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Relationship of glycaemic index with cardiovascular risk factors: analysis of the National Diet and Nutrition Survey for people aged 65 and older

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Abstract

Objective: To identify associations between dietary glycaemic index (GI) and weight, body mass index and other risk factors for cardiovascular disease (CVD) – waist-to-hip ratio (WHR), lipoprotein fractions, triacylglycerols (TAG) and blood pressure (BP) – in an older British population.

Design: Cross-sectional dietary, anthropometric and biochemical data from the National Diet and Nutritional Survey for adults aged over 65 years were reanalysed using a hierarchical regression model. Associations between body weight, CVD risk factors, and dietary factors including GI and fibre intakes were explored among 1152 healthy older people living in the UK between 1994 and 1995.

Results: In the unadjusted model, GI was significantly and directly associated with TAG ($\beta = 0.008 \pm 0.003$) and diastolic BP ($\beta = 0.325 \pm 0.164$) in males. These relationships were attenuated and non-significant after adjustment for potential confounding factors. WHR ($\beta = 0.003 \pm 0.001$) and TAG ($\beta = 0.005 \pm 0.002$) were significantly predicted by GI in males and females combined. The association with WHR was attenuated by adjustment for sex, age, region and social class; the relationship with TAG was non-significant after adjustment for other potential dietary confounders.

Conclusion: After controlling for potential confounders, no clear links were detected between GI and body weight or other CVD risk factors. This study provides little evidence for advising the consumption of a low-GI diet in the elderly to prevent weight gain or improve other CVD risk factors.

Keywords
Glycaemic index
Dietary fibre
Elderly
Weight
Cardiovascular risk factors
Cross-sectional survey

The 2003 World Health Report attributes 29.2% of global deaths to cardiovascular disease (CVD)[1]. Overall, CVD is estimated to cost the UK close to £26 billion per year: 56% from direct health-care costs, 24% from reduced productivity and 16% as a result of informal care[2]. Advancing age is the principal non-modifiable risk factor for this disease[3] and, given that the UK population is ageing[4], research efforts must focus on primary prevention to tackle the growing public health burden. High blood pressure (BP), smoking and high total cholesterol (TC) levels are the principal modifiable risk factors for CVD mortality[5], although risk is not limited to these factors.

A wealth of evidence is available describing the CVD risk attributable to adiposity (estimated, for example, using body mass index (BMI; $kg\,m^{-2}$)[6] or waist-to-hip ratio (WHR)[7]); serum TC, low-density lipoprotein cholesterol (LDL-C)[8] and high-density lipoprotein cholesterol (HDL-C)[9]; serum triacylglycerols (TAG)[10]; and dietary fats[11,12]. Less is known about the potential role of dietary carbohydrate[13].

The glycaemic index (GI) ranks carbohydrate-containing foods based on the increase in blood glucose following their consumption relative to that following consumption of an equi-carbohydrate portion of a reference food (white bread or glucose)[14]. This index is therefore more informative than measuring the quantity of carbohydrate consumed alone. The exaggerated glucose and insulin responses that occur when consuming a high-GI diet could increase the risk of CVD via a number of mechanisms. These include oxidative stress as a result of

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postprandial hyperglycaemia and independent effects of hyperinsulinaemia on lipid profile, blood pressure, coagulation factors, inflammatory mediators and endothelial function[15].

Table 1[16–29] summarises previous research investigating the relationship between GI and CVD risk factors. Two publications have reviewed the efficacy of low-GI diets for weight loss; however their conclusions differ, highlighting the controversy surrounding low-GI diets. Raben[18] concluded that there was no evidence to support the use of low-GI diets for long-term weight control. Of the 20 longer-term studies reviewed (duration ranging from 6 days to 6 months) mean weight loss was 1.5 kg on the low-GI diets compared with 1.6 kg (not significant, NS) on the high-GI diets. When including only the nine studies that were well-controlled for energy, macronutrient and fibre intake, the mean weight loss was greater on the low-GI diets (-3.0 ± 1.8 vs. -2.8 ± 1.1 kg, NS). This latter finding echoes those reported by Brand-Miller *et al.*[9]. Four medium-term (duration range 5–18 weeks) human intervention studies were examined, with two demonstrating greater weight or fat loss on low- vs. high-GI diets and two showing no significant difference. Findings from animal models that support a beneficial role of low-GI diets for weight loss were also included. For example, rats fed a diet based on high-GI starch, but otherwise matched for energy and macronutrient intakes, had increased visceral fat stores and reduced rates of lipolysis[30]. Brand-Miller *et al.*[19] suggested that animal studies provide mechanistic evidence and contradict the assumption that all calories are equal in terms of effect on weight gain. Suggestive evidence is available from well-controlled, sufficiently powered studies to suggest that decreased dietary GI is associated with a reduced risk from CVD; however, as reasoned in both review articles, longer-term randomised controlled trials (RCTs) are necessary to elucidate this relationship in different population groups and to investigate the effects of conventional energy-restricted vs. *ad libitum* low-GI diets on weight loss for example.

A 10% reduction in LDL-C ($P < 0.05$)[21], elevations in HDL-C (0.08–0.25 mmol l^{-1} greater in the lowest quintile, $P < 0.05$)[23,26] and reductions in TAG (-37.2 vs. -19.1%, $P = 0.005$)[22] have been observed with low- vs. high-GI diets. Two meta-analyses (of 15 and 16 RCTs, respectively) examining the effects of low-GI diets on blood lipid levels found evidence for a beneficial effect of low-GI diets on TC and limited evidence for a reduction in LDL-C in subjects with type 2 diabetes. No benefit was observed for either HDL-C or TAG[16,17]. However, many of the studies reviewed were methodologically flawed, under-powered or of short duration.

No standard definitions exist to quantify 'low'- and 'high'-GI diets, although a difference of at least 10–15 units is observed in most publications[17]. Between-study differences in these definitions may contribute to the inconsistent results of clinical trials to date. Many low-GI foods (e.g. granary bread, pasta, beans and pulses) are high in fibre and inadequate control for differences in fibre intake – a potentially cardioprotective factor[31,32] – may confound studies of dietary GI and cardiovascular health[33]. The glycaemic load (GL), calculated as total amount of daily carbohydrate (g) × GI, is a measure of the overall postprandial glycaemic effect of a food or meal. High-GL diets have also been associated with an increased risk of coronary heart disease (CHD)[24] and detrimental effects on CVD risk factors[29]. In the current study the effect of GI alone is examined. Relationships between other carbohydrate-related dietary variables (fibre, GL, carbohydrate intake) and CVD risk factors are currently being analysed for this cohort and will be the subject of future publications.

