and oil meals) by economic value to avoid double counting. We used country-specific feed requirements for terrestrial animals [1] to derive the cropland and bluewater footprints for meat and dairy, and we used global feed requirements for aquaculture [6] and projections of the ratio between wild-caught and farmed fish production [7,8] to derive the cropland and bluewater footprints for fish and seafood.

As control variable for nitrogen-related pollution, we used the surplus of reactive nitrogen (labelled nitrogen application in text), a measure that accounts for all inputs and offtakes of nitrogen.[15] For that purpose, Springmann et al [148] constructed a region-specific nitrogen budget module based on Lassaletta and colleagues.[13,16] Data on fertilizer application rates of nitrogen and phosphorous were adopted from the International Fertilizer Industry Association.[12] Data on symbiotic fixation rates were adapted from Lassaletta and colleagues [13,14]. In a sensitivity analysis, we also analysed the impact in terms of direct nitrogen application (synthetic N application plus N fixation by legumes) and found generally small differences between using nitrogen application and nitrogen surplus as control variables at a global level.[148]

Scenarios

We used the food system model to estimate the environmental impacts of the food system in 2050 on GHG emissions, cropland use, bluewater use, and nitrogen and phosphorus application. For estimating the environmental impacts in absence of dedicated mitigation measures (a scenario we term business-as-usual projection), we paired footprints of current intensity to future projections of food demand along a middle-of-the-road socio-economic development pathway (SSP2).[17–19] Additional socio-economic pathway, including a more optimistic pathway with higher income and lower population growth (SSP1), and a more pessimistic pathway with lower income and greater population growth (SSP3) are analysed elsewhere.[148]

We then analysed the option space for reducing the environmental pressures of the food system by constructing scenarios of changes in food loss and waste, technological change, and dietary change (Table 1). Estimates of food loss and waste were based on percentage values reported by the FAO [20]. In the scenario focused on food loss and waste (*waste/2*), we assumed that food losses at the production side and food waste at the consumption side are reduced by half, a goal in line with the Sustainable Development Goals for 2030.

The scenarios of technological change include projected efficiency gains in emissions intensities, agricultural yields, feed conversion, water use, and nitrogen and phosphorus application. In our analysis of technological measures, we differentiate between measures of medium and high ambition (*tech*, *tech+*). For the scenarios describing changes in emissions intensities of foods, we incorporated the mitigation potential of bottom-up changes in management practices and technologies by using marginal abatement cost curves [21] and the value of the social cost of carbon (SCC) in 2050.[22] The mitigation options included changes in irrigation, cropping and fertilization that reduce methane and nitrous oxide emissions for rice and other crops, as well as changes in manure management, feed conversion and feed additives that reduce enteric fermentation in livestock. We used SCC values of 72 USD/tCO_2 (associated with a rate of discounting future climate damages by 3%) for the scenario of medium ambition (*tech*), and implemented all available mitigation options (equivalent to using a SCC of above 99 USD/tCO_2-eq) for the scenario of high ambition (*tech+*).

Efficiency gains in agricultural yields, water management, and feed conversion were based on IMPACT projections.[1] For water management, we relied on an integrated hydrological model within IMPACT that operates at the level of watersheds and accounts for management changes that increase basin efficiency, storage capacity, and better utilization of rainwater.[1] For agricultural yields, the gains in land-use efficiency matched estimates of yield-gap closures of about 75% between current yields and yields that are feasible in a given agro-climatic zone.[23] The potential efficiency gains in nitrogen and phosphorus application rates included rebalancing of fertilizer application rates between over and under-applying regions in line with closing yield gaps.[23] In the ambitious technology scenario (*tech+*), we increased yield-gap closures to 90% based on data by Mueller and colleagues,[23] and assumed additional improvements in nitrogen use efficiency of 30%, in line with targets suggested by the Global Nitrogen Assessment,[24] and a recycling rate of phosphorus of 50%.[25]

The scenarios of dietary change include shifts towards nutritionally balanced dietary patterns that reflect the current evidence on healthy eating.[26–28] The scenarios include the reference diet outlined in chapter 1, as well as more specialised dietary patterns, including pescatarian, vegetarian, and vegan diets. We aimed to preserve the regional character of each dietary pattern by maintaining the regional composition of specific foods within broader categories, such as preferences for specific staple crops (wheat, maize, rice, etc) and fruits (temperate, tropical). Baseline intakes of food and energy were calculated from food availability projections of the IMPACT model by using region-specific factors of food waste and ratios of the edible portions of foods.[20]

The reference diet includes at least 500 g/d of fruits and vegetables of different colours and groups (the composition of which is determined by regional preferences), at least 100 g/d of plant-based protein sources (legumes, soybeans, nuts), modest amounts of animal-based proteins, such as poultry, fish, milk, and eggs, and limited amounts of red meat (1 portion per week), refined sugar (<5% of total energy), vegetable oils that are high in saturated fat (in particular palm oil), and starchy foods which have a relatively high glycaemic index.

Based on the reference diet, we constructed the more specialised diet scenarios in line with dietary guidelines and observed dietary patterns in specialised cohorts.[29,30] For the pescatarian diets, meat-based protein sources in the flexitarian diets were replaced (on a kcal basis) to two thirds by fish and seafood, and one third by fruits and vegetables; for the vegetarian diets, they were replaced to two thirds by plant-based proteins, and one third by fruits and vegetables; and for the vegan diets, all animal-based protein sources were replaced to two thirds by plant proteins, and one third by fruits and vegetables.

The main analysis of this report focuses on changes in dietary composition and only moderately constrain total energy intake to 2500 kcal/d. An analysis that takes into account global recommendations on bodyweight and physical activity levels is described elsewhere in full.[148] Here we consider a scenario that limits total energy intake to recommended levels as a sensitivity analysis. For that purpose, we used estimates of energy intake based on the calorie needs of a moderately active population of US characteristics for height divided into 5-year age groups [31], something that can be seen as an upper bound. Calorie needs reach a maximum of 2500 kcal/d for ages 19-25 (averaged between men and women), but are reduced to 2000 kcal for ages 66 and older. The average calorie needs differed by region based on its age composition, and ranged around 2100 kcal/d.

Data availability
The results generated during the current study will be uploaded to the Oxford University Research Archive (ORA) upon acceptance.

Biodiversity analysis
To project species extinctions due to human land use within a region, ecological models such as species-area relationship (SAR) have often been employed, which in turn can inform conservation intervention (Chaudhary & Brooks, 2017). Given the current and original (before human intervention) extent of the natural habitat in a region, the SARs project the eventual (equilibrium) number of extinctions that will take place (that might take decades or centuries to unfold) given no mitigation measures are taken and current land use mix stays the same. Species extinctions lag behind land use changes or habitat degradation by community "relaxation" period, during which species progressively disappear over time (Wearn et al., 2012). During this time delay ('window of conservation opportunity', Wearn et al., 2012), it is possible to take conservation actions such as restoring the habitat through reforestation to ensure species that are otherwise "committed" to extinction are saved. The projections from SARs thus can help flag the global regions where a high number of species extinctions are expected in the near future and where conservation actions are needed. The SARs can also be employed to project and compare the biodiversity outcomes of alternative future land use pathways.

Countryside species-area relationship (SAR)
For each taxonomic group g, countryside SAR predicts the number of species loss (S_{lost}) caused by all (cumulative) land uses within a region j as (Chaudhary & Brooks, 2017):

$$S_{lost,g,j}^{regional} = S_{org,g,j} - S_{org,g,j} \cdot \left(\frac{A_{new,j} + \sum_{i=1}^{n} h_{g,i,j} \cdot A_{i,j}}{A_{org,j}}\right)^{z_j} \qquad (1)$$

Here $S_{org,g,j}$ is the original number of species of taxon g (g =1:3; mammals, birds and amphibians) occurring in the ecoregion before any human intervention, $A_{new,j}$ is the natural habitat area in the ecoregion currently (m^2), $A_{i,j}$ is the current area of land use type (i =1:4; cropland, pasture, urban and secondary vegetation) in m^2, $A_{org,j}$ is the total ecoregion area, z_j is the SAR exponent for the ecoregion and $h_{g,i,j}$ is the affinity of taxon g to the land use type i in ecoregion j. Note that, $S_{new,g,j} = S_{org,g,j} - S_{lost,g,j}$ is the equilibrium number of species that would eventually remain if land use change ceased at current levels and no conservation/mitigation measures are adopted. Also, in order to predict permanent extinctions, one needs to replace the species richness ($S_{org,g,j}$) by the number of endemic species ($S_{end,g,j}$) in the region (Chaudhary & Brooks, 2017; Chaudhary & Kastner, 2016; Chaudhary et al. 2017).

