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Health effects of adopting low greenhouse gas emission diets in the UK

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ABSTRACT

Objective: Dietary changes which improve health are also likely to be beneficial for the environment by reducing emissions of greenhouse gases (GHG). However, previous analyses have not accounted for the potential acceptability of low GHG diets to the general public. This study attempted to quantify the health effects associated with adopting low GHG emission diets in the UK.

Design: Epidemiological modelling study.

Setting: UK.

Participants: UK population.

Intervention: Adoption of diets optimised to achieve the WHO nutritional recommendations and reduce GHG emissions while remaining as close as possible to existing dietary patterns.

Main outcome: Changes in years of life lost due to coronary heart disease, stroke, several cancers and type II diabetes, quantified using life tables.

Results: If the average UK dietary intake were optimised to comply with the WHO recommendations, we estimate an incidental reduction of 17% in GHG emissions. Such a dietary pattern would be broadly similar to the current UK average. Our model suggests that it would save almost 7 million years of life lost prematurely in the UK over the next 30 years and increase average life expectancy by over 8 months. Diets that result in additional GHG emission reductions could achieve further net health benefits. For emission reductions greater than 40%, improvements in some health outcomes may decrease and acceptability will diminish.

Conclusions: There are large potential benefits to health from adopting diets with lower associated GHG emissions in the UK. Most of these benefits can be achieved without drastic changes to existing dietary patterns. However, to reduce emissions by more than 40%, major dietary changes that limit both acceptability and the benefits to health are required.

Strengths and limitations of this study

- This paper presents a novel application of an optimisation approach used to estimate dietary changes in the UK which would meet nutritional and climate change mitigation targets.
- The method generates dietary patterns at a greater level of detail than previous assessments.
- The work also accounts for the potential acceptability of the modelled diets through the use of data on consumer behaviour.
- The resulting impacts on mortality in the UK have been modelled using disease-specific time lag curves.
- The limitations of the study mostly relate to inadequacies of the available data on food consumption and greenhouse gas emissions related to the diet.

INTRODUCTION

Meeting targets for reducing greenhouse gas (GHG) emissions in many countries is likely to require significant changes to diets.[1 2] It is now relatively well established that dietary changes that reduce GHG emissions are also likely to be desirable from the standpoint of their nutritional content[3] and health outcomes.[4 5] Previous work has modelled the potential beneficial effects of broad and often radical dietary changes on health and GHG emissions, typically based on greatly reduced consumption of animal source foods and higher consumption of fruit and vegetables.[4 6–10] However, there is a need for more detailed information on the specific composition of healthy and low-emission diets, which will help to prioritise policy action and interventions aimed at promoting healthier eating.[11] Further, given the possible public resistance, it is important to know how radical dietary changes need to be to achieve health and climate change mitigation targets.

The recent WHO Global Burden of Disease (GBD) estimates suggest that dietary risk factors account for a tenth of the global disease burden.[12] In the UK, current average diets fail to meet multiple UK dietary recommendations and do not contain the recommended daily amounts of many

micronutrients.[13] Diet-related ill health in the UK is estimated to cost the National Health Service (NHS) around £6 billion annually.[14] Little work has been carried out on defining realistic diets for the UK population that are good for health while reducing GHG emissions. A recent estimate has suggested that increasing the likely acceptability of the changed diets to the UK population reduces the level of emissions reduction that could be achieved by almost two-thirds.[7]

We present here work that aims to balance improving health, reducing GHG emissions and maintaining realistic diets in the UK, finding a point at which the combined benefits are maximised. It is designed to quantify the potential health benefits of a range of dietary changes that would help meet the UK's commitment to reduce GHG emissions. We have constrained the modelled diets to be as close as possible to current food consumption, thus increasing the likelihood of acceptability. This paper provides estimates of health effects, whereas the GHG emissions resulting from the dietary changes are presented elsewhere.[15]

METHODS

We modelled a range of 'optimised' modifications to the average UK dietary pattern to achieve compliance with the WHO nutritional recommendations and GHG emission targets. The potential health impacts which would result from adopting the new optimised dietary patterns were estimated using life tables. The methods used to model the diets are summarised below but described in greater detail elsewhere.[15]

Dietary data

Information on current average diets in the UK was obtained from the National Diet and Nutrition Survey (NDNS), based on 4-day food diaries for 1571 adults. All foods in the survey were included in the model. The data were aggregated to calculate average daily consumption of 42 representative (compositionally similar) food groups for males and females. The data were also used to obtain the average nutritional contents for each food group.

Greenhouse gas emissions data

GHG emissions associated with each food group were estimated using a Life Cycle Inventory (LCI) compiled from the published literature.[2 6 16–21] Where possible, we included information on food losses from production, handling, sales, cooking and consumer waste. Where LCI emissions were not available, we based our estimates on information on representative items contained within that group.

Optimisation of future diets

We used an optimisation method to generate modified average dietary intake patterns for the UK which met the WHO nutritional recommendations.[22] The optimisations were performed in the statistical software R[23] using the package Alabama.[24] To increase the likelihood that the diets would be acceptable to the population, each simulation minimised the deviation from the existing UK dietary pattern by minimising the sum of squared percentage differences from the current consumption, with the contributions from individual food groups weighted according to their price responsiveness and their share of the diet (reflecting a simplified measure of welfare—the degree to which people are unwilling to modify their consumption). All optimisation models were constrained to ensure that the resultant dietary patterns complied with the WHO recommendations and maintained the total calories and proportion of liquids in the diets. Simulations were performed to identify:

1. Optimised average dietary intake for the UK that met the WHO nutritional recommendations without any specification for GHG emissions reduction; and
2. Average dietary intake patterns optimised to achieve target reductions in dietary GHG emissions of 10%, 20%, 30%, 40%, 50% and 60% while meeting the WHO recommendations.[15]

Health impact modelling

We modelled the impact on mortality in the UK associated with the modified dietary patterns resulting from changes in consumption of fruit, vegetables and red and processed meat. We conducted a literature search for meta-analyses relating food or nutrient consumption to non-communicable disease outcomes, and also used information published by the 2010 GBD study.[12] The selected health outcomes were coronary heart disease, stroke, type 2 diabetes and cancers of the mouth/pharynx/larynx, oesophagus, lung, stomach and colon/rectum (table 1).[25–31]

The health impact calculations were performed using the life table model, IOMLIFET,[32] implemented in R.[23] Separate life tables were constructed for males and females, given their different underlying mortality rates. Age-specific and sex-specific data on population size, all-cause mortality and disease-specific mortality from the Office for National Statistics, General Register Office for Scotland and Northern Ireland Statistics and Research Agency were combined to create input data for the UK. To highlight the effects of the dietary modifications, we assume here that the diets are adopted instantly and underlying mortality rates remain constant for the duration of follow-up. For the analysis, the exposure–response functions were assumed to be log-linear and, in cases where several dietary exposures affected the same disease, the risks were multiplied together. The model outputs were changes in years of life lost (YLL) for the UK population over 20 and 30 years.

To account for time lags between dietary changes and changes in health outcomes, time-varying functions based on cumulative distribution functions of normally distributed variables (s-shaped curves) were used.