Evidence for an association between dietary GI and CVD risk in free-living settings is conflicting, and data are particularly limited in elderly populations. Here, data from the National Diet and Nutrition Survey of adults aged 65 years and over (referred to hereafter as NDNS) are examined to determine if low dietary GI is associated with lower body weight, BMI, WHR, BP and a more favourable serum cholesterol profile.

Subjects and design

The NDNS[34] is a comprehensive, cross-sectional survey which collected data on dietary habits and nutritional status. All individuals who agreed to participate completed a descriptive interview detailing general eating habits, medications, socio-economic and health status before they were invited to take part in further data collection. All subsequent interviews (e.g. 4-day weighed intake record, blood and urine collections) were optional. Only data collected from free-living, non-institutionalised participants are considered here; no subjects were following a weight-loss diet at the time of the survey.

NDNS methods are reported in detail elsewhere[34]. Briefly, anthropometric measurements were carried out at the participant's home, with their shoes removed, wearing light clothing. Height was measured to the nearest 1 mm using a portable, digital telescopic stadiometer; weight to the nearest 100 g using Soehnle Quantratronic digital scales. BMI was calculated as weight (kg) divided by the square of height (m^2)[35]. Duplicate waist and hip measurements were made using an insertion tape, and WHR was calculated from the mean of two readings. Three supine BP measurements were taken from the right arm at intervals of 1 min using an automated sphygmomanometer (DINAMAP; GE Health Systems). Blood samples were collected after an overnight fast and analysed for TC, HDL-C, LDL-C and TAG concentrations[34].

Participants were issued with Soehnle Quanta digital food scales and two 4-day dietary record sheets: one for use at home and a simplified form for use outside home.

Table 1 Summary of previous research investigating the relationship between GI and CVD risk factors

Study category	Study type	Author and date	Number of subjects	Subject characteristics	Outcome measure(s)	Intervention period	Difference in GI, mean (SD) or range	Results	Significance
Intervention	Meta-analysis	Opperman *et al.* (2004)[16]	396	Adults with T1DM (105)	Change in fructosamine, HbA1c, HDL-C, LDL-C, TC and TG conc	12 days to 6 months	24 (9) units	↓ in fructosamine in low-GI groups (-0.1 mmol l^{-1})	$P = 0.05$
				Adults with T2DM (228)			21 (7) units	↓ in HbA1c in low-GI groups (-0.27%)	$P = 0.03$
				Healthy adults (17)			22 (9) units	No difference in HDL-C conc (-0.03 mmol l^{-1})	$P = 0.23$
				Adults with CHD (46)			21 (10) units	↓ in LDL-C conc in low-GI groups (-0.15 mmol l^{-1})	$P = 0.06$
							22 (8) units	↓ in TC conc in low-GI groups (-0.33 mmol l^{-1})	$P < 0.001$
							20 (9) units	No difference in TG conc (0.03 mmol l^{-1})	$P = 0.73$
	Meta-analysis	Kelly *et al.* (2004)[17]	325	Adults with at least 1 major risk factor for CHD or diagnosed with CHD	Change in weight, fasting glucose, fasting insulin, TC, HDL-C, LDL-C, TG and HbA1c conc	4 weeks to 6 months	5 to 32 units	Pooled ↓ in TC of -0.17mmol l^{-1} for the low- vs. the high-GI group	$P = 0.03$
								No difference in change in HDL-C between diets	
								No difference in change in LDL-C between diets	
								No difference in change in TG conc between diets	
								No difference in change in body weight between diets	
								No difference in change in fasting glucose, fasting insulin or HbA1c between diets	
	Review – medium-term human studies	Raben (2002)[18]	538	Normal weight, overweight or obese children and adults	Change in weight	6 days to 6 month	Not provided	No difference in mean weight loss between low- and high-GI diets (-1.5 kg vs. -1.6 kg)	$P > 0.05$
	Review – medium-term human studies	Brand-Miller *et al.* (2002)[19]	170	Overweight or obese children and adults	Change in weight, WC, fat mass	5 weeks to 4 months	Not provided	3 of 4 studies observed a greater ↓ in weight, BMI or fat mass on low- vs. high-GI diets	$P < 0.05$
								1 of 4 showed no difference in weight loss between diets	$P > 0.05$
	Meta-analysis	Brand-Miller *et al.* (2003)[20]	356	Adults with T2DM	Change in HbA1c and fructosamine conc	12 days to 12 months	Not provided	Low-GI diets reduced HbA1c by 0.43% points more than high-GI diets	$P < 0.05$
								Low-GI diets reduced glycated proteins (HbA1c and fructosamine) by 7.4% more than high-GI diets	

Table 1. *Continued*

Study category	Study type	Author and date	Number of subjects	Subject characteristics	Outcome measure(s)	Intervention period	Difference in GI, mean (SD) or range	Results	Significance
	Parallel randomised study	Sloth *et al.* (2004)[21]	45	Healthy overweight females	Change in weight, fat mass, fasting insulin, insulin resistance, β- cell function, NEFA, TC, HDL-C, LDL-C and TG conc	10 weeks	17 units for test foods provided	No difference in change in weight or fat mass between diets	$P > 0.05$
								No difference in fasting insulin, insulin resistance, β-cell function, TG, NEFA or HDL-C conc	$P > 0.05$
								Greater ↓ in LDL-C on ow-GI diet	$P < 0.05$
								Greater ↓ in TC on low-GI diet	$P = 0.06$
	Parallel randomised study	Ebbeling *et al.* (2005)[22]	23	Healthy obese young adults	Weight, TG, PAI-1, TC, insulin sensitivity, BP	12 months	6.6 units at 12 months	No difference in change in weight between diets	$P > 0.05$
								No difference in change in TG, PAI-1, TC, insulin sensitivity or BP	$P > 0.05$
								Greater ↓ in TG concentration in low-GI group (−37.2% vs. −19.1%)	$P = 0.005$
								PAI-1 ↓ in intervention group (−39.0%) and ↑ in the conventional group (33.1%)	$P = 0.004$
Epidemiological	Cross-sectional study	Frost *et al.* (1999)[23]	1420	Healthy adults	TC, HDL-C and LDL-C conc	N/A	N/A	Inverse relationship between GI and HDL-C	$P = 0.02$ for males, $P < 0.0001$ for females
								No significant relationship between GI and TC or LDL-C	
	Prospective study, 10-year follow-up period	Liu *et al.* (2000)[24]	75 521	Healthy adult females	CHD incidence	N/A	N/A	Direct relationship between GL and CHD risk	$P < 0.0001$ for linear trend
	Prospective study, 10-year follow-up period and cross-sectional study	van Dam *et al.* (2000)[25]	646 (prospective); 394 (cross-sectional)	Elderly males	CHD incidence; metabolic risk factors	N/A	N/A	No relationship between GI and CHD risk	$P = 0.70$ for linear trend
								No relationship between GI and TC, HDL-C, TG, (fasting or post-load) glucose or insulin conc	
	Cross-sectional study	Ford & Liu (2001)[26]	13 907	Healthy adults	HDL-C conc	N/A	N/A	Inverse relationship between GI/GL and HDL-C	$P < 0.001$ for linear trend
								15-unit ↑ in GI associated with a ↓ in HDL-C of 0.06 mmol l^{-1}	

