BMJ open accessible medical research

PRESS RELEASE

Differences in coronary heart disease, stroke and cancer mortality rates between England, Wales, Scotland and Northern Ireland: the role of diet and nutrition

Peter Scarborough,[1] Robert David Morgan,[2] Premila Webster,[3] Mike Rayner[1]

To cite: Scarborough P, Morgan RD, Webster P, *et al.* Differences in coronary heart disease, stroke and cancer mortality rates between England, Wales, Scotland and Northern Ireland: the role of diet and nutrition. *BMJ Open* 2011;**1**:e000263. doi: 10.1136/bmjopen-2011-000263

► Prepublication history and additional appendix for this paper are available online. To view these files please visit the journal online (http://bmjopen.bmj.com).

Received 20 July 2011
Accepted 14 September 2011

[1]British Heart Foundation Health Promotion Research Group, Department of Public Health, University of Oxford, Oxford, UK
[2]Department of Medicine, John Radcliffe Hospital, Oxford, UK
[3]Department of Public Health, University of Oxford, Oxford, UK

Correspondence to
Dr Peter Scarborough; peter.scarborough@dph.ox.ac.uk

ABSTRACT

Introduction: It is unclear how much of the geographical variation in coronary heart disease (CHD), stroke and cancer mortality rates within the UK is associated with diet. The aim of this study is to estimate how many deaths from CHD, stroke and cancer would be delayed or averted if Wales, Scotland and Northern Ireland adopted a diet equivalent in nutritional quality to the English diet.

Methods: Mortality data for CHD, stroke and 10 diet-related cancers for 2007–2009 were used to calculate the mortality gap (the difference between actual mortality and English mortality rates) for Wales, Scotland and Northern Ireland. Estimates of mean national consumption of 10 dietary factors were used as baseline and counterfactual inputs in a macrosimulation model (DIETRON). An uncertainty analysis was conducted using a Monte Carlo simulation with 5000 iterations.

Results: The mortality gap in the modelled scenario (achieving the English diet) was reduced by 81% (95% credible intervals: 62% to 108%) for Wales, 40% (33% to 51%) for Scotland and 81% (67% to 99%) for Northern Ireland, equating to approximately 3700 deaths delayed or averted annually. For CHD only, the mortality gap was reduced by 88% (69% to 118%) for Wales, 58% (47% to 72%) for Scotland, and 88% (70% to 111%) for Northern Ireland.

Conclusion: Improving the average diet in Wales, Scotland and Northern Ireland to a level already achieved in England could have a substantial impact on reducing geographical variations in chronic disease mortality rates in the UK. Much of the mortality gap between Scotland and England is explained by non-dietary risk factors.

ARTICLE SUMMARY

Article focus

- Scotland, Wales and Northern Ireland experience excess cardiovascular and cancer mortality compared to England.
- How much of this excess mortality is associated with differences in diet and nutrition in the four countries of the UK?

Key messages

- Modelled results suggest that if Wales and Northern Ireland achieved an average diet equivalent in nutritional quality to the average diet in England, then 81% of the excess cardiovascular and cancer mortality experienced in these countries would be removed.
- If Scotland achieved an average diet equivalent in nutritional quality to the average diet in England, then 40% of the excess cardiovascular and cancer mortality would be removed.
- For Wales, Scotland and Northern Ireland, changes in diet would have the biggest impact on inequalities in coronary heart disease mortality.

Strengths and limitations of this study

- The macrosimulation model used for the analysis is parameterised using meta-analyses of cohort and case–control studies, and considers 10 different dietary factors and 10 mortality outcomes.
- Uncertainty analysis, allowing parameter estimates to vary stochastically according to distributions reported in the literature, allow for an assessment of the uncertainty of the presented results.
- The model is parameterised from meta-analyses of observational studies, and therefore it is not possible to exclude the possibility of residual confounding.

INTRODUCTION

Within the UK there is considerable geographical variation in morbidity and mortality associated with chronic disease.[1–5] This variation is apparent for coronary heart disease (CHD),[5–8] stroke[4 9 10] and, to a lesser extent, cancer.[10 11] Previous attempts to quantify the impact of modifiable risk factors on these geographical variations have only

been able to partially explain them,[5 8] and have been restricted to individual cohorts that may not represent the experience of the whole population. It would be helpful to quantify the impact of behavioural risk factors on these geographical inequalities in order to prioritise public health interventions.

It is well established that a poor diet (ie, high in saturated fat and salt, and low in fibre, fruit and vegetables) is associated with increased risk of CHD, stroke and certain cancers, such as oesophageal, stomach and colorectal cancer.[6 12] A number of reports have demonstrated consistent geographical differences in the nutritional qualities of national diets within the UK.[13 14] It has been speculated that differences in dietary quality could be responsible for much of the differences in health experienced in the different countries of the UK, but no studies have so far attempted to quantify the full impact of diet on geographical health variations within the UK.[15 16]

A macrosimulation model (DIETRON) has been developed that quantifies the change in population mortality from CHD, stroke and 10 diet-related cancers that would be expected given a change in the average dietary quality within a population. Previous analysis using DIETRON suggested that 33 000 deaths would be delayed or averted per year in the UK if recommended dietary intakes for fats, fruit and vegetables, salt and fibre were achieved.[17] The lowest potential reduction in estimated mortality occurred in England (13.8%) compared with Wales (15.0%), Scotland (18.3%) and Northern Ireland (18.9%), suggesting that health inequalities within the UK would be narrowed if dietary quality were standardised. In this paper, we use the DIETRON model to quantify how much of the CHD, stroke and cancer 'mortality gap' between England, Scotland, Wales and Northern Ireland would be closed if dietary quality were standardised to the current level achieved in England.

METHODS

The DIETRON model[17] was used to estimate the annual number of deaths from CHD, stroke and 10 diet-related cancers (mouth/larynx/pharynx, oesophagus, stomach, lung, colon, gallbladder, pancreas, breast, endometrial and kidney) that would be delayed or averted in Wales, Scotland and Northern Ireland if the nutritional quality of the average diets in these countries were equivalent to the average diet in England. The DIETRON model uses age- and gender-specific estimates of relative risks drawn from meta-analyses of trials, cohort studies and case–control studies.[12 18–24] The model estimates a change in risk for an individual as a result of changes in dietary quality, and this change in risk is applied to a population to estimate the percentage difference in cause-specific mortalities that would be expected under the assumptions that (a) relative risks are combined multiplicatively, and (b) the relationship between the nutritional quality of a diet, risk factors and CHD, stroke and cancer follows a dose–response relationship. The DIETRON model used in this paper is a slight adaptation of the model described elsewhere[17] in that a parameter has been included that models change in body weight as a result of changes in both energy intake and physical activity levels.[25] A complete description of the DIETRON model, including the underlying assumptions, parameters used in the model and the supporting literature, is provided in the supplementary appendix.

Dietary data

The Family Food Survey (FFS) annual reports and datasets for 2007, 2008 and 2009 published by the UK Department for Environment, Food and Rural Affairs provided information regarding geographical variations in the energy and nutritional intake derived from household food and drink, and food and drink eaten outside of the home in England, Wales, Scotland and Northern Ireland. The FFS is a subset of the Family Expenditure Survey and estimates of food consumption and nutritional quality of diets are based on food purchases. The unit of data collection is the household, and hence estimates used in this analysis are for average household nutrient intake, adjusted for the size of the household. The nutrients and food components included as inputs in the DIETRON model are: total energy intake (kcal per day), fruit (g per week), vegetables excluding potatoes (g per week), salt (g per day), total fat (% energy), saturated fat (% energy), poly-unsaturated fat (% energy), mono-saturated fat (% energy), dietary cholesterol (% energy) and non-starch polysaccharide fibre (g per day). The DIETRON model requires an estimate of average trans fat consumption, which is not available from the FFS data. For these analyses, it was assumed that there was no variation in trans fat consumption between countries, and average consumption was assumed to be 1.4 g per day, which is the estimated level of consumption in the 2008/2009 National Diet and Nutrition Survey (NDNS).[26]

Mortality data

Mortality data for CHD (ICD-10: I20–25), stroke (ICD-10: I60–69) and diet-related cancers (ICD-10: C00–14, C15, C16, C18–20, C23, C25, C33–34, C50, C54–55 and C64), stratified by gender and 5-year age band, were acquired from the Office for National Statistics for England and Wales, the General Register Office for Scotland and the Northern Ireland Statistics and Research Agency for 2007, 2008 and 2009. Age- and gender-stratified population data for the same years were supplied by the same sources.