Table 1 Dietary exposure-response pathways used in the health impact model

Dietary exposure	Health outcome	Relative risk (95% CI)	Source
Fruit	Coronary heart disease	0.93 (0.89 to 0.96) per 80 g increase per day	Dauchet *et al*[26]
	Stroke	0.89 (0.85 to 0.93) per 80 g increase per day	Dauchet *et al*[25]
	Oral cancer (mouth/pharynx/larynx)	0.72 (0.59 to 0.87) per 100 g increase per day	Marmot *et al*[31]
	Oesophagus cancer	0.56 (0.42 to 0.74) per 100 g increase per day	Marmot *et al*[31]
	Lung cancer	0.94 (0.90 to 0.97) per 80 g increase per day	Marmot *et al*[31]
	Stomach cancer	0.67 (0.59 to 0.76) per 100 g increase per day	Marmot *et al*[31]
Non-starchy vegetables	Coronary heart disease	0.89 (0.83 to 0.95) per 80 g increase per day	Dauchet *et al*[26]
	Stroke	0.97 (0.92 to 1.02) per 80 g increase per day	Dauchet *et al*[25]
	Oral cancer (mouth/pharynx/larynx)	0.72 (0.63 to 0.82) per 50 g increase per day	Marmot *et al*[31]
	Oesophagus cancer	0.87 (0.72 to 1.05) per 50 g increase per day	Marmot *et al*[31]
	Stomach cancer	0.70 (0.62 to 0.79) per 100 g increase per day	Marmot *et al*[31]
Red meat	Colorectal cancer	1.29 (1.04 to 1.60) per 100 g increase per day	Marmot *et al*[31]
	Type 2 diabetes	1.19 (1.04 to 1.37) per 100 g increase per day	Pan *et al*[28]
	Stroke	1.21 (1.10 to 1.33) per 100 g increase per day	Micha *et al*[29]
Processed meat	Colorectal cancer	1.21 (1.04 to 1.42) per 50 g increase per day	Marmot *et al*[31]
	Type 2 diabetes	1.51 (1.25 to 1.83) per 100 g increase per day	Pan *et al*[28]
	Coronary heart disease	1.37 (1.11 to 1.68) per 50 g increase per day	Micha *et al*[29]

The shapes of the functions were informed by empirical evidence of the effects of dietary interventions on various causes of mortality over time.[33–36] The assumed lags for coronary heart disease, stroke and type 2 diabetes reach a maximum impact after approximately 10 years and for cancers after 30 years, with no change in cancer risk for the first 10 years (see web materials).

We assessed the sensitivity of the health impact model results in two areas:

1. To test the sensitivity of the results to key parameters, we generated upper and lower health impact estimates using high and low estimates for GHG emissions for each food group based on evidence from the literature, combined with high and low estimates for the relative risks based on published 95% CIs from the source meta-analyses (table 1). These simulations were performed for a 20% GHG reduction only.
2. Since evidence of the independence of health effects due to fruit and vegetable consumption is currently unclear,[37] including both may lead to double counting. There is also evidence that relative risks linking vegetable consumption and coronary heart disease may be overestimated.[26] As a 'structural test' of the model, we therefore repeated the main analysis including effects due to (i) vegetables but not fruit and (ii) fruit but not vegetables.

Further details of the methods are contained in online supplementary web materials.

RESULTS

According to our optimisation model, in order to conform to the WHO nutritional recommendations, the UK diets would need to contain less red meat, dairy products, eggs and sweet and savoury snacks, but more cereals, fruit and vegetables.[15] Modelled changes for all food groups can be found in the online supplementary web materials. Since the required changes to average male diets are greater than those for females, the resulting dietary exposure changes are correspondingly greater (table 2). When no dietary GHG reduction is required (the diet is optimised solely to meet the WHO guidelines), there is a large increase in consumption of fruit and a somewhat smaller increase in consumption of vegetables. However, as GHG emissions are reduced, there is a shift in the optimised diets, with increasingly more vegetables and less fruit (though still more than in current diets). As emissions are progressively reduced, consumption of red and processed meats in the optimised diets is also reduced and eventually removed altogether.

Translation of the optimised dietary patterns into estimated health impacts shows that even diets not constrained to reduce GHG emissions can result in significant beneficial effects on health since the new diets meet the WHO recommendations (table 3). This scenario, with no GHG reduction target, produced an incidental reduction in emissions of over 17%. Although this is short of the needed reduction in emissions from the food and agriculture sector as suggested by the UK Climate Change Committee,[1] our model suggests that the optimised diets would result in a saving of more than 6.8 million YLL over 30 years. This would represent a gain of 12 months of life expectancy for the current birth cohort of males and more than 4 months for females (approximately 8 months on average). Around 70% of this impact is from coronary heart disease and there is also substantial benefit for stroke. Cancer benefits would be likely to be relatively modest initially (due to the initial latency period), but these would become more significant over the longer term, as demonstrated by the impacts over 30 years.

For simulations requiring incremental increases in the level of required GHG emissions mitigation, our results suggest that reducing emissions would lead to a net

Table 2 Modelled changes in average dietary intakes in the UK (relative to current diets) for different levels of GHG reduction

GHG reduction		Modelled change (and % change) from current average diet (g/day)							
		Fruit		Non-starchy vegetables		Red meat		Processed meat	
Target (%)	Achieved (%)	Males	Females	Males	Females	Males	Females	Males	Females
0*	17.2	+110.2 (+80.4%)	+96.8 (+66.7%)	+53.3 (+53.6%)	+57.0 (+56.4%)	−16.2 (−38.0%)	−4.5 (−15.8%)	−37.4 (−63.0%)	−3.2 (−8.8%)
10	18.0	+109.0 (+79.5%)	+94.9 (+65.4%)	+54.5 (+54.9%)	+58.9 (+58.3%)	−16.1 (−37.8%)	−3.4 (−12.1%)	−38.0 (−64.1%)	−3.8 (−10.5%)
20	21.9	+109.4 (+79.8%)	+97.2 (+67.0%)	+54.2 (+54.5%)	+56.6 (+56.0%)	−17.1 (−40.0%)	−11.7 (−41.3%)	−36.2 (−61.0%)	−5.9 (−16.1%)
30	30.0	+107.1 (+78.2%)	+86.1 (+59.3%)	+56.4 (+56.7%)	+67.8 (+67.0%)	−27.2 (−63.9%)	−21.7 (−76.7%)	−37.7 (−63.6%)	−5.9 (−16.4%)
40	40.0	+95.4 (+69.6%)	+77.6 (+53.5%)	+68.2 (+68.6%)	+76.2 (+75.4%)	−35.6 (−83.6%)	−24.8 (−87.4%)	−43.0 (−72.5%)	−11.5 (−31.7%)
50	50.0	+84.6 (+61.7%)	+68.9 (+47.5%)	+78.9 (+79.4%)	+84.9 (+84.0%)	−42.6 (−100.0%)	−27.0 (−95.5%)	−56.0 (−94.4%)	−23.2 (−64.0%)
60	60.0	+36.7 (+26.7%)	+45.2 (+31.2%)	+126.9 (+127.6%)	+108.6 (+107.4%)	−42.6 (−100.0%)	−28.3 (−100.0%)	−59.3 (−100.0%)	−36.3 (−100.0%)

*Diet optimised to achieve the WHO guidelines with no GHG reduction target.
GHG, greenhouse gases.

benefit for health which increases until the emissions are reduced radically, at which point the health benefits may decline (figure 1). In particular, benefits for stroke and cancers peak at a GHG reduction of 50% and are lower for greater reductions. While the 60% GHG reduction still results in large savings of over 8.9 million YLL (30 years), the diet only barely meets the WHO guidelines and its composition is limited to a relatively few food groups. There may also be adverse effects due to reduced consumption of calcium and vitamins (see web materials).

Figure 2 shows the additional health benefits for each outcome as the achieved GHG reduction increases relative to the optimised diet in which no GHG reduction is required. Again, it can be seen that the total health benefit increases as GHG emissions are reduced. However, the additional impact at 60% GHG reduction is only 30% greater than that achieved without a GHG reduction target. In addition, benefits for stomach cancer, oesophageal cancer and lung cancer decrease consistently as GHG emissions are reduced due to reductions in fruit consumption.

Table 3 Modelled health impacts (cumulative reduction in Years of Life Lost, YLL) associated with dietary changes in the UK over 20 and 30 years for a scenario with no GHG reduction target

Health outcome	Cumulative reduction in YLL*	
	Over 20 years	Over 30 years
Coronary heart disease	2 098 200	4 810 400
Stroke	428 000	947 700
Oral cancer	14 600	136 400
Oesophageal cancer	33 900	313 100
Lung cancer	26 600	247 600
Stomach cancer	22 100	200 600
Colorectal cancer	15 900	144 600
Type 2 diabetes	18 900	42 400
Total	2 658 200	6 842 800

*Figures rounded to the nearest 100.

Sensitivity analysis

Sensitivity analysis demonstrates that the majority of the uncertainty relates to the exposure–response functions rather than the GHG emissions (figure 3). The results provide an indication of the ranges around our central model estimates. Since the impacts were modelled using relative risks based on published meta-analyses, the central exposure–response estimates are most likely to be closest to the 'true' associations.