Table 1. *Continued*

Study category	Study type	Author and date	Number of subjects	Subject characteristics	Outcome measure(s)	Intervention period	Difference in GI, mean (SD) or range	Results	Significance
	1-year longitudinal analysis	Ma et al. (2005)[47]	572	Healthy adults	Change in BMI	N/A	N/A	Direct relationship between GI and BMI 5-unit ↑ in GI associated with ↑ of 0.75 BMI units from cross-sectional data and ↑ of 0.04 units from longitudinal data	Cross-sectional: $P = 0.01$ Longitudinal: $P = 0.02$
	Cross-sectional study	Liese *et al.* (2005)[28]	979	Healthy adults (66%), IGT adults (33%)	Insulin sensitivity, fasting insulin, acute insulin response, disposition index, BMI and WC	N/A	N/A	No relationship between GI and any outcome variable	$P > 0.05$
	Cross-sectional study	Slyper *et al.* (2005)[29]	32	Healthy subjects aged 11–25 years	TC, HDL-C and LDL-C	N/A	N/A	Inverse relationship between GL and HDL-C	$P < 0.05$

GI – glycaemic index; CVD – cardiovascular disease; SD – standard deviation; T1DM – type 1 diabetes mellitus; T2DM – type 2 diabetes mellitus; CHD – coronary heart disease; IGT – impaired glucose tolerance; HbA1c – glycated haemoglobin; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol; TC – total cholesterol; TG – triglycerides; conc – concentration(s); WC – waist circumference; NEFA – non-esterified fatty acids; PAI-1 – plasminogen-activator inhibitor-1; BP – blood pressure; BMI – body mass index; N/A – not applicable; ↓, decrease; ↑, increase; GL – glycaemic load.

A description/brand of all food and drink consumed at home was recorded alongside the weight of food served and any leftovers. For food consumed away from home – where weighing was inconvenient – participants recorded a description, price and place of purchase. Trained interviewers then purchased and weighed the items. The feasibility study revealed significant differences in nutrient intake between different days of the week, so a diary placement pattern (i.e. completing a set number or proportion of diaries on each day of the week) was used in the final survey to ensure that, for example, weekday vs. weekend variation in intakes was accounted for[36]. Details from both diaries were matched to nutrient composition information (55 nutrients) using a nutrient databank of over 3500 foods, compiled by the Ministry of Agriculture, Fisheries and Foods[37].

GI calculation

A GI value (reference food = glucose) was added to the NDNS database for each of the 2323 carbohydrate-containing food codes recorded in the diet diaries. Values were obtained from the international table of GI and GL values[38] and other published sources[39,40], using means of multiple entries when available. Where no published data were available, published analogues were used (for example, the mean GI value for 'oranges' was imputed for 'clementines') or GI values were calculated from recipes using the mixed-meal formula[41]:

$$\mathrm{GI_{food}} = \sum_{\text{ingredient}} \frac{\mathrm{GI_{ingredient}} \times \text{Quantity of carbohydrate in the ingredient (g)}}{\text{Total quantity of carbohydrate in the food (g)}}.$$

To calculate the average GI of each diet, the following formula was applied[42]:

$$\mathrm{GI_{diet}} = \sum_{\text{food}} \frac{\mathrm{GI_{food}} \times \text{Quantity of carbohydrate in the food (g)}}{\text{Total quantity of carbohydrate in the diet (g)}}.$$

Many of the foods for which GI values were estimated contributed only a small proportion of the total carbohydrate consumed by each individual. To assess the relative importance of GI values obtained from various sources, the mean percentage of an individual's total carbohydrate intake attributable to each source of GI data (international tables, other published data, estimations and calculations) was calculated.

Validation of dietary intake

Basal metabolic rate (BMR), calculated using standard regression equations based on age, sex and weight (Schofield equations)[43], was compared with energy intake to identify underreporting. A habitual energy intake estimate less than 1.2 × BMR, considered incompatible with energy balance[44], was used here to define low energy reporters (LERs). In the feasibility study, a 24-hour urine sample was taken and *p*-aminobenzoic acid tablets were given to participants to check the completeness of the sample. Protein intakes were validated by comparing urinary and dietary nitrogen (N) concentration: ratios of urinary N/dietary N were 0.92 for males and 0.87 for females, close to the permitted range for validity (0.70–0.90)[36].

Statistical analysis

To control for the potentially confounding effect of energy intake, all nutrient data are presented as energy-adjusted variables. HDL-C, TC/HDL-C ratio, TAG and Englyst fibre intake were log-transformed to normalise the distributions. The ratio of polyunsaturated to saturated fatty acid intake (P/S) was calculated and used in subsequent analyses to improve the stability of the regression model. Differences in means between LERs and non-LERs and between males and females were compared with unpaired *t*-tests for continuous variables and by χ^2 tests for categorical variables.

To inform regression analyses and identify potential collinearity, a Spearman correlation matrix, controlled for energy intake (males and females separately and combined), was constructed for nutrient and physical activity level (PAL) data.

Associations of GI with CVD risk factors (weight, $n = 993$; BMI, $n = 983$; WHR, $n = 993$; BP, $n = 960$; lipoprotein sub-fractions, $n = 784$; TAG, $n = 1100$) were analysed using multiple linear regression. The basic model for all dependent variables was adjusted for age, region and social class (non-manual or manual, classified by main occupation prior to retirement). Analyses were carried out separately for males and females and combined (with adjustment for sex in combined analysis).

