Dietary pattern and depressive symptoms in middle age.

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Dietary pattern and depressive symptoms in middle age: the Whitehall II study

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Running Title: **Diet and depressive symptoms**

ABSTRACT

Background**:** Studies on diet and depression have focused primarily on individual nutrients. This paper examines the association between dietary patterns and depression using an overall diet approach.

Method : Analyses were carried on 3486 participants (26.2% women, mean age 55.6 years) from the Whitehall II prospective cohort, in which two dietary patterns were identified: "whole food" (vegetables/fruits/fish) and "processed food" pattern (eg sweetened desserts/fried food/processed meat/refined grains/ high fat dairy products). Self-reported depression was assessed five years later using the CES-D scale.

Results: After adjusting for potential confounders, participants in the highest tertile of the "whole food" pattern had lower odds of CES-D depression [Odds Ratio=0.74 (95% CI:0.56-0.99)] than those in the lowest tertile. In contrast, high consumption of "processed food" was associated with an increased odds of CES-D depression [Odds Ratio=1.58 (95% CI:1.11-2.23)].

Conclusion: In middle-aged participants, a "processed food" dietary pattern is a risk factor for CES-D depression five years later, while a "whole food" pattern is protective.

Declaration of interest: none

Research on the association between diet and depression has focused primarily on nutrients such as fatty acids (1-4) and nutrients involved in the homocysteine pathway, e.g. vitamin B6, B9 and B12 (2, 5-7), but the results have been inconclusive. Recent years have seen a move away from analyzing associations between isolated nutrients and health to consideration of the effects of dietary patterns (8). For example, a meta-analysis published in 2008 showed that a greater adherence to a Mediterranean dietary pattern (high intake of fruits, vegetable, and fish and low intake of meat and dairy product) was associated with a lower incidence of Parkinson's and Alzheimer's disease (9). However, the health outcomes of that meta-analysis did not include depression and, to the best of our knowledge, no previous prospective study has investigated the association between dietary patterns and the occurrence of depressive symptoms. Thus, the objective of this study was to examine the association between dietary patterns derived from a food frequency questionnaire using factor analysis and depression using the CES-D scale in a large British middle aged population, the Whitehall II study. We were able to control for a large range of socio-demographic variables, health behaviors and health parameters including chronic diseases and cognitive functioning.

METHODS

The target population for the Whitehall II study was all London-based office staff, aged 35–55 years, working in 20 civil service departments (10). Baseline screening (Phase 1) took place during 1985-1988 (N=10,308), and involved a clinical examination and a self-administered questionnaire containing sections on demographic characteristics, health, lifestyle factors, work characteristics, social support and life events. The clinical

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examination included measures of blood pressure, anthropometry, biochemical measurements, neuroendocrine function, and subclinical markers of cardiovascular disease. Subsequent phases of data collection have alternated between postal questionnaire alone [Phases 2 (1989-1990), 4 (1995-1996), 6 (2001) and 8 (2006)] and postal questionnaire accompanied by a clinical examination [Phases 3 (1991-1993), 5 (1997-1999) and 7 (2002-2004)]. Analyses reported in this study were restricted to the 3486 white European participants with data on dietary patterns and all covariates at phase 5 and depression at phase 7. Black (n=175) and Asian (n=331) participants were excluded due to differences in eating patterns.

After complete description of the study to the subjects, written informed consent was obtained; the University College London ethics committee approved the study.

Dietary assessment at phase 5 and determination of dietary pattern:

A machine-readable Food Frequency Questionnaire (11) based on the one used in the US Nurses Health Study (12) was sent to the participants. The food list (127 items) in the FFQ was anglicized, and foods commonly eaten in the UK were added (13). A common unit or portion size for each food was specified, and participants were asked how often, on average, they had consumed that amount of the item during the previous year. Response to all items was on a 9-point scale, ranging from 'never or less than once per month' to 'six or more times per day'. The selected frequency category for each food item was converted to a daily intake.