Analyses which accounted only for impacts on health due to consumption of either vegetables *or* fruit reduced the total impacts by around 41% (vegetables only) and 17% (fruit only) for the scenario with no GHG reduction target. As the emissions are progressively reduced, the optimal balance of total fruit and vegetable consumption gradually shifts towards increasing vegetable consumption, since vegetables are associated with lower GHG emissions than fruits on average (figure 4). Counting only impacts due to vegetables therefore leads to additional increases in benefits as GHG emissions are reduced. On the other hand, only counting fruit reduces the benefits for several cancers at greater levels of GHG reduction.

DISCUSSION

Our results show that substantial benefits for health and climate change mitigation can be achieved in the UK by

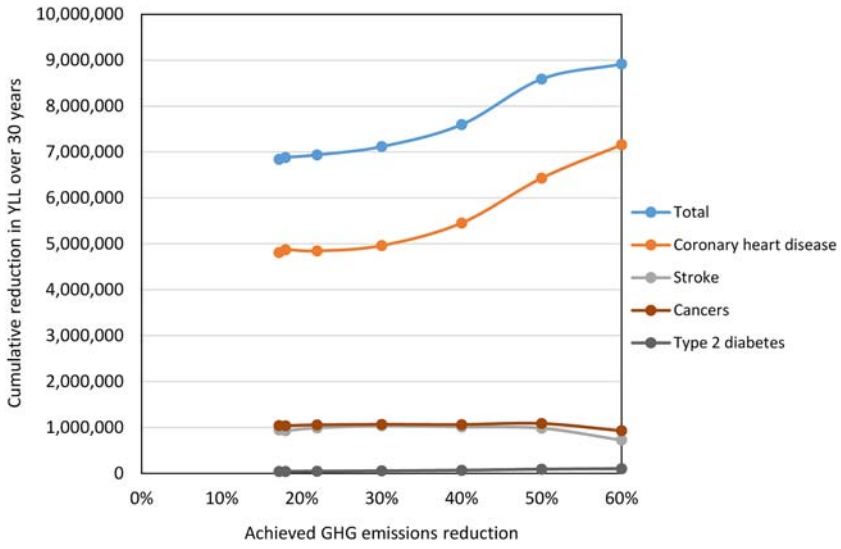

Figure 1 Modelled health impacts associated with dietary changes in the UK for different levels of greenhouse gases (GHG) reduction.

modifying existing diets so that they meet nutritional requirements while also reducing GHG emissions. We have also demonstrated that this can be achieved in ways which maintain the likely acceptability of diets for emission reductions up to 40%. The new diets would contain fewer animal products and savoury snacks and more fruit, vegetables and cereals. Even diets requiring no reduction in GHG emissions were found to result in an incidental reduction of over 17%. Our model suggests that such changes to the UK diets would save almost 7 million YLL over 30 years and increase life expectancy at birth by around 8 months, primarily from benefits to coronary heart disease. Additional health benefits were found to accrue as the GHG emissions associated with diets were progressively reduced. However, the health gains of incremental emission reductions would be lower than those obtained purely by optimising diets to meet health targets alone. Furthermore, if emissions are reduced radically, the optimised diets favour consumption of vegetables over consumption of fruit, since emissions associated with vegetables are lower on average. Therefore, benefits for some health outcomes may begin to reduce and the overall health benefit appears to reach a plateau, driven primarily by the fact that there is no more avoidable meat in the diet.

In general, the level of deviation in the diets from current diets (% deviation from the current diet after normalisation by price elasticities and food expenditure shares) remained low and relatively constant when dietary GHG emissions were reduced by 40% or less. However, as emissions were reduced further, the deviation from the current diet increased dramatically, suggesting that diets in which emissions are reduced by more than 40% are unlikely to be acceptable in the UK unless preferences change in the future.[15] This suggests a natural limit to how much can be achieved for health and climate change mitigation by dietary change alone.

Previous studies have attempted to quantify GHG emission reductions associated with dietary changes using similar optimisation methods to ours.[38] However,

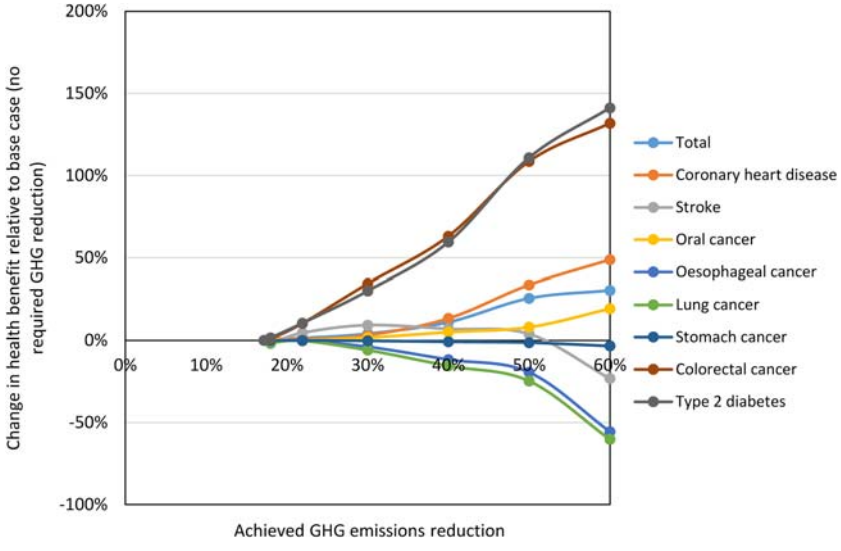

Figure 2 Relative changes in modelled health impacts for incremental increases in greenhouse gases (GHG) reduction target.

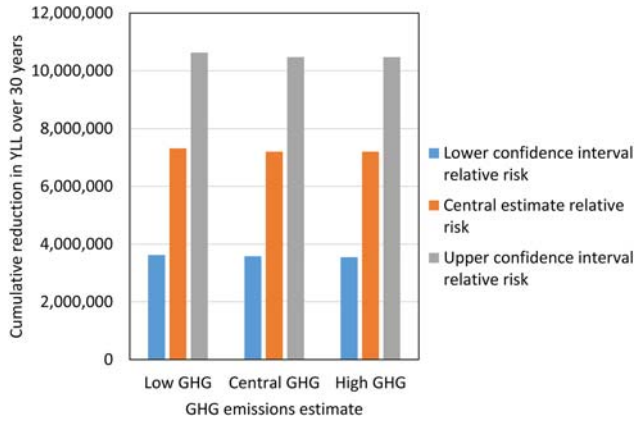

Figure 3 Sensitivity of modelled health impacts (20% greenhouse gases (GHG) reduction) to low/high estimates of GHG emissions and exposure–response functions.

our study has used a novel application of an optimisation approach to model the detailed composition of the entire diet and attempted to maintain its acceptability implicitly through incorporation of information on consumer behaviour (price elasticity and expenditure share). Our method therefore has the considerable advantage of generating more realistic, detailed diets for the UK. This does not, however, necessarily ensure that our dietary scenarios would be acceptable for consumption. In those few studies where the associated health impacts were estimated, the analysis is usually based on hypothetical scenarios in which the nutritional composition of the diet is adjusted to meet dietary or GHG goals.[4 5 8 9] In all cases, these hypothetical scenarios were shown to lead to considerable benefits for health and GHG reduction.

The limitations of the study relate primarily to inadequacies of the available input data and some of the assumptions (as with all modelling analyses). It is most likely that food consumption reported in the NDNS is an underestimate of actual intake but this will not affect the relative contributions of different food groups.[39–41] The NDNS is the most recent data set available for the UK and almost certainly the most accurate. For GHG emissions, we used complete LCI emissions specific to the UK where possible. Our estimates are likely to be somewhat different from other estimates since they incorporate emissions from production, processing, packaging, transport, storage and waste, which are often not included. New advice from the United Nations' Intergovernmental Panel on Climate Change Working Group 1 suggests that different climate active pollutants and GHG should not be combined to produce a single carbon dioxide equivalent (CO_2e) measure of relative climate forcing because they have different effects over a range of time periods.[42] However, the literature to date largely follows earlier convention.