Traditionally used classic SAR (Brooks et al. 2002) is a special case of countryside SAR, when h = 0, i.e. the converted land use is totally hostile and assumed to not host any species. Unlike classic SAR that assumes no species can survive in human-modified landscapes and therefore often overestimates the extinctions due to land use change, countryside SAR accounts for the fact that some species are tolerant to human land uses. Chaudhary & Brooks (2017) recently showed that countryside SAR performs better than classic SAR in predicting species extinctions for 804 terrestrial ecoregions.

Species extinctions due to conversion of an additional m^2 of land
We first calculate the characterization factors (CFs) providing number of species projected to go extinct due to conversion of one additional m^2 of primary habitat (e.g. natural forest) into cropland in each of the

804 terrestrial ecoregion j (Olson et al. 2001) by taking the partial differentiation of countryside SAR with respect to area of land use *i.* (Chaudhary et al. 2015).

$$CF_{crop,j} = \frac{\partial S_{loss,g,j}}{\partial A_{crop,j}} = \frac{\left(1 - h_{g,crop,j}\right) \cdot S_{end,g,j} \cdot z_j}{A_{org,j}} \cdot \left[\left(\frac{A_{new,j} + \sum_{i=1}^{4} h_{g,i,j} \cdot A_{i,j}}{A_{org,j}}\right)^{z_j - 1}\right] \quad (2)$$

We obtained number of endemic species ($S_{end,g,j}$) per ecoregion from IUCN species range maps (IUCN, 2017) and the z-values (z_j) from Drakare et al. (2006). The area parameters per ecoregion ($A_{org,j}$, $A_{new,j}$ and $A_{i,j}$) were derived by overlaying global land use map of Hoskins et al. (2016) with ecoregion boundaries and the taxon affinity to different land uses in each ecoregion ($h_{g,i,j}$) were derived from species habitat classification scheme of IUCN Red List (see Chaudhary & Brooks, 2017 for details on model parameterization). Reptiles and plants were excluded from these analyses because not all species have their range maps available through IUCN. Further, we calculate country-specific CFs by weighting the ecoregion CFs with the area of each land use type within each country's different ecoregions.

Finally, the country-specific CFs calculated above are multiplied with projected cropland expansion area per country till 2050 (obtained from IMPACT model in m^2) to derive number of endemic species projected to go extinct in each country.

Using above model, it is possible that that some nations will show biodiversity gains if instead of expansion, the net cropland area is reduced due to abandonment of cropland used currently. In these cases, instead of negative species extinctions, we set the species gains equal to zero following van Vuuren et al. (2006). In other words, we assume that the SAR can be applied in one direction only, i.e. habitat loss leads to inferred extinction of species, but an abandonment of human land use and the consequent increase in regenerating area does not lead to a similar increase in species, as timescales examined are extremely short (few decades) compared to evolutionary timescale (many centuries).

We also calculate CFs for an additional scenario where instead of natural (primary) undisturbed habitat, cropland expansion is assumed to occur at the cost of existing secondary vegetation by replacing $\left(1 - h_{g,crop,j}\right)$ above with $\left(h_{g,secveg,j} - h_{g,crop,j}\right)$. In Supplementary Table 9 we combine the scenarios of cropland expansion on natural and managed habitat. For that purpose, we used global GIS data on the extend of total and pristine forests to inform the extend by which cropland can be expanded to managed forests.

Global changes in biodiversity loss do not necessarily agree well with global changes in cropland change, because it is the specific location of cropland change that matters for biodiversity. To show that closer alignment is possible, we devised another set of runs in which we optimize regional land-use changes for biodiversity conservation. For that purpose, we constructed an optimization algorithm which reallocated crop production amongst countries such that biodiversity loss was minimized, subject to suitability and production constraints. The suitability constraint only allowed production to increase for a certain crop where that crop has been previously produced. The production constraints included limiting expansion of projected production for specific crops in 2050 to 30%, and keeping total production in a country to below its arable land area.

We compare the projected rate of endemic extinctions of mammals, birds and amphibians in the units 'extinctions per million species years (E/MSY)' for the period 2005-2050 with their recent rate of extinctions (period 1500-2000) as reported by Pimm et al. (2014) and Ceballos et al. (2015). The recent rate of extinctions for the period 1500-2000 vary from 10-50 E/MSY for the three species groups (Ceballos et al. 2015).

The species extinction numbers we calculate can be considered as an underestimation due to three main factors. First, our projected extinctions are based on power-law countryside species area relationship that does not account for the effects of habitat fragmentation that usually accompanies habitat loss (Hanski et al., 2013) or the effects of geometry of area loss (Keil et al., 2015). Second, we do not account for the impact that agricultural inputs and runoff (e.g. fertilizer, pesticides, etc.) would have on on-farm and off-farm biodiversity. Finally, we only calculate the endemic species extinctions but it might be that cropland expansion leads to habitat loss of certain non-endemics in each ecoregion that they occur in. The SAR approach that we apply is not able to quantify such extinctions (Chaudhary & Brooks, 2017). Accounting for these three additional factors would increase our estimates of global biodiversity loss due to cropland expansion in 2050.

Supplementary Table 7. Environmental impact per serving of major food groups (global averages). GHG emissions include CH_4 and N_2O. The environmental footprints for livestock and fish/seafood relate to the impacts of feed, with the exception of GHG emissions for which livestock has a direct component. The footprints used in the analysis differ by region. Colours indicate environmental impacts from low – green to light green; medium – orange; high - red.

Food item	GHG emissions ($10kgCO_2$/serving)	Cropland use ($10m^2$/serving)	Water use ($10m^3$/serving)	Nitrogen use (10gN/serving)	Phosphorus use (10gP/serving)
wheat	0.10	1.51	0.22	12.93	1.98
rice	0.53	1.58	0.48	16.49	2.34
maize	0.08	0.89	0.07	10.25	1.60
other grains	0.13	2.76	0.07	7.36	1.22
roots	0.08	0.76	0.05	3.99	0.78
legumes	0.08	3.86	0.33	0.00	0.00
soybeans	0.04	1.38	0.05	0.96	2.06
nuts & seeds	0.21	1.92	0.13	4.28	0.63
vegetables	0.05	0.41	0.07	8.12	1.42
fruits (temperate)	0.11	1.65	0.47	17.82	2.67
fruits (tropical)	0.13	1.32	0.45	14.38	2.21
fruits (starchy)	0.15	1.18	0.16	8.76	1.50
sugar	0.01	0.07	0.05	0.89	0.15
palm oil	0.26	0.43	0.00	3.13	0.50
vegetable oil	0.09	1.44	0.07	5.98	1.61
beef	35.74	4.64	0.24	30.01	5.89
lamb	36.33	6.86	0.54	30.27	5.43
pork	3.21	6.69	0.38	56.68	9.75
poultry	1.55	7.25	0.44	55.22	9.92
eggs	0.79	3.43	0.22	25.61	4.40
milk	2.93	3.21	0.19	15.18	3.79
shellfish	0.08	0.40	0.04	3.69	0.89
fish (freshwater)	0.33	1.66	0.11	18.46	3.98
fish (demersal)	0.02	0.14	0.01	1.32	0.32
fish (pelagic)	0.00	0.00	0.00	0.00	0.00

Supplementary Panel 3. Livestock on leftovers

Most environmental life cycle assessments conclude that products from ruminants (e.g. cattle, buffalo, sheep, and goats) are the most GHG emissions intensive of all animal products, and that ruminants that are raised on grass (i.e. grazing animals) are the highest emitters of methane and use vast tracts of land. But it has been argued that this conclusion is overly simplistic and based on a narrow set of metrics – such as GHG emissions per unit of meat or milk output. While emissions and overall land use may be high for grazing animals, ruminants can be reared on land unsuited for other food producing purposes and on by-products from crop production. In addition, in mixed farming systems the animals recycle nutrients and re-fertilise soils with their dung, thus fostering a new generation of crops and pasture. In contrast animals reared in intensive systems, and particularly monogastrics such pigs and poultry are fed grains whose production requires quality arable land that could instead be used to feed humans.[149] When measuring cropland environmental footprints, poultry has higher footprints than cows for land and water use, and N/P pollution because of the amount of grain used globally to feed poultry.[150] This is despite the much higher feed-conversion ratios for cows.