Analysis

The 'mortality gap' between England, Wales, Scotland and Northern Ireland was defined as the difference between the actual annual number of deaths in Wales, Scotland and Northern Ireland and the annual number of deaths that would be expected in each country if they achieved English age-, gender- and cause-specific mortality rates. For example, in Scotland in 2007 there

were 850 deaths from CHD in women aged 80–84. However, if the English 2007 CHD mortality rate for women aged 80–84 were applied to the Scottish population, then there would be only 678 deaths in this age–sex group. Therefore, the 2007 'mortality gap' between Scotland and England for CHD in women aged 80–84 was 172 deaths.

Estimates of dietary quality and age-, gender- and cause-specific number of deaths for Wales, Scotland and Northern Ireland were used as baseline inputs for the DIETRON model. Estimates of dietary quality for England were used as the counterfactual input for DIETRON. The output of the model was the annual number of deaths delayed or averted in Wales, Scotland and Northern Ireland under the counterfactual scenario of achieving a dietary quality equivalent to that in England. The estimated number of deaths delayed or averted for each country was used as a numerator to calculate the percentage of the mortality gap closed for each country under the hypothetical counterfactual scenario of achieving a diet equivalent to that in England.

Uncertainty analysis

A Monte Carlo simulation was performed to estimate 95% credible intervals around the results. These credible intervals are based on the 2.5th and 97.5th percentiles of results generated from 5000 iterations of the DIETRON models, where the estimates of relative risks used to parameterise the model were allowed to vary stochastically according to the distributions reported in the literature. For example, the DIETRON model uses a relative risk of 0.81 for CHD mortality for every 10 g/d increase in fibre intake. This parameter is taken from a meta-analysis of 10 cohort studies adjusted for age, energy intake, smoking status, body mass index, physical activity, education, alcohol intake, multiple vitamin use, raised cholesterol, raised blood pressure and dietary saturated fat, polyunsaturated fat and cholesterol.[24] The meta-analysis reported confidence intervals which were used to estimate the log-normal distribution over which the actual relative risk is likely to lie. The DIETRON parameter for fibre-CHD was allowed to vary stochastically according to this log-normal distribution.

RESULTS

National dietary variation

Table 1 shows the difference in dietary quality between England, Wales, Scotland and Northern Ireland in the years 2007, 2008 and 2009, and average estimated intakes from these 3 years. These data show that the populations of Scotland and Northern Ireland consume a poorer diet than that of England; for example, between 2007 and 2009 Northern Ireland consumed 4% more saturated fat and 7% more salt per day, and approximately 20% less fruit and vegetables per week than England. In contrast, the differences between the Welsh and English diets were small and not consistently unidirectional; for example, in 2008 and 2009 the average Welsh diet contained more vegetables than the average English diet, but also more saturated fat and salt.

Deaths from CHD, stroke and cancer delayed or averted

Table 2 shows the mortality gap for CHD, stroke and diet-related cancers in Wales, Scotland and Northern Ireland in comparison with England. Between 2007 and 2009, this was 3723 deaths for Wales, 15 719 deaths for Scotland and 2329 deaths for Northern Ireland.

The DIETRON model suggests that if Scotland had adopted a diet equivalent to the English diet between 2007 and 2009, 6353 (95% credible intervals (CI) 5217 to 7957) deaths from CHD, stroke and diet-related cancer would have been delayed or averted. This accounts for 40% (95% CI 33% to 51%) of the mortality gap for CHD, stroke and diet-related cancers between Scotland and England.

The modelled result of achieving the English diet was similar for Wales and Northern Ireland, and was substantially higher than the result for Scotland. For Wales, the mortality gap for CHD, stroke and diet-related cancer was reduced by 81% (95% CI 62% to 108%) in the counterfactual scenario, and for Northern Ireland the mortality gap was reduced by 81% (95% CI 67% to 99%).

Figure 1 shows the percentage of the mortality gap closed under the counterfactual scenario for each country for CHD, stroke and diet-related cancers separately. In Scotland, the largest reduction in the mortality gap was for CHD (58% reduction; 95% CI 47% to 72%). For Wales, 88% (95% CI 69% to 118%) of the mortality gap for CHD was closed, and 88% (95% CI 70% to 111%) of the mortality gap for CHD in Northern Ireland was closed. In total, 6285 (95% CI 5034 to 7988) deaths from CHD, 2351 (95% CI 1080 to 3714) from stroke and 2612 (95% CI 2072 to 3302) from cancer were delayed or averted in the counterfactual scenario.

For Wales, Scotland and Northern Ireland, the dietary factors most associated with excess cardiovascular and cancer mortality are total energy intake and fruit and vegetable consumption.

DISCUSSION

This study suggests four key findings. First, that diet has a substantial impact on geographical variations in mortality from CHD, stroke and various cancers within the UK. Second, that the differences in mortality from CHD, stroke and diet-related cancers within the UK are less affected by diet in Scotland compared with Wales and Northern Ireland. Third, that if Wales, Scotland and Northern Ireland adopted the English diet, the majority of deaths delayed or averted would be from CHD. And finally, that total energy intake and fruit and vegetable consumption are the principle dietary determinants of geographical variations in mortality. However, the results are based on modelling hypothetical scenarios and rely on observational data, which implies that residual confounding cannot be ruled out. With that caveat,

Table 1 Energy and nutrient intakes derived from food and drink by country (includes both household and purchases eaten outside the home)

	Energy (kcal/day)	Total fat (g/day)	Saturated FAs (g/day)	MUFAs (g/day)	PUFAs (g/day)	Cholesterol (mg/day)	Fibre (g/day)	Salt (g/day)	Fruit (g/week)	Vegetables* (g/week)
2009										
England	2289	94.9	35.7	35.8	17.1	261	15.1	7.0	1213	1171
Wales	2364	98.1	37.7	36.7	17.0	270	15.2	7.4	1211	1242
Scotland	2370	97.6	37.2	36.7	17.2	258	15.1	7.6	1205	963
NI	2424	100.1	38.3	37.4	17.7	273	15.1	7.8	1061	897
2008										
England	2252	93.9	35.4	34.9	17.3	260	14.9	6.9	1277	1190
Wales	2439	101.2	38.8	37.7	17.9	287	16.3	7.5	1245	1226
Scotland	2333	96.0	37.0	35.2	17.4	264	14.9	7.4	1211	944
NI	2320	94.6	36.2	35.1	16.8	266	15.2	7.3	1095	909
2007										
England	2306	95.0	35.9	35.2	17.6	273	15.2	7.1	1320	1208
Wales	2361	97.4	37.3	35.9	17.8	273	15.6	7.3	1176	1151
Scotland	2422	100.6	39.1	37.1	17.8	282	14.9	7.6	1199	945
NI	2378	97.0	36.7	36.3	17.6	269	15.1	7.5	1076	900
2007–2009†										
England	2282	94.6	35.7	35.3	17.3	265	15.1	7.0	1270	1190
Wales	2388	98.9	37.9	36.8	17.6	278	15.7	7.4	1211	1206
Scotland	2375	98.1	37.8	36.3	17.5	268	15.0	7.5	1205	951
NI	2374	97.2	37.1	26.3	17.4	269	15.1	7.5	1077	902

*Vegetable consumption does not include potatoes.
†Mean values for 2007–2009.
NI, Northern Ireland; FAs, fatty acids; MUFAs, mono-unsaturated fatty acids; PUFAs, polyunsaturated fatty acids.