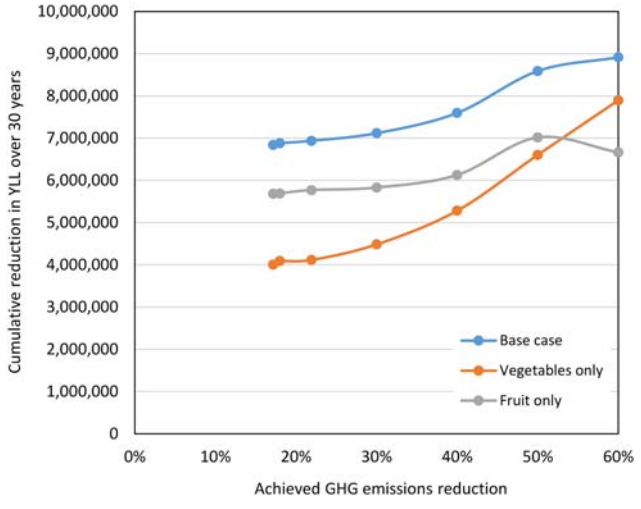

Figure 4 Sensitivity of modelled health impacts to inclusion of effects due to fruit and vegetables for different levels of greenhouse gases (GHG) reduction.

We selected a relatively limited subset of health outcomes for modelling despite evidence of dietary associations for other outcomes.[12 30] In general, epidemiological relationships reported in the literature are not adjusted for consumption of other foods, so we purposely limited the range of modelled outcomes to avoid double counting. We also did not take into account relationships between saturated and unsaturated fat consumption and coronary heart disease because it would be likely to be confounded by the (modelled) relationship between meat consumption and coronary heart disease. We have modelled impacts only on mortality, whereas actual benefits to health would be considerably greater were corresponding impacts on morbidity also included. We also used current mortality rates, although these are declining and may continue to do so in the future (similarly for cardiovascular disease). However, future trends are unclear, for example, because of increases in obesity.[43] Ultimately, our estimates should be treated as indicative of broader patterns rather than precise estimates of the total potential impact.

This study has shown that substantial health benefits could be achieved in the UK by making relatively modest dietary changes which would also benefit the environment. These cobenefits are largely achieved by reducing the consumption of animal products (and switching away from high-emission meats such as beef and lamb and towards lower emission meats such as pork and chicken) and savoury processed foods, while increasing consumption of cereals, fruit and vegetables. However, the health benefits and acceptability of such diets is likely to peak at around a 40% reduction in GHG emissions—greater reductions than this would be likely to result in unacceptable diets and progressively reduced health gains (though still improved relative to current diets). Consequently, more dramatic emission reductions may be required from other sectors of society.

The results show that radical dietary changes such as veganism are not necessary in order for there to be large reductions in GHG emissions and quantifiable

benefits to health. Indeed, making dietary changes that are too restrictive in terms of emissions reduction is likely to place limits on the health benefits that can be achieved by restricting consumption of some healthy foods (such as fruit). Instead, encouraging people in the UK to modify their diets to contain fewer animal products and processed foods and more cereals, fruit and vegetables would produce tangible benefits to both health and the environment.

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Contributors JM contributed to the study design, analysed the data and revised the paper. He is the guarantor of the work. RG contributed to the study design, conducted data management and analysis and drafted the paper. ADD, AH and AM assisted with the study design and revised the draft paper. ZC advised on modelling and revised the draft paper. JS provided data, conducted data management and analysis and revised the draft paper. PW initiated the project, designed the study and revised the draft paper.

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Data sharing statement All researchers involved in the work had full access to all of the data in the study and can take responsibility for the integrity of the data and the accuracy of the data analysis.

Independence of researchers All researchers involved in the work were independent of the funder.

REFERENCES

1. CCC. *Building a low-carbon economy—the UK's contribution to tackling climate change*. London: Committee on Climate Change, 2008.
2. Audsley E, Brander M, Chatterton J, *et al*. *How low can we go? An assessment of greenhouse gas emissions from the UK food system and the scope for reduction by 2050*. WWF-UK, 2009.
3. Stehfest E, Bouwman L, van Vuuren DP, *et al*. Climate benefits of changing diet. *Clim Change* 2009;95:83–102.
4. Scarborough P, Allender S, Clarke D, *et al*. Modelling the health impact of environmentally sustainable dietary scenarios in the UK. *Eur J Clin Nutr* 2012;66:710–15.
5. Friel S, Dangour AD, Garnett T, *et al*. Public health benefits of strategies to reduce greenhouse-gas emissions: food and agriculture. *Lancet* 2009;374:2016–25.
6. Berners-Lee M, Hoolohan C, Cammack H, *et al*. The relative greenhouse gas impacts of realistic dietary choices. *Energ Policy* 2012;43:184–90.
7. Macdiarmid JI, Kyle J, Horgan GW, *et al*. Sustainable diets for the future: can we contribute to reducing greenhouse gas emissions by eating a healthy diet? *Am J Clin Nutr* 2012;96:632–9.
8. Scarborough P, Nnoaham KE, Clarke D, *et al*. Modelling the impact of a healthy diet on cardiovascular disease and cancer mortality. *J Epidemiol Community Health* 2012;66:420–6.
9. Aston LM, Smith JM, Powles JW. Impact of a reduced red and processed meat dietary pattern on disease risks and greenhouse gas emissions in the UK: a modelling study. *BMJ Open* 2012;2:e001072.
10. Tukker A, Goldbohm RA, de Koning A, *et al*. Environmental impacts of changes to healthier diets in Europe. *Ecol Econ* 2011;70:1776–88.
11. Capacci S, Mazzocchi M, Shankar B, *et al*. Policies to promote healthy eating in Europe: a structured review of policies and their effectiveness. *Nutr Rev* 2012;70:188–200.
12. Lim SS, Vos T, Flaxman AD, *et al*. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2224–60.
13. Bates B, Lennox A, Prentice A, *et al*. *National Diet and Nutrition Survey: Headline results from Years 1, 2 and 3 (combined) of the Rolling Programme (2008/2009–2010/11)*. London: Department of Health, Food Standards Agency and NatCen Social Research, 2012.
14. Rayner M, Scarborough P. The burden of food related ill health in the UK. *J Epidemiol Community Health* 2005;59:1054–7.
15. Green R, Milner J, Dangour AD, *et al*. UK food-based greenhouse gas emissions are reduced through realistic and healthy dietary change. *Climatic Change* 2015;129:253–65.
16. Weiss F, Leip A. Greenhouse gas emissions from the EU livestock sector: a life cycle assessment carried out with the CAPRI model. *Agric Ecosyst Environ* 2012;149:124–34.
17. Vieux F, Darmon N, Touazi D, *et al*. Greenhouse gas emissions of self-selected individual diets in France: changing the diet structure or consuming less? *Ecol Econ* 2012;75:91–101.
18. Hammerschlag K, Venkat K. *Meat-eater's guide to climate change and health: lifecycle assessments—methodology and results*. Washington, DC, USA: Environmental Working Group, 2011.
19. Garnett T. Where are the best opportunities for reducing greenhouse gas emissions in the food system (including the food chain)? *Food Policy* 2011;36:S23–32.
20. Foster C, Green K, Bleda M, *et al*. *Environmental impacts of food production and consumption: a report to the department for environment, food and rural affairs*. Defra, London, UK: Manchester Business School, 2006.
21. Venkat K. The climate change and economic impacts of food waste in the United States. *Int J Food System Dynamics* 2011;2:431–46.
22. WHO. *Diet, Nutrition, and the Prevention of Chronic Disease: Report of a Joint WHO/FAO Expert Consultation*. Geneva, Switzerland: World Health Organization, 2003, Contract No.: WHO Technical Report Series 916.
23. R Core Team. *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Contract No.: ISBN 3-900051-07-0. http://www.R-project.org/
24. Varadhan R. Alabama: Constrained nonlinear optimization. 2012. http://CRAN.R-project.org/package=alabama
25. Dauchet L, Amouyel P, Dallongeville J. Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies. *Neurology* 2005;65:1193–7.
26. Dauchet L, Amouyel P, Hercberg S, *et al*. Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies. *J Nutr* 2006;136:2588–93.
27. WCRF. *Food, nutrition, physical activity, and the prevention of cancer: a global perspective*. Washington, DC: World Cancer Research Fund/American Institute for Cancer Research, 2007.
28. 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. *Am J Clin Nutr* 2011;94:1088–96.
29. Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke and dibaetes mellitus—a systematic review. *Circulation* 2010;121:2271–83.
30. Kelly JH, Sabate J. Nuts and coronary heart disease: an epidemiological perspective. *Brit J Nutr* 2006;96:S61–7.
31. Marmot M, Atinmo T, Byers T, *et al*. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. *World Cancer Res Fund/Am Inst Cancer* 2007;46:312–14.
32. Miller B, Hurley J. Life table methods for quantitative impact assessments in chronic mortality. *J Epidemiol Community Health* 2003;57:200–6.
33. Capewell S, O'Flaherty M. Can dietary changes rapidly decrease cardiovascular mortality rates? *Eur Heart J* 2011;32:1187–9.

