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

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