Evidence-based confounding factors were added sequentially in additional models for each dependent variable (e.g. BMI when BP was the dependent)[45]. In subsequent models, reported potential confounders were also added (e.g. P/S ratio when BP was the dependent)[46]. To determine if the amount of fibre consumed affected the relationship of dietary GI to the dependent variables, an interaction term (GI × Englyst fibre) was included in additional regression models.

All analyses were performed with SPSS (version 12.0; SPSS Inc.). Analyses were carried out on the full sample and repeated with the exclusion of LERs. *P*-values < 0.05 (two-tailed) were considered statistically significant.

Results

Study population

Of the free-living sample ($n = 2172$ individuals), 75% ($n = 1632$) provided a full or partial interview (responding sample) and 78% ($n = 1275$) of the responding sample also provided a 4-day weighed dietary intake record. Data collected from those who reported being unwell during the dietary recording period, with appetite/eating patterns affected (8% males, $n = 50$; 11% females, $n = 73$), were excluded. This resulted in a maximum study population of 1152 individuals (53% of the initial sample), of which 50.5% were male (age 75.9 ± 7.0 years, BMI 26.3 ± 3.6 kg m^{-2}; mean ± standard deviation, SD) and 49.5% were female (age 77.6 ± 8.0 years, BMI 26.6 ± 4.8 kg m^{-2}; mean ± SD). Selected demographic, lifestyle, anthropometric, biochemical and nutrient characteristics are presented by sex in Table 2. Some 49.2% ($n = 478$) of individuals (39.7% males, 59.4% females; $P < 0.001$) were defined as LERs. The exclusion of these participants did not materially affect the outcomes, and thus only the analyses performed on the full dataset are presented.

Source of GI values

GI values were assigned to 2323 individual food codes, contributing 73% of the total weight of carbohydrate consumed by participants. Of these, 235 (10%) were assigned a mean value from multiple entries in the international tables, and 358 (15%) were obtained from a single entry in the international tables[38]. Values for 1580 (68%) codes were estimated from analogous foods with published GI values, while 96 (4%) were calculated by the mixed-meal method[42]. The 54 remaining GI values were sourced from other published data[39,40]. On average, GI values assigned from a mean value from the international tables contributed 43.5 ± 14.2% (mean ± SD) of an individual's total carbohydrate intake; values obtained from a single entry 24.7 ± 13.0%; analogues 26.3 ± 11.7%; and mixed-meal calculated GI values 1.8 ± 3.3%. The remaining GI values, obtained from other published sources[39,40], contributed on average 3.5 ± 3.4% of total carbohydrate intake.

Correlation analyses

Correlation matrices for males and females are presented in Tables 3 and 4, respectively. Dietary GI did not correlate significantly with weight, BMI, WHR, BP or any lipoprotein fraction in either sex. Dietary GI was inversely associated with Englyst fibre intakes in males and females ($r = -0.34$ and $r = -0.32$, respectively).

Regression analyses

Associations of dietary GI with weight, BMI, WHR and other CVD risk factors, with and without control for potential confounders, are reported for males and females in Tables 5 and 6, respectively.

In males, prior to controlling for potential confounders, dietary GI was directly associated with 10g TAG concentration ($\beta = 0.008 \pm 0.003$). There was also a direct relationship between GI and diastolic BP ($\beta = 0.325 \pm 0.164$). However, after adjusting for age, social class and region (Model 2), neither association was statistically significant. In females, dietary GI was not significantly associated with any of the anthropometric variables or CVD risk factors assessed, either before or after adjustment for confounders.

When the analyses were repeated in males and females combined (data not shown), WHR was significantly predicted by dietary GI ($\beta = 0.003 \pm 0.001$; $P < 0.001$) in Model 1. However, after controlling for sex, age, social class and region, the relationship was no longer significant. Subsequent models further reduced the strength of the relationship. Dietary GI predicted (log) TAG concentration ($\beta = 0.005 \pm 0.002$; $P = 0.017$); the association remained significant after control for sex, age, social class and region (Model 2), and PAL and BMI (Model 3) ($\beta = 0.004 \pm 0.002$; $P = 0.049$). Adjusting for alcohol (units week^{-1}) (Model 4), carbohydrate (g day^{-1}) (Model 5) and Englyst fibre (g day^{-1}) (Model 6) weakened the association ($\beta = 0.02 \pm 0.02$; $P = 0.362$), which became non-significant.

When the interactions between fibre and GI were considered, it was apparent that in females the non-significant relationships between dietary GI and weight and between dietary GI and BMI were dependent on fibre intake. The addition of a GI × fibre interaction term to the regression model revealed significant inverse associations between GI and weight ($\beta = -0.995 \pm 0.360$; $P = 0.008$) and GI and BMI ($\beta = -0.302 \pm 0.145$; $P = 0.038$). Significant and borderline-significant interactions were detected between fibre and GI in males for HDL-C, TC/HDL-C ratio and TAG, indicating that the observed relationships (non-significant) of dietary GI and these outcome measures are also dependent on fibre intake.

Discussion

Carbohydrate is absorbed rapidly from high-GI (relative to low-GI) foods, causing higher postprandial glucose and insulin responses[47]. This encourages carbohydrate rather than fat oxidation, stimulates lipolysis and increases visceral non-esterified fatty acid release in the late postprandial period, promoting body fat gain. Low-GI carbohydrates have been associated with greater satiety and hence reductions in subsequent energy intake[48]. Thus, we hypothesised that low-GI diets would be associated with lower body weight, BMI, WHR, BP and a more favourable cholesterol profile in a free-living population. Our study found a high-GI diet to be associated with higher TAG concentrations and higher

Table 2 Demographic, lifestyle, anthropometric, biochemical and dietary intake data by sex for all participants (all significant differences are highlighted)

