A high-fat, high-glycaemic index, low-fibre dietary pattern is prospectively associated with type 2 diabetes in a British birth cohort

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Key words: Dietary pattern, type 2 diabetes, dietary fibre, glycaemic index

Abstract

The combined association of dietary fat, glycaemic index (GI) and fibre with type 2 diabetes has rarely been investigated. The objective was to examine the relationship between a high-fat, high-GI, low-fibre dietary pattern across adult life and type 2 diabetes risk using reduced rank regression. Data were from the MRC National Survey of Health and Development. Repeated measures of dietary intake estimated using 5-day diet diaries were available at age 36, 43 and 53 for 1180 subjects. Associations between longitudinal changes in dietary pattern z-scores and type 2 diabetes incidence (n=106) from 53 to 60-64 years were analysed. The high-fat, high-GI, low-fibre dietary pattern was characterised by low intakes of fruit, vegetables, low-fat dairy products, and whole grain cereals, and high intakes of white bread, fried potatoes, processed meat and animal fats. Higher scores for the dietary pattern were significantly associated with increased type 2 diabetes odds among women but not among men. Among women, for each 1 SD unit increase in dietary pattern z-score between 36 and 53 years the OR for type 2 diabetes was 1.67 (95% CI: 1.20, 2.43) independently of changes in BMI and waist circumference in the same periods. The effect size was larger among initially (age 36) overweight (OR: 2.90, 95%CI: 1.04, 8.11) compared to normal-BMI women (OR: 1.48, 95%CI: 0.94, 2.32). A high-fat, high-GI low-fibre dietary pattern was associated with increased type 2 diabetes risk in middle-aged British women. Long-term increases in this dietary pattern, independently of BMI and waist circumference, were particularly detrimental for overweight women.

Introduction

Increasing evidence points to the role of certain dietary factors as key players in metabolic abnormalities, not only as contributors to body weight, a prominent risk factor for type 2 diabetes, but also as independent risk factors. For example, studies support the beneficial role of dietary fibre in reducing post-prandial glycaemic response, improving insulin resistance and reducing inflammation (1, 2). Conversely, high glycaemic index (GI) foods induce postprandial hyperglycaemia, which has been linked to type 2 diabetes risk (3, 4). Evidence also shows that increased fat intake can promote insulin resistance and inflammatory responses (5, 6). However these dietary factors have been rarely examined simultaneously in relation to type 2 diabetes risk.

Over the past decade, dietary pattern analyses have increasingly been used to study associations between diet and disease risk. Dietary patterns may better describe the 'real world' eating habits of free-living people, where nutrients are consumed together, and not in

isolation (7), and can therefore be used to create food-based public health guidance that is easier to interpret than nutrient-based advice. Dietary patterns are the sum of the effect of different foods the likely synergistic effects of different foods and nutrients that make up the total diet (8).

Empirically-defined dietary patterns defined as 'healthy' and high in fruit, vegetables and wholegrain foods, and low in red meat, added sugar and fried foods, have been linked with a reduced type 2 diabetes risk (9-13), however the mechanisms or pathways between 'healthy' dietary patterns and type 2 diabetes risk are as yet, unclear. Reduced rank regression (RRR) (14) is a hypothesis-based statistical approach to identifying dietary patterns. The few studies so far that have applied RRR to examine diet and type 2 diabetes risk have mainly investigated dietary patterns related to inflammatory pathways (15-17). To our knowledge, no study has used RRR to investigate dietary patterns characterised by dietary GI, fibre and fat intake to date, yet separately, these dietary factors have been linked with diabetes risk. Furthermore, despite the increasing popularity of studying dietary patterns, most cohort studies use only a single measure of dietary intake at baseline. It is important to study how changes in these patterns over the lifecourse affect disease risk and to what extent changing diet alters disease risk.

The aim of this study was to identify an RRR-derived dietary pattern characterised according to dietary fibre, GI and dietary fat, as these have been independently linked to increased type 2 diabetes risk, and to assess its longitudinal association with type 2 diabetes risk in the MRC National Survey of Health and Development. It was hypothesised that repeated measures of a dietary pattern characterised by high intakes of fibre and low intakes of fat and low GI would be longitudinally and positively associated with type 2 diabetes risk over the life course, and independently of body weight and waist circumference.