According to nutrient profile and culinary use of food items, the 127 items of the FFQ were grouped in 37 predefined food groups, (by adding food items within each

group) (Appendix 1) (14). Dietary patterns were identified using principal component analysis of these 37 groups. The factors were rotated by an orthogonal transformation (Varimax rotation function in SAS; SAS Institute, Cary, NC) to achieve a simple structure, allowing greater interpretability. Two dietary patterns were identified using multiple criteria: the diagram of Eigen values, the Scree plot, the interpretability of the factors and the percentage of variance explained by the factors (Appendix 2). The factor score for each pattern was calculated by summing intakes of all food groups weighted by their factor loadings. Factors loadings represent correlation coefficients between the food groups and the dietary pattern. The first pattern was heavily loaded by high intake of vegetables, fruits and fish, labelled the "whole food" pattern. The second pattern, labelled "processed food", was heavily loaded by high consumption of sweetened desserts, chocolates, fried food, processed meat, pies, refined grains, high fat dairy products and condiments. Each participant received a factor score for each identified pattern. Factor analysis does not group individuals into clusters, instead all individuals contribute to both factors and it is the homogeneity between food items that defines the factors. The validity and the reliability of this version of the FFQ in terms of nutrient and food consumption have been documented in detail in our cohort (11) and in other UK cohort (13). To assess the validity of the dietary patterns resulting from this "a posteriori" food grouping, we reran the principal component analyses using the 127 individual food items and the results obtained were similar.

Center for Epidemiologic Studies Depression Scale (CES-D) measured at phase 7

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The CES-D scale is a short self-report scale designed to measure depressive symptoms in the general population. The 20 items of the scale measure symptoms associated with depression and have been validated against longer scales (15). Participants were asked to score the frequency of occurrence of specific symptoms during the previous week on a four point scale (0 = "less than one day", 1 = "1-2 days", 2 = "3-4 days" and 3 = "5-7 days"). These were summed to yield a total score between 0 and 60. Participants scoring more than 15 were defined as cases of CES-D depression (15).

Covariates at phase 5

Socio-demographic variables consisted of age, gender, marital status, employment grade and education. The British civil service employment grade, defined on the basis of salary, social status and level of responsibility, included 3 levels, with grade 1 representing the highest level and grade 3 the lowest. Highest educational attainment was grouped into five levels (no academic qualification, lower secondary education, higher secondary education, university degree, higher university degree. Health behaviours measured were smoking (non smoker, former, current smoker) and physical activity converted to metabolic equivalent (MET)-scores (16) and categorized as "mildly energetic" (MET values below 3), "moderately energetic" (MET values ranging from 3 to 6) and "vigorous" (MET values of 6 or above) physical activity. Health status was ascertained using a number of measures: prevalence of Coronary Heart Disease (CHD), based on clinically verified events, included non fatal myocardial infarction and definite angina; self-reported stroke or transient ischemic attack; diabetes (diagnosed according to the WHO definition); hypertension (systolic or diastolic blood pressure ≥ 140 or ≥ 90 mm

Hg respectively or use of hypertensive drugs); use of antidepressants; and cognitive functioning assessed by the Alice Heim (AH) 4-I, composed of a series of 65 verbal and mathematical reasoning items of increasing difficulty. Low cognitive score was defined as performances in the lowest quintile. For sensitivity analyses, we used the General Health Questionnaire (GHQ) (17), assessed both at phase 3 and phase 5 of the study which captured common mental disorders and included the 4-item depression subscale. All items were scored from 0 to 3 and then summed, cut-off points of 4 out of 12 were used to identify depression cases.

Statistical analysis

Neither natural thresholds nor clinically based thresholds are defined for the factor score measures of the two dietary patterns. We divided both scores into thirds based on their distribution in order to allow a robust estimation of self-reported depression across levels of dietary patterns that was not be driven by extreme values. Logistic regression was used to model the association between the tertiles of the two dietary patterns and CES-D depression. In the first model (M1), the analyses were adjusted for age, gender and energy intake; in the second model (M2) they were also adjusted for employment grade, educational level, marital status, smoking and physical activity. In the final model (M3), the analyses were further adjusted for health measures. Interactions between each dietary pattern and the covariates were tested and were found not to be statistically significant. To examine whether the association between dietary pattern and CES-D depression was robust, we ran two sensitivity analyses, the first adjusting for additional covariates, such as dyslipidemia (LDL cholesterol ≥ 4.1 mmol/L or use of lipid-lowering drugs) and BMI (calculated from measured high and weight, kg/m^2) in a sub sample for

whom these data were available and the second excluding individuals on anti-depressive treatment or who had GHQ-depression at phase 5. All analyses were conducted using the SAS software, version 9 (SAS Institute).