Table 2 Deaths from coronary heart disease (CHD), stroke and cancer delayed or averted and percentage of mortality gap closed as a result

	Deaths from CHD, stroke and diet-related cancers	Expected deaths from CHD, stroke and diet-related cancers*	Mortality gap	Deaths delayed or averted if English diet achieved (95% credible intervals)	% Mortality gap closed (95% credible intervals)
Wales					
2009	12 733	11 620	1113	591 (434 to 824)	53 (40 to 74)
2008	13 367	12 122	1245	1493 (1096 to 2088)	120 (88 to 168)
2007	13 569	12 204	1365	591 (540 to 782)	43 (40 to 57)
Total†	39 669	35 946	3723	3005 (2324 to 4013)	81 (62 to 108)
Scotland					
2009	22 765	17 934	4831	1674 (1364 to 2134)	35 (28 to 44)
2008	23 774	18 235	5539	1892 (1546 to 2358)	34 (28 to 43)
2007	24 214	18 865	5349	2746 (2287 to 3376)	51 (43 to 63)
Total†	70 753	55 034	15 719	6353 (5217 to 7957)	40 (33 to 51)
Northern Ireland					
2009	5836	4931	905	727 (593 to 910)	80 (66 to 101)
2008	6142	5429	713	499 (402 to 607)	70 (56 to 85)
2007	6170	5459	711	584 (482 to 707)	82 (68 to 99)
Total†	18 148	15 819	2329	1890 (1570 to 2310)	81 (67 to 99)

*Expected deaths estimated using English age-, gender- and cause-specific mortality rates.
†Estimates combine mortality data over 3 years, and use mean dietary data (see table 1).

these results suggest that relatively small (and achievable) improvements in the diet of Wales, Scotland and Northern Ireland could have a substantial impact on geographical variations in health within the UK. Identifying the specific dietary factors that produce the greatest reduction in health inequalities between countries may help to produce specific national level interventions. For example, the results suggest that fiscal initiatives aimed at increasing the cost of foods high in saturated fat (so called 'fat taxes') may be best placed to reduce geographical inequalities in health if they are paired with subsidies for fruit and vegetables. Previous modelling work has suggested that pairing fat taxes with subsidies for fruit and vegetables increases the effectiveness of fiscal initiatives.[27 28]

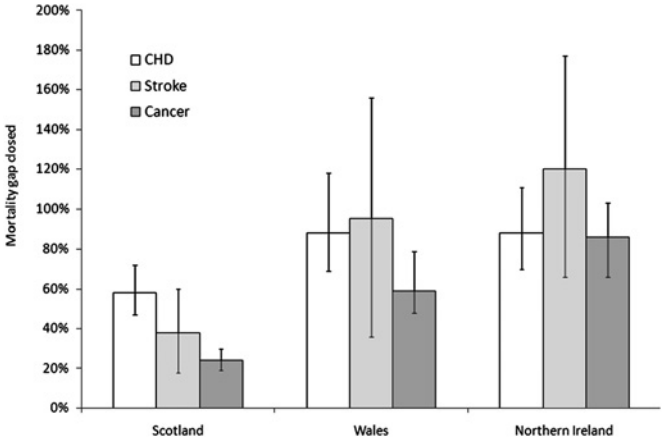


Figure 1 Reduction in mortality gap (mean and 95% credible intervals) for coronary heart disease (CHD), stroke and cancer in Scotland, Wales and Northern Ireland.

The main outcome of our analyses was the percentage of the mortality gap that would be closed if Wales, Scotland or Northern Ireland achieved a diet similar to that of England. This outcome is dependent upon two factors: the size of the existing mortality gap and the difference in dietary quality between the countries. Considering these two factors separately allows for a greater understanding of the results presented in this paper. The mortality gap between England and Wales is small (only 3723 deaths between 2007 and 2009, 9% of all CHD, stroke and diet-related cancer deaths in Wales in this period), and the difference in dietary quality between England and Wales is also small. In comparison, the random error associated with estimates of dietary quality in England and Wales is relatively large, which explains why the primary outcome '% mortality gap closed by diet' is inconsistent in the 3 years studied, and why the credible intervals around the final estimate of 81% are wide. In comparison, the mortality gap between England and Northern Ireland is wider (13% of all CHD, stroke and diet-related cancer deaths) and the difference in dietary quality is also wider. Therefore, the random error associated with the estimates of dietary quality is relatively smaller, and the estimates of '% mortality gap closed by diet' are consistent in 2007, 2008 and 2009, and credible intervals are narrower. The difference in dietary quality between England and Scotland is of comparable size to the difference between England and Northern Ireland, but the mortality gap is much wider (22% of all CHD, stroke and diet-related cancer deaths). As such, the '% mortality gap closed by diet' is much smaller (40%), but the results are consistent in 2007, 2008 and 2009 and credible intervals are narrow.

Comparison with other studies

A number of well established cohort studies have been published showing that established risk factors such as systolic blood pressure and smoking account for a significant proportion of geographical inequalities in cardiovascular disease within the UK.[4 5 8 12 29] However, these cohort studies do not directly address the impact of diet on geographical variation. For example, the British Regional Heart Study (a cohort of over 7000 men between 40 and 59 years of age recruited in 1978–80) found that variation in systolic blood pressure explained 27% of geographical variation in CHD incidence in Great Britain, body mass index explained 6%, and serum cholesterol levels had no affect.[5 12 29] Although not all of the variations in blood pressure, body mass index and serum cholesterol are due to dietary factors, the results reported in this paper are broadly in agreement, that is, they suggest that variation in saturated fat consumption has very little impact on variation in mortality rates, whereas variations in salt and energy consumption have a substantial impact.

It is now well accepted that intrinsic, albeit undetermined, risk factors are responsible for significant inequalities between Scotland and other countries such as England, referred to as the 'Scottish effect'.[5 30–33] This study adds to the conclusion that geographical variation in known cardiovascular risk factors only partially explains this effect. Our analysis suggests that adopting an English-equivalent diet in Scotland would reduce the mortality gap between these countries by less compared to either Wales or Northern Ireland.

The results of these analyses are dependent upon the estimates of dietary quality that were taken from the FFS. An alternative data source would have been the NDNS, which estimates dietary quality for individuals using a weighed food diary. There were a number of reasons why we chose not to use the NDNS in this analysis. First, the most recent estimates for adult consumption data from the NDNS were published in 2000/2001. Although additional NDNS data were published in 2009,[26] this represented only a single year of data collection from a 3-year rolling programme and estimates from the full sample are not available until 2012. Second, the 'adult' NDNS survey does not include adults above 64 years of age, the age group in whom the majority of cardiovascular and cancer incidents occur. And third, the NDNS is limited to Great Britain (ie, England, Scotland and Wales). Nevertheless, given these limitations the results of the 2000/2001 NDNS are generally supportive of the dietary trends shown in the FFS; for example, NDNS shows that diets in Scotland are of poorer quality than in England and Wales in terms of salt, fat content, dietary cholesterol, and fruit and vegetable consumption.[13]

Strengths and limitations of the study

Cardiovascular disease and cancer are the most common causes of mortality in the UK.[34] This study attempts to estimate the impact of dietary intake on these wide-reaching conditions. We illustrate that the majority of deaths delayed or averted if the English diet were adopted throughout the UK would occur in CHD, which provides a platform from which to potentially direct public health policy. The results that are reported here are based on a diet that has already been obtained in England, and is therefore realistically achievable by other nations. Wherever possible, the DIETRON model is based on estimates of relative risk that have been adjusted for non-dietary behavioural risk factors (eg, smoking and alcohol consumption),[17] and hence the outcomes reported here are likely to be primarily due to diet rather than confounding by other socially patterned behaviours. However, the DIETRON model is based on a synthesis of results mainly from observational studies, so residual confounding cannot be ruled out.