Materials and methods

Participants

The MRC National Survey of Health and Development (NSHD) is a socially stratified sample of 5362 individuals (2547 males and 2815 females) born during one week in March 1946 in England, Scotland and Wales. The cohort has been followed-up 23 times to date, and the response rate throughout the study has been good, ranging between 78% at age 16-35 years and 95% at age 0-4 years (18). At the latest data collection in 2006-2010 at age 60-64, 53% of the original cohort (N=2856) was eligible for inclusion after exclusion of those who had died (n=778), lived abroad (n=584), had previously refused consent (n=594) or were

untraceable (n=550). The 2661 individuals who responded (49% of the original cohort) had remained broadly representative of the white British population born in the early post-war years (19). The present analysis includes data on diet at age 36, 43 and 53 and incident type 2 diabetes diagnosed between age 53 and 60-64. Survey respondents who recorded at least 3-days of a food record were included in these analyses. The number of respondents completing diet diaries for at least 3 days was 2441 at age 36, 3187 at age 43 and 1776 at age 53 corresponding to respectively 45%, 59% and 33% of the original cohort. At all ages, individuals who completed diet diaries were more likely to be female, to be more educated, less likely to be in manual employment, and to be smokers. We restricted all analyses to individuals with complete data on diet as well as all variables needed. Complete data on diet, diabetes and all covariables were available for 1804 individuals at age 36, 2267 at age 43 and 1478 at age 53.

Dietary data

Study members were asked to complete a 5-day food diary at age 36, 43 and 53, detailing all foods and drinks consumed over 5 consecutive days (20). Survey members were given guidance on household measures and photographs of portion sizes to aid completion by a research nurse who visited them at home. Food diaries were checked before coding and calculation of average daily nutrient intakes using an in-house program developed at the MRC Human Nutrition Research Unit (21), which linked food diaries with contemporaneous British food composition data. Food intakes were collapsed into 45 food groups defined according to differences in GI and content of fat and fibre (**Table 1**). Dietary fibre density (g/1000kcals) and fat density (g/1000kcals) were calculated as total daily g fibre (non starch polysaccharide) or g fat divided by total daily energy intake (kcal) and multiplied by 1000. Glycaemic index values were assigned to each food using the methodology described in detail by Aston et al. (22). Briefly, all food codes with total carbohydrate >0.1g per 100g were assigned a GI value, based on five levels of data confidence relating to source of the data used, with level 1 being the highest. The average GI of the daily diet was calculated by assigning a glycaemic load (GL) value for each food item, then summing the GL values for the day and dividing this by the total daily carbohydrates in grams (23).

To assess dietary misreporting, the ratio of energy intake (EI) to estimated energy requirement (EER) was calculated according to an individualised method (24). EERs based on individual physical activity levels were calculated using equations from the Institute of Medicine of the National Academies (25). To account for the variability of the methods used to estimate EI and EER, a 95% confidence interval (CI) for EI:EER was calculated (26). The

95% CI of EI:EER for the NSHD was 0.54 and 1.46. Individuals reporting EI less than 54% of their EER were classified as underreporters, those reporting more than 146% as overreporters. The percentage of plausible EI reporters was 83% at age 36, 84% at age 43 and 88% at age 53. EI underreporting was higher among overweight people and decreased with higher dietary pattern z-score. At age 36, 21% of overweight or obese people (n=126 out of 581) were EI underreporters compared with 9% of normal-weight people. At age 43 16% of those overweight (n/N=168/1046) underreported their EI compared to 9% among those with a normal weight. The corresponding percentages of EI underreporting at age 53 were 4% (n/N=38/875) and 2%. At age 43 EI underreporting was higher among those diagnosed with type 2 diabetes between age 53 and 60-64 (20% compared with 13% among the rest of the sample). Therefore, EI misreporting was included as a covariable in all analyses.

Type 2 diabetes

Ascertainment of type 2 diabetes at age 53 was based on validated self-report. Self -reported diabetes was determined in response to a direct question and from all relevant medical information that study members reported (hospital attendances and medications). The validity of self-reported diabetes was assessed using general practitioners records, with a positive predictive value of 95% (27). One hundred cases of prevalent diabetes at age 53 were excluded from these analyses. At age 60-64 diabetes was ascertained by both self-reported information and by analyses of fasting blood glucose and HbA1c from 50-ml blood samples collected between 2006 and 2011 in 5 clinical research facilities. A diagnosis of diabetes was established if fasting plasma glucose was equal or greater than 7mmol/L or HbA1c was equal or greater than 6.5% (48 mmol⁄mol) (28).

Covariables

Occupational social class, educational attainment, smoking and physical activity, based on interview and questionnaire data, were included as possible confounding factors. Body Mass Index (BMI) and waist circumference (WC) were included as mediating variables as it was hypothesized that body weight would partially explain the association between diet and type 2 diabetes.