	Males	Females
Demographics		
Region		
Scotland and Northern	34.4	34.9
Central and South West	42.1	37.5
London and South East	23.5	27.5
Social class		
Non-manual	45.1	46.6
Manual	54.9	53.4
Age (years)	75.9 ± 7.0*	77.6 ± 8.0
Lifestyle		
Smoking status		
Non (never & ex)	82.9	87.5
< 20 cigarettes day^{-1}	11.0	10.2
≥ 20 cigarettes day^{-1}	6.0	2.3
Self-reported alcohol intake level		
Non-drinker	7.2	25.3
Ex-drinker	10.8	8.2
Very low	11.5	20.5
Low	35.5	31.1
Moderate	16.2	6.8
Fairly high	8.3	4.0
High	4.5	0.9
Very high	2.6	0.4
Drinker, units unknown	3.4	2.8
Estimated alcohol intake (units week^{-1})	10.5 ± 15.0**	3.0 ± 5.0
Maximum physical activity level		
Vigorous (e.g. running or jogging)	0.5	0.4
Moderate (e.g. cycling, keep fit, swimming, $\geq$20 min brisk walking)	79.7	85.4
Light (e.g. dancing, golf, yoga, $\geq$20 min slow walking)	18.4	13.5
Inactive	1.4	0.7
BMR	1521.5 ± 157.1**	1263.6 ± 121.4
EI/BMR ratio	1.26 ± 0.30**	1.13 ± 0.27
Anthropometry		
Height (cm)	169.0 ± 6.9**	155.4 ± 6.7
Weight (kg)	75.1 ± 12.0**	64.1 ± 12.4
BMI (kg m^{-2})	26.3 ± 3.6	26.6 ± 4.8
WHR	0.93 ± 0.06**	0.84 ± 0.07
Blood pressure		
Systolic (mmHg)	150.1 ± 21.9*	154.5 ± 23.8
Diastolic (mmHg)	79.7 ± 12.7*	76.7 ± 13.5
Biochemical		
TC (mmol l^{-1})	5.5 ± 1.1**	6.3 ± 1.5
TAG (mmol l^{-1})	1.6 ± 0.9	1.6 ± 0.8
HDL-C (mmol l^{-1})	1.2 ± 0.5**	1.4 ± 0.5
LDL-C (mmol l^{-1})	4.3 ± 1.2**	4.9 ± 1.5
Dietary intake		
Energy intake (kcal)	1891.2 ± 454.8**	1415.3 ± 338.0
CHO (% of food energy)	49.1 ± 7.0	49.4 ± 6.3
Starch (% of food energy)	27.2 ± 5.8	27.3 ± 5.3
Sugar (% of food energy)	22.0 ± 7.1	22.0 ± 6.8
Englyst fibre (g per 1000 kcal)	7.1 ± 2.8**	7.7 ± 3.1
Protein (% of food energy)	15.1 ± 3.0**	15.9 ± 3.6
Fat (% of food energy)	35.0 ± 5.7**	36.7 ± 6.2
SFA (% of food energy)	14.5 ± 3.6**	15.8 ± 4.1
MUFA (% of food energy)	10.8 ± 2.0	11.0 ± 2.1
n−3 PUFA (% of food energy)	0.77 ± 0.36*	0.82 ± 0.36
n−6 PUFA (% of food energy)	4.8 ± 2.1	4.8 ± 2.2
P/S ratio	0.42 ± 0.22	0.40 ± 0.24
TFA (% of food energy)	1.5 ± 0.5**	1.6 ± 0.5
GI	59.4 ± 3.6**	58.2 ± 3.8

BMR – basal metabolic rate; EI – energy intake; BMI – body mass index; WHR – waist-to-hip ratio; TC – total cholesterol; TAG – triacylglycerols; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol; CHO – carbohydrate; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids; P/S ratio – polyunsaturated fat/saturated fat; TFA – *trans* fatty acids; GI – glycaemic index.
Data are presented as % (categorical variables) or mean ± standard deviation (continuous variables).
Significant difference between males and females (unpaired Student's *t*-test for continuous variables, χ^2 test for categorical variables): *$P < 0.05$, ** $P < 0.01$.

Table 3 Correlation matrix of nutrient variables for males controlled for energy intake (kcal)

	CHO (g)	Sugar (g)	Starch (g)	Englyst fibre (g)	Protein (g)	Fat (g)	SFA (g)	MUFA (g)	*n*–3 PUFA (g)	*n*–6 PUFA (g)	P/S ratio	TFA (g)	Chol (mg)	EtOH (units week^{-1})	PAL
GI	0.01	−0.16**	0.20**	−0.34**	−0.17**	−0.04	−0.06	−0.02	−0.02	0.04	0.07	0.00	0.00	0.06	0.04
CHO (g)		0.67**	0.43**	0.26**	−0.16**	−0.45**	−0.34**	−0.48**	−0.18**	−0.03	0.09*	−0.04	−0.34**	−0.46	0.03
Sugar (g)			−0.38**	−0.05	−0.35**	−0.42**	−0.18**	−0.44**	−0.26**	−0.24**	−0.12**	−0.09*	−0.20**	−0.19**	0.04
Starch (g)				0.39**	0.22**	−0.05	−0.22**	−0.05	0.08*	0.26**	0.27**	0.06	−0.19**	−0.34**	−0.01
Englyst fibre (g)					0.35**	−0.15**	−0.24**	−0.16**	0.13**	0.19**	0.25**	−0.17**	−0.18**	−0.17**	−0.11**
Protein (g)						0.02	−0.07	0.06	0.27**	0.06	0.08*	−0.12**	0.25**	−0.09*	−0.06
Fat (g)							0.78**	0.86**	0.19**	0.24**	−0.12**	0.52**	0.33**	−0.27**	0.07
SFA (g)								0.53**	−0.09*	−0.33**	−0.63**	0.48**	0.34**	−0.21**	0.07
MUFA (g)									0.28**	0.18**	−0.07	0.40**	0.33**	−0.15**	0.06
n–3 PUFA (g)										0.16**	0.26**	−0.04	0.03	−0.02	−0.06
n–6 PUFA (g)											0.85**	−0.08*	−0.17**	−0.14**	−0.02
P/S ratio												−0.29**	−0.23**	−0.01	−0.06
TFA (g)													0.15**	−0.31**	0.03
Chol (mg)														0.00	0.09*
EtOH (units week^{-1})															−0.02

CHO – carbohydrate; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids; P/S ratio – polyunsaturated fat/saturated fat; TFA – *trans* fatty acids; Chol – cholesterol; EtOH – alcohol; PAL – physical activity level; GI – glycaemic index.
Significant correlation: *$P < 0.05$, **$P < 0.01$.

