

**Dietary energy density predicts the risk of clinically incident type 2 diabetes:
The EPIC-Norfolk study**

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Objective: Accumulating evidence suggests that energy dense foods predispose to obesity, and such foods may also be associated with increased risk of type 2 diabetes, but there is limited evidence. Our aim was to investigate whether there is an independent association between dietary energy density and incidence of diabetes.

Research Design and Methods: Population-based prospective study, the EPIC (European Prospective Investigation of Cancer)-Norfolk Cohort Study of persons aged 40-79 years at baseline. We calculated energy density for overall diet (all solids and drinks) using food frequency questionnaire. During 12 years of follow-up, we documented 725 clinically incident cases of diabetes among 21,919 participants without diabetes, cancer or cardiovascular disease at baseline.

Results: Baseline energy density (age, sex, baseline BMI adjusted) was higher in those who developed type 2 diabetes (Mean 3.08 kJ/g, 95% CI 3.03-3.13) than those who remained non-diabetic (3.01 kJ/g, 3.00-3.02) (P=0.012). Energy density was positively associated with incident diabetes (odds ratio, OR, 1.21 per unit increase, 95% CI 1.06-1.38) adjusted for known risk factors. There was a 60% higher risk of diabetes (OR 1.60, 95% CI 1.19-2.16) in the highest quintile of energy density (range 3.55-7.97 kJ/g) compared with the lowest quintile (range 1.04-2.43 kJ/g) in adjusted analysis.

Conclusions: This is the first large population-based prospective study to report that an energy dense diet may be associated with increased risk of developing diabetes, independently of baseline obesity. The potential public health impact of a low energy dense diet on reducing the risk of diabetes deserves further study.

Dietary energy density (DED) is defined as the amount of energy able to be metabolized per unit weight or volume of food (1). Thus, assuming energy expenditure is held constant, high energy density of a given volume of food consumed would lead to increased energy intake and weight gain, as demonstrated by both short-term and long-term intervention trials [reviewed by Yao et al.(1)]. Energy-dense foods were also reported to be associated with body fatness in children: for instance, at age 7 years a unit (1kJ/g) rise in DED increased the odds of excess adiposity at 9 years by 36% (OR 1.36, 95% CI 1.09–1.69) (2). Mendoza et al also found that DED was independently associated elevated fasting insulin and the metabolic syndrome in a cross-sectional setting (3). It is plausible that such foods may also be associated with increased risk of type 2 diabetes, but there is limited evidence. In the Finnish Diabetes Prevention Study (DPS), overweight men and women with impaired glucose tolerance receiving standard care or intensive dietary and exercise counselling, showed positive, but not significant, association between energy density and risk of developing diabetes after 3 years of follow-up (hazard ratio 1.74, 95% CI 0.89-3.37) (4). To the best of our knowledge, this is the only published study regarding DED and the risk of diabetes.

Therefore, the purpose of our study was to investigate the association of dietary energy density with clinically incident diabetes in a population-based cohort study including both men and women, appropriately adjusted for a comprehensive range of lifestyle factors, social factors and dietary factors.

RESEARCH DESIGN AND METHODS

The European Prospective Investigation of Cancer (EPIC)- Norfolk Cohort Study recruited a total of 25,639 volunteers, aged

40-79 years, from general practices in Norwich and surrounding towns in Norfolk between 1993 and 1997, and has been described in detail elsewhere (5). Briefly, it was a population-based cohort study where participants completed a baseline health check, and follow-up constituted a postal questionnaire at 18 months, a second health check in 1998-2000, and a further postal questionnaire in 2002-2004. For the current analysis, we excluded participants with diagnosed prevalent diabetes, cancer, or cardiovascular disease at baseline because they may have altered their diet as a result of their condition. In addition, participants with missing food frequency questionnaire, or with more than 10 missing dietary items, as well as participants in the top 0.5% and bottom 0.5% of the ratio of self-reported energy intake to basal metabolic rate (EI:BMR) (6) were excluded from the analysis. After these exclusions, the analysis included 21,919 volunteers (9,781 men and 12,138 women) from the entire cohort. The study was approved by the local research ethics committee and participants gave written informed consent.