Data on lifetime occupational head of household social class at age 53 (or earlier if this was unavailable) according to the UK Registrar-General (29) was coded into 6 categories: I professional, II managerial and technical, IIINM skilled non-manual, IIIM skilled manual, IV partly-skilled manual, V unskilled manual. The highest level of educational qualification achieved by age 26 was grouped into 3 categories: none (none attempted), intermediate (GCE

'O' level or equivalent, or vocational) or advanced (GCE A level or equivalent, or degree or equivalent).

Physical activity at 36, 43 and 53 years was coded as inactive (no participation), moderately active (participated one to four times) and most active (participated five or more times), in the previous month (36 years), per month (43 years) and in the previous 4 weeks (53 years). Smoking at each follow up was categorised as current, ex and never smoker. Use of prescribed medicines was assessed at each follow-up by a questionnaire. The latest information available (at age 53) on prescribed medication for hypertension and dyslipidaemia was used in all models. At all ages (36, 43, 53), a trained research nurse measured height, weight and WC using standard protocols. BMI was calculated from weight (in kilograms) divided by height (in meters) squared.

Statistical analyses

RRR was used to identify dietary pattern z-scores. RRR derives dietary patterns by extracting successive linear combinations of predictor variables (food groups) that explain as much variation as possible in another set of response variables, which are hypothesised to be on the pathway between the predictor variables and the outcome (14). Dietary fibre density (g/1000kcal), GI (units) and total dietary fat density (g/1000kcal) were chosen as the response variables because, based on previous literature, they were hypothesised to be important dietary determinants of the risk of type 2 diabetes. The function PROC PLS in the software SAS was used to conduct all RRR analyses.

Initially, exploratory RRR analyses were conducted separately using dietary data collected at age 36, 43 and 53. Each study member received a z-score calculating the degree to which their dietary intake reflected this dietary pattern at age 36, 43 and 53. To assess longitudinal associations between dietary patterns and type 2 diabetes a z-score for exactly the same dietary pattern (based on the same covariance matrix) at 36, 43 and 53 years was required. To achieve this, confirmatory RRR analyses (30) were used to calculate dietary pattern z-scores at 36 and 43 years of age using scoring weights from the first dietary pattern identified at 53 years. This dietary pattern was chosen because it explained a greater variation in response variables (38%) compared to those at age 36 and 43.

Multivariable logistic regression models were used to examine prospective associations between quintiles of dietary pattern z-scores at 36, 43 and 53 years of age and type 2 diabetes risk between age 53 and 60-64 years. The diet z-score quintiles were entered as a categorical variable, with the lowest quintile used as the reference category. Analyses were adjusted for social class, education, smoking, physical activity, medication, EI and EI misreporting

(Model 1) and subsequently for BMI and WC (Model 2). Interactions between the dietary pattern z-score and gender were tested using multiplicative interaction models.

To examine changes in dietary pattern z-scores over the lifecourse in relation to Type 2 Diabetes risk, a conditional model of change (31) was used to estimate the association between periods of changes in dietary pattern z-scores and the odds of type 2 diabetes. Dietary pattern z-score changes for the periods 36–53, 36–43 and 43–53 years were calculated conditional on earlier z-score using the residual method. Z-score changes were obtained by regressing each z-score measure on the earlier measures and saving the residuals. These residuals represent the change in dietary pattern z score above or below what is expected given an earlier z-score. A positive change z-score value reflects a deterioration of diet quality; conversely a negative change z-score represents an improvement of the diet. It has been reported that the detrimental effect of a high GI diet might be more pronounced among overweight people who are often more insulin resistant than normal weight individuals (32, 33). Therefore, to test this hypothesis, longitudinal analyses were also presented stratified by initial (age 36) BMI. Interaction by sex and BMI was tested in multiplicative interaction models.

Results

RRR derives as many dietary patterns as there are response variables, which in this case were three. At all ages the first dietary pattern derived from RRR analyses explained the greatest variation in all three response variables (total variation accounted for was 29.8% at age 36, 31.8% at age 43 and 37.9% at age 53) compared with the second and third patterns, which explained around 12-15% and 5% respectively. Therefore, only the first dietary pattern was analysed further. Characteristics of the 3 RRR-derived dietary patterns at age 36, 43 and 53 are show in **Supplementary Table 1**. Results are presented separately for men and women because a significant interaction between the dietary pattern z-score and sex was observed at age 43 ($p=0.01$), although the interaction terms at age 36 ($p=0.85$) and 53 ($p=0.14$) were not significant at the 5% level.