RESULTS

Compared to the 6943 individuals still alive at Phase 7, the 3486 participants included in the analyses were more likely to be men (73.8% vs. 66.7%), and less likely to be in the low occupational grade (8.3% vs. 18.0%) or have no academic qualification (8.0% vs. 10.0%). 5990 individuals had a CES-D assessment at phase 7, compared to those excluded from the present analyses, the prevalence of CES-D depression was lower in our study sample (11.9% vs. 19.2%). Concerning dietary habits, participants included in analyses were more likely to be in the highest tertile of both "whole food" and "processed food" dietary patterns. At phase 7, 416 participants were defined as CES-D cases (score>15). Characteristics of the participants as a function of the presence of CES-D depression are presented in Table 1.

Please insert Table 1 here

Factors associated with tertiles of the two dietary patterns, "whole food" and "processed food" at phase 5 are shown in Table 2.

Please insert Table 2 here

Table 3 shows the association between the two dietary pattern scores categorised in tertiles at phase 5 and CES-D depression at phase 7. Participants with the highest intake of "whole food" were less likely to report CES-D depression (Odds Radio (OR) = 0.64 95% Confidence Interval (CI) = 0.49-0.83 after adjusting for age, gender and energy intake, Model 1). This association was not much attenuated after adjustment for all covariates, (OR = 0.74, 95% CI: 0.56-0.99, Model 3 in the upper panel). By contrast, in the fully adjusted analyses (M3) participants with a high intake of "processed food" had higher odds of CES-D depression compared to those with the lowest intake (OR = 1.58, 95% CI: 1.11-2.23, Model 3 in the lower panel).

Please insert Table 3 here

Sensitivity analyses

Additional analyses were undertaken on a subsample with data on BMI and dyslipidemia (n=2702) at phase 5. Among them, 323 participants had CES-D depression at phase 7. High "whole food" intake at phase 5 remained associated with lower odds of subsequent CES-D depression at phase 7 (OR= 0.75, 95% CI: 0.54-1.03) while participants with high "processed food" scores had higher odds of CES-D depression (OR= 1.76, 95% CI: 1.19-2.62) after adjustment for all potential confounders, including BMI and dyslipidemia.

In an attempt to elucidate whether the association shown in table 3 was due to an effect of diet on depression and not the reverse, the analysis was repeated after excluding

the 427 participants who identified themselves as depressive at phase 5. As CES-D scale was unavailable at Phase 5,self-reported depression identified based on a score greater than or equal to 4 in the GHQ depression subscale (n=374) or reported antidepressant treatment (n=81). As can be seen in Table 4, among the remaining 3059 individuals of whom 265 had CES-D depression at phase 7 the results are comparable to those reported in table 3, reinforcing our original observation that poor diet is a risk factor for self-reported depression.

A further test of the reverse causality hypothesis examined GHQ depression at phase 3 (n=397) as a predictor of dietary pattern at phase 5. We found no evidence (p=0.24 for the "whole food" pattern and p=0.92 for the "processed food" pattern) to suggest that dietary patterns at phase 5 were worse among those participants who were GHQ depression cases at phase 3.

Please insert Table 4 here

DISCUSSION

We examined associations between two distinct dietary patterns, "whole food" (rich in fruit, vegetables and fish) and "processed food" (rich in processed meat, chocolates, sweet desserts, fried food, refined cereals and high fat dairy products) and CES-D depression 5 years later in a middle aged population. The "whole food" pattern was associated with lower odds of subsequent CES-D depression and the "processed food" pattern with higher odds of CES-D depression. These associations were robust to adjustments for a range of health parameters and behavioural factors.