34. Franco M, Ordunez P, Caballero B, *et al*. Impact of energy intake, physical activity, and population-wide weight loss on cardiovascular disease and diabetes mortality in Cuba, 1980–2005. *Am J Epidemiol* 2007;166:1377–80.
35. Harashima E, Nakagawa Y, Urata G, *et al*. Time-lag estimate between dietary intake and breast cancer mortality in Japan. *Asia Pac J Clin Nutr* 2007;16:193–8.
36. Tsuji K, Harashima E, Nakagawa Y, *et al*. Time-lag effects of dietary fiber and fat intake ratio on Japanese colon cancer mortality. *Biomed Environ Sci* 1996;9:223–8.
37. He FJ, Nowson CA, Lucas M, *et al*. Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. *J Hum Hypertens* 2007;21:717–28.
38. Wilson N, Nghiem N, Ni Mhurchu C, *et al*. Foods and dietary patterns that are healthy, low-cost, and environmentally sustainable: a case study of optimization modeling for New Zealand. *PLoS ONE* 2013;8:e59648.
39. Macdiarmid JI, Blundell J. Assessing dietary intake: who, what and why of under-reporting. *Nutr Res Rev* 1998;11:231–53.
40. Hill RJ, Davies PSW. The validity of self-reported energy intake as determined using the doubly labelled water technique. *Brit J Nutr* 2001;85:415–30.
41. Black AE. The sensitivity and specificity of the Goldberg cut-off for EI:BMR for identifying diet reports of poor validity. *Eur J Clin Nutr* 2000;54:395–404.
42. IPCC. Climate Change 2013: The Physical Science Basis. IPCC Working Group I Contribution to Fifth Assessment Report (AR5). Intergovernmental Panel on Climate Change, 2013.
43. Nichols M, Townsend N, Scarborough P, *et al*. Trends in age-specific coronary heart disease mortality in the European Union over three decades: 1980–2009. *Eur Heart J* 2013;34:3017–27.

SUPPLEMENTARY WEB MATERIALS

This document contains further details of methods and results to accompany the paper *Health effects of adopting low greenhouse gas emission diets in the UK: modelling study*.

Methods

This section contains further details of the methods used in the paper.

Dietary data

The UK National Diet and Nutrition Survey (NDNS) is a rolling programme of cross-sectional surveys using a 4 day food diary.(1) We used data from Waves 1-3 (2008-2011) of the survey, which includes data from 1,571 adults.

The day-level dietary data were used to obtain nutritional information for each food entry on each day of the NDNS. Foods were aggregated into sub-groups according to the NDNS classification (148 groups). The nutritional information for each food entry within a sub-group was then averaged to achieve a nutritional classification for each sub-group, along with the mean g consumed per sitting and the number of sittings for each food group in the dataset.

This information was then used to calculate the average nutritional content for each of the food groups. This was done by calculating proportional weights for each sub-group within a food category (e.g. the weight for cream within the 'milk and milk products' category) using the portion size (in g) and the number of portions of the sub-group that had been eaten. Finally, these weights were applied to all nutritional information in order to calculate weighted averages of nutritional content for each food category.

The individual food-level consumption data were used to obtain the total consumption of each food sub-group over the 4 day diary period among all individuals. These sub-groups were then combined to create 42 food categories, and the total consumption was divided by 4 to give the daily average consumption of each food group. Non-consumers of each food group were included in the averages.

Average intake of nutrients for each individual was taken from the individual-level consumption data. For macronutrients, the recommendations state that intake should be as a proportion of total calories, and we therefore converted grams of macronutrients consumed to calories as a proportion of total calories, using the accepted content figures of 9 kcal per g fat, 4 kcal per g protein, and 4 kcal per g carbohydrate.

Greenhouse gas emissions

Estimates of greenhouse gas (GHG) emissions associated with each of the 42 food groups were calculated using a Life Cycle Inventory (LCI) compiled from the relevant literature in the UK and Europe.(2-6) In some cases a full LCI of emissions was not available,(3-5) so extrapolations from the literature(2, 3, 6-8) were used to extend the estimates across the full life cycle of the food. We also estimated food losses from production, handling and sales, from cooking meals and from consumer waste, extrapolated from estimates of waste in the US.(9) For food groups where specific emissions estimates were not available in the literature, representative items included in the food group for which emissions data were available were used as a reference point for all foods within the group.

Optimization method

To produce the potential future diets, optimizations were performed in the statistical software R(10) using the package Alabama which optimizes smooth nonlinear objective functions with constraints.(11) Optimizations were performed separately for males and females given their different diets.

We modelled potential future diets which achieve nutrition and GHG emission targets but also which minimize deviation from the current average diet for men and women. For a given food group i, the loss of welfare W_i resulting from consumption greater or less than the ideal level for health is proportional to the share of expenditure for that food group s_i and inversely proportional to the price elasticity of demand ε_i

$$\Delta W_i \propto \frac{s_i}{\varepsilon_i}\left(\frac{\Delta X_i}{X_i}\right)^2$$

where X_i is the current consumption for food group i and ΔX_i is the difference between current and ideal consumption for food group i. The analysis therefore seeks to find the combination of foods that minimizes the weighted deviations of squared percentage consumption from the desired levels, where each deviation is weighted by s_i/ε_i. For the 42 food groups identified from the NDNS, we attempted to find the solution of

$$\min_{\{\Delta X_i; i=1..42\}}\left[\sum_{i=1}^{42}\frac{s_i}{\varepsilon_i}\left(\frac{\Delta X_i}{X_i}\right)^2\right]$$

whilst ensuring that the resultant diet complied with WHO recommendations and maintained the total calories and proportion of liquids in the diets. The ideal consumption of food i is given by $X_i^* = X_i + \Delta X_i^*$ where ΔX_i^* is the solution for food i. Initial estimates of future consumption for each food group (i.e. initial estimates of the solution of the above equation) were generated randomly. The values of s_i were determined directly from the NDNS dietary survey. Values of ε_i were obtained from Tiffin et al. (2011).(12) Ideal consumption levels of different nutrients in the diet were determined from WHO nutritional guidelines, shown in Table S1.(13)

Table S1. Nutritional content of current UK diet for males and females compared to WHO guideline values

Food group / nutrient	WHO guideline	Current UK diet	
		Males	Females
Total energy (kcal)	-	2,010	1,560
Total fat (% total energy)	15-30%	29.91%	31.38%
Saturated fat (% total energy)	<10%	11.25%	11.96%
Polyunsaturated fat (% total energy)	6-10%	4.87%	5.09%
N6 polyunsaturated fat (% total energy)	5-8%	4.05%	4.22%
N3 polyunsaturated fat (prop total energy)	1-2%	0.82%	0.88%
Trans fat (% total energy)	<1%	0.68%	0.74%
Monounsaturated fat (prop total energy)	(remaining)	10.60%	10.96%
Carbohydrate (% total energy)	55-75%	50.01%	53.66%
Free sugars (% total energy)	<10%	16.19%	15.57%
Protein (% total energy)	10-15%	14.78%	15.16%
Cholesterol (mg)*	<300 mg	-	-
Sodium (g)	<2 g	2.20	1.69
Fruit and vegetables (g)	≥400 g	236.50	246.20

*Not modelled

In order to find solutions which achieved both nutrition and GHG emission reduction targets, constraints were specified which varied depending on the scenario. Primarily, we explored the effect of 0%, 10%, 20%, 30%, 40%, 50% and 60% reductions in GHG emissions associated with food (with the required minimum reduction specified as a constraint). Further constraints were applied to avoid unrealistic solutions. These included:

- Total calories equal to present day average;
- Total liquids (excluding alcohol) equal to present day;
- Tea, coffee and mineral water cannot more than double;
- Consumption of each food group must be ≥0.

Each simulation was repeated 100 times to increase the probability of finding an overall minimum solution rather than local minima.