The dietary pattern used for confirmatory analyses was negatively associated with dietary fibre density ($r=-0.70$) and positively associated with fat density ($r=0.44$) and GI ($r=0.55$). A higher z-score for this dietary pattern signifies a diet higher in GI and fat and lower in fibre. Factors loadings for this dietary pattern are shown in **Figure 1**. A positive factor loading indicated that, as the intake of that food increased, so did the dietary pattern z-score; whereas foods with a negative factor loading decreased the z-score. The dietary pattern was

characterised by low intake of fruit, vegetables, low-fat yogurt, wholemeal bread, high-fibre cereals and high intakes of white bread, processed meat, fried potatoes, butter and animal fat and added sugar. Fifty-seven per cent of the variation in dietary pattern z-score was explained by the top five and bottom five factor loadings, with fresh fruit explaining the most variation (23%), then white bread (8%), vegetables (6%), low-fat yogurt (5%), and processed meat (4%).

At all ages people with higher z-scores for the high fat, high GI, low fibre dietary pattern were significantly more likely to be in manual employment, to be smokers, physically inactive and to have no educational qualifications (**Table 2**). BMI and WC were positively associated with higher dietary pattern z-scores at age 53. Those with higher z-scores had greater intakes of energy (kcal), fat density, alcohol and a greater average daily GI as well as lower intakes of dietary fibre density.

The number of incident cases of type 2 diabetes diagnosed between age 53 and 60-64 was 166 (94 among men and 72 among women). A significant interaction was observed between dietary pattern scores at age 43 years and sex on type 2 diabetes (p=0.02) and between dietary pattern scores changes and diabetes (p=0.01). The dietary pattern was significantly associated with increased odds of diabetes among women at age 43 and 53 (**Table 3**). Among women there was an increasing trend in OR for type 2 diabetes with increasing quintile of dietary pattern z-score. Those women in the highest z-score quintile at 43 years had an OR for type 2 diabetes of 5.45 (2.01, 14.79); women in the highest quintile at 53 years had an OR of 3.22 (1.08, 9.54). After adjustment for BMI and WC the associations remained at age 43 (p for trend across quintiles <0.01) but were no longer significant at age 53 (p=0.05) (Table 3). No associations at the 5% level were observed for men.

Analyses of z-scores changes in dietary pattern and type 2 diabetes were conducted for those who provided diet diaries at all three data collection years and had non-missing values for all covariables (N=1180). There were no significant differences in average score change between men and women. People who developed type 2 diabetes between age 53 and 60-64 on average increased their dietary pattern z-score, with an overall change between age 36 and 53 of 0.26 standard deviation (SD) units, compared to a change of -0.06 SD units for the rest of the sample (p <0.01). The difference in mean dietary pattern z-score change from age 36 to 43 (and consequently from age 36 to 53) between those who later developed diabetes and those who did not was greater in women than men (**Figure 2**).

Multivariable regression models (**Table 4**) showed that, independently of simultaneous changes in BMI and WC, increases in dietary pattern z-score between age 36 and 43 were significantly associated with type 2 diabetes risk among women (OR 1.63, 95%CI: 1.08,

2.46) but not men; increase between age 43 and 53 were of borderline significance among women. In analyses stratified by baseline BMI, the effect size for a 1 SD unit increase in z-score between age 36 and 53 was larger among overweight women (OR: 2.90, 95%CI: 1.04, 8.11) compared to normal-BMI women (OR: 1.48, 95%CI: 0.94, 2.32). However, the test for interaction was not significant ($p>0.05$) and therefore any differences between the groups should be interpreted with caution.

Discussion

In this analysis of a large UK birth cohort, we identified a high fat, high GI, low fibre dietary pattern that was prospectively associated with type 2 diabetes risk. This dietary pattern was characterised by a high consumption of white bread, processed meat, fried potatoes, butter, animal fats and added sugar, and a low intake of fruits, vegetables, low-fat yogurt and high-fibre cereals. Higher z-scores for this dietary pattern at age 43 and 53 were associated with an increased risk of type 2 diabetes diagnosed between age 53 and 60-64 among women, but not among men. Among women, a gradually increasing z-score representing an increasingly unhealthy diet over the life course (36 to 53 years) was strongly associated with type 2 diabetes. This association was independent of a wide range of potential confounders, including other health-related behaviours, and of the potential mediation of BMI and WC. Dietary glycaemic index and fibre act on satiety signals while foods high in fat are very energy-dense therefore affecting energy intake. Thus, it was expected that a dietary pattern high in fat and GI and low in fibre would act partly through its effect on energy intake and weight gain. The fact that an independent association between dietary pattern and diabetes remained after adjustment for energy intake and BMI and WC changes suggests that this pattern also acts through alternative pathways. The postprandial hyperglycaemia induced by high GI foods can affect β-cell functions and insulin resistance both directly and indirectly by inducing a counter-regulatory hormone response, which increases circulating levels of free fatty acids (3, 34). Free fatty acids, which are elevated when excess calories and fat are consumed, increase insulin resistance by disrupting insulin signals in the gut and promote β-cell dysfunction though their lipotoxic effect in the pancreas (35). Dietary fibre might reduce type 2 diabetes risk though its anti-inflammatory properties and its effect on glycaemia (36). Our results also show that this dietary pattern is more detrimental in women who were already overweight as young adults. This is consistent with some studies (31, 32) reporting that the detrimental effect of dietary GI might be more pronounced in overweight and obese people, who are often more insulin resistant than normal weight individuals. However, we could not address this since insulin resistance was not measured directly in this study.