Previously, dietary patterns have been investigated in relation to many health outcomes. However, the research on depression has mostly focused on the risk for depression associated with single nutrients. Our results suggest a protective effect of an overall diet rich in fruits, vegetables and fish while an overall diet rich in processed meat, chocolates, sweetened desserts, fried food, refined cereals and high fat dairy products seemed to be deleterious for depression. These findings are in line with a recent meta-analysis showing that adherence to a diet characterized by a high intake of fruits, vegetable, and fish and low intake of meat and dairy product (Mediterranean diet) was associated with lower overall mortality, mortality from cancer and cardiovascular diseases and lower incidence of neurodegenerative diseases (9).

There are several plausible mechanisms underlying the association we observed between the "whole food" pattern and self reported depression. The high amount of antioxidants in fruits and vegetables (18) could be protective as some studies have shown higher antioxidant levels to be associated with lower depression risk (19). The potential

protective effect of the "whole food" diet could also come from the folate, found in large amounts in some cruciferous vegetables (broccoli, cabbage, Brussels sprouts), leafy vegetables (spinach), other green vegetables (asparagus, avocado) and dried legume (lentil, chick pea) (20). It has been suggested that low levels of folate might increase the risk of depression and result in reduced availability of S-adenosylmethionine, a universal methyl donor, which can result in impaired formation of myelin, neurotransmitters and membrane phospholipids (21). In line with this a large study on Finnish middle aged men found an increased risk of depression (7) in participants with lower dietary intake of folate. However, some studies have found no association between folate levels and depression in elderly populations (6). A further plausible mechanism involves fish consumption. The "whole food" dietary pattern include a high intake of fish and there is evidence suggesting an association between high fish consumption and low incidence of depression (22). This protective effect of fish consumption has been traditionally attributed to its high long chain omega 3 poly-unsaturated fatty acids content (23). These are a major component of neuron membranes and have vascular and anti-inflammatory properties. Evidence of this association has come from observational studies that have shown an inverse association between n-3 fatty acid levels measured in blood or estimated from intake and depression (3, 4). Finally, it is also possible that the protective effect of diet on depression comes from the cumulative and synergic effect of nutrients from different sources of foods rather than from the effect of one isolated nutrient.

The deleterious effect of "processed food" on self reported depression is a novel finding. The "processed food" diet in our study was composed of sweet desserts, fried food, processed food, refined grain products, high fat dairy products and pies. A previous

cross sectional study has shown a correlation between sugar consumption and the annual rate of depression in 6 countries (24). Furthermore the "processed food" diet is very close to the "Western" pattern defined in the American population (8) which has been shown to be associated with higher risk of CHD (8) and inflammation (25). Several lines of investigation have suggested that CHD (26) and inflammation (27) are involved in the pathogenesis of depression. However, further studies are needed to better understand the association between "processed food" intake, the inflammation process and depression.

There are several limitations to the present study. First, reverse causation with depression affecting the dietary pattern rather than the other way around remains an alternative interpretation to the observed associations. To test this issue, we undertook sensitivity analyses and found no significant association between previous reports of depression (phase 3, 1991-93), using the GHQ depression subscale, and dietary patterns assessed 6 year later. This suggests that depression did not predict dietary behaviour in our study. Furthermore, we also showed that our main finding - the association between dietary patterns (phase 5, 1997-99) and CES-D depression at phase 7 (2002-04) - remains significant after excluding participants who reported depression at phase 5, assessed using the GHQ depression subscale and report of antidepressant treatment. We were not able to use the CES-D to exclude prior depression as it was only introduced at phase 7 of the study. Even if the use of different tools to assess self reported depression may decrease sensitivity of these analyses, results using the GHQ to exclude participants with prior depression show that the estimates of the association between dietary patterns and subsequent CES-D depression were similar to those reported in this paper. Thus, reverse causation seems an unlikely explanation for our findings.

Second, some bias due to selective retention of participants is possible as we found socio-economic position, depression and dietary patterns to be associated with the likelihood of being included in the analyses. If anything, this could contribute to an over-estimation of the association between a "whole food" pattern and self reported depression on account of the over-representation of individuals who are not depressed and follow a health conscious diet.