Health impact model

The health impact calculations were performed using a version of the life table model, IOMLIFET,(14) implemented in R.(10) The model estimates survival patterns in the population over time based on age-specific mortality rates. To perform an impact assessment, the underlying mortality rates are adjusted (using knowledge of the change in exposure combined with the exposure-response function) and the resulting life table is compared against the baseline life table.

Table S2 maps the modelled health outcomes to WHO International Classification of Diseases (ICD-10) codes. These codes are needed for the disease-specific mortality data used in the model.

Table S2. Underlying cause of death classifications (ICD-10) used for each health outcome

Health outcome	ICD-10 underlying cause of death classification	
	Codes	Underlying causes
Coronary heart disease	I20 – I25	Ischaemic heart diseases
Stroke	I61 – I64	Intracerebral haemorrhage; Other nontraumatic intracranial haemorrhage; Cerebral infarction; Stroke not specified as haemorrhage or infarction
Oral cancer (mouth/pharynx/larynx)	C00 – C10, C12 – C14, C32	Malignant neoplasms of lip, oral cavity and pharynx (excluding Malignant neoplasm of nasopharynx)*; Malignant neoplasm of larynx
Oesophageal cancer	C15	Malignant neoplasm of oesophagus
Lung cancer	C33 – C34	Malignant neoplasm of trachea, bronchus and lung
Stomach cancer	C16	Malignant neoplasm of stomach
Colorectal cancer	C18 – C20, C21.8	Malignant neoplasm of colon; Malignant neoplasm of rectosigmoid junction; Malignant neoplasm of rectum; Overlapping lesion of rectum, anus and anal canal[+]
Type 2 diabetes	E11	Non-insulin-dependent diabetes mellitus

* Malignant neoplasm of nasopharynx (ICD-10 C11) excluded since this was considered separately in Marmot et al. (2007)(15)

[+] Overlapping lesion of rectum, anus and anal canal (ICD-10 C21.8) included for consistency with Cancer Research UK (http://www.cancerresearchuk.org/cancer-info/cancerstats/types/bowel/survival/bowel-cancer-survival-statistics)

Age- and sex-specific data on population size, all-cause mortality and disease-specific mortality for ages 0 to 105 were obtained from the Office for National Statistics (England and Wales), the General Register Office for Scotland (Scotland) and the Office for National Statistics/Northern Ireland Statistics and Research Agency (Northern Ireland). These data were added together at each age to create data for the UK. The disease-specific mortality data were not available in single-year-of-age format. Therefore, this was generated from the age-grouped data by linear interpolation. Separate life tables were created for each outcome (to allow quantification of the impact due to that outcome alone).

Changes in risk were applied at all ages in the life tables. For the analysis, the exposure-response functions were assumed to be log-linear. To calculate the change in mortality risk ΔR associated with a modelled change in dietary exposure δE

$$\Delta R = exp\left[\frac{log(RR_{\Delta E})}{\Delta E} \times \delta E\right]$$

where $RR_{\Delta E}$ is the relative risk associated with a change in exposure ΔE (i.e. the relative risk reported in the literature). For example, for a 110.5 g increase in fruit consumption the change in the risk of oeshophageal cancer is

$$\Delta R = exp\left[\frac{log(0.56)}{100} \times 110.5\right] = 0.53$$

Therefore, the age-specific mortality rates in the life table would be multiplied by 0.53. In cases where several dietary exposures affect the same disease risk, the risks were multiplied together. So, the change in oral cancer risk equals the change in oral cancer risk due to changes in fruit consumption multiplied by the change in oral cancer risk due to changes in non-starchy vegetable consumption. For example, for a 110.5 g increase in fruit consumption and a 53.0 g increase in non-starchy vegetable consumption, the change in the risk of oral cancer would be

$$\Delta R = exp\left[\frac{log(0.72)}{100} \times 110.5\right] \times exp\left[\frac{log(0.72)}{50} \times 53.0\right] = 0.49$$

To account for time lags between dietary changes and changes in health outcomes, time-varying functions based on cumulative distribution functions of normally distributed variables (s-shaped or sigmoidal curves) were used in the model. The shapes of the functions were informed by empirical evidence of the effects of dietary interventions on various causes of mortality over time.(16-19) The assumed lags for coronary heart disease, stroke, and type 2 diabetes reach a maximum impact after approximately 10 years (Figure S1) and for cancers after around 30 years, with no change in cancer risk for the first 10 years (Figure S2).

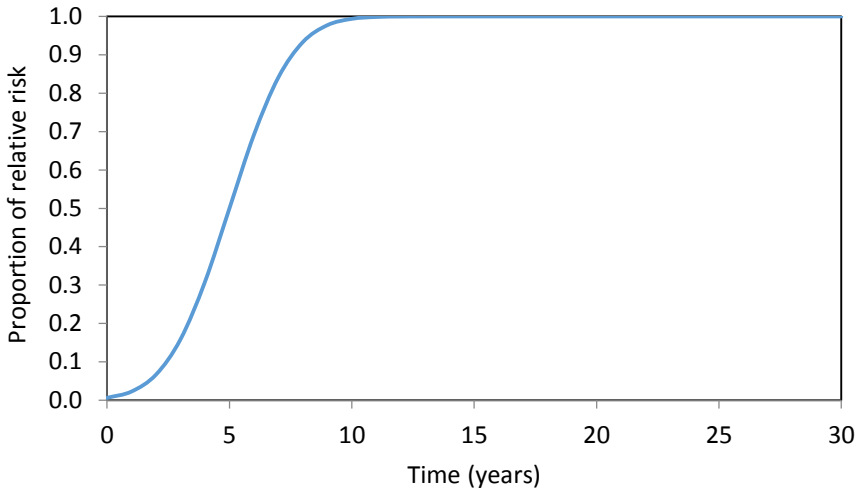

Figure S1. Time lag function used for coronary heart disease, stroke and type 2 diabetes

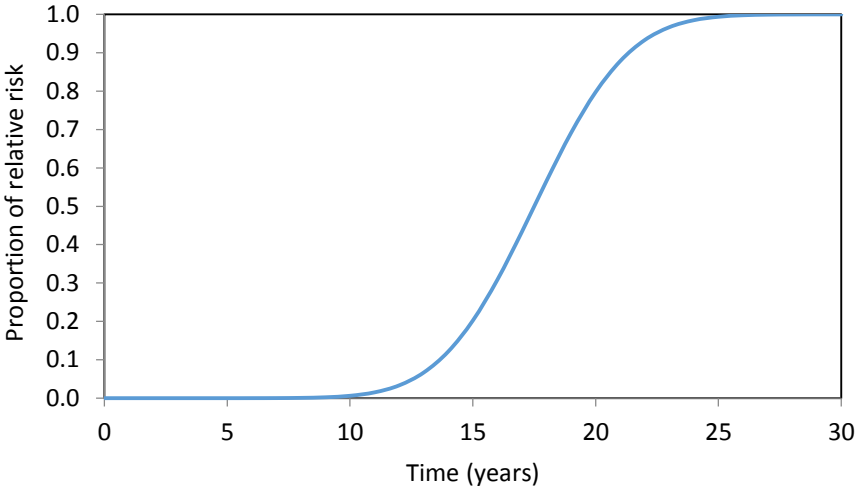

Figure S2. Time lag function used for all cancer outcomes

Results

This sections contains additional results relevant to the paper.

Optimized diets

Tables S3 and S4 show the full optimized diets for each GHG reduction target (42 food groups).