The stronger association between the dietary pattern and diabetes among women could be due to several reasons. There might be biological sex differences in the responses to certain nutrients and the way these are disposed of and stored in the postprandial state. For example it is known that sex-specific hormones can influence insulin receptors and lipid removal (37), and that men oxidise a higher percentage of ingested fat than women (38). It is unlikely that the gender difference could be due to different food choices since there were no major gender differences in intake of the main foods characterizing the dietary patterns. Hormonal changes associated with menopause might also explain the higher relative risk for type 2 diabetes with longer-term increases in dietary pattern z-score in women; it is possible that the cumulative influence of an unhealthy diet (as well as other lifestyle factors) on metabolic functions could come into play in the peri-menopausal years, which is when women become more susceptible to chronic diseases associated with aging (39).

Few cohort studies of this type have investigated men and women separately. In the Melbourne Collaborative Cohort Study (11) the association between a dietary pattern characterized by meats and fatty fried foods and diabetes was significantly stronger among women, whose risk for the disease was nearly 4-fold in the highest quintile of intake compared to the lowest quintile. Conversely, the risk among men in the highest quintile of intake was 2-fold compared to the lowest quintile and borderline significant (11). In the Nurses' Health Studies (15) the relative risk of diabetes from intakes of a RRR-derived dietary pattern high in processed meat, refined grains and soft drinks was particularly high; on the other hand a similarly characterised dietary pattern showed comparatively weaker associations in the Health Professionals Follow up Study, which included male study members (12).

Previous studies have found that protective dietary patterns identified with factor and cluster analyses, often labelled 'healthy' or 'prudent', tend to include fruits, vegetables, whole grains, whole bread and low-fat dairy products, whereas, dietary patterns associated with increased type 2 diabetes risk tend to be high in red and processed meat, refined grains, fried foods, high-fat dairy products and sugar (9-13). However, these dietary patterns were identified used purely exploratory methods which do not necessarily identify disease-specific dietary patterns and therefore, their mechanisms of action may be difficult to elucidate. On the contrary, this study used RRR and incorporated hypothesised knowledge about pathways to disease, thus providing insight into the possible biological pathways that link these food groups with type 2 diabetes. This allowed us to investigate the synergistic action of dietary fibre, GI and dietary fat, individual factors for which there is increasing evidence of a link

with type 2 diabetes. Furthermore, food-based public health recommendations based on key diabetes-relevant nutrients can be provided.

We should address various strengths and weaknesses of this study. Unlike most other prospective cohort studies, which rely on food frequency questionnaires (FFQs), the NSHD uses diet diaries, which do not rely on dietary recall. Prospectively recorded diet diaries correlate significantly better with biomarkers of intake, and are subject to substantially less regression dilution than FFQs (40). GI values were assigned by rigorous methodology and, where possible, GI values were sourced from the UK or from European studies. This ensured that the GI values in the NSHD were country-specific and as accurate as possible.

A particular strength was the use of repeated measures of dietary intake to investigate adult life course changes in dietary patterns and type 2 diabetes risk; this has rarely been addressed in epidemiological studies, and most studies of dietary patterns assume that eating behaviours remain stable over the adult life course. Other strengths of this study were the use of a validated diabetes outcome measure and inclusion of energy misreporting.

On the other hand, loss to follow-up in NSHD might have introduced some degree of bias. Those providing dietary data were healthier and more likely to be women compared to those who did not complete diet diaries. Loss to follow-up of those less socially advantaged and less healthy may have resulted in under-estimation of effect sizes (41), although we have no reason to suspect that this would have altered the pattern of these associations. Reflecting the ethnic make-up of Britain in the 1940s, the NSHD is comprised exclusively of Caucasians. Therefore, the findings from this thesis might not be generalizable to cohorts of different ethnic groups. It is also important to recognise the potential measurement error associated with dietary assessment. The use of conditional change models might be associated with error when applied to repeated measures that are measured with some degree of error, as it is with diet.