Third, a further limit of the study is the use of a semi-quantitative food questionnaire that only covers specific foods and is recognized to be less precise than dietary assessment by diary questionnaire. However, we have shown previously in this study population that nutrient intake estimated by the FFQ method is well correlated with biomarker levels and with intake estimates from the generally more accurate 7 day-diary (11). The factor analyses approach used to identify these patterns involves several arbitrary decisions such as the consolidation of food items into food groups: the number of factors extracted the methods of rotation and the labelling of the factors. However, the two major eating patterns identified – the "whole food" the "processed food " diet were similar to the "prudent" and "Western" patterns determined in large American prospective cohorts (8).

Fourth, the Whitehall II study participants are mainly office-based civil servants, not fully representative of the British population, and analyses were restricted to "white" participants which may limit the generalizability of the findings.

Finally, we cannot exclude the possibility of residual confounding in the analysis due to unmeasured or imprecisely measures factors. It is possible, for example, that a healthy diet is only one component of an overall healthy lifestyle which affords

protection against depression. However, the effects of dietary patterns on depressive symptoms did not substantially attenuate after adjustment for other indicators of a healthy lifestyle, such as smoking, physical activity and body mass, and a range of other potential confounders. This provides evidence against the interpretation that we have found a spurious association which is simply a by-product of an overall healthy lifestyle.

Despite these limitations, our study is unique in expanding the focus in this field of research from single nutrients and single foods to overall diet patterns. Our study provides evidence of a robust association between two dietary patterns - the "whole food and the "processed food" patterns - and depressive symptoms in a large prospective Caucasian middle aged British cohort. The results suggest that fruits, vegetables and fish consumption afford protection against the onset of depressive symptoms five years later, while a diet rich in processed meat, chocolates, sweet desserts, fried food, refined cereals and high fat dairy products increased vulnerability. These findings suggest that existing healthy eating policies will generate additional benefits to health and well-being, and that diet should be considered as a potential target for the prevention of depressive disorders.

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Tables

Table 1: Characteristics of Whitehall participants at phase 5 (2002-2004) according to the presence of CES-D depression at phase 7.

	CES-D Depression *		
	No N=3070	Yes n=416	
	% or M (SD)**	% or M (SD)**	p-value for heterogeneity
Women	25.3	33.2	0.0006
Age (year)	55.7(6.0)	54.5 (6.2)	0.0002
Single or Divorced	18.4	31.7	$<10^{-4}$
No Academic qualification	7.6	11.5	0.09
Low employment grade	7.8	12.0	$<10^{-4}$
Current Smoker	7.3	14.7	$<10^{-4}$
Low level of physical activity	12.0	18.3	$<10^{-4}$
Diabetes	2.5	5.0	0.003
Hypertension	25.1	22.6	0.26
Coronary heart disease	5.4	7.7	0.06
Self reported stroke	0.59	0.72	0.74
Antidepressant drug use	1.9	5.5	$<10^{-4}$
Low AH4 cognitive score ($< 1^{st}$ quintile)	11.9	14.4	0.14
Total energy (kcal/day)	2269(660)	2275(759)	0.85

* Depression assessed with CES-D questionnaire. Participants scoring more than 15 were classified as having 'CES-D depression'

** for continuous variables, means + standard deviations are given.

Table 2: Associations between dietary pattern at phase 5 and covariates at phase 5