The uncertainty analysis that was used to generate the credible intervals for this paper was based on 5000 iterations of the DIETRON model, allowing the relative risks derived from the literature to vary stochastically according to their reported distribution. This uncertainty analysis incorporates the degree of uncertainty generated by the DIETRON model, but does not account for the uncertainty associated with the estimates of dietary quality generated by the FFS. Therefore, the credible intervals developed for this paper are underestimates of the true uncertainty of the results reported here.

Further work and unanswered questions

The results reported here show the effect of diet on geographical variations in health within the constituent countries of the UK. To be a true guide for policymakers, these results should be compared with equivalent estimates of the impact of other behavioural risk factors (smoking, alcohol consumption and physical inactivity). This would provide a comparable set of estimates of the likely impact of successful public health interventions on reducing inequalities. These modelled results should be supported by evidence of the effectiveness and cost-effectiveness of interventions to increase healthy life styles in order to provide policy makers with the evidence to make informed decisions.

Funding This work was supported by the British Heart Foundation (grant numbers PPC/Jul05/5b and PPC/JAN05/6biii).

Competing interests None.

Contributors PS contributed to study conception, analysis design, acquisition of data, interpretation of results, drafting of the manuscript, and final approval of the manuscript. RM contributed to acquisition of data, analysis, interpretation of results, drafting of the manuscript, and final approval of the manuscript. PW contributed to study conception, interpretation of results, drafting of the manuscript, and final approval of the manuscript. MR contributed to study conception, interpretation of results, drafting of the manuscript, and final approval of the manuscript.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement No additional data are available.

REFERENCES

1. Boyle PJ, Gatrell AC, Duke-Williams O. Do area-level population change, deprivation and variations in deprivation affect individual-level self-reported limiting long-term illness? *Soc Sci Med* 2001;53:795–9.
2. Boyle PJ, Curtis S, Graham E, *et al*. *The Geography of Health Inequalities in the Developed World: Views from Britain and North America*. London: Ashgate, 2004.
3. Congdon P, Shouls S, Curtis S. A multi-level perspective on small-area health and mortality: a case study of England and Wales. *Int J Popul Geogr* 1997;3:243–63.
4. Lawlor DA, Bedford C, Taylor M, *et al*. Geographical variation in cardiovascular disease, risk factors, and their control in older women: British Women's Heart and Health Study. *J Epidemiol Community Health* 2003;57:134–40.
5. Morris RW, Whincup PH, Lampe FC, *et al*. Geographic variation in incidence of coronary heart disease in Britain: the contribution of established risk factors. *Heart* 2001;86:277–83.
6. Marmot M, Elliott P. *Coronary Heart Disease Epidemiology: From Aetiology to Public Health*. Oxford: OUP, 2005.
7. Elford J, Phillips AN, Thomson AG, *et al*. Migration and geographic variations in ischaemic heart disease in Great Britain. *Lancet* 1989;1:343–6.
8. Kim LG, Carson C, Lawlor DA, *et al*. Geographical variation in cardiovascular incidence: results from the British Women's Heart and Health Study. *BMC Public Health* 2010;10:696.
9. Bhatnagar P, Scarborough P, Smeeton NC, *et al*. The incidence of all stroke and stroke subtype in the United Kingdom, 1985 to 2008: a systematic review. *BMC Public Health* 2010;10:539.
10. Maheswaran R, Strachan DP, Dodgeon B, *et al*. A population-based case-control study for examining early life influences on geographical variation in adult mortality in England and Wales using stomach cancer and stroke as examples. *Int J Epidemiol* 2002;31:375–82.
11. Mullee MA, De SB, Romanengo M, *et al*. Geographical variation in breast cancer survival rates for women diagnosed in England between 1992 and 1994. *Br J Cancer* 2004;90:2153–6.
12. World Cancer Research Fund/American Institute for Cancer Research. *Food, Nutrition, Physical Activity, and Prevention of Cancer: a Global Perspective*. Washington DC: AICR, 2007.
13. Office for National Statistics. *The National Diet and Nutrition Survey: Adults Aged 19 to 64 Years*. London: Office for National Statistics, 2002.
14. Office of National Statistics. *Family Food in 2008*. London: The Stationery Office, 2008.
15. Shelton NJ. Regional risk factors for health inequalities in Scotland and England and the "Scottish effect". *Soc Sci Med* 2009;69:761–7.
16. Speight S, Wardle H, Bromley C. Fruit and vegetable consumption and eating habits. In: Bromley C, Sproston K, Shelton N, eds. *The Scottish Health Survey 2003*. Edinburgh: Scottish Executive, 2005.
17. 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*. Published Online First: 15 December 2010. doi:10.1136/jech.2010.114520.
18. Clarke R, Frost C, Collins R, *et al*. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *BMJ* 1997;314:112–17.
19. 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.
20. 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.
21. He FJ, MacGregor GA. How far should salt intake be reduced? *Hypertension* 2003;42:1093–9.
22. Lewington S, Clarke R, Qizilbash N, *et al*. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet* 2002;360:1903–13.
23. Lewington S, Whitlock G, Clarke R, *et al*. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet* 2007;370:1829–39.
24. Pereira MA, O'Reilly E, Augustsson K, *et al*. Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. *Arch Intern Med* 2004;164:370–6.
25. Christiansen E, Garby L. Prediction of body weight changes caused by changes in energy balance. *Eur J Clin Invest* 2002;32:826–30.
26. Office for National Statistics. *National Diet and Nutritional Survey. Headline results from year 1 of rolling programme (2008/09)*. London: Food Standard Agency, 2010.
27. Mytton O, Gray A, Rayner M, *et al*. Could targeted food taxes improve health? *J Epidemiol Community Health* 2007;61:689–94.
28. Nnoaham KE, Sacks G, Rayner M, *et al*. Modelling income group differences in the health and economic impacts of targeted food taxes and subsidies. *Int J Epidemiol* 2009;38:1324–33.
29. Morris RW, Whincup PH, Emberson JR, *et al*. North-south gradients in Britain for stroke and CHD: are they explained by the same factors? *Stroke* 2003;34:2604–9.
30. The Scottish Executive. *The Chief Medical Officer's Annual Report Health in Scotland 2004*. Edinburgh: Scottish Executive, 2005.
31. Hanlon P, Lawder RS, Buchanan D, *et al*. Why is mortality higher in Scotland than in England and Wales? Decreasing influence of socioeconomic deprivation between 1981 and 2001 supports the existence of a 'Scottish Effect'. *J Public Health* 2005;27:199–204.
32. Mitchell R, Fowkes G, Blane D, *et al*. High rates of ischaemic heart disease in Scotland are not explained by conventional risk factors. *J Epidemiol Community Health* 2005;59:565–7.
33. Walsh D, Whyte B, Gordon DS. Changing places? A comparative analysis of area-based health trends in Scotland through the 1980s and 1990s. *Public Health* 2007;121:889–97.
34. Scarborough P, Bhatnagar P, Wickramasinghe K, *et al*. *Coronary Heart Disease Statistics 2010 Edition*. London: British Heart Foundation, 2010.