In conclusion, a dietary pattern characterised by high fat, high GI and low fibre intakes was prospectively associated with type 2 diabetes risk among women and this association was independent of energy intake, BMI and WC. This association was robust when the dietary pattern was examined longitudinally over the life course (36 to 53 years), suggesting that the cumulative effects of changes in diet over a long-term period are particularly important for type 2 diabetes for women.

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Conflict of interest

None.

Authorship

Authors' contributions were as follows—SP and GLA: conceived and designed the study; SP: analysed the data; all authors interpreted the data and oversaw the study; SP: wrote the first draft of the manuscript. All authors revised and contributed to the final manuscript.

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Table 1. Description of food groups included in the dietary pattern analyses

Food group name	Foods included
Pizza	Pizza
Pasta	Pasta & pasta dishes
Rice	Rice & rice dishes
Cereals_other	Cereals other than pasta, bread and rice
High-fibre cereals	Breakfast/oat cereals with fibre content equal or >3g per 40g portion;
Low-fibre cereals	Low-fibre cereals and breakfast bars
White bread	White bread
Wholemeal bread	Wholemeal, granary and brown bread
Crisp & other bread	Crisp bread (e.g. Rivita, grissini) and other bread
Biscuit, pastry, cakes	Biscuits, pastries, buns, pies and cakes
Whole milk	Whole milk (cow or goat)
Skimmed milk	Skimmed milk, semi-skimmed milk and milk 1%
Low-fat dairy desserts	Low fat dairy desserts, low-fat ice-cream and flavoured milk
Full-fat yogurt	Full-fat yogurt
Low-fat yogurt	Low-fat yogurt
Full fat dairy dessert	Full fat dairy desserts, ice-cream and milk pudding
Cream	Cream
Butter and animal fat	Butter and animal fat
Cheese	Cheese
Eggs	Eggs
Oils	Vegetable Oils
Plant fat solid	Plant based fats (solid)
Plant fat solid low fat	Plant based fats (solid), such as margarine
Fish	White fish, oily fish and shellfish
Red meat, offal	Beef, lamb, pork and other red meat (including dishes)
White meat	Chicken, turkey and other game birds (including dishes)
Processed meat	Bacon, ham, meat pies, sausages and other processed meats
Vegetables	Raw and cooked vegetables
Pulses	Pulses, lentils and baked beans
Fruit	Fresh, canned and dried fruits
Potatoes	Potatoes (not fried or roasted)
Fried potatoes	Fried and roasted potatoes
Nuts and seeds	Nuts and seeds (including peanut butter)
Soups	Canned, fresh and dried soup
Dressing & sauces	Dressings, mayonnaise, cooking sauces and other sauces
Jam and chutney	Jam, marmalade, chutney and pickles
Table sugar	Sucrose
Honey and syrup	Honey, syrup and other sugars (not pure sugar)
Confectionery	Chocolate products, sugar-based products, sorbets and lollies
Savoury snacks	Savoury biscuits, potato-, cereal- and vegetable-based snacks
Alcoholic drinks	Wine, beer, spirits, Alco pops
Squashes & juices	Squashes & fruit concentrate, fruit juice drinks
Pure fruit juice	Pure fruit juice and smoothies
Soft drinks	Carbonated soft drinks
Coffee & tea	Coffee, tea, powdered beverages (e.g. ovaltine)

Table 2. Study population characteristics by quintiles of the high-fat, high-GI, low-fibre dietary pattern z-score at age 36 (N=1804), 43 (N=2267) and 53 (N=1478)