	Dietary pattern at phase 5							
	"Whole food" dietary pattern				"Processed food" dietary pattern			
Variables measures at phase 7	Tertile 1[†] n=1162	Tertile 2[†] n=1162	Tertile 3[†] n=1162	p-value for trend	Tertile 1[†] n=1162	Tertile 2[†] n=1162	Tertile 3[†] n=1162	p-value for trend
Women	21.6	27.2	30.0	$<10^{-4}$	39.9	25.4	13.4	$<10^{-4}$
Age* (year)	54.9(6.0)	55.8(6.0)	56.1(6.1)	$<10^{-4}$	55.6(5.8)	56.0(6.1)	55.5(6.2)	0.005
Single / Divorced	27.5	20.0	20.5	0.0006	26.2	22.8	19.0	$<10^{-4}$
No academic qualification	9.6	8.3	6.2	$<10^{-4}$	7.8	8.4	7.9	0.23
Low employment grade	9.2	8.3	7.5	$<10^{-4}$	8.9	8.9	7.2	0.46
Current Smoker	11.1	8.1	5.4	$<10^{-4}$	6.6	8.9	9.2	0.10
Low level of physical activity	17.1	11.2	9.9	$<10^{-4}$	14.6	13.2	10.3	0.005
Diabetes	2.7	2.1	3.4	0.17	3.1	2.7	2.5	0.67
Hypertension	23.7	26.2	24.6	0.34	25.3	25.6	23.7	0.52
Coronary heart diseases	7.1	5.2	4.8	0.04	5.3	5.6	6.1	0.71
Self reported stroke	0.69	0.69	0.43	0.68	0.69	0.60	0.52	0.87
Antidepressant drugs use	3.1	1.6	2.3	0.06	2.6	2.2	2.1	0.77
Low AH4 Cognitive score	21.9	21.0	16.5	0.002	20.2	19.0	20.2	0.70
Total energy * (kcal/day)	1994(569)	2228(571)	2587(729)	$<10^{-4}$	1765(412)	2200(426)	2843(647)	$<10^{-4}$

[*] For continuous variable, Mean (M) with its standard deviation are given.
[†] Tertile 1, 2 and 3 represent individuals in the lowest, intermediate and highest thirds of the dietary factor score.

Table 3: Associations between dietary pattern scores at phase 5 and CES-D depression at phase 7 (n=3486).

	Lowest tertile	Intermediate tertile			Highest tertile		
	OR	OR	95% CI	p-value for trend	OR	95% CI	p-value for trend
"Whole food" dietary pattern							
Model 1	1	0.62	0.48-0.79	0.0002	0.64	0.49-0.83	0.001
Model 2	1	0.68	0.52-0.89	0.004	0.74	0.56-0.98	0.03
Model 3	1	0.71	0.54-0.92	0.01	0.74	0.56-0.99	0.04
"Processed food" dietary pattern							
Model 1	1	1.28	0.97-1.69	0.08	1.75	1.25;2.45	0.001
Model 2	1	1.22	0.92-1.62	0.17	1.58	1.12-2.23	0.009
Model 3	1	1.22	0.92-1.62	0.17	1.58	1.11-2.23	0.01

Model 1: Adjusted for gender, age and energy intake
Model 2: Model 1 + adjustment for marital status, employment grade, education, physical activity and smoking habits
Model 3: Model 2 + adjustment for hypertension, diabetes, cardio-vascular diseases, self reported stroke, use of anti-depressive drugs, and cognitive functioning.

Table 4: Associations between dietary pattern scores at phase 5 and CES-D depression at phase 7 after excluding participants identified as depressive* at phase 5 (total n=3059).

| | Lowest tertile | Intermediate tertile | | | Highest tertile | | |
	OR	OR	95% CI	p-value for trend	OR	95% CI	p-value for trend
"Whole food" dietary pattern							
Model 1	1	0.63	0.46-0.87	0.005	0.66	0.47-0.92	0.01
Model 2	1	0.70	0.50-0.96	0.03	0.74	0.52-1.04	0.08
Model 3	1	0.68	0.50-0.94	0.02	0.73	0.51-1.02	0.07
"Processed food" dietary pattern							
Model 1	1	1.44	1.02-2.02	0.04	1.83	1.20-2.79	0.004
Model 2	1	1.41	1.00-2.00	0.05	1.76	1.14-2.70	0.01
Model 3	1	1.38	0.98-1.95	0.06	1.69	1.10-2.60	0.02

Model 1: adjusted for gender, age and energy intake
Model 2: Model 1 + adjusted for marital status, employment grade and level of education, physical activity and smoking habits
Model 3: Model 2 + adjusted for hypertension, diabetes, cardio-vascular diseases, self reported stroke, use of anti-depressive drugs, and cognitive functioning