Appendix: Description of the DIETRON model Much of the description of the DIETRON model that appears in this appendix is taken from an earlier publication outlining the development of the model (Scarborough et al., 2010). The DIETRON model is based on a conceptual framework that leads from consumption of foods and nutrients through to biological risk factors for ill health, through to adverse health outcomes. Food components (e.g. fruit, vegetables, saturated fat etc.) were included in the model as inputs if either (1) a quantitative estimate of the association between the food component and CHD, stroke or cancer has been derived from a meta-analysis of cohort or case-control studies, or (2) a quantitative estimate of the association between the food component and a biological risk factor has been derived from a meta-analysis of randomised trials, cohort or case-control studies, and a further quantitative assessment of the association between the biological risk factor and CHD, stroke or cancer has been derived from a meta-analysis of cohort or case-control studies. An additional condition was assigned for links with cancer: that the association be defined as 'probable' or 'convincing' by the World Cancer Research Fund (AICR / WCRF, 2007). The parameterisation of the relationship between physical activity levels, total energy intake and body mass index was not provided by a meta-analysis, but from an equation reported in the literature derived from the principles of conservation of energy (Christiansen and Garby, 2002).

Meta-analyses were identified through computerised database and hand searching, and priority was given to meta-analyses of trials, followed by cohort studies and then case-control studies. Where age- and sex-specific quantitative estimates where available they were included in the model. The DIETRON model relies on four key assumptions, which are detailed in table A1. Details on the meta-analyses parameterising the links in the DIETRON model are provided in table A2. The parameters used in the DIETRON model are provided in table A3. In some instances, meta-analyses were available that would allow us to either model the association between food component and health outcome directly or via a biological risk factor. In all such instances, we chose to model via the biological risk factor in order to follow the general principle that, where data are available, it is advantageous to generate structural models of disease risk that incorporate the underlying system under investigation (Murray et al., 2003). The connections included in the DIETRON model are displayed in figures A1-A6, where a dashed line indicates an inverse relationship, a solid line indicates a positive relationship, and a dotted line indicates a U-shaped relationship. All of the relationships in the DIETRON model are assumed to follow a log-linear dose –response relationship, with the exception of the relationships between obesity levels and health outcomes. Here, the model calculates a shift in the distribution of BMI within a population under a counterfactual scenario, and models the total amount of risk in the counterfactual scenario on the basis of risk levels at different levels of BMI. In this way, the DIETRON model can accommodate non-linear risk relationships between obesity and health outcomes.
Figure A1: Links between fruit and vegetables and health outcomes in the DIETRON model Figure A2: Links between fibre and health outcomes in the DIETRON model Figure A3: Links between fatty acids and health outcomes in the DIETRON model Figure A4: Links between salt and health outcomes in the DIETRON model Figure A5: Links between physical activity and total energy intake and health outcomes in the DIETRON model Figure A6: Conceptual framework underlying the DIETRON model
Table A1: Assumptions incorporated in the DIETRON model

1	Wherever possible, it is advantageous to generate structural models of disease risk that incorporate the underlying system under investigation.
2	Changes in risk associated with changes in more than one food component are combined multiplicatively (e.g. if changing food component X reduces risk of a health outcome by 10% and changing food component Y reduces risk by 12%, then changing X and Y together reduces risk by 100%*(1-(1-0.10)*(1-0.12)) = 20.8%
3	With the exception of obesity, changes in risk in the DIETRON model follow a log-linear dose-response relationship (e.g. a change in consumption of fruit and vegetables from 2 to 3 portions per day has the same effect as a change in consumption from 7 to 8 portions per day).

1	Wherever possible, it is advantageous to generate structural models of disease risk that incorporate the underlying system under investigation.
4	Changes between baseline and counterfactual food consumption distributions will be made by all individuals within the population equally (i.e. a population shift in the overall distribution of consumption).

Table A2 Meta-analyses used to parameterise the DIETRON model

Food component / risk factor	Outcome	Meta-analysis details	Adjustments	Log-linear dose-response relationship observed?	Source
Fruit	CHD (I20-25)	Six cohort studies (3,446 events)	Age, smoking, obesity	Yes	(Dauchet et al., 2006)
	Stroke (I60-69)	Five cohort studies (1,853 events)	Age, hypertension, smoking, obesity	Yes	(Dauchet et al., 2005)
	Mouth, pharynx, larynx cancer (C00-14)	Seven case-control studies	Smoking	No	(AICR / WCRF, 2007)
	Oesophagus cancer (C15)	Eight case-control studies	–	–	(AICR / WCRF, 2007)
	Lung cancer (C34)	Fourteen cohort studies	Smoking	Yes	(AICR / WCRF, 2007)
	Stomach cancer (C16)	Eight cohort studies	–	No	(AICR / WCRF, 2007)
Vegetables	CHD	Seven cohort studies (3,833 events)	Age, smoking, obesity	Yes	(Dauchet et al., 2006)
	Stroke	Four cohort studies (933 events)	Age, hypertension, smoking, obesity, blood cholesterol, physical activity, energy intake, alcohol intake	No	(Dauchet et al., 2005)
	Mouth, pharynx, larynx cancer	Four case-control studies	Sex, smoking, alcohol intake	Yes	(AICR / WCRF, 2007)
	Oesophagus cancer	Five case-control studies	–	–	(AICR / WCRF, 2007)
	Stomach cancer	Seven cohort studies	–	No	(AICR / WCRF, 2007)
Fibre	CHD	Ten cohort studies (2,011 CHD deaths)	Age, energy intake, smoking, obesity, physical activity, education, alcohol intake, multiple vitamin use, raised cholesterol, hypertension, dietary saturated fat, PUFA and cholesterol	Yes	(Pereira et al., 2004)
Total fat, saturated fat, MUFA, PUFA, dietary cholesterol	Total serum cholesterol	227 dietary intervention studies with diets persisting at least two weeks	Age, weight, other dietary fat measures	Yes	(Clarke et al., 1997)
Trans fats	Total serum cholesterol	40 dietary intervention studies with diets persisting at least two weeks	Age, weight, other dietary fat measures	Yes	(Clarke et al., 1997)

Food component / risk factor	Outcome	Meta-analysis details	Adjustments	Log-linear dose-response relationship observed?	Source
Salt	Stomach cancer	Two cohort studies.	–	Yes	(AICR / WCRF, 2007)
	Blood pressure	28 randomised controlled trials in hypertensive and normotensive individuals	All potentially confounding factors	Yes	(He and MacGregor, 2002; He and MacGregor, 2003)
Physical activity, total energy intake	Obesity (body mass index)	Equation derived from principles of conservation of energy	n/a	Yes	(Christiansen and Garby, 2002)
Total serum cholesterol	CHD	61 cohort studies (33,744 events)	Age, sex	Yes	(Prospective Studies Collaboration, 2007)
	Stroke	61 cohort studies (11,663 events)	Age, sex	No	(Prospective Studies Collaboration, 2007)
Blood pressure	CHD	61 cohort studies (34,283 events)	Blood cholesterol, diabetes, weight, alcohol intake, smoking	Yes	(Prospective Studies Collaboration, 2002)
	Stroke	61 cohort studies (11,960 events)	Blood cholesterol, diabetes, weight, alcohol intake, smoking	Yes	(Prospective Studies Collaboration, 2002)
Obesity	CHD	57 cohort studies	Age, sex, smoking	Risk at each stage of distribution modelled (see Table A3)	(Prospective Studies Collaboration, 2009)
	Stroke	57 cohort studies	Age, sex, smoking	Risk at each stage of distribution modelled (see Table A3)	(Prospective Studies Collaboration, 2009)
	Oesophagus cancer	Four case-control studies	–	–	(AICR / WCRF, 2007)
	Pancreas cancer (C25)	17 cohort studies	Smoking	Yes	(AICR / WCRF, 2007)
	Colorectum cancer (C18)	28 cohort studies	–	Yes	(AICR / WCRF, 2007)
	Breast cancer (C50)	16 cohort studies	–	–	(AICR / WCRF, 2007)
	Endometrial cancer (C54.1)	15 cohort studies	–	Yes	(AICR / WCRF, 2007)
	Kidney cancer (C64)	Seven cohort studies.	Smoking	Yes	(AICR / WCRF, 2007)
	Gallbladder cancer (C23)	Four cohort studies.	–	–	(AICR / WCRF, 2007)