Ascertainment of cases of type 2 diabetes—Multiple sources of case ascertainment for new-onset diabetes till the end of 2005 were as follows: self-report of doctor-diagnosed diabetes from the second health check or follow-up health and lifestyle questionnaires; self-report of diabetes-specific medication in either of the two follow-up questionnaires or medication brought to the follow-up health check. In addition, external sources of information through record linkage included listing of any EPIC-Norfolk participant in the general practice diabetes register, local hospital diabetes register, hospital admissions data at that hospital screened for any diabetes-related admissions among study participants, and Office of National Statistics mortality data with coding

for diabetes. Participants who gave a self-report of history of diabetes but could not be confirmed against any other sources of ascertainment were not included as a confirmed case of diabetes.

Assessment of diet—Information on diet was collected using a 130-item validated EPIC-FFQ (food frequency questionnaire) at baseline (6,7). Participants were asked how often they had consumed a commonly used unit or portion size of each food on average during the previous year, choosing from 9 possible frequency responses ranging from “never or less than once per month” to “more than 6 times per day”. Nutrient intakes (per day) were computed by multiplying the frequency response by the nutrient content of the specified portion size using the CAFÉ (Compositional Analyses from Frequency Estimates) program (6). Values for nutrients were derived from the UK food composition database ‘McCance and Widdowson’s The Composition of Foods’ and its supplements (6). The energy and weight of each food item were summed for all solid food and beverages (not including pure water consumption) to get the total energy intake (kJ/day) and the total weight of foods (g/day). Dietary energy density (DED) was calculated as the available dietary energy per unit weight of foods (kJ/g).

$$\text{DED} = \frac{\sum_{i=1}^n \text{Energy}_i}{\sum_{i=1}^n \text{Quantity}_i}$$

Assessment of non-dietary factors—A detailed health and lifestyle questionnaire was completed at baseline (1993-1997). It included questions on demography, personal and family history of type 2 diabetes among first-degree relatives, smoking, physical activity, occupation, education, and medication (8). A menstrual/menopausal history was recorded in women, and those with their last menstrual period ≥ 2 years previously were coded as postmenopausal. Physical activity level was assessed by a four-

point physical activity index according to occupational and leisure-time physical activity (9). Smoking status was coded as never, former or current. At the health check visit, clinical examination was performed using a standard protocol as previously described (8). Anthropometric measurement included height (cm), weight (kg), waist and hip circumference (cm). BMI was calculated as body weight divided by squared height (kg/m^2). A further postal questionnaire was sent in 2004 to collect self-reported body weight, and weight change was calculated as follow-up weight minus baseline weight.

Statistical Analysis—Results were expressed as mean (SD) and for categorical variables by frequency and percentage. Differences between men and women in baseline characteristics and dietary energy density were tested using *t*-tests for continuous variables and by chi-squares tests for categorical variables. The independent variable (DED) was defined both as a continuous variable as well as a categorical variable with 5 categories (quintiles). Associations between DED and risk of developing type 2 diabetes were examined using two separate approaches. In the first approach we constructed three logistic regression models: the first model was adjusted for age, sex and baseline BMI; the second model was further adjusted for known risk factors for diabetes including lifestyle factors [family history of diabetes (yes, no), physical activity (inactive, moderate inactive, moderate active, active), smoking (current, former, never), and occupational status (professional, managerial and technical, skilled non-manual, skilled manual, partly skilled, unskilled), or education (no qualifications, O level, A level, degree)]; and the third model further included dietary factors [alcohol consumption (yes, no) and total energy intake (continuous)]. The third model was further adjusted for the percent of energy from dietary fat instead of total energy

intake to examine if the observed association was independent of fat intake. To explore the effect of central obesity, we also adjusted for baseline waist circumference in addition to baseline BMI, or instead of baseline BMI. We also further adjusted model 3 for weight change between the baseline and follow-up postal questionnaire in 2004. The same analyses were repeated in men and women separately using sex-specific quintiles of DED. In women, the model was further adjusted for menopausal status and use of hormone replacement therapy. In the second approach, in order to reduce the confounding effect of total energy intake, we repeated the analysis using calorie-adjusted DED computed as the residuals from the regression model with total energy intake as the independent variable and absolute DED as the dependent variable (10).

We also performed the following sensitivity analyses. We attempted to identify plausible under-reporters of total energy intake using two different published methods. One method used the Goldberg cut-off point of 1.35 for the ratio of reported energy intake (EI) to predicted basal metabolic rate (BMR) (11). BMR was estimated using published equations based on gender, age, weight and height (12). The other method was based on the ratio of reported energy intake (EI) to estimated energy requirement (EER) (13), which takes into account each individual's basal metabolic rate and physical activity level (14). In the current analysis, we adopted the range of EI:EER between 0.8-1.2 as plausible energy intake reporters.