*Cases defined as depressive using the General Health Questionnaire (GHQ) depression subscale (n=374) or those taking antidepressant drugs (n=81)

Appendix 1 : Food groups used for factor analyses

Foods or Food groups	Food items
Red Meat	Beef, beef burgers, pork, lamb
Poultry	Chicken or other poultry
Processed meats	Bacon, Ham, corned beef, Spam, luncheon meats, sausages
Organ meat	Liver
Fish	White fish, oily fish and shellfish
Refined grain	White bread and rolls, cream cracker, cheese biscuits, crisp bread, Refined grain ready-to-eat cereals, white pasta, white rice
Whole grain	Brown bread and rolls, wholemeal bread and rolls, wholemeal pasta, brown rice, whole grain ready-to-eat cereals
Eggs	Eggs
Butter	Butter
Margarine	Margarines, spread
High fat dairy	Full cream milk, Channel Island milk, Coffee whitener, Single or clotted cream, cheese, ice cream
Low fat dairy	Skimmed milk, sterilized milk, dried milk, yoghurt, cottage cheese
Soya product	Soya milk, tofu, Soya bean curd, Soya meat, TVP, vege-burger
Liqueurs/Spirits	Port, sherry, Liqueurs, spirits
Wine	Wine
Beer	Beers, ciders
Hot drinks	Tea, Regular coffee, Decaffeinated coffee, Cocoa, hot chocolate, Chicory
Fruits	Apples, pears, oranges, mandarins, grapefruit, bananas, grapes, melon, peaches, plums, apricots, strawberries, raspberries, tinned fruit, dried fruits
Fruit juice	100 % Real fruit juice
Leafy vegetables	Spinach, salads
Cruciferous vegetables	Broccoli, kales, Brussels spouts, cabbage, cauliflower, coleslaw
Other vegetables	Carrots, marrow, courgettes, parsnip, leeks, mushroom, peppers onion, garlic
Tomatoes	Tomatoes
Peas and dried Legume	Beans, peas, baked beans, dried lentils
Soup	Vegetable soup, meat soup
Nuts	Peanuts, other nuts, peanut butter
Potatoes	Boiled, mashed potatoes, jacket potatoes, potato salad
Quiche/Pie	Quiche, meat pie
Pizza/Lasagne	Pizza, Lasagne
Fried food	Chips or French fries, Roast potatoes, Fish fingers, fried fish in batter
Snacks	Crisps
Desserts/biscuits	Sweet biscuits, cakes, buns, pastries, fruits pies, tarts, crumbles, milk pudding, sponge puddings
Chocolate and sweets	Chocolate bars, sweets, toffees, sugar added to tea, coffee, jam, marmalade, honey.
Sugar beverages	Fizzy soft drinks, fruit squash
Low calorie beverages	Low calorie or diet fizzy soft drinks
Condiments	Sauce, tomato ketchup, pickles, marmites
Salad dressing	French vinaigrette, salad cream

Appendix 2: Factor loading† for high loading items (≥0.40) ‡ on the two dietary patterns

	1st Pattern : "whole food" pattern	2nd Pattern : "processed food" pattern
Leafy vegetables	0.66	-
Other vegetables	0.64	-
Tomatoes	0.59	-
Fruits	0.57	-
Cruciferous vegetables	0.49	-
Salad Dressing	0.49	-
Fish	0.44	-
Desserts/biscuits	-	0.55
Processed meats	-	0.52
Fried food	-	0.50
Chocolates, and sweets	-	0.50
Refined grain	-	0.46
Quiche/Pie	-	0.44
High fat dairy	-	0.44
Condiments	-	0.43

† The 2 dietary patterns were derived using principal component analysis. Factor loadings issued from orthogonal rotation, represent the correlation between the factors and individual items from food group.

‡ The factor score for each pattern was calculated by summing intakes of the 37 predefined food groups (see Appendix 1) weighted by their factor loadings. In order to simplify interpretation of the 2 patterns, values < 0.40 were not listed in the table but are used in the construction of the factors.