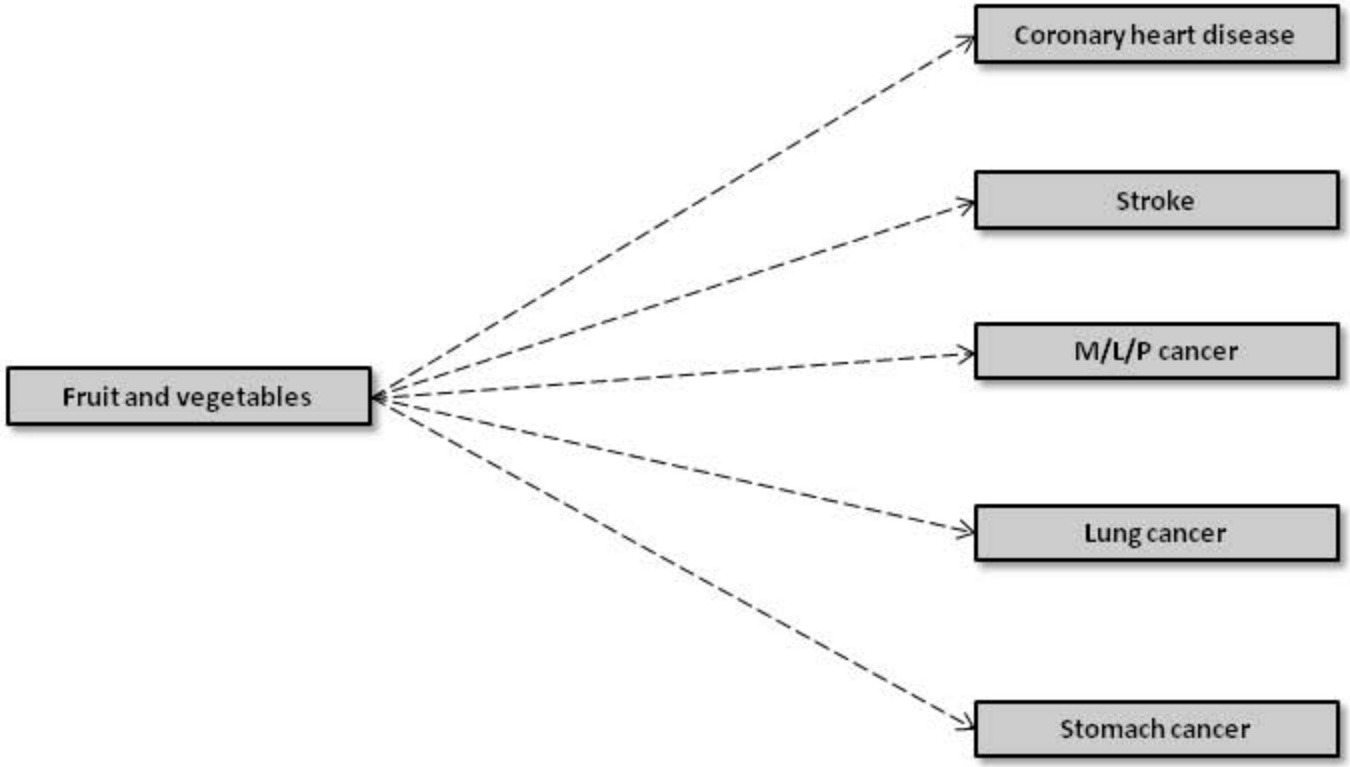
- indicates that either the adjusted factors or the dose-response relationship were not reported. ICD-10 codes are provided in brackets for the first entry of each disease. MUFA – mono-unsaturated fatty acids; PUFA – poly-unsaturated fatty acid. Number of events is provided where reported in the source document. Table A3: Parameters used in the DIETRON model

Food component / biological risk factor	Outcome	Unit of change	Relative risk (95% confidence intervals)
Fruit	CHD	106g/day increase	0.93 (0.89, 0.96)
	Stroke	106g/day increase	0.89 (0.85, 0.93)
	M/L/P cancer	100g/day increase	0.72 (0.59, 0.87)
	Oesophagus cancer	100g/day increase	0.56 (0.42, 0.74)
	Lung cancer	80g/day increase	0.94 (0.90, 0.97)
	Stomach cancer	100g/day increase	0.95 (0.89, 1.02)
Vegetables	CHD	106g/day increase	0.89 (0.83, 0.95)
	Stroke	106g/day increase	0.97 (0.92, 1.02)
	M/L/P cancer	50g/day increase	0.72 (0.63, 0.82)
	Oesophagus cancer	50g/day increase	0.87 (0.72, 1.05)
	Stomach cancer	100g/day increase	0.98 (0.91, 1.06)
Fibre	CHD	10g/day increase	0.81 (0.72, 0.92)
Salt	Stomach cancer	1g/day increase	1.08 (1.00, 1.17)
Serum cholesterol	CHD	1mmol/l decrease	Under 49: 0.44 (0.42, 0.48) 50-59: 0.58 (0.56, 0.61) 60-69: 0.72 (0.69, 0.74) 70-79: 0.82 (0.80, 0.85) Over 79: 0.85 (0.82, 0.89)
	Stroke	1mmol/l decrease	Under 59: 0.90 (0.84, 0.97) 60-69: 1.02 (0.97, 1.08) 70-79: 1.04 (0.99, 1.09) Over 79: 1.06 (1.00, 1.13)
Blood pressure	CHD	20mmHg SBP decrease	Under 49: 0.49 (0.45, 0.53) 50-59: 0.50 (0.49, 0.52) 60-69: 0.54 (0.53, 0.55) 70-79: 0.60 (0.58, 0.61) Over 79: 0.67 (0.64, 0.70)
	Stroke	20mmHg SBP decrease	Under 49: 0.36 (0.32, 0.40) 50-59: 0.38 (0.35, 0.40) 60-69: 0.43 (0.41, 0.45) 70-79: 0.50 (0.48, 0.52) Over 79: 0.67 (0.63, 0.71)
Body mass index	CHD	5kg/m^2 increase	Men, BMI 15-25: 1.27 (1.16, 1.39) Women, BMI 15-25: 1.01 (0.86, 1.18) Men, BMI 25-50: 1.42 (1.35, 1.48) Women, BMI 25-50: 1.35 (1.28, 1.43)
	Stroke	5kg/m^2 increase	BMI 15-25: 0.92 (0.82, 1.03) BMI 25-50: 1.39 (1.31, 1.48)
	Oesophagus cancer	1kg/m^2 increase	1.11 (1.07, 1.15)
	Pancreas cancer	5kg/m^2 increase	1.14 (1.07, 1.22)
	Colorectum cancer	1kg/m^2 increase	1.03 (1.02, 1.04)
	Breast cancer	2kg/m^2 increase	Under 60: 0.94 (0.92, 0.95) Over 60: 1.03 (1.01, 1.04)
	Endometrial cancer	5kg/m^2 increase	1.52 (1.35, 1.72)
	Kidney cancer	5kg/m^2 increase	1.31 (1.24, 1.39)
	Gallbladder cancer	5kg/m^2 increase	1.23 (1.15, 1.32)
Food component	**Outcome**	**Unit of change**	**Regression parameter (95% confidence intervals**
Total fat	Total serum cholesterol (mmol/l)	1% of total calories increase	0.020 (0.010, 0.030)
Saturated fat	Total serum cholesterol (mmol/l)	1% of total calories increase	0.052 (0.046, 0.058)