All statistics were performed using STATA statistical software (version 9.2; Statacorp, College Station, TX, USA).

RESULTS

During a median of 10.2 years of follow-up (range 7.6-12.8 years), we documented 725 new cases of type 2 diabetes among 21,919 participants. At baseline,

participants, who later developed diabetes during follow-up, consumed a more energy dense diet than those who did not develop diabetes (age, sex and baseline BMI adjusted mean DED 3.08 kJ/g (95% CI 3.03-3.13) versus 3.01 kJ/g (3.00-3.02), $P=0.009$). Case participants were less physically active (inactive: 42.2% versus 28.4%; and active: 16.0% versus 19.1%, $P < 0.001$), more obese (BMI 29.7 versus 26.2 kg/m², waist circumference 99.4 versus 87.5 cm, obesity prevalence 40.4% versus 13.8%, all $P < 0.001$), and more likely to have a positive family history of diabetes than those who did not develop diabetes. Table 1 shows the range of DED across the five DED quintiles. Waist circumference, total energy intake and fat intake were significantly higher across categories of increasing DED. In contrast, BMI, prevalence of overweight and obesity, alcohol consumption, proportion of smokers, and occupational socio-economic status were lower across increasing DED quintiles.

There was an increased risk of diabetes associated with dietary energy density as shown in Table 2. This was the case both for the continuous association per unit increase in energy density, or when we examined the population divided into 5 groups according to the quintiles of energy density. Compared with the lowest quintile of DED (range 1.04-2.43 kJ/g), there was a 60% higher risk of clinically incident diabetes in the highest quintile of DED (range 3.55-7.97 kJ/g) (OR 1.60, 95% CI 1.19-2.16). Adjusting for percentage of energy from fat instead of total energy intake, and/or baseline waist circumference instead of baseline BMI did not materially change the results (data not shown). Adjusting for weight change did not materially change the results (OR 1.23 per unit increase of DED, 95% CI 1.03-1.47, and OR 1.52, 95% CI 1.03-2.24 in the highest quintile of energy density compared with the lowest group, p for trend 0.021, in model 3 with additional adjustment for weight

change). There was no significant interaction between DED and either BMI or waist circumference, or between DED and sex on the risk of diabetes (all $P > 0.86$). The results were similar in men and women if we used sex-specific quintiles, even when further adjusting for menopausal status and hormone replacement therapy in women (data not shown).

In analyses using calorie-adjusted DED (see methods), the association with the risk of clinically incident type 2 diabetes was unchanged. The DED was positively associated with an increased risk of diabetes in adjusted analyses (OR 1.21, 95% CI 1.06-1.38, model 3).

To address the issue of possible under-reporting regarding self-reported dietary intake, we performed a sensitivity analysis in plausible energy reporters using two different methods in identifying under-reporters. In the first method, which uses the Goldberg cut-off point (EI:BMR 1.35), we identified 11,242 potential under-reporters. In the second method, which takes into account the physical activity level of each individual, we identified 5,819 persons outside the range of EI:EER 0.8-1.2. Exclusion of under-reporters using both methods did not materially change the results. (OR 1.23, 95% CI 1.004-1.519 for EI:BMR ≥ 1.35 ; OR 1.20, 95% CI 1.02-1.42 for EI:EER 0.8-1.2)

To illustrate the intake pattern of an energy-diluted diet, we summarized the average intake of food groups by the quintile of DED (Table 3). Compared with the highest DED (energy-dense) quintile, participants in the lowest DED (energy-diluted) group consumed significantly more fresh fruit, more vegetables, less meat, less processed meat, less soft drinks, more alcoholic drinks, more non-energy containing beverages, and lower percent of energy from fat.

DISCUSSION

In this prospective study, we found a positive association between dietary energy density and the development of incident diabetes independent of baseline BMI, total energy intake, fat intake and lifestyle factors. Indeed, the risk was elevated by 60% in the highest energy density group compared with the lowest DED category. This finding if confirmed in other populations, will be of potential importance in understanding the aetiology of type 2 diabetes, and may have public health relevance in its prevention.