Table S3. Optimized diets in 42 food groups for UK adult males for different levels of GHG reduction

Food group	Average consumption for different GHG reduction targets (g/day)							
	Current diet	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Beef	24.2	13.8	14.0	13.6	5.0	0.0	0.0	0.0
Processed beef	25.1	18.2	18.2	18.7	17.7	16.3	3.3	0.0
Pork	9.7	6.0	6.0	4.5	5.5	6.6	0.0	0.0
Processed pork	34.2	3.7	3.0	4.4	3.9	0.0	0.0	0.0
Lamb	7.9	5.8	5.7	6.6	4.3	0.0	0.0	0.0
Other red meat	0.8	0.8	0.7	0.7	0.6	0.4	0.0	0.0
Poultry	37.2	23.3	23.2	23.5	26.8	24.9	1.2	2.7
Processed white meat	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Fish	24.3	30.0	30.2	28.5	29.2	25.4	20.9	0.0
Milk and milk products	194.8	114.0	125.6	117.0	108.1	58.7	6.5	0.0
Cheese	16.3	2.6	3.4	2.0	2.6	3.4	0.0	0.0
Ice cream	5.1	4.2	4.1	4.6	4.2	4.0	1.4	0.0
Eggs	20.9	4.8	3.8	4.7	10.1	20.5	15.6	3.5
Bread	99	145.4	145.8	145.6	142.7	146.1	154.9	147.9
Pasta and pizza	42.7	45.7	45.6	46.6	47.2	49.7	49.5	40.8
Breakfast cereals	28.6	39.5	37.3	39.7	40.3	42.3	61.8	97.8
Rice	27.1	51.1	51.3	51.4	48.2	33.1	35.5	5.6
Other cereals	6.7	14.4	13.8	14.6	15.4	19.2	29.3	43.8
Unprocessed potatoes	73.9	123.0	121.4	122.6	121.0	115.9	118.7	87.3
Processed potatoes	24.5	31.8	33.5	32.6	32.9	31.2	36.7	41.5
Other vegetables	84.8	132.4	132.0	131.9	134.0	140.7	146.0	164.7
Beans and pulses	14.6	20.3	21.9	21.6	21.7	26.8	32.3	61.6
Tomatoes	45.4	90.6	90.6	90.4	87.7	74.5	49.9	0.0
Fruit	91.7	156.7	155.4	156.1	156.6	158.0	171.7	173.7
Butter	3.7	1.0	0.0	1.3	1.6	1.4	0.0	0.0
Margarine and low fat spread	9.3	21.8	21.7	22.0	20.9	16.4	13.3	0.0
Cooking oil	0.3	0.3	0.4	0.3	0.3	0.4	0.5	0.9
Biscuits	13.6	14.1	16.0	13.4	14.8	18.7	21.9	23.1
Buns and cakes	18.5	20.1	18.4	19.2	21.5	26.4	32.2	37.9
Chocolate and sweets	9.9	8.6	9.0	9.3	9.4	10.8	10.8	11.6
Sugar and sweet spreads	10.7	7.1	8.4	7.4	9.2	11.9	9.3	1.7
Crisps and savoury snacks	7.2	7.2	7.6	6.3	7.5	9.9	13.1	13.8

Puddings and pies	19.1	12.6	12.2	12.8	16.5	21.2	12.0	0.0
Soups	36.8	0.9	0.3	0.0	0.0	0.0	0.0	0.0
Preserves	27.6	2.6	1.8	4.7	3.2	0.0	0.0	0.0
Soft drinks	246.3	65.9	55.0	42.6	21.7	0.0	0.0	0.0
Alcoholic drinks	426.4	426.4	426.4	426.4	426.4	426.4	426.4	426.4
Fruit juice	62	50.1	45.2	41.2	29.4	0.0	0.0	0.0
Coffee	265.4	25.9	0.0	0.0	0.0	0.8	0.0	0.0
Mineral water	66.2	102.1	1.7	1.2	1.6	6.2	0.0	0.0
Tea	406.8	802.7	944.8	961.7	994.0	1,039.7	1,046.7	1,046.7
Nuts and seeds	2.9	5.7	5.9	6.0	5.5	8.5	12.4	30.4

Table S4. Optimized diets in 42 food groups for UK adult females for different levels of GHG reduction

Food group	Average consumption for different GHG reduction targets (g/day)							
	Current diet	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Beef	17.2	13.9	14.4	7.6	0.0	0.0	0.0	0.0
Processed beef	15.6	14.2	16.1	14.9	13.4	11.8	7.8	0.0
Pork	5.3	5.0	5.1	5.1	4.4	3.4	1.1	0.0
Processed pork	20.7	19.0	16.4	15.6	16.9	13.0	5.3	0.0
Lamb	5.6	4.8	5.2	3.7	2.0	0.0	0.0	0.0
Other red meat	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.0
Poultry	30.1	30.2	29.9	28.6	25.1	14.9	0.0	0.0
Processed white meat	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Fish	23.4	26.8	27.8	26.7	25.9	23.2	16.4	1.6
Milk and milk products	182.7	113.2	125.6	115.2	112.2	69.6	0.0	0.0
Cheese	12.9	6.5	4.8	6.7	7.0	2.8	0.0	0.0
Ice cream	5.1	3.8	3.6	4.2	4.3	4.3	2.9	0.0
Eggs	17.3	12.7	12.4	14.7	15.5	15.2	10.3	0.0
Bread	69.4	92.4	92.7	95.3	96.7	112.1	131.9	133.6
Pasta and pizza	31.9	31.9	31.7	33.0	32.7	34.1	31.2	19.9
Breakfast cereals	27	32.0	31.3	31.7	32.4	41.0	48.6	68.8
Rice	23.4	29.3	29.2	26.7	24.8	23.7	14.6	0.0
Other cereals	6.4	8.7	8.3	9.4	12.4	17.0	23.5	40.1
Unprocessed potatoes	98.1	122.1	120.7	119.8	119.2	115.4	122.0	68.3
Processed potatoes	18.2	19.8	17.9	20.0	20.7	21.6	23.5	29.6
Other vegetables	88.6	137.7	139.6	136.0	144.8	150.7	153.7	156.2
Beans and pulses	12.5	20.4	20.5	21.7	24.0	26.6	32.2	53.5
Tomatoes	43.1	83.7	82.8	82.7	72.9	61.3	42.2	28.2
Fruit	102	158.2	157.2	159.5	158.3	161.4	171.8	162.1
Butter	3.6	0.0	0.0	0.0	0.5	0.0	0.0	0.0
Margarine and low fat spread	6.3	11.9	12.2	11.5	10.4	9.2	8.9	3.3
Cooking oil	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.4
Biscuits	13.3	11.5	12.7	12.6	15.1	17.8	20.3	27.2
Buns and cakes	16.2	15.0	14.8	18.4	19.2	22.5	26.5	36.6
Chocolate and sweets	8.8	7.7	7.8	8.3	8.6	9.4	10.2	6.9
Sugar and sweet spreads	5.9	4.8	5.0	5.8	6.3	8.0	9.5	5.4
Crisps and savoury snacks	5.6	5.6	5.8	5.7	6.6	7.4	8.8	13.0

Puddings and pies	14.2	11.2	10.9	12.4	12.8	12.0	7.5	0.0
Soups	36.1	36.1	35.8	20.5	0.0	0.0	0.0	0.0
Preserves	24.3	6.5	5.7	6.3	6.2	0.0	0.0	0.0
Soft drinks	187	61.2	60.2	31.0	21.4	0.0	0.0	0.0
Alcoholic drinks	117.8	117.8	117.8	117.8	117.8	117.8	117.8	117.8
Fruit juice	46.9	37.6	37.6	28.7	21.3	0.0	0.0	0.0
Coffee	246.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Mineral water	81.4	0.0	0.0	0.0	0.1	1.5	0.0	0.0
Tea	445.6	908.7	909.6	947.7	964.6	1,005.9	1,007.4	1,007.4
Nuts and seeds	2.2	3.2	3.2	3.5	4.0	5.0	6.9	14.3

Modelled changes in nutrients

Tables S5 and S6 show the resulting changes in nutrients and food groups (relative to the current diet) used for the health impact assessment.