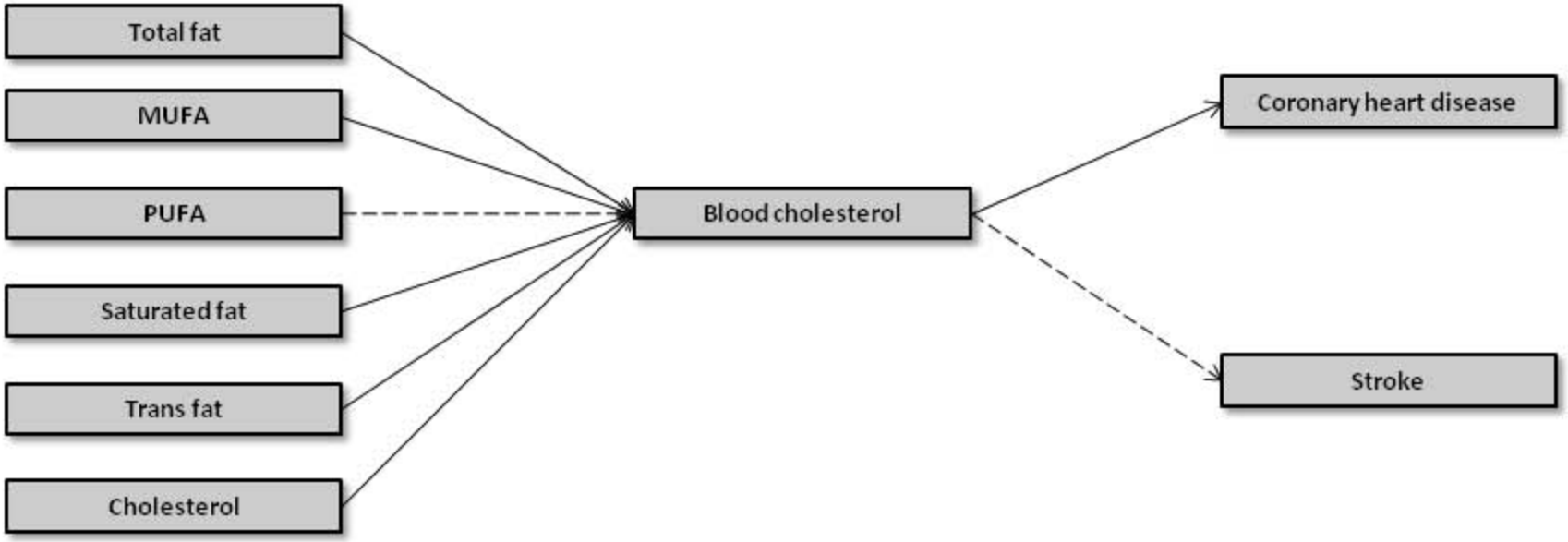
Food component / biological risk factor	Outcome	Unit of change	Relative risk (95% confidence intervals)
MUFAs	Total serum cholesterol (mmol/l)	1% of total calories increase	0.005 (-0.001, 0.011)
PUFAs	Total serum cholesterol (mmol/l)	1% of total calories increase	−0.026(−0.034,−0.018)
Dietary cholesterol	Total serum cholesterol (mmol/l)	1% of total calories increase	0.001 (0.001, 0.001)
Trans fats	Total serum cholesterol (mmol/l)	1% of total calories increase	0.038 (0.018, 0.058)
Salt	Systolic blood pressure (mmHg)	3g/day reduction	−2.50(−2.85,−2.15)
Total energy intake / physical activity level	Change in body weight (kg)	1MJ/PAL increase	Men: 17.7 Women: 20.7

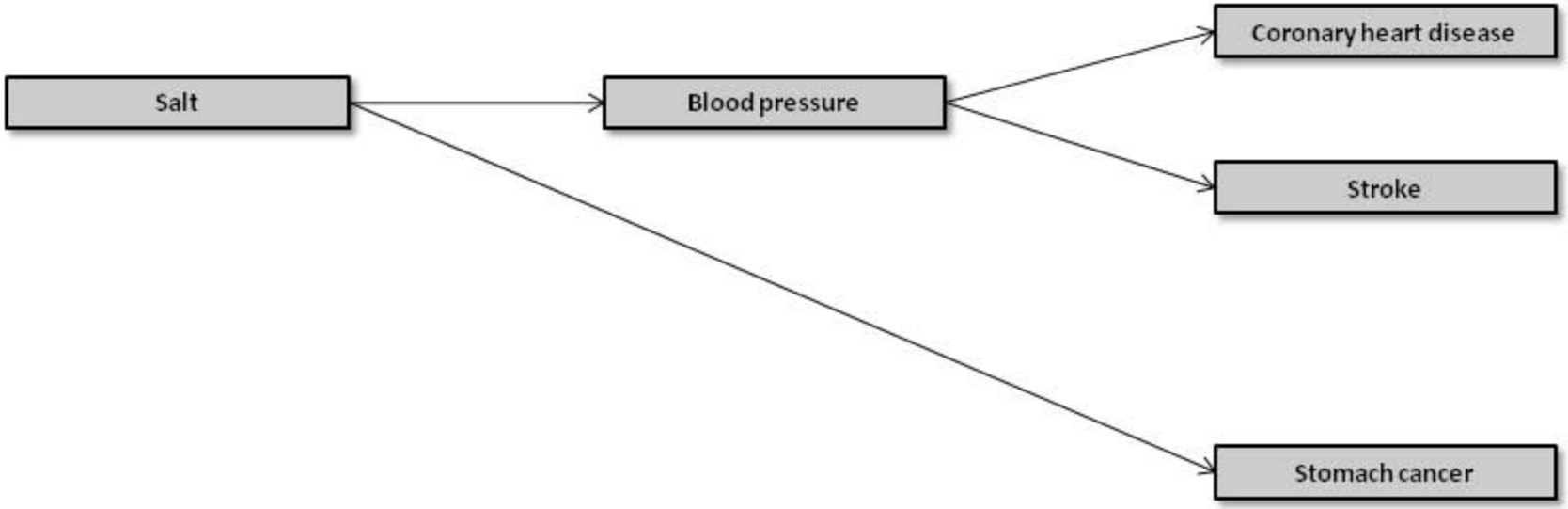
References: *American Institute for Cancer Research (AICR) and World Cancer Research Fund (WCRF). Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington DC: AICR, 2007.*