Several studies[151-153] have explored how much animal protein from ruminants and monogastrics might be available to feed a global population of 9 billion in 2050 if a ‘livestock on leftovers’[154] approach were adopted. This approach limits the availability of animal protein globally to what can be produced by raising animals on a) grassland unsuited to crop production; b) by-products arising from agricultural crop production; and c) food waste. The studies make slightly different assumptions but nevertheless, they all yield an approximately similar answer to 'how much' animal protein could be made available globally using a 'livestock on leftovers' approach.

The per capita availability of animal protein these studies estimate varies from 11 to 32 g/person/day. The mean is about 21 g protein/person/day, which is approximately 100 g of raw bone-free meat/person/day but no milk – or 50 g of meat and 300 ml of milk. These are the figures before allowing for losses and waste so actual availability may be lower. The availability of ruminant protein specifically from grazing only systems (e.g. grass-fed beef) is substantially lower, amounting to between 7–19 g protein/person/day or approximately 65 g of raw bone-free meat. However, all of these estimates of animal protein availability using a 'livestock on leftovers' approach to feed a global population of 9 billion people fall within the ranges for the reference diet (Table 1).

Supplementary Table 8. Scenario analysis of biodiversity impacts (species loss per million species years). The colours illustrate whether environmental impacts transgress food production boundaries: green - below lower range value; light green - below or equal to boundary but above lower range value; orange - above boundary but below upper range value; red – above upper range value.

			Energy intake of 2500 kcal/d				Energy intake of 2100 kcal/d			
Production (2050)	Waste (2050)	Diet (2050)	Expansion to natural habitats		Expansion to managed habitats		Expansion to natural habitats		Expansion to managed habitats	
			BMK	OPT	BMK	OPT	BMK	OPT	BMK	OPT
BAU	full waste	BAU	1067	153	36	2	1067	153	36	2
BAU	full waste	reference	1309	145	45	2	994	120	34	2
BAU	full waste	pescatarian	1313	143	46	2	1002	118	35	2
BAU	full waste	vegetarian	1374	148	48	2	1062	122	37	2
BAU	full waste	vegan	1431	152	50	2	1125	128	39	2
BAU	halve waste	BAU	716	105	24	1	716	105	24	1
BAU	halve waste	reference	940	100	32	2	647	81	22	1
BAU	halve waste	pescatarian	940	97	33	2	652	78	22	1
BAU	halve waste	vegetarian	1000	102	35	2	713	83	24	1
BAU	halve waste	vegan	1051	104	36	2	772	90	26	1
PROD	full waste	BAU	237	68	7	1	237	68	7	1
PROD	full waste	reference	414	62	14	1	371	54	13	1
PROD	full waste	pescatarian	426	61	15	1	390	54	14	1
PROD	full waste	vegetarian	462	63	15	1	427	56	15	1
PROD	full waste	vegan	507	66	17	1	475	59	16	1
PROD	halve waste	BAU	103	41	3	0	103	41	3	0
PROD	halve waste	reference	270	38	9	1	246	33	8	1
PROD	halve waste	pescatarian	281	38	9	1	262	34	9	1
PROD	halve waste	vegetarian	317	40	10	1	299	36	10	1
PROD	halve waste	vegan	358	44	12	1	342	40	11	1
PROD+	full waste	BAU	292	61	10	1	292	61	10	1
PROD+	full waste	reference	414	56	14	1	252	47	8	1
PROD+	full waste	pescatarian	424	54	15	1	266	46	9	1
PROD+	full waste	vegetarian	456	55	16	1	301	47	10	1
PROD+	full waste	vegan	494	56	17	1	346	49	12	1
PROD+	halve waste	BAU	196	38	7	0	196	38	7	0
PROD+	halve waste	reference	290	34	10	0	170	28	5	0
PROD+	halve waste	pescatarian	298	32	10	0	181	27	6	0
PROD+	halve waste	vegetarian	330	34	11	0	215	29	7	0
PROD+	halve waste	vegan	366	37	12	0	259	33	8	0

Supplementary Table 9. Biodiversity loss presented as a single data set where PROD scenarios assume that land is expanded first into secondary habitat (eg, logged forests and plantations) or other managed ecosystems (eg, pastures and rangelands) and then to intact forests. For PROD+ we assumed that land use is optimized across regions such that it minimizes impacts on biodiversity with the constraint that not more land can be used than is arable in a given country, and that production of a specific crop in a country cannot increase by more than 30% relative to the benchmark value for that crop, country and year.

Production (2050)	Waste (2050)	Diet (2050)	Biodiversity loss (E/MSY)
Food Production Boundary			**10 (1-80)**
Baseline in 2010			**100-1000**
BAU	full waste	BAU	1,043
BAU	full waste	reference	1,270
BAU	full waste	pescatarian	1,266
BAU	full waste	vegetarian	1,324
BAU	full waste	vegan	1,362
BAU	halve waste	BAU	684
BAU	halve waste	reference	885
BAU	halve waste	pescatarian	873
BAU	halve waste	vegetarian	932
BAU	halve waste	vegan	960
PROD	full waste	BAU	206
PROD	full waste	reference	351
PROD	full waste	pescatarian	349
PROD	full waste	vegetarian	382
PROD	full waste	vegan	400
PROD	halve waste	BAU	50
PROD	halve waste	reference	102
PROD	halve waste	pescatarian	98
PROD	halve waste	vegetarian	129
PROD	halve waste	vegan	140
PROD+	full waste	BAU	37
PROD+	full waste	reference	34
PROD+	full waste	pescatarian	29
PROD+	full waste	vegetarian	29
PROD+	full waste	vegan	28
PROD+	halve waste	BAU	21
PROD+	halve waste	reference	19
PROD+	halve waste	pescatarian	14
PROD+	halve waste	vegetarian	15
PROD+	halve waste	vegan	13

Supplementary References

1. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and mortality: results from 2 prospective cohort studies. *Arch Intern Med* 2012; **172**(7): 555-63.
2. Bernstein AM, Pan A, Rexrode KM, et al. Dietary protein sources and the risk of stroke in men and women. *Stroke* 2012; **43**(3): 637-44.
3. Bernstein AM, Sun Q, Hu FB, Stampfer MJ, Manson JE, Willett WC. Major dietary protein sources and risk of coronary heart disease in women. *Circulation* 2010; **122**(9): 876-83.
4. Sinha R, Cross AJ, Graubard BI, Leitzmann MF, Schatzkin A. Meat intake and mortality: a prospective study of over half a million people. *Arch Intern Med* 2009; **169**(6): 562-71.
5. Sánchez-Pimienta TG, Batis C, Lutter CK, Rivera JA. Sugar-Sweetened Beverages Are the Main Sources of Added Sugar Intake in the Mexican Population–5. *The Journal of nutrition* 2016; **146**(9): 1888S-96S.
6. Rivera JA, Pedraza LS, Aburto TC, et al. Overview of the Dietary Intakes of the Mexican Population: Results from the National Health and Nutrition Survey 2012–3. *The Journal of nutrition* 2016; **146**(9): 1851S-5S.
7. Aburto TC, Pedraza LS, Sánchez-Pimienta TG, Batis C, Rivera JA. Discretionary Foods Have a High Contribution and Fruit, Vegetables, and Legumes Have a Low Contribution to the Total Energy Intake of the Mexican Population–4. *The Journal of nutrition* 2016; **146**(9): 1881S-7S.
8. Appel LJ, Sacks FM, Carey VJ, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *Jama* 2005; **294**(19): 2455-64.
9. Orlich MJ, Singh PN, Sabate J, et al. Vegetarian dietary patterns and mortality in Adventist Health Study 2. *JAMA Intern Med* 2013; **173**(13): 1230-8.
10. Satija A, Bhupathiraju SN, Rimm EB, et al. Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: results from three prospective cohort studies. *PLoS Med* 2016; **13**(6): e1002039.
11. Satija A, Bhupathiraju SN, Spiegelman D, et al. Healthful and unhealthful plant-based diets and the risk of coronary heart disease in U.S. Adults. *J Am Coll Cardiol* 2017; **70**(4): 411-22.
12. Abete I, Romaguera D, Vieira AR, Lopez de Munain A, Norat T. Association between total, processed, red and white meat consumption and all-cause, CVD and IHD mortality: a meta-analysis of cohort studies. *Br J Nutr* 2014; **112**(5): 762-75.
13. Chen GC, Lv DB, Pang Z, Liu QF. Red and processed meat consumption and risk of stroke: a meta-analysis of prospective cohort studies. *Eur J Clin Nutr* 2013; **67**(1): 91-5.
14. Feskens EJ, Sluik D, van Woudenbergh GJ. Meat consumption, diabetes, and its complications. *Curr Diab Rep* 2013; **13**(2): 298-306.
15. Etemadi A, Sinha R, Ward MH, et al. Mortality from different causes associated with meat, heme iron, nitrates, and nitrites in the NIH-AARP Diet and Health Study: population based cohort study. *BMJ* 2017; **357**: j1957.
16. 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. *The American journal of clinical nutrition* 2011; **94**(4): 1088-96.
17. Pan A, Sun Q, Bernstein AM, Manson JE, Willett WC, Hu FB. Changes in red meat consumption and subsequent risk of type 2 diabetes mellitus: three cohorts of US men and women. *JAMA Intern Med* 2013; **173**(14): 1328-35.
18. Kromhout D, Keys A, Aravanis C, et al. Food consumption patterns in the 1960s in seven countries. *The American journal of clinical nutrition* 1989; **49**: 889-94.
19. Chan DS, Lau R, Aune D, et al. Red and processed meat and colorectal cancer incidence: meta-analysis of prospective studies. *PLoS One* 2011; **6**(6): e20456.
20. Bouvard V, Loomis D, Guyton KZ, et al. Carcinogenicity of consumption of red and processed meat. *Lancet Oncol* 2015; **16**(16): 1599-600.