	Q1		Q2		Q3		Q4		Q5		P‡
	n		n		n		n		n		
36 y	361		361		361		361		360		
43 y	454	3.8	453	3.8	454	3.8	453	3.8	453	3.8	
53 y	296		296		295		296		295		
	%		%		%		%		%		
Type 2 diabetes*	7.7		9.7		8.8		10.2		10.2		0.22
	7.9		6.6		10.5		10.1		12.1		<0.01
	5.7		7.4		7.4		11.4		13.2		<0.001
Male sex	49.0		49.3		46.5		44.0		48.3		0.59
	46.7		50.7		48.7		45.2		47.6		0.53
	44.9		43.2		45.7		49.6		42.7		0.46
Manual SEP	23.2		23.5		26.5		32.4		43.0		<0.001
	21.5		27.1		28.1		34.4		45.0		<0.001
	23.6		23.6		27.8		33.7		41.0		<0.001
No education	21.6		21.6		30.1		36.0		48.8		<0.001
	24.0		26.9		31.7		37.0		49.6		<0.001
	17.2		25.0		26.4		33.1		43.7		<0.001
Physically Inactive	29.0		28.8		32.4		37.6		44.1		<0.001
	37.8		42.1		48.9		55.8		63.5		<0.001
	34.8		36.8		48.8		50.5		44.3		<0.001
Current smoker	16.3		21.0		23.5		28.2		43.3		<0.001
	12.7		20.0		22.4		27.5		45.4		<0.001
	6.0		10.1		11.5		19.9		35.9		<0.001
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
BMI (kg/m^2)	24.3	3.8	23.8	3.2	23.7	3.2	24.0	3.5	23.5	3.1	0.07
	25.1	3.7	25.0	3.6	25.3	3.8	25.2	3.7	25.3	4.4	0.30
	26.3	4.3	26.7	4.1	27.1	4.5	27.2	4.6	27.4	4.9	<0.01
WC (cm)	82.1	11.6	82.4	11.7	81.3	11.7	82.9	12.3	82.2	12.0	0.35
	83.0	12.3	83.4	11.8	84.5	12.0	84.1	12.4	84.7	12.8	<0.01
	87.2	12.7	89.0	12.9	90.5	12.6	91.1	12.8	91.3	13.3	<0.001
Energy, kcal	1785	569	1940	574	2004	550	2065	551	2373	679	<0.001
	1833	567	1934	568	2078	571	2129	569	2424	708	<0.001
	1867	476	1913	456	1999	474	2031	501	2117	514	<0.001
EE:EER	0.65	0.1	0.71	0.1	0.75	0.1	0.78	0.2	0.91	0.2	<0.001
	0.69	0.1	0.74	0.2	0.80	0.2	0.84	0.2	0.97	0.2	<0.001
	0.70	0.1	0.73	0.1	0.78	0.1	0.78	0.1	0.83	0.2	<0.001
Fibre density†	8.4	3.4	6.6	2.3	5.7	2.0	5.1	2.0	4.3	1.6	<0.001
	7.6	2.8	6.0	2.0	5.6	1.6	5.1	1.2	4.6	1.1	<0.001
	9.8	3.3	8.2	2.3	7.0	2.1	6.4	1.9	5.5	1.5	<0.001
Glycaemic Index	61.3	10.0	63.1	3.0	64.4	3.3	65.5	3.6	66.5	2.9	<0.001
	60.4	4.8	63.0	4.7	63.8	4.5	65.1	4.2	66.8	3.8	<0.001
	58.8	3.5	60.3	3.2	61.6	3.3	63.5	3.3	64.9	3.5	<0.001
Fat density	41.6	5.9	43.7	5.4	44.3	5.9	44.6	5.2	45.1	4.9	<0.001
	38.6	7.3	41.7	5.6	43.3	5.6	44.9	5.7	46.5	5.1	<0.001
	32.6	6.8	36.1	5.6	38.8	5.7	39.4	5.4	42.3	5.9	<0.001
Alcohol† (g/d)	5.5	15.3	6.7	17.7	7.5	18.0	8.2	21.4	6.2	21.3	0.01
	5.4	15.9	7.7	18.0	6.4	17.6	6.1	17.3	6.8	15.9	<0.01
	7.5	17.4	8.1	17.5	9.7	21.9	12.3	25.6	9.2	26.6	0.01

*Diagnosed between age 53 and 60-64. †Median ± IQR. ‡Test for trend by linear or logistic regression with control for sex.

Table 3. Associations at each age between a high fat, high GI, low fibre dietary pattern z-score and incident type 2 diabetes between age 53 and 60-64