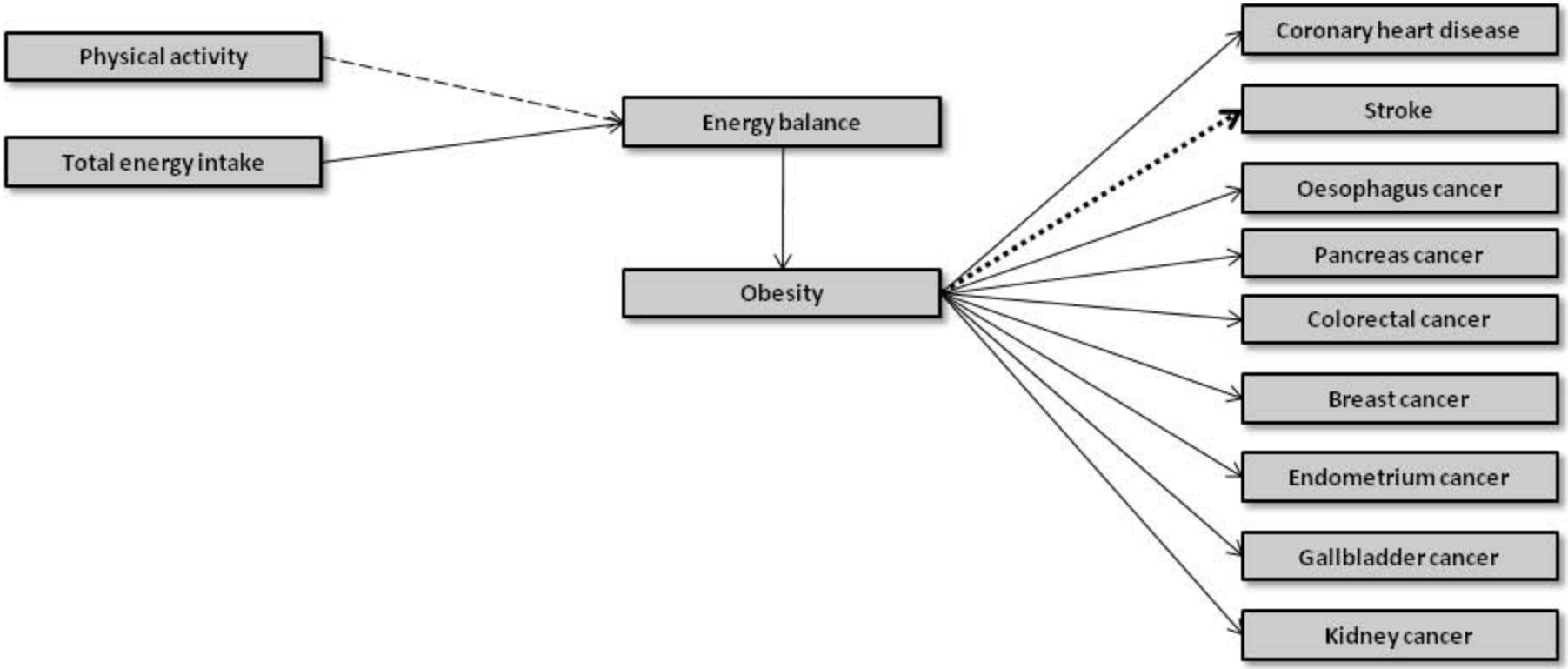
Christiansen E, Garby L. Prediction of body weight changes caused by changes in energy balance. Eur J Clin Investigation, 2002;32:826-830.
Clarke R, Frost C, Collins R, et al. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. BMJ. 1997;314:112.
Dauchet L, Amouyel P, Dallongeville J. Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies. Neurology. 2005;65:1193-1197.
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;136:2588-2593.
He F, MacGregor G. Effect of modest salt reduction on blood pressure: a meta-analysis of randomized trials. Implications for public health. J Hum Hypertens. 2002;16:761-770.
He F, MacGregor G. How far should salt intake be reduced? Hypertension. 2003;42:1093-1099.
Murray C, Ezzati M, Lopez A, et al. Comparative quantification of health risks: conceptual framework and methodological issues. Population Health Metrics. 2003:1;1-20.
Pereira M, O'Reilly E, Augustsson K, et al. Dietary fiber and risk of coronary heart disease. A pooled analysis of cohort studies. Arch Intern Med. 2004;164:370-376.
Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet. 2002;360:1903-1913.
Prospective Studies Collaboration. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. Lancet. 2007;370:1829-1839.
Prospective Studies Collaboration. Body-mass index and cause-specific mortality in 900,000 adults: collaborative analyses of 57 prospective studies. Lancet. 2009;373:1083-1096.
Scarborough P, Nnoaham K, Clarke D, et al. Modelling the impact of a healthy diet on cardiovascular disease and cancer mortality. J Epidemiol Comm Health, 2010.doi:10.1136/jech.2010.114520

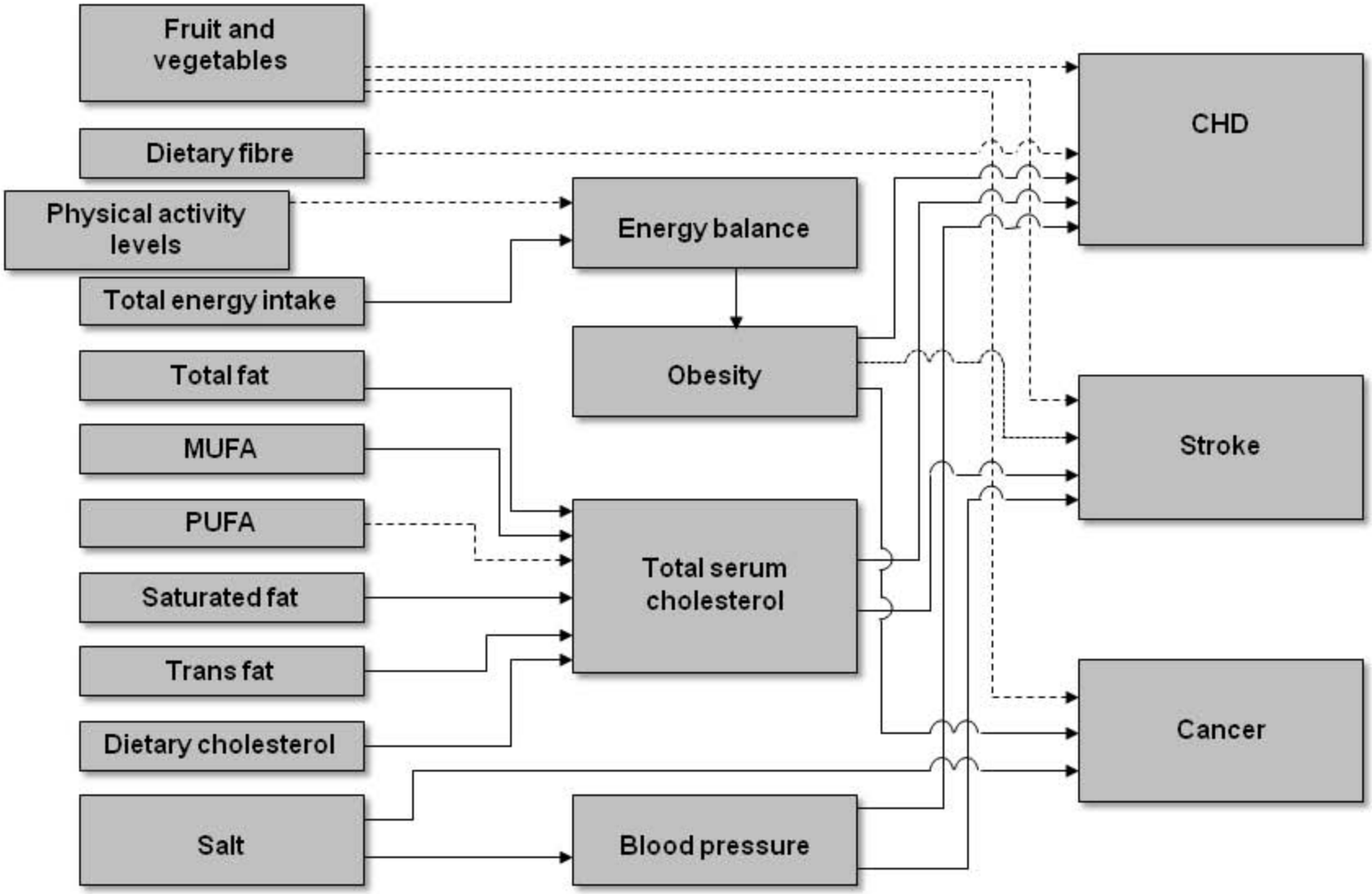

Fruit and vegetables
Coronary heart disease
Stroke
M/L/P cancer
Lung cancer
Stomach cancer


Fibre
Coronary heart disease


Total fat
MUFA
PUFA
Saturated fat
Trans fat
Cholesterol
Blood cholesterol
Coronary heart disease
Stroke


Salt
Blood pressure
Coronary heart disease
Stroke
Stomach cancer


Physical activity
Total energy intake
Energy balance
Obesity
Coronary heart disease
Stroke
Oesophagus cancer
Pancreas cancer
Colorectal cancer
Breast cancer
Endometrium cancer
Gallbladder cancer
Kidney cancer


Fruit and vegetables
Dietary fibre
Physical activity levels
Total energy intake
Total fat
MUFA
PUFA
Saturated fat
Trans fat
Dietary cholesterol
Salt
Energy balance
Obesity
Total serum cholesterol
Blood pressure
CHD
Stroke
Cancer