21. Farvid MS, Cho E, Chen WY, Eliassen AH, Willett WC. Adolescent meat intake and breast cancer risk. *Int J Cancer* 2015; **136**(8): 1909-20.
22. Song M, Fung TT, Hu FB, et al. Association of animal and plant protein intake with all-cause and cause-specific mortality. *JAMA Intern Med* 2016; **176**(10): 1453-63.
23. Lee JE, McLerran DF, Rolland B, et al. Meat intake and cause-specific mortality: a pooled analysis of Asian prospective cohort studies. *The American journal of clinical nutrition* 2013; **98**(4): 1032-41.
24. Talaei M, Wang Y-L, Yuan J-M, Pan A, Koh W-P. Meat, dietary heme iron, and risk of type 2 diabetes mellitus: the Singapore Chinese Health Study. *Am J Epidemiol* 2017; **186**(7): 824-33.
25. Bischoff-Ferrari HA, Dawson-Hughes B, Baron JA, et al. Calcium intake and hip fracture risk in men and women: a meta-analysis of prospective cohort studies and randomized controlled trials. *The American journal of clinical nutrition* 2007; **86**(6): 1780-90.
26. Feskanich D, Bischoff-Ferrari HA, Frazier AL, Willett WC. Milk consumption during teenage years and risk of hip fractures in older adults. *JAMA Pediatr* 2014; **168**(1): 54-60.
27. Guo J, Astrup A, Lovegrove JA, Gijsbers L, Givens DI, Soedamah-Muthu SS. Milk and dairy consumption and risk of cardiovascular diseases and all-cause mortality: dose–response meta-analysis of prospective cohort studies. *Eur J Epidemiol* 2017; **32**(4): 269-87.
28. Aune D, Navarro Rosenblatt DA, Chan DS, et al. Dairy products, calcium, and prostate cancer risk: a systematic review and meta-analysis of cohort studies. *The American journal of clinical nutrition* 2015; **101**(1): 87-117.
29. Chen M, Sun Q, Giovannucci E, et al. Dairy consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. *BMC Med* 2014; **12**: 215.
30. Mozaffarian D, Rimm E. Fish intake, contaminants, and human health: evaluating the risks and the benefits. *JAMA* 2006; **296**(15): 1885-99.
31. Virtanen JK, Mozaffarian D, Chiuve SE, Rimm EB. Fish consumption and risk of major chronic disease in men. *The American journal of clinical nutrition* 2008; **88**(6): 1618-25.
32. Oken E, Radesky JS, Wright RO, et al. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. *Am J Epidemiol* 2008; **167**(10): 1171-81.
33. Del Gobbo LC, Imamura F, Aslibekyan S, et al. omega-3 Polyunsaturated Fatty Acid Biomarkers and Coronary Heart Disease: Pooling Project of 19 Cohort Studies. *JAMA Intern Med* 2016; **176**(8): 1155-66.
34. Rong Y, Chen L, Zhu T, et al. Egg consumption and risk of coronary heart disease and stroke: dose-response meta-analysis of prospective cohort studies. *BMJ* 2013; **346**: e8539.
35. Iannotti LL, Lutter CK, Stewart CP, et al. Eggs in Early Complementary Feeding and Child Growth: A Randomized Controlled Trial. *Pediatrics* 2017; **140**(1).
36. Kris-Etherton PM, Hu FB, Ros E, Sabate J. The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. *J Nutr* 2008; **138**(9): 1746S-51S.
37. Sabate J, Oda K, Ros E. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. *Arch Intern Med* 2010; **170**(9): 821-7.
38. Grosso G, Estruch R. Nut consumption and age-related disease. *Maturitas* 2016; **84**: 11-6.
39. Mayhew AJ, de Souza RJ, Meyre D, Anand SS, Mente A. A systematic review and meta-analysis of nut consumption and incident risk of CVD and all-cause mortality. *Br J Nutr* 2016; **115**(2): 212-25.
40. Luo C, Zhang Y, Ding Y, et al. Nut consumption and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a systematic review and meta-analysis. *The American journal of clinical nutrition* 2014; **100**(1): 256-69.
41. Bao Y, Han J, Hu FB, et al. Association of nut consumption with total and cause-specific mortality. *N Engl J Med* 2013; **369**(21): 2001-11.
42. Estruch R, Ros E, Salas-Salvadó J, et al. Primary prevention of cardiovascular disease with a mediterranean diet supplemented with extra-virgin olive oil or nuts. *New England Journal of Medicine* 2018.

43. Kushi LH, Meyer KA, Jacobs DR, Jr. Cereals, legumes, and chronic disease risk reduction: evidence from epidemiologic studies. *The American journal of clinical nutrition* 1999; **70**(3 Suppl): 451S-8S.
44. 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. *The American journal of clinical nutrition* 2014; **100**(1): 278-88.
45. Lee SA, Shu XO, Li H, et al. Adolescent and adult soy food intake and breast cancer risk: results from the Shanghai Women's Health Study. *The American journal of clinical nutrition* 2009; **89**(6): 1920-6.
46. Zong G, Gao A, Hu FB, Sun Q. Whole Grain Intake and Mortality From All Causes, Cardiovascular Disease, and Cancer: A Meta-Analysis of Prospective Cohort Studies. *Circulation* 2016; **133**(24): 2370-80.
47. Dehghan M, Mente A, Zhang X, et al. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. *Lancet* 2017; **390**(10107): 2050-62.
48. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *The American journal of clinical nutrition* 2003; **77**(5): 1146-55.
49. Jeppesen J, Schaaf P, Jones G, Zhou MY, Chen YD, Reaven GM. Effects of low-fat, high-carbohydrate diets on risk factors for ischemic heart disease in postmenopausal women. *The American journal of clinical nutrition* 1997; **65**: 1027-33.
50. Liu S, Willett WC, Stampfer MJ, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *The American journal of clinical nutrition* 2000; **71**: 1455-61.
51. Muraki I, Rimm EB, Willett WC, Manson JE, Hu FB, Sun Q. Potato Consumption and Risk of Type 2 Diabetes: Results From Three Prospective Cohort Studies. *Diabetes Care* 2016; **39**(3): 376-84.
52. Borgi L, Rimm EB, Willett WC, Forman JP. Potato intake and incidence of hypertension: results from three prospective US cohort studies. *BMJ* 2016; **353**: i2351.
53. Bertoia ML, Mukamal KJ, Cahill LE, et al. Changes in Intake of Fruits and Vegetables and Weight Change in United States Men and Women Followed for Up to 24 Years: Analysis from Three Prospective Cohort Studies. *PLoS Med* 2015; **12**(9): e1001878.
54. 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. *BMJ* 2014; **349**: g4490.
55. Aune D, Giovannucci E, Boffetta P, et al. Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality-a systematic review and dose-response meta-analysis of prospective studies. *Int J Epidemiol* 2016.
56. Muraki I, Imamura F, Manson JE, et al. Fruit consumption and risk of type 2 diabetes: results from three prospective longitudinal cohort studies. *BMJ* 2013; **347**: f5001.
57. Hung HC, Joshipura K, Jiang R, et al. Fruit and vegetable intake and the risk of major chronic disease. *J Natl Cancer Inst* 2004; **21**(21): 1577-84.
58. Boffetta P, Wichmann J, Ferrari P, et al. Fruit and vegetable intake and overall cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC). *J Natl Cancer Inst* 2010; **102**(8): 529-37.
59. Wang DD, Li Y, Chiuve SE, et al. Association of Specific Dietary Fats With Total and Cause-Specific Mortality. *JAMA Intern Med* 2016.
60. Prentice RL, Caan B, Chlebowski RT, et al. Low-fat dietary pattern and risk of invasive breast cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *Jama* 2006; **295**(6): 629-42.
61. Jakobsen MU, O'Reilly EJ, Heitmann BL, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies–. *The American journal of clinical nutrition* 2009; **89**(5): 1425-32.