Table 4 Correlation matrix of nutrient variables for females controlled for energy intake (kcal)

	CHO (g)	Sugar (g)	Starch (g)	Englyst fibre (g)	Protein (g)	Fat (g)	SFA (g)	MUFA (g)	*n* − 3 PUFA (g)	*n* − 6 PUFA (g)	P/S ratio	TFA (g)	Chol (mg)	EtOH (units week^{-1})	PAL
GI	− 0.01	− 0.32**	0.39**	− 0.32**	− 0.18**	0.11*	0.00	0.17**	0.05	0.10*	0.09*	0.09*	0.04	− 0.05	0.01
CHO (g)		0.67**	0.34**	0.22**	− 0.24**	− 0.77**	− 0.53**	− 0.68**	− 0.23**	− 0.16**	0.07	− 0.25**	− 0.52**	− 0.18**	0.06
Sugar (g)			− 0.47**	0.07	− 0.22**	− 0.51**	− 0.20**	− 0.52**	− 0.25**	− 0.29**	− 0.14**	− 0.23**	− 0.24**	− 0.05	0.06
Starch (g)				0.16**	0.00	− 0.26**	− 0.37**	− 0.15**	0.04	0.17**	0.26**	0.00	− 0.31**	− 0.15**	0.00
Englyst fibre (g)					0.36**	− 0.38**	− 0.35**	− 0.36**	0.11*	0.09*	0.27**	− 0.28**	− 0.22**	− 0.03	− 0.06
Protein (g)						− 0.28**	− 0.30**	− 0.17**	0.15**	0.04	0.21**	− 0.32**	0.22**	0.04	− 0.10*
Fat (g)							0.76**	0.84**	0.14**	0.15**	− 0.20**	0.50**	0.40**	− 0.16**	0.01
SFA (g)								0.44**	− 0.16**	− 0.43**	− 0.68**	0.43**	0.34**	− 0.13**	0.05
MUFA (g)									0.27**	0.17**	− 0.08	0.42**	0.37**	− 0.11*	− 0.03
n − 3 PUFA (g)										0.23**	0.31**	− 0.10*	0.12*	0.06	− 0.09*
n − 6 PUFA (g)											0.84**	− 0.15**	− 0.11*	− 0.02	− 0.04
P/S ratio												− 0.31**	− 0.17**	0.05	− 0.06
TFA (g)													0.12**	− 0.14**	0.03
Chol (mg)														0.00	0.02
EtOH (units week^{-1})															0.08

CHO – carbohydrate; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids; P/S ratio – polyunsaturated fat/saturated fat; TFA – *trans* fatty acids; Chol – cholesterol; EtOH – alcohol; PAL – physical activity level; GI – glycaemic index.
Significant correlation: *$P < 0.05$, **$P < 0.01$.

Table 5 Results of the linear regression analysis of GI and CVD risk factors in males

	Model 1†		Model 2‡		Model 3		Model 4		Model 5		Model 6	
	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P
Weight§	0.004 ± 0.151	0.98	−0.085 ± 0.149	0.57	−0.077 ± 0.149	0.60	−0.084 ± 0.149	0.58	−0.054 ± 0.157	0.73		
BMI§	0.008 ± 0.046	0.86	−0.030 ± 0.046	0.52	−0.031 ± 0.046	0.51	−0.032 ± 0.047	0.49	−0.005 ± 0.049	0.92		
WHR§	0.001 ± 0.001	0.09	0.001 ± 0.001	0.29	0.001 ± 0.001	0.33	0.001 ± 0.001	0.31	0.001 ± 0.001	0.30		
TC¶	−0.003 ± 0.016	0.84	−0.010 ± 0.017	0.55	−0.005 ± 0.017	0.75	−0.005 ± 0.017	0.75	0.000 ± 0.017	0.99	−0.008 ± 0.017	0.63
LDL-C¶	0.000 ± 0.016	0.99	−0.008 ± 0.017	0.63	−0.005 ± 0.017	0.78	−0.005 ± 0.017	0.75	−0.002 ± 0.017	0.88	−0.009 ± 0.018	060
+ HDL-C‖	−0.003 ± 0.002	0.21	−0.002 ± 0.002	0.36	−0.001 ± 0.02	0.52	−0.002 ± 0.002	0.35	−0.001 ± 0.002	0.49	−0.001 ± 0.002	0.57
+ TC/HDL-C ratio‖	0.002 ± 0.002	0.44	<0.001 ± 0.002	0.90	<0.001 ± 0.002	0.99	0.001 ± 0.002	0.81	<0.001 ± 0.002	0.91	0.001 ± 0.002	0.91
+ TAG††	0.008 ± 0.003	0.02*	0.006 ± 0.003	0.07	0.005 ± 0.003	0.10	0.005 ± 0.003	0.12	0.005 ± 0.003	0.13	0.003 ± 0.003	0.35
SBP‡‡	0.409 ± 0.278	0.14	0.246 ± 0.287	0.39	0.261 ± 0.288	0.37	0.219 ± 0.288	0.45	0.184 ± 0.290	0.52	0.073 ± 0.306	0.81
DBP‡‡	0.325 ± 0.164	0.05*	0.164 ± 0.167	0.33	0.180 ± 0.167	0.28	0.159 ± 0.167	0.34	0.148 ± 0.168	0.38	0.026 ± 0.177	0.88

GI – glycaemic index; CVD – cardiovascular disease; SE – standard error; BMI – body mass index; WHR – waist-to-hip ratio; TC – total cholesterol; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; TAG – triacylglycerols; SBP – systolic blood pressure; DBP – diastolic blood pressure.

+ indicates dependent variables that were log-transformed to normalise before use in regression analysis.

* Significant at $P < 0.05$.

† Model 1 shows unadjusted influence of GI on dependent variable.

‡ Model 2, for all variables, is adjusted for age, social class and region.

§ For weight, BMI and WHR, Model 3 is adjusted for physical activity and energy intake; fat (g day^{-1}) is added to Model 4; and Englyst fibre (g day^{-1}) to Model 5.

¶ For TC and LDL-C, Model 3 is adjusted for physical activity, energy intake and BMI; Model 4 is additionally controlled for fat intake (g day^{-1}); smoking, polyunsaturated fat/saturated fat ratio, cholesterol intake (mg day^{-1}) and *trans* fatty acids (g day^{-1}) are added in Model 5; and Engylst fibre (g day^{-1}) in Model 6.

‖ HDL-C and TC/HDL-C ratio are adjusted for physical activity, energy intake and BMI in Model 3; fat intake (g day^{-1}), polyunsaturated fat/saturated fat ratio and alcohol intake (units week^{-1}) in Model 4; and additionally for smoking, cholesterol intake (mg day^{-1}) and monounsaturated fat intake (g day^{-1}) in Model 5 and Englyst fibre (g day^{-1}) in Model 6.