	Quintiles of dietary patterns z-score					
	1	2	3	4	5	P‡
Men						
Age 36 (N=856)						
No. of cases	16	20	16	20	22	
OR (95% CI)						
Model 1*	1.00	1.52 (0.70, 3.29)	1.27 (0.56, 2.89)	1.39 (0.61, 3.14)	1.58 (0.65, 3.85)	0.44
Model 2†	1.00	1.46 (0.67, 3.18)	1.23 (0.53, 2.83)	1.36 (0.59, 3.11)	1.48 (0.60, 3.66)	0.51
Age 43 (N=1080)						
No. of cases	28	20	26	24	26	
OR (95% CI)						
Model 1*	1.00	0.71 (0.35, 1.42)	1.21 (0.63, 2.32)	1.01 (0.50, 2.04)	1.10 (0.53, 2.28)	0.54
Model 2†	1.00	0.68 (0.33, 1.40)	1.23 (0.62, 2.42)	1.01 (0.49, 2.09)	1.08 (0.51, 2.28)	0.55
Age 53 (N=669)						
No. of cases	11	12	12	18	18	
OR (95% CI)						
Model 1	1.00	1.01 (0.41, 2.47)	0.96 (0.39, 2.36)	1.44 (0.62, 3.36)	1.66 (0.67, 4.09)	0.17
Model 2†	1.00	0.94 (0.37, 2.35)	0.92 (0.37, 2.33)	1.29 (0.54, 3.06)	1.58 (0.62, 3.98)	0.22
Women						
Age 36 (N=948)						
No. of cases	11	16	14	18	13	
OR (95% CI)						
Model 1*	1.00	1.95 (0.81, 4.52)	2.02 (0.84, 4.84)	2.32 (0.99, 5.46)	2.01 (0.77, 5.27)	0.11
Model 2†	1.00	2.27 (0.93, 5.54)	2.33 (0.94, 5.78)	2.53 (1.05, 6.09)	2.26 (0.83, 6.10)	0.11
Age 43 (N=1187)						
No. of cases	8	11	23	20	29	
OR (95% CI)						
Model 1*	1.00	1.77 (0.62, 5.07)	3.78 (1.46, 9.79)	3.74 (1.42, 9.81)	5.45 (2.01, 14.79)	<0.001
Model 2†	1.00	1.77 (0.61, 5.14)	3.56 (1.36, 9.35)	3.77 (1.41, 10.02)	4.95 (1.77, 13.84)	<0.01
Age 53 (N=809)						
No. of cases	5	9	10	17	19	
OR (95% CI)						
Model 1*	1.00	1.92 (0.62, 5.91)	1.74 (0.55, 5.43)	3.10 (1.05, 9.12)	3.22 (1.08, 9.54)	0.01
Model 2†	1.00	1.94 (0.59, 6.49)	1.64 (0.49, 5.49)	2.82 (0.89, 8.97)	2.83 (0.88, 9.09)	0.05

*Model 1: Adjusted for socioeconomic position, education, energy intake, energy underreporting, smoking, physical activity, medication

†Model 2: As Model 1 + adjusted for BMI and waist circumference; ‡p for trend across quintiles of z-score

Table 4. **Associations between changes in dietary pattern z-score through the adult life course and type 2 diabetes between age 53 and 60-64, stratified by BMI**

		Men (N=524)		Women (N=655)	
	N	OR (95% CI)	P value	OR (95% CI)	P value
Dietary pattern z-score change					
Multivariate adjusted*					
All	524			655	
36 to 43 y		1.09 (0.75, 1.57)	0.63	1.63 (1.08, 2.46)	0.01
43 to 53 y		1.14 (0.80, 1.63)	0.44	1.45 (0.98, 2.15)	0.05
36 to 53 y		1.19 (0.84, 1.68)	0.30	1.65 (1.12, 2.42)	0.01
Baseline BMI <25	317			522	
36 to 43 y		0.84 (0.56, 1.52)	0.56	1.53 (0.91, 2.56)	0.10
43 to 53 y		0.73 (0.35, 1.53)	0.41	1.43 (0.80, 2,57)	0.22
36 to 53 y		1.32 (0.73, 2.38)	0.93	1.48 (0.94, 2.32)	0.08
Baseline BMI ≥25	208			133	
36 to 43 y		1.09 (0.67, 1.79)	0.70	2.53 (0.95, 6.74)	0.06
43 to 53 y		1.32 (0.87, 2.01)	0.18	1.73 (0.94, 3.18)	0.07
36 to 53 y		1.11 (0.71, 1.76)	0.62	2.90 (1.04, 8.11)	0.04

*Adjusted for socioeconomic position, education, energy intake, energy underreporting, smoking, physical activity, conditional BMI change and conditional waist circumference change.

OR of type 2 diabetes for a 1 SD increase in dietary patterns z-score in each interval conditional on previous dietary pattern z-score

Legends for figures

Figure 1. Factor loadings for the high-fat, high-GI, low-fibre dietary pattern in the NSHD used in confirmatory dietary pattern analyses

Figure 2. Mean change in dietary pattern z-score across the adult life course (36-53 years) by type 2 diabetes diagnosis (diagnosed between age 53 to 60-64 years) and sex. Student t-test was used to test for differences in z-score changes; age 36-43: P=0.50 for men and <0.01 for women; age 43-53: P=0.39 for men and <0.01 for women; age 36-53: P=0.29 for men and <0.001 for women.