62. 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. *Circulation* 2014; **130**(18): 1568-78.
63. Chowdhury R, Warnakula S, Kunutsor S, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med* 2014; **160**(6): 398-406.
64. Sun Y, Neelakantan N, Wu Y, Lote-Oke R, Pan A, van Dam RM. Palm Oil Consumption Increases LDL Cholesterol Compared with Vegetable Oils Low in Saturated Fat in a Meta-Analysis of Clinical Trials. *J Nutr* 2015; **145**(7): 1549-58.
65. Kabagambe EK, Baylin A, Ascherio A, Campos H. The type of oil used for cooking is associated with the risk of nonfatal acute myocardial infarction in costa rica. *J Nutr* 2005; **135**(11): 2674-9.
66. de Lorgeril M, Renaud S, Mamelle N, et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease [Erratum in: *Lancet* 1995;345:738]. *Lancet* 1994; **343**: 1454-9.
67. Chen M, Li Y, Sun Q, et al. Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. *The American journal of clinical nutrition* 2016; **104**(5): 1209-17.
68. Knopp RH, Walden CE, Retzlaff BM, et al. Long-term cholesterol-lowering effects of 4 fat-restricted diets in hypercholesterolemic and combined hyperlipidemic men: The Dietary Alternatives Study. *J Am Med Assoc* 1997; **278**: 1509-15.
69. Tobias DK, Chen M, Manson JE, Ludwig DS, Willett W, Hu FB. Effect of low-fat diet interventions versus other diet interventions on long-term weight change in adults: a systematic review and meta-analysis. *Lancet Diabetes Endocrinol* 2015; **3**(12): 968-79.
70. Chiavaroli L, de Souza RJ, Ha V, et al. Effect of Fructose on Established Lipid Targets: A Systematic Review and Meta-Analysis of Controlled Feeding Trials. *J Am Heart Assoc* 2015; **4**(9): e001700.
71. Barclay AW, Petocz P, McMillan-Price J, et al. Glycemic index, glycemic load, and chronic disease risk--a meta-analysis of observational studies. *The American journal of clinical nutrition* 2008; **87**(3): 627-37.
72. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ* 2012; **346**: e7492.
73. Malik VS, Popkin BM, Bray GA, Despres JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care* 2010; **33**(11): 2477-83.
74. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* 2014; **174**(4): 516-24.
75. Maslova E, Rytter D, Bech BH, et al. Maternal protein intake during pregnancy and offspring overweight 20 y later. *The American journal of clinical nutrition* 2014; **100**(4): 1139-48.
76. Brantsaeter AL, Olafsdottir AS, Forsum E, Olsen SF, Thorsdottir I. Does milk and dairy consumption during pregnancy influence fetal growth and infant birthweight? . *Food and Nutrition Research* 2012; **56**(20050).
77. Sánchez PH, Ruano C, De Irala J, Ruiz-Canela M, Martinez-Gonzalez M, Sánchez-Villegas A. Adherence to the Mediterranean diet and quality of life in the SUN Project. *European journal of clinical nutrition* 2012; **66**(3): 360.
78. Bhushan A, Fondell E, Ascherio A, Yuan C, Grodstein F, Willett W. Adherence to Mediterranean diet and subjective cognitive function in men. *European journal of epidemiology* 2017: 1-12.
79. Morris MC, Tangney CC, Wang Y, Sacks FM, Bennett DA, Aggarwal NT. MIND diet associated with reduced incidence of Alzheimer's disease. *Alzheimer's & dementia: the journal of the Alzheimer's Association* 2015; **11**(9): 1007-14.
80. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med* 1997; **336**(16): 1117-24.
81. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the dietary approaches to stop hypertension (DASH) diet. *N Engl J Med* 2001; **344**: 3-10.

82. Chiuve SE, Sampson L, Willett WC. The association between a nutritional quality index and risk of chronic disease. *Am J Prev Med* 2011; **40**(5): 505-13.
83. Wang DD, Li Y, Chiuve SE, Hu FB, Willett WC. Improvements in US diet helped reduce disease burden and lower premature deaths, 1999–2012; overall diet remains poor. *Health Affairs* 2015; **34**(11): 1916-22.
84. Schwingshackl L, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. *Journal of the Academy of Nutrition and Dietetics* 2015; **115**(5): 780-800. e5.
85. Onvani S, Haghighatdoost F, Surkan P, Larijani B, Azadbakht L. Adherence to the Healthy Eating Index and Alternative Healthy Eating Index dietary patterns and mortality from all causes, cardiovascular disease and cancer: a meta-analysis of observational studies. *Journal of Human Nutrition and Dietetics* 2017; **30**(2): 216-26.
86. Cespedes EM, Hu FB, Tinker L, et al. Multiple Healthful Dietary Patterns and Type 2 Diabetes in the Women's Health Initiative. *Am J Epidemiol* 2016; **183**(7): 622-33.
87. Mehta RS, Song M, Nishihara R, et al. Dietary Patterns and Risk of Colorectal Cancer: Analysis by Tumor Location and Molecular Subtypes. *Gastroenterology* 2017.
88. Hou L, Li F, Wang Y, et al. Association between dietary patterns and coronary heart disease: a meta-analysis of prospective cohort studies. *Int J Clin Exp Med* 2015; **8**(1): 781-90.
89. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med* 2003; **348**: 2599-608.
90. Samieri C, Sun Q, Townsend MK, et al. The association between dietary patterns at midlife and health in aging: an observational study. *Ann Intern Med* 2013; **159**(9): 584-91.
91. Sotos-Prieto M, Bhupathiraju SN, Mattei J, et al. Association of Changes in Diet Quality with Total and Cause-Specific Mortality. *N Engl J Med* 2017; **377**(2): 143-53.
92. Micha R, Michas G, Mozaffarian D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes--an updated review of the evidence. *Curr Atheroscler Rep* 2012; **14**(6): 515-24.
93. Zheng J, Huang T, Yu Y, Hu X, Yang B, Li D. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. *Public Health Nutr* 2012; **15**(4): 725-37.
94. Aune D, Keum N, Giovannucci E, et al. Nut consumption and risk of cardiovascular disease, total cancer, all-cause and cause-specific mortality: a systematic review and dose-response meta-analysis of prospective studies. *BMC medicine* 2016; **14**(1): 207.
95. Chiuve SE, Fung TT, Rimm EB, et al. Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr* 2012; **142**(6): 1009-18.
96. Liese AD, Krebs-Smith SM, Subar AF, et al. The Dietary Patterns Methods Project: Synthesis of Findings across Cohorts and Relevance to Dietary Guidance–4. *The Journal of nutrition* 2015; **145**(3): 393-402.
97. Sotos-Prieto M, Bhupathiraju SN, Mattei J, et al. Association of changes in diet quality with total and cause-specific mortality. *New England Journal of Medicine* 2017; **377**(2): 143-53.
98. Khatibzadeh S, Kashaf MS, Micha R, et al. A global database of food and nutrient consumption. *Bulletin of the World Health Organization* 2016; **94**(12): 931.
99. Imamura F, Micha R, Khatibzadeh S, et al. Dietary quality among men and women in 187 countries in 1990 and 2010: a systematic assessment. *The Lancet Global health* 2015; **3**(3): e132-42.
100. Del Gobbo LC, Khatibzadeh S, Imamura F, et al. Assessing global dietary habits: a comparison of national estimates from the FAO and the Global Dietary Database–. *The American journal of clinical nutrition* 2015; **101**(5): 1038-46.
101. Mozaffarian D, Fahimi S, Singh GM, et al. Global Sodium Consumption and Death from Cardiovascular Causes. *New England Journal of Medicine* 2014; **371**(7): 624-34.
102. School HM. The Nurses' Health Study. http://www.channing.harvard.edu/nhs/ (accessed May 24 2015).