Our study adds new information regarding the association of dietary intake and incident diabetes. Dietary energy density is one way to assess the nature of the overall diet rather than assessing individual nutrients or foods. Diets that are high in fat tend to be energy-dense (15), because fat is the most energy-dense nutrient and fat content varies substantially between individual foods. In the Finnish DPS, compared with the low-fat/high-fibre group, the risk of developing diabetes increased by 89% in the high-fat/low-fibre group (4). Similarly, in the Health Professionals' Study, which followed 42,504 men for 12 years, total fat intakes were associated with a higher risk of type 2 diabetes, but this association disappeared after adjustment for BMI (16). However, several other factors other than fat intake also contribute to the energy density of the diet. The other important determinant of energy density is water, which has zero energy content and the proportion of which varies substantially among commonly consumed foods. Fibre also has the potential to influence DED because of its minimal energy content and the capacity to bind water, but fibre has a much smaller range of concentrations in common foods. Therefore, it is necessary to evaluate the nature of the overall diet using dietary energy density rather than the proportion of individual nutrients or foods.

The concept of energy density from the whole diet is appealing because it is simple both in terms of its calculation, and in terms of its “utility” and understanding by the general public. This approach takes into account the complex interactions among nutrients and foods in the context of a free-living population. On the other hand, it cannot identify particular components responsible for an energy dense diet, and thus is less informative in terms of biological relations between individual dietary components and disease risk. However, DED is weighed energy density of individual components of the overall diet. Therefore, foods which have high energy per unit of weight contribute to a more energy-dense diet. Since the two most important determinants of DED are fat and water content, dry foods with high fat content are especially energy dense, while watery foods low in fat constitute an energy-diluted diet. Generally, in our study less energy dense diets consist of more fresh fruit and vegetables, less meat and processed meat, more non energy-containing beverages, and lower energy from fat.

There are also several other methods of calculating DED (17,18). Previous studies reported that different DEDs have different associations with health outcomes i.e. BMI and body composition(19,20). The method we chose included all solid foods and all beverages (except water-not available in our study), because dietary habits influence every aspect of the diet, and adopting healthy diets should include not only the solid, but also the liquid components, which have been reported to have an important role in the development of obesity and type 2 diabetes (21).

Our study is the first study, to our knowledge, to examine the association between dietary energy density and clinically incident type 2 diabetes in a large population-based cohort setting of >21,000 persons with long follow-up in both men and women. There was a non-significant positive

association between DED and the risk of incident type 2 diabetes at 3 years of follow-up in the Finnish Diabetes Prevention Study, but that study was small with a total sample size of 522, and limited to a high risk group with overweight/obesity and impaired glucose tolerance (4). We report a significant and positive association between DED and clinically incident diabetes in a low-risk free-living general population of the EPIC-Norfolk study. Additional strengths of our study were that we examined the exposure variable of DED both as a continuous and a categorical variable. We also found that energy-dense diets were predictive of diabetes independently of a comprehensive range of risk factors and confounders including baseline BMI and total energy intake. In addition, we accounted for the potential effect of under-reporting, which is a major drawback of self-report dietary assessment (11,13,22). Our case ascertainment of diagnosed new-onset diabetes was thorough, with self-report information supplemented by external sources i.e. general practice records, hospital records as well as death certificates. Thus a further strength of our study is that new cases of diabetes were identified through sources of data that did not depend on the follow-up participation rate.

Limitations of our study merit consideration. Dietary intake in our study was assessed by semi-quantitative food frequency questionnaire with its associated limitations. For one thing, there is an issue of potential under-reporting in self-report dietary intake. Our results, however, were robust with and without under reporters identified with two published methods taking into account individual characteristics. For another, the FFQ was designed to cover the most commonly consumed foods and represent the usual dietary pattern of each individual. Thus foods not in the questionnaire were not included in the calculation of DED. We could only ascertain diagnosed incident cases of

diabetes. The presence of any undiagnosed cases in the cohort will reduce the number of new cases and have the effect of attenuating any observed associations. Our population is predominantly of European-Caucasian origin (99.1%), thus our findings cannot be considered equally valid in other groups. Our cohort, though representative of the general population with respect to clinical and anthropometric characteristics, may have included greater healthier lifestyle choices, for instance, smoking prevalence was lower (12%) than the national average (~27% in 1998, Health Survey for England) (5). This would have led to a possible underestimation of the observed effect, and hence our strong positive association is noteworthy.