†† TAG is adjusted for physical activity, energy intake and BMI in Model 3, as well as alcohol intake (units week^{-1}) in Model 4, carbohydrate intake (g day^{-1}) in Model 5 and Englyst fibre (g day^{-1}) in Model 6.

‡‡ For SBP and DBP, Model 3 is adjusted for physical activity, energy intake and BMI; in Model 4 alcohol intake (units week^{-1}) is added; Model 5 includes smoking status, polyunsaturated fat/saturated fat ratio and cholesterol intake (mg day^{-1}); and Englyst fibre (g day^{-1}) was added in Model 6.

Table 6 Results of the linear regression analysis of GI and CVD risk factors in females

	Model 1†		Model 2‡		Model 3		Model 4		Model 5		Model 6	
	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P	$B \pm$ SE	P
Weight§	−0.061 ± 0.156	0.70	−0.049 ± 0.156	0.75	−0.086 ± 0.157	0.58	−0.084 ± 0.157	0.60	−0.089 ± 0.167	0.60		
BMI§	0.028 ± 0.061	0.65	0.020 ± 0.063	0.75	0.013 ± 0.063	0.84	0.009 ± 0.064	0.89	−0.005 ± 0.068	0.94		
WHR§	0.001 ± 0.001	0.33	0.001 ± 0.001	0.42	0.001 ± 0.001	0.45	0.001 ± 0.001	0.46	0.000 ± 0.001	0.94		
TC¶	0.009 ± 0.022	0.68	0.009 ± 0.023	0.71	0.009 ± 0.023	0.71	0.007 ± 0.023	0.76	0.006 ± 0.024	0.80	0.009 ± 0.026	0.73
LDL-C¶	0.013 ± 0.022	0.56	0.011 ± 0.023	0.63	0.010 ± 0.023	0.65	0.009 ± 0.023	0.71	0.007 ± 0.024	0.78	0.009 ± 0.026	0.73
+ HDL-C‖	<0.001 ± 0.002	0.92	0.001 ± 0.002	0.76	0.001 ± 0.002	0.68	0.001 ± 0.002	0.62	0.001 ± 0.002	0.63	0.002 ± 0.002	0.42
+ TC/HDL-C ratio‖	<0.001 ± 0.002	0.87	−0.001 ± 0.002	0.82	−0.001 ± 0.002	0.76	−0.001 ± 0.003	0.66	−0.001 ± 0.003	0.74	−0.001 ± 0.003	0.59
+ TAG††	0.004 ± 0.003	0.10	0.003 ± 0.003	0.36	0.002 ± 0.003	0.47	0.002 ± 0.003	0.47	0.002 ± 0.003	0.47	0.001 ± 0.003	0.86
SBP‡‡	0.210 ± 0.322	0.52	0.110 ± 0.329	0.74	0.165 ± 0.329	0.62	0.159 ± 0.329	0.63	0.209 ± 0.332	0.53	0.159 ± 0.361	0.66
DBP‡‡	−0.075 ± 0.183	0.68	−0.116 ± 0.188	0.54	−0.126 ± 0.189	0.51	−0.126 ± 0.190	0.51	−0.083 ± 0.190	0.66	−0.122 ± 0.207	0.56

GI – glycaemic index; CVD – cardiovascular disease; SE – standard error; BMI – body mass index; WHR – waist-to-hip ratio; TC – total cholesterol; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; TAG – triacylglycerols; SBP – systolic blood pressure; DBP – diastolic blood pressure.

+ indicates dependent variables that were log-transformed to normalise before use in regression analysis.

† Model 1 shows unadjusted influence of GI on dependent variable.

‡ Model 2, for all variables, is adjusted for age, social class and region.

§ For weight, BMI and WHR, Model 3 is adjusted for physical activity and energy intake; fat (g day^{-1}) is added to Model 4; and Englyst fibre (g day^{-1}) to Model 5.

¶ For TC and LDL-C, Model 3 is adjusted for physical activity, energy intake and BMI; Model 4 is additionally controlled for fat intake (g day^{-1}); smoking, polyunsaturated fat/saturated fat ratio, cholesterol intake (mg day^{-1}) and *trans* fatty acids (g day^{-1}) are added in Model 5; and Engylst fibre (g day^{-1}) in Model 6.

‖ HDL-C and TC/HDL-C ratio are adjusted for physical activity, energy intake and BMI in Model 3; fat intake (g day^{-1}), polyunsaturated fat/saturated fat ratio and alcohol intake (units week^{-1}) in Model 4; and additionally for smoking, cholesterol intake (mg day^{-1}) and monounsaturated fat intake (g day^{-1}) in Model 5 and Englyst fibre (g day^{-1}) in Model 6.

†† TAG is adjusted for physical activity, energy intake and BMI in Model 3, as well as alcohol intake (units week^{-1}) in Model 4, carbohydrate intake (g day^{-1}) in Model 5 and Englyst fibre (g day^{-1}) in Model 6.

‡‡ For SBP and DBP, Model 3 is adjusted for physical activity, energy intake and BMI; in Model 4 alcohol intake (units week^{-1}) is added; Model 5 includes smoking status, polyunsaturated fat/saturated fat ratio and cholesterol intake (mg day^{-1}); and Englyst fibre (g day^{-1}) was added in Model 6.

diastolic BP in males. In the combined analysis of both sexes the association with TAG persisted and a direct relationship between GI and WHR was identified. These relationships were attenuated by increasing levels of adjustment for potential confounding factors, and became statistically non-significant. No significant relationships were observed for weight, BMI or lipoprotein concentrations.

Previous intervention studies investigating the efficacy of high- vs. low-GI diets for weight loss report conflicting findings[18,19]. Liese *et al.* studied 979 normal (67%) and 'glucose tolerance impaired' (33%) adults and found no association between GI (as assessed by food-frequency questionnaire, FFQ) and BMI or waist circumference[28]. This lack of association was attributed to the sample size and the exclusion of people with pre-existing CVD or diabetes, thus resulting in a cohort less susceptible to environmental exposure[13]. In contrast, Ma *et al.* found a direct association between GI (7-day dietary recall) and BMI in a longitudinal study of 572 healthy adults[27]. Other studies have found significant, direct relationships between GI and weight, yet these tend to include obese individuals or younger adults[49,50]. Fat mass increases and lean mass decreases with age, independent of body weight change. This change comprises increases in visceral adipose tissue, intramyocellular and intramuscular lipid and liver fat, and decreases in lean soft tissue and in fat-free mass[51]. These changes may render an elderly population less responsive to dietary changes and comparisons of studies with different population ages invalid.