103. Health HTHCSoP. The Health Professionals Follow-Up Study. https://www.hsph.harvard.edu/hpfs/ (accessed May 24 2015).
104. Murray CJ, Lopez AD. On the comparable quantification of health risks: lessons from the Global Burden of Disease Study. *Epidemiology* 1999; **10**(5): 594-605.
105. Organization WH. WHO mortality database. Geneva, Switzerland; 2018.
106. Fanning AL, O'Neill DW. Tracking resource use relative to planetary boundaries in a steady-state framework: A case study of Canada and Spain. *Ecological indicators* 2016; **69**: 836-49.
107. Dearing JA, Wang R, Zhang K, et al. Safe and just operating spaces for regional social-ecological systems. *Global Environmental Change-Human and Policy Dimensions* 2014; **28**: 227-38.
108. Cole MJ, Bailey RM, New MG. Tracking sustainable development with a national barometer for South Africa using a downscaled "safe and just space" framework. *Proceedings of the National Academy of Sciences* 2014; **111**(42): E4399-E408.
109. Frischknecht R, Stolz P, Tschümperlin L. National environmental footprints and planetary boundaries: from methodology to policy implementation 59th LCA forum, Swiss Federal Institute of Technology, Zürich, June 12, 2015. *The International Journal of Life Cycle Assessment* 2016; **21**(4): 601-5.
110. Dao Q-H, Peduzzi P, Chatenoux B, De Bono A, Schwarzer S, Friot D. Environmental limits and Swiss footprints based on Planetary Boundaries. 2015.
111. Pisano U, Berger G. Planetary boundaries for SD: from an international perspective to national applications: ESDN Quarterly Report, 2015.
112. Häyhä T, Cornell SE, Hoff H, Lucas P, Vurren Dv. Operationalizing the concept of a safe operating space at the EU level - first steps and explorations. Stockholm: Stockholm Resilience Center, 2018.
113. Fang K, Heijungs R, Duan Z, de Snoo GR. The environmental sustainability of nations: benchmarking the carbon, water and land footprints against allocated planetary boundaries. *Sustainability* 2015; **7**(8): 11285-305.
114. O'Neill DW, Fanning AL, Lamb WF, Steinberger JK. A good life for all within planetary boundaries. *Nature Sustainability* 2018; **1**(2): 88.
115. Hoornweg D, Hosseini M, Kennedy C, Behdadi A. An urban approach to planetary boundaries. *Ambio* 2016; **45**(5): 567-80.
116. McLellan R, Iyengar L, Jeffries B, Oerlemans N. Living planet report 2014: species and spaces, people and places: World Wide Fund for Nature; 2014.
117. NI T. Living Planet Report 2016. 1986.
118. Whiteman G, Walker B, Perego P. Planetary boundaries: Ecological foundations for corporate sustainability. *Journal of Management Studies* 2013; **50**(2): 307-36.
119. Butz C, Liechti J, Bodin J, Cornell SE. Towards defining an environmental investment universe within planetary boundaries. *Sustainability Science* 2018; **13**(4): 1031-44.
120. Jägermeyr J, Pastor A, Biemans H, Gerten D. Reconciling irrigated food production with environmental flows for Sustainable Development Goals implementation. *Nature communications* 2017; **8**: 15900.
121. Campbell B, Beare D, Bennett E, et al. Agriculture production as a major driver of the Earth system exceeding planetary boundaries. *Ecology and Society* 2017; **22**(4).
122. Heck V, Hoff H, Wirsenius S, Meyer C, Kreft H. Land use options for staying within the Planetary Boundaries–Synergies and trade-offs between global and local sustainability goals. *Global Environmental Change* 2018; **49**: 73-84.
123. Hoff H. Understanding the nexus. background paper for the Bonn2011 Conference: the water, energy and food security nexus. *Stockholm Environment Institute, Stockholm* 2011.
124. Erb K-H, Lauk C, Kastner T, Mayer A, Theurl MC, Haberl H. Exploring the biophysical option space for feeding the world without deforestation. *Nature Communications* 2016; **7**: 11382.
125. Carpenter SR, Bennett EM. Reconsideration of the planetary boundary for phosphorus. *Environmental Research Letters* 2011; **6**(1).

126. De Vries W, Kros J, Kroeze C, Seitzinger SP. Assessing planetary and regional nitrogen boundaries related to food security and adverse environmental impacts. *Current Opinion in Environmental Sustainability* 2013; **5**(3-4): 392-402.
127. Kahiluoto H, Kuisma M, Kuokkanen A, Mikkilä M, Linnanen L. Taking planetary nutrient boundaries seriously: can we feed the people? *Global Food Security* 2014; **3**(1): 16-21.
128. Gerten D, Hoff H, Rockström J, Jägermeyr J, Kummu M, Pastor AV. Towards a revised planetary boundary for consumptive freshwater use: role of environmental flow requirements. *Current Opinion in Environmental Sustainability* 2013; **5**(6): 551-8.
129. Bogardi JJ, Fekete BM, Vörösmarty CJ. Planetary boundaries revisited: a view through the 'water lens'. *Current Opinion in Environmental Sustainability* 2013; **5**(6): 581-9.
130. Mace GM. Whose conservation? *Science* 2014; **345**(6204): 1558-60.
131. Barnosky AD, Hadly EA, Bascompte J, et al. Approaching a state shift in Earth's biosphere. *Nature* 2012; **486**(7401): 52.
132. Rothman DH. Thresholds of catastrophe in the Earth system. *Science Advances* 2017; **3**(9): e1700906.
133. Diamond ML, de Wit CA, Molander S, et al. Exploring the planetary boundary for chemical pollution. *Environment international* 2015; **78**: 8-15.
134. Persson LM, Breitholtz M, Cousins IT, de Wit CA, MacLeod M, McLachlan MS. Confronting unknown planetary boundary threats from chemical pollution. ACS Publications; 2013.
135. Sala S, Goralczyk M. Chemical footprint: A methodological framework for bridging life cycle assessment and planetary boundaries for chemical pollution. *Integrated environmental assessment and management* 2013; **9**(4): 623-32.
136. Itsuki CH, KAWAI T. Bayesian Uncertainty Analysis of the Global Dynamics of Persistent Organic Pollutants: Towards Quantifying the Planetary Boundaries for Chemical Pollution. 2011.
137. Waters CN, Zalasiewicz J, Summerhayes C, et al. The Anthropocene is functionally and stratigraphically distinct from the Holocene. *Science* 2016; **351**(6269): aad2622.
138. Barnosky AD. Transforming the global energy system is required to avoid the sixth mass extinction. *MRS Energy & Sustainability* 2015; **2**.
139. Gasser T, Guivarch C, Tachiiri K, Jones C, Ciais P. Negative emissions physically needed to keep global warming below 2 C. *Nature communications* 2015; **6**: 7958.
140. Rogelj J, Popp A, Calvin KV, et al. Scenarios towards limiting global mean temperature increase below 1.5° C. *Nature Climate Change* 2018: 1.
141. Williamson P. Scrutinize CO2 removal methods: the viability and environmental risks of removing carbon dioxide from the air must be assessed if we are to achieve the Paris goals. *Nature* 2016; **530**(7589): 153-6.
142. Smith P, Davis SJ, Creutzig F, et al. Biophysical and economic limits to negative CO2 emissions. *Nature Climate Change* 2016; **6**(1): 42-50.
143. Zomer RJ, Trabucco A, Coe R, Place F. Trees on Farm:

Analysis of Global Extent and Geographical Patterns of Agroforestry. ICRAF. Nairobi: ICRAF, 2009.
144. Griscom BW, Adams J, Ellis PW, et al. Natural climate solutions. *Proceedings of the National Academy of Sciences* 2017; **114**(44): 11645-50.
145. Ramankutty N, Evan AT, Monfreda C, Foley JA. Farming the planet: 1. Geographic distribution of global agricultural lands in the year 2000. *Global Biogeochemical Cycles* 2008; **22**(1).
146. Steffen W, Richardson K, Rockstrom J, et al. Planetary boundaries: guiding human development on a changing planet. *Science* 2015; **347**(6223).
147. Tilman D, Clark M, Williams DR, Kimmel K, Polasky S, Packer C. Future threats to biodiversity and pathways to their prevention. *Nature* 2017; **546**(7656): 73.
148. Springmann M, Clark M, D'Croz D, et al. Changes in food management, technology and diets to stay within planetary boundaries. *Nature* 2018; **562**: 519-525.
149. Garnett T, Roos E, Little D. Lean, mean, green, obscene…? What is efficiency, and is it sustainable?: Food Climate Research Network, University of Oxford, 2015.