Table S5. Modelled changes in health-relevant nutrients for UK males for different levels of GHG reduction

Nutrient / food group	Change for different GHG reduction targets						
	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Trans fatty acids (% total energy)	-0.18%	-0.18%	-0.18%	-0.19%	-0.22%	-0.33%	-0.42%
Fibre (g)	5.0	5.0	5.1	5.1	5.8	7.7	10.4
Sodium (g)	-0.2	-0.20	-0.2	-0.2	-0.2	-0.2	-0.2
Calcium (mg)	-110.1	-95.8	-112.1	-115.3	-141.4	-187.3	-155.4
Iron (mg)	1.0	0.9	1.0	1.0	1.0	1.9	3.9
Vitamin B12 (µg)	-1.1	-1.1	-1.1	-1.2	-1.5	-2.2	-2.8
Fruit (g)[a]	110.2	109.0	109.4	107.1	95.4	84.6	36.7
Non-starchy vegetables (g)[b]	53.3	54.5	54.2	56.4	68.2	78.9	126.9
Red meat (g)[c]	-16.2	-16.1	-17.1	-27.2	-35.6	-42.6	-42.6
Processed meat (g)[d]	-37.4	-38.0	-36.2	-37.7	-43.0	-56.0	-59.3
Nuts (g)	2.8	3.0	3.1	2.6	5.6	9.5	27.5

[a]Includes food groups: tomatoes, fruit
[b]Includes food groups: other vegetables, beans and pulses
[c]Includes food groups: beef, pork, lamb, other red meat
[d]Includes food groups: processed beef, processed pork, processed white meat

Table S6. Modelled changes in health-relevant nutrients for UK females for different levels of GHG reduction

Nutrient / food group	Change for different GHG reduction targets						
	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Trans fatty acids (% total energy)	-0.15%	-0.16%	-0.17%	-0.19%	-0.27%	-0.36%	-0.44%
Fibre (g)	3.7	3.6	3.8	4.0	5.1	6.5	7.9
Sodium (g)	0.1	0.1	0.1	0.0	0.0	0.0	0.0
Calcium (mg)	-76.2	-72.1	-66.6	-59.7	-97.6	-161.6	-133.9
Iron (mg)	0.6	0.7	0.7	0.8	1.2	1.4	2.6
Vitamin B12 (µg)	-0.6	-0.5	-0.6	-0.7	-1.1	-1.9	-2.4
Fruit (g)[a]	96.8	94.9	97.2	86.1	77.6	68.9	45.2
Non-starchy vegetables (g)[b]	57.0	58.9	56.6	67.8	76.2	84.9	108.6
Red meat (g)[c]	-4.5	-3.4	-11.7	-21.7	-24.8	-27.0	-28.3
Processed meat (g)[d]	-3.2	-3.8	-5.9	-5.9	-11.5	-23.2	-36.3
Nuts (g)	1.0	1.0	1.4	1.8	2.8	4.7	12.1

[a]Includes food groups: tomatoes, fruit
[b]Includes food groups: other vegetables, beans and pulses
[c]Includes food groups: beef, pork, lamb, other red meat
[d]Includes food groups: processed beef, processed pork, processed white meat

Modelled health impacts

Tables S7 and S8 show the modelled health impacts for each outcome over 20 and 30 years.

Table S7. Modelled health impacts for each outcome over 20 years for different levels of GHG reduction

Health outcome	Cumulative reduction in years of life lost						
	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Coronary heart disease	2,098,236	2,125,063	2,114,439	2,164,704	2,379,812	2,806,775	3,126,928
Stroke	428,000	419,593	447,662	467,631	457,648	444,234	328,723
Oral cancer	14,573	14,639	14,596	14,819	15,306	15,747	17,373
Oesophageal cancer	33,927	33,588	33,824	32,598	29,956	27,313	15,067
Lung cancer	26,617	26,242	26,540	25,045	22,487	20,035	10,545
Stomach cancer	22,074	22,058	22,070	22,009	21,887	21,773	21,321
Colorectal cancer	15,893	16,041	17,522	21,341	25,892	33,113	36,786
Type 2 diabetes	18,903	19,196	20,930	24,648	30,262	40,049	45,872
Total	2,658,223	2,676,420	2,697,582	2,772,795	2,983,249	3,409,039	3,602,615

Table S8. Modelled health impacts for each outcome over 30 years for different levels of GHG reduction

Health outcome	Cumulative reduction in years of life lost						
	0% GHG reduction	10% GHG reduction	20% GHG reduction	30% GHG reduction	40% GHG reduction	50% GHG reduction	60% GHG reduction
Coronary heart disease	4,810,412	4,871,642	4,844,374	4,959,769	5,451,598	6,429,843	7,158,336
Stroke	947,731	929,254	990,689	1,035,374	1,013,498	984,054	727,100
Oral cancer	136,385	137,007	136,597	138,684	143,243	147,376	162,606
Oesophageal cancer	313,053	309,924	312,096	300,811	276,416	252,014	138,895
Lung cancer	247,577	244,069	246,871	232,816	209,038	186,238	98,262
Stomach cancer	200,587	200,434	200,546	199,988	198,883	197,843	193,740
Colorectal cancer	144,639	145,992	159,517	194,330	235,816	301,671	335,203
Type 2 diabetes	42,391	43,046	46,833	55,108	67,596	89,365	102,220
Total	6,842,776	6,881,367	6,937,522	7,116,879	7,596,088	8,588,404	8,916,362

References

1. Bates B, Lennox A, Prentice A, Bates C, Swan G. National Diet and Nutrition Survey: Headline results from Years 1, 2 and 3 (combined) of the Rolling Programme (2008/2009 - 2010/11). London: Department of Health, Food Standards Agency and NatCen Social Research, 2012.
2. Audsley E, Brander M, Chatterton J, Murphy-Bokern D, Webster C, Williams A. How low can we go? An assessment of greenhouse gas emissions from the UK food system and the scope for reduction by 2050. WWF-UK, 2009.G
3. Berners-Lee M, Hoolohan C, Cammack H, Hewitt CN. The relative greenhouse gas impacts of realistic dietary choices. Energ Policy. 2012;43:184-90.
4. Weiss F, Leip A. Greenhouse gas emissions from the EU livestock sector: a life cycle assessment carried out with the CAPRI model. Agric Ecosyst Environ. 2012;149:124-34.
5. Vieux F, Darmon N, Touazi D, Soler LG. Greenhouse gas emissions of self-selected individual diets in France: changing the diet structure or consuming less? Ecol Econ. 2012;75:91-101.
6. Hammerschlag K, Venkat K. Meat-Eater's Guide to Climate Change and Health: Lifecycle Assessments - Methodology and Results. Washington, DC, USA: Environmental Working Group, 2011.
7. Garnett T. Where are the best opportunities for reducing greenhouse gas emissions in the food system (including the food chain)? Food Policy. 2011;36:S23-S32.
8. Foster C, Green K, Bleda M, Dewick P, Evans B, Flynn A, et al. Environmental Impacts of Food Production and Consumption: A Report to the Department for Environment, Food and Rural Affairs. Defra, London, UK: Manchester Business School, 2006.
9. Venkat K. The climate change and economic impacts of food waste in the United States. International Journal on Food System Dynamics. 2011;2:431-46.
10. R Core Team. R: A language and environment for statistical computing. Contract No.: ISBN 3-900051-07-0 http://www.R-project.org/. Vienna, Austria: R Foundation for Statistical Computing, 2012.
11. Varadhan R. Alabama: Constrained nonlinear optimization. http://CRAN.R-project.org/package=alabama. 2012.
12. Tiffin R, Balcombe K, Salois M, Kehlbacher A. Estimating Food and Drink Elasticities. Reading: University of Reading and Defra, 2011.
13. WHO. Diet, Nutrition, and the Prevention of Chronic Disease: Report of a Joint WHO/FAO Expert Consultation. Geneva, Switzerland: World Health Organization, 2003.
14. Miller B, Hurley J. Life table methods for quantitative impact assessments in chronic mortality. J Epidemiol Community Health. 2003;57:200-6.
15. Marmot M, Atinmo T, Byers T, Chen J, Hirohata T, Jackson A, James W, Kolonel L, Kumanyika S, Leitzmann C. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. World Cancer Res Fund/Am Inst Cancer. 2007;46:312-4.
16. Capewell S, O'Flaherty M. Can dietary changes rapidly decrease cardiovascular mortality rates? Eur Heart J. 2011;32:1187-9.
17. Franco M, Ordunez P, Caballero B, Tapia Granados JA, Lazo M, Bernal JL, et al. Impact of energy intake, physical activity, and population-wide weight loss on cardiovascular disease and diabetes mortality in Cuba, 1980-2005. Am J Epidemiol. 2007;166:1377-80.
18. Harashima E, Nakagawa Y, Urata G, Tsuji T, Shirataka M, Matsumura Y. Time-lag estimate between dietary intake and breast cancer mortality in Japan. Asia Pac J Clin Nutr. 2007;16:193-8.
19. Tsuji K, Harashima E, Nakagawa Y, Urata G, Shirataka M. Time-lag effects of dietary fiber and fat intake ratio on Japanese colon cancer mortality. Biomed Environ Sci. 1996;9:223-8.