Ingestion of high-GI carbohydrates results in reduced insulin sensitivity, and potentially a reduction in HDL-C and reciprocal increase in TAG concentration[52]. Low-GI diets improve insulin sensitivity[53], therefore higher HDL-C and lower TAG concentrations might be expected in individuals following such diets. Unlike previous population studies[23,26], we failed to observe an association between dietary GI and HDL-C. Our results are consistent with those from the Zutphen Elderly Study[25], which examined the association between high-GI diets and hyperinsulinaemia, hyperglycaemia, dyslipidaemia and CHD risk in 394 males (64–84 years). The lack of association was attributed to the exclusion of males with a history of diabetes or myocardial infarction[13]; however, no such exclusions were applied here. The disparity in age may explain these differences, as TC and LDL-C levels have been shown to increase[54], while fractional clearance of LDL-C[55] and the number of LDL receptors in hepatic tissue decline[56], with age.

The NDNS had a low response rate (~50%), resulting in a sample size half that estimated to observe significant associations, and one potentially biased by differences in diet, lifestyle and CVD risk factors between responders and non-responders. A high proportion of LERs (40% males, 60% females) was identified and insufficient information is available to determine whether under- or misreporting was evident, or if reported low intakes were valid. Under-reporting may not affect foods or nutrients systematically[57,58]; thus, although energy adjustment controls for differences in energy intake, percentage intakes from macronutrients may remain inaccurate. Protein intake was validated in the NDNS feasibility study through comparisons of urinary with dietary nitrogen concentrations; however, no biomarkers exist for carbohydrate or fibre and therefore validation of GI calculations is not routine.

Our dietary GI values ranged from 40 to 72 units (glucose = 100), with a normal distribution. In the Zutphen study[25] values ranged from 39 to 65 units, with a right skewed distribution. Frost *et al.*[23] previously associated low-GI diets with higher HDL-C concentrations in healthy middle-aged adults. The total range of GI was not presented, however data were normally distributed and quintile means were similar to those of the current study. The lack of variation in dietary GI is unlikely to be responsible for the lack of association observed here; therefore GI calculation estimates were considered. The majority of values were obtained from international tables[38], many of which are sourced outside the UK; thus estimates for processed foods (e.g. Special K) may be invalid. Between-subject variation (for example, between diabetic and non-diabetic test groups)[59], methodological differences, and differences in preparation, processing, variety and ripeness may also introduce inaccuracies. Using the mean of multiple estimates, we attempted to improve the reliability of GI estimates, but undoubtedly inaccuracies remain that could attenuate observed associations[60].

GI values estimated from analogues contributed to approximately 26% carbohydrate. Where recipes and mixed-meal calculations are used, effects of protein and fat from non-carbohydrate-containing foods on insulin secretion or gastric emptying are ignored[61]. One study of eight healthy subjects demonstrated that variations in the energy (395–610 kcal), fat (8–24 g) and protein (12–25 g) content of a meal have little effect on the postprandial insulin and glucose response[62], the amount of carbohydrate and the GI explaining 90% of the observed variation in glucose response. Flint *et al.*[41] disagree however. Their study of 28 subjects consuming 13 test meals found no association between the estimated GI and that obtained using *in vivo* GI measurements. The meals all contained 50 g available carbohydrate with varying amounts of fibre (1–24 g), fat (3–42 g) and protein (5–28 g), and hence differing energy contents (270–715 kcal)[41].

The current study is limited by its cross-sectional design. We are trying to detect true associations between dietary intake estimated from a 4-day record (which may or may not represent long-term habitual intake) and various CVD risk factors, which may have been influenced by diet for more than 60 years, and which are also subject to other environmental[63] and genetic[64] influences. Notably, dietary

intakes differ between adulthood and old age due to changes in physiology, dentition, and intended weight gain or loss. Problems with dentition may cause elderly individuals to eat softer, lower-fibre foods (e.g. white bread (GI = 70) vs. grainy/seeded bread (GI = 54)) and could explain why our results do not support those of other studies; however, no food group analysis was completed here.

The physiological changes associated with ageing might limit or mask the beneficial effect of low-GI diets on body weight or CVD risk factors. The prospective Nurses' Health Study investigated the effect of GI on CVD risk[24] among >75 000 US women followed-up for 10 years. The highest quintile of GI (assessed by FFQ, relative to the lowest category) was associated with a relative risk of 1.28 ($P = 0.02$) for total CHD. We have examined risk factors for CVD independently and found no significant beneficial effect; however, small additive effects (undetectable in an epidemiological study of this size) on individual risk factors might combine to produce a measurable effect on disease incidence in the long term.

Our results provide limited evidence for a beneficial effect of low-GI diets with regard to body weight, BMI or CVD risk factors in an older British population. The relationship between GI and CVD risk may not exist in this population, or may be confounded by factors such as pre-existing CVD (58% taking cardiovascular medications) or diabetes (15% taking endocrine medications). Furthermore, regression analyses may be under-powered and subject to attenuation of dietary GI–CVD risk factor associations, the result of regression dilution bias introduced by unreliable dietary GI estimates[60].

No data are available on frequencies of individual or groups of foods consumed. Dairy products and high-fibre foods have low GI[38], but may affect anthropometric variables, BP or dyslipidaemia differently due to variability in fibre, micronutrient, antinutrient and antioxidant levels for example. Our final regression analysis found interactions between fibre and GI in some outcome variables, indicating that a high dietary GI may be more important in low- compared with high-fibre diets. The interrelationships between GI, fibre and CVD risk warrant further research. A factorial RCT could provide key carbohydrate-containing foods and compare the CVD risk profiles of four groups following combinations of low- or high-GI and low- or high-fibre diets. Alternatively, large RCTs could be used to examine the effect of GI on anthropometric and CVD risk factors, while prospective studies assessing food intake longitudinally with anthropometric, biochemical and CVD outcomes would be informative.

This study shows some associations between dietary GI and measures of obesity, BP and lipoprotein profile in an older British population, prior to adjusting for potential confounding factors. However, there is very limited evidence in an elderly population to justify advice to lower dietary GI for CVD prevention.

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