150. Herrero M, Havlík P, Valin H, et al. Biomass use, production, feed efficiencies, and greenhouse gas emissions from global livestock systems. *Proceedings of the National Academy of Sciences* 2013; **110**(52): 20888-93.
151. Schader C, Muller A, Scialabba NE-H, et al. Impacts of feeding less food-competing feedstuffs to livestock on global food system sustainability. *Journal of The Royal Society Interface* 2015; **12**(113): 20150891.
152. Van Zanten H, Meerburg B, Bikker P, Herrero M, de Boer I. Opinion paper: The role of livestock in a sustainable diet: a land-use perspective. *Feed sources for livestock: recycling towards a green planet* 2016: 169.
153. Röös E, Bajželj B, Smith P, Patel M, Little D, Garnett T. Greedy or needy? Land use and climate impacts of food in 2050 under different livestock futures. *Global Environmental Change* 2017; **47**: 1-12.
154. Garnett T. Livestock-related greenhouse gas emissions: impacts and options for policy makers. *environmental science & policy* 2009; **12**(4): 491-503.

name	Foods	USDA code	amount	calor	prot	tfat	carbo	aofib	calc	iron	magn	ph	k	zn	vitc	b1	b2	niacin	b6	fdfol	fol98	b12	bcar	rae	vitd	satfat	monfat	poly fat	f182, LA	f183, ALA	f225, EPA	f226, DHA	chol
wheat.red	WHEAT,HARD RED SPRING	20071.0	**116.0**	381.6	17.9	2.2	78.9	14.2	29.0	4.2	143.8	385.1	394.4	3.2	0.0	0.6	0.1	6.6	0.4	49.9	49.9	0.0	5.8	0.5	0.0	0.4	0.3	0.9	0.8	0.0	0.0	0.0	0.0
br.rice.raw	RICE,BROWN,LONG-GRAIN,RAW	20036.0	**116.0**	429.2	9.2	3.4	89.6	4.1	26.7	1.7	165.9	386.3	258.7	2.3	0.0	0.5	0.1	5.9	0.6	23.2	23.2	0.0	0.0	0.0	0.0	0.7	1.2	1.2	1.2	0.0	0.0	0.0	0.0
pot.raw	POTATO,FLESH & SKN,RAW	11352.0	**50.0**	38.5	1.0	0.1	8.8	1.1	6.0	0.4	11.5	28.5	212.5	0.2	9.9	0.0	0.0	0.6	0.2	7.5	7.5	0.0	0.5	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
spin.raw	RAW SPINACH	11457.0	**100.0**	23.0	2.9	0.4	3.6	2.2	99.0	2.7	79.0	49.0	558.0	0.5	28.1	0.1	0.2	0.7	0.2	194.0	194.0	0.0	5626.0	468.8	0.0	0.1	0.0	0.2	0.0	0.1	0.0	0.0	0.0
yel.sqs	BAKED, UNSALTED WINTER SQUASH	11644.0	**33.0**	12.2	0.3	0.1	2.9	0.9	7.3	0.1	4.3	6.3	79.5	0.1	3.2	0.0	0.0	0.2	0.1	6.6	6.6	0.0	921.7	115.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
carrot.c	BOILED, DRAINED, UNSALTED CARROTS	11125.0	**33.0**	11.6	0.3	0.1	2.7	1.0	9.9	0.1	3.3	9.9	77.6	0.1	1.2	0.0	0.0	0.2	0.0	4.6	4.6	0.0	2749.6	281.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
tom	RIPE, RED TOMATOES	11529.0	**34.0**	6.1	0.3	0.1	1.3	0.4	3.4	0.1	3.7	8.2	80.6	0.1	4.7	0.0	0.0	0.2	0.0	5.1	5.1	0.0	152.7	14.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
onions	RAW ONIONS	11282.0	**33.0**	13.2	0.4	0.0	3.1	0.6	7.6	0.1	3.3	9.6	48.2	0.1	2.4	0.0	0.0	0.0	0.0	6.3	6.3	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
zuke	BOILED, DRAINED, UNSALTED SUMMER SQUASH	11478.0	**33.0**	5.0	0.4	0.1	0.9	0.3	5.9	0.1	6.3	12.2	87.1	0.1	4.3	0.0	0.0	0.2	0.0	9.2	9.2	0.0	221.1	18.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
peppers	RAW GREEN PEPPERS	11333.0	**34.0**	6.8	0.3	0.1	1.6	0.6	3.4	0.1	3.4	6.8	59.5	0.0	27.3	0.0	0.0	0.2	0.1	3.4	3.4	0.0	70.7	6.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
apple	RAW APPLES WITH SKIN	9003.0	**66.0**	34.3	0.2	0.1	9.1	1.6	4.0	0.1	3.3	7.3	70.6	0.0	3.0	0.0	0.0	0.1	0.0	2.0	2.0	0.0	17.8	1.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
orang	RAW ORANGES	9200.0	**66.0**	31.0	0.6	0.1	7.8	1.6	26.4	0.1	6.6	9.2	119.5	0.0	35.1	0.1	0.0	0.2	0.0	19.8	19.8	0.0	46.9	7.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
ban	RAW BANANAS	9040.0	**68.0**	60.5	0.7	0.2	15.5	1.8	3.4	0.2	18.4	15.0	243.4	0.1	5.9	0.0	0.0	0.5	0.3	13.6	13.6	0.0	17.7	2.2	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
milk	WHOLE MILK	1077.0	**250.0**	152.5	7.8	8.0	12.0	0.0	282.5	0.1	25.0	210.0	330.0	0.9	0.0	0.1	0.4	0.3	0.1	12.5	12.5	1.1	17.5	114.0	127.5	4.7	2.0	0.5	0.3	0.2	0.0	0.0	25.0
lentils.raw	LENTILS,RAW	16069.0	**25.0**	88.0	6.2	0.3	15.9	2.7	8.8	1.6	11.8	70.3	169.3	0.8	1.1	0.2	0.1	0.7	0.1	119.8	119.8	0.0	5.8	0.5	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0
beans.raw	BEANS,NAVY,MATURE SEEDS,RAW	16037.0	**25.0**	84.3	5.6	0.4	15.2	3.8	36.8	1.4	43.8	101.8	296.3	0.9	0.0	0.2	0.0	0.6	0.1	91.0	91.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.1	0.1	0.0	0.0	0.0
nuts.raw (peanuts)	PEANUTS,ALL TYPES,RAW	16087.0	**25.0**	141.8	6.5	12.3	4.0	2.1	23.0	1.1	42.0	94.0	176.3	0.8	0.0	0.2	0.0	3.0	0.1	60.0	60.0	0.0	0.0	0.0	0.0	1.6	6.1	3.9	3.9	0.0	0.0	0.0	0.0
almonds	OIL ROASTED, UNSALTED ALMONDS	12065.0	**12.5**	75.9	2.7	6.9	2.2	1.3	36.4	0.5	34.3	58.3	87.4	0.4	0.0	0.0	0.1	0.5	0.0	3.4	3.4	0.0	0.1	0.0	0.0	0.5	4.3	1.7	1.7	0.0	0.0	0.0	0.0
cashews	OIL ROASTED, UNSALTED CASHEWS	12086.0	**12.5**	72.5	2.1	6.0	3.7	0.4	5.4	0.8	34.1	66.4	79.0	0.7	0.0	0.0	0.0	0.2	0.0	3.1	3.1	0.0	0.0	0.0	0.0	1.1	3.2	1.1	1.1	0.0	0.0	0.0	0.0
soy.bn.raw	SOYBEANS,MATURE SEEDS,RAW	16108.0	**25.0**	111.5	9.1	5.0	7.6	2.3	69.3	3.9	70.0	176.0	449.3	1.2	1.5	0.2	0.2	0.4	0.1	93.8	93.8	0.0	3.3	0.3	0.0	0.7	1.1	2.8	2.5	0.3	0.0	0.0	0.0
beef.raw	BEEF,GROUND,85% LN MEAT / 15% FAT,RAW	23567.0	**7.0**	15.1	1.3	1.1	0.0	0.0	1.1	0.1	1.3	12.0	20.7	0.3	0.0	0.0	0.0	0.3	0.0	0.4	0.4	0.2	0.0	0.3	0.2	0.4	0.5	0.0	0.0	0.0	0.0	0.0	4.8
chix.raw	CHICKEN,BROILERS OR FRYERS,MEAT & SKN,RAW	5006.0	**29.0**	62.4	5.4	4.4	0.0	0.0	3.2	0.3	5.8	42.6	54.8	0.4	0.5	0.0	0.0	2.0	0.1	1.7	1.7	0.1	0.0	11.9	2.9	1.2	1.8	0.9	0.8	0.0	0.0	0.0	21.8
pork.raw	PORK,FRSH,COMP (LEG,LOIN,SHLDR,&SPARERIBS),LN&FAT,RAW	10187.0	**7.0**	15.1	1.3	1.0	0.0	0.0	1.3	0.1	1.5	14.0	23.5	0.1	0.0	0.1	0.0	0.3	0.0	0.4	0.4	0.0	0.0	0.1	0.0	0.4	0.5	0.1	0.1	0.0	0.0	0.0	4.7
eggs	RAW WHOLE EGG	1123.0	**13.0**	18.6	1.6	1.2	0.1	0.0	7.3	0.2	1.6	25.7	17.9	0.2	0.0	0.0	0.1	0.0	0.0	6.1	6.1	0.1	0.0	20.9	10.7	0.4	0.5	0.2	0.2	0.0	0.0	0.0	48.4
dk.fish	DRY HEAT COOKED SOCKEYE SALMON	NA	**14.0**	23.9	3.3	1.1	0.0	0.0	8.5	0.1	4.7	41.6	58.0	0.1	0.2	0.0	0.0	1.5	0.1	2.8	2.8	0.5	0.1	3.0	41.7	0.2	0.4	0.4	0.0	0.0	0.0	0.1	8.4
oth.fish	DRY HEAT COOKED ATLANTIC COD	NA	**14.0**	15.5	3.0	0.3	0.0	0.0	2.1	0.1	4.1	38.1	48.3	0.1	0.0	0.0	0.0	0.5	0.0	1.2	1.2	0.3	0.0	1.1	7.5	0.1	0.1	0.1	0.0	0.0	0.0	0.0	8.4
palm	OIL,VEGETABLE,PALM	4055.0	**6.8**	60.1	0.0	6.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.4	2.5	0.6	0.6	0.0	0.0	0.0	0.0
soy.90	OIL,SOYBEAN,SALAD OR COOKING	4044.0	**8.0**	70.8	0.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.2	1.8	4.6	4.1	0.6	0.0	0.0	0.0
rapeseed	VEGETABLE OIL,CANOLA	4582.0	**8.0**	70.8	0.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	5.1	2.3	1.5	0.7	0.0	0.0	0.0
o	OIL,OLIVE,SALAD OR COOKING	4053.0	**8.0**	70.8	0.0	8.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.1	5.8	0.8	0.8	0.1	0.0	0.0	0.0
sn	OIL,VEGETABLE,SUNFLOWER,LINOLEIC,(APPROX. 65%)	4506.0	**8.0**	70.8	0.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.8	1.6	5.3	5.3	0.0	0.0	0.0	0.0
pn	OIL,PEANUT,SALAD OR COOKING	4042.0	**8.0**	70.8	0.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.4	3.7	2.6	2.6	0.0	0.0	0.0	0.0
bu	SALTED BUTTER	1001.0	**0.0**	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
lard	LARD	4002.0	**4.0**	36.1	0.0	4.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.1	1.6	1.8	0.4	0.4	0.0	0.0	0.0	3.8
sug	GRANULATED SUGAR	19335.0	**31.0**	120.0	0.0	0.0	31.0	0.0	0.3	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Totals				2499.9	90.1	105.6	317.3	42.9	717.8	20.2	732.5	1883.9	4100.7	13.7	128.5	2.4	1.7	25.6	2.8	741.3	741.3	2.3	9857.5	1067.9	194.6	22.7	44.6	31.3	28.3	2.5	0.0	0.2	125.2
name	**shrt_desc**	**ndb_no**	**amount**	**calor**	**prot**	**tfat**	**carbo**	**aofib**	**calc**	**iron**	**magn**	**ph**	**k**	**zn**	**vitc**	**b1**	**b2**	**niacin**	**b6**	**fdfol**	**fol98**	**b12**	**bcar**	**rae**	**vitd**	**satfat**	**monfat**	**poly**	**f182**	**f183**	**f225**	**f226**	**chol**
% Energy																																	

calor	Total Calories kcal
prot	Protein gm
tfat	Total Fat gm
carbo	Carbohydrates gm
aofib	AOAC Fiber gm
calc	Calcium mg
iron	Iron mg
heme	Heme iron mg
magn	Magnesium mg
ph	Phospherous mg
k	Potassium mg
sodium	Sodium mg
zn	Zinc mg
cu	Copper mg
mn	Manganese mg
vitc	Vitamin C mg
b1	Thiamine mg
b2	Riboflavin mg
niacin	Niacin mg
panto	Pantothenic Acid mg
b6	Pyridoxine mg
fdfol	Food Folate
folic	Folic Acid
fol98	Total Folate SR12 mcg
dfe	Dietary Folate Equivalents
b12	Vitamin B12 mcg
acar	Alpha Carotene mcg
bcar	Beta Carotene mcg
bcryp	Beta Cryptoxanthin mcg
lyco	Lycopene mcg
lut	Lutein and Zeaxanthin mcg
zeax	Zeaxanthin mcg
rae	Retinol Activity Equivalents mcg
vitd	Vitamin D IU
e02mg	Vitamin E mg atoco Conversion (food+supplement)
ubtoco	Beta Tocopherol mg
ugtoco	Gamma Tocopherol mg
udtoco	Delta Tocopherol mg
uaT3	Alpha Tocotrienol mg
ubT3	Beta Tocotrienol mg
ugT3	Gamma Tocotrienol mg
udT3	Delta Tocotrienol mg
uttoco	mg Total Tocopherols without supplement 2008
satfat	Total Saturated Fat gm
f40	Butyric fatty acid gm
f60	Caproic fatty acid gm
f80	Caprylic fatty acid gm
f100	Capric fatty acid gm
f120	Lauric fatty acid gm
f140	Myristic fatty acid gm
f160	Palmitic fatty acid gm
f180	Stearic fatty acid gm
f200	Eicosanoic Acid gm
f220	Docosanoic Acid gm
f240	Tetracosanoic Acid gm USDA 2006
monfat	Total Monounsaturated Fat gm
f141	Tetradecenoic Acid gm
f151	Pentadecenoic Acid gm
f161	Palmitoleic fatty acid gm
f171	Heptadecenoic Acid gm
f181	Oleic fatty acid gm
f201	Eicosenoic fatty acid gm
f221	Erucic fatty acid
f241	Nervonic fatty acid
poly	Total Polyunsaturated Fat gm
f182	Linoleic fatty acid gm
f183	Linolenic fatty acid gm
f184	Parinaric fatty acid
f203	Eicosatrienoic Acid gm
f204	Arachadonic fatty acid gm
f205	Eicosapentaenoic EPA fatty acid gm
f225	Docosapentaenoic 22:5 fatty acid gm
f226	Docosahexaenoic DHA fatty acid gm
chol	Cholesterol mg
trypto	Tryptophan gm
thr	Threonine gm
iso	Isoleucine gm
leu	Leucine gm
lys	Lysine gm
meth	Methionine gm
cys	Cystine gm
phenyl	Phenylalanine gm
phenyla	Phenylalanine
tyro	Tyrosine gm
val	Valine gm
arg	Arginine gm
hist	Histidine gm
ala	Alanine gm
asp	Aspartic Acid gm
aspa	Aspartic Acid
glut	Glutamic Acid gm
gly	Glycine gm
pro	Proline gm
ser	Serine gm
h2o	H20