The mechanism for the association between energy density and type 2 diabetes is not yet fully understood. Humans tend to eat in a way that maintains a constant volume of food intake because stomach distension triggers afferent vagal signals of fullness (1). Therefore, consumption of foods with high ED will result in excess energy intake because of the small volume of food in relation to its energy content. Foods that are high in ED also tend to be more palatable which is associated with increased food intake, the so-called “passive over-eating”, which would probably result in overweight and body composition change(1,2,20), both of which have been reported to be important risk factors for the development of diabetes. However, in our study, DED was positively associated with increased total energy intake as expected (Table 1), but the association between DED and risk of incident diabetes was independent of total energy intake, baseline BMI, and self-reported weight change. However, weight change data is only available till the follow-up questionnaire in 2004, while our end-point (diabetes) ascertainment is till end of 2005. Future studies are needed to elucidate the mechanism of association between high DED and development of type 2 diabetes, including

an understanding of whether it is mediated by weight gain.

In conclusion, we have shown prospectively that higher dietary energy density at baseline predicts the risk of incident diabetes independently of baseline BMI, total energy intake, and other known risk factors. This finding has potential implications for preventing type 2 diabetes through adopting a healthier lifestyle and merits further research, including confirmation in other studies.

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Table 1 Baseline characteristics by quintile of dietary energy density: EPIC Norfolk Study

	Dietary energy density quintiles										P value
	1		2		3		4		5		
Dietary energy density (kJ/g)	1.04-2.43		2.43-2.78		2.78-3.12		3.12-3.55		3.55-7.97		
Age (years)	57.2	(8.6)	57.9	(9.1)	58.5	(9.3)	58.5	(9.5)	58.6	(9.6)	<0.001
Weight (kg)	73.4	(13.4)	73.0	(13.0)	73.5	(13.0)	73.4	(13.2)	73.5	(12.9)	0.331
Height (cm)	165.4	(8.8)	166.0	(9.0)	167.0	(9.2)	167.7	(9.3)	168.5	(9.1)	<0.001
Waist (cm)	87.0	(12.5)	87.2	(12.4)	88.1	(12.2)	88.3	(12.2)	88.8	(12.0)	<0.001
BMI (kg/m ²)	26.8	(4.1)	26.4	(3.9)	26.3	(3.8)	26.0	(3.8)	25.8	(3.7)	<0.001
Energy intake (kJ/day)	6493.0	(1568.4)	7713.2	(1720.7)	8526.5	(1836.5)	9405.6	(2099.5)	10904.8	(2705.9)	<0.001
Fat intake (g/day)	50.0	(15.9)	65.2	(18.2)	75.9	(20.3)	87.3	(23.9)	106.6	(32.2)	<0.001
Energy from fat (%)	29.0	(5.8)	31.8	(5.2)	33.5	(5.2)	34.9	(5.0)	36.6	(5.2)	<0.001
Alcohol (g/day)	10.0	(15.7)	9.9	(14.0)	8.7	(12.6)	7.9	(11.3)	6.8	(9.8)	<0.001
Occupational status, N (%)											<0.001
professional	305	(7.1)	330.00	(7.7)	303	(7.0)	292	(6.8)	262	(6.1)	
managerial & technical	1,709	(39.7)	1,646	(38.3)	1,617	(37.5)	1,540	(35.7)	1387	(32.3)	
skilled non-manual	683	(15.9)	730	(17.0)	731	(17.0)	683	(15.9)	717	(16.7)	
skilled manual	943	(21.9)	910	(21.2)	944	(21.9)	1,048	(24.3)	1,115	(25.9)	
partly skilled	525	(12.2)	547.00	(12.7)	548	(12.7)	578	(13.4)	634	(14.7)	
unskilled	125	(2.9)	128.00	(3.0)	153	(3.6)	153	(3.6)	173	(4.0)	
Smoking, N (%)											<0.001
current	610	(14.0)	509	(11.7)	470	(10.8)	494	(11.4)	484	(11.2)	
former	1810	(41.6)	1785	(41.0)	1761	(40.5)	1835	(42.2)	1718	(39.7)	
never	1935	(44.4)	2063	(47.4)	2115	(48.7)	2016	(46.4)	2131	(49.2)	
Physical activity, N (%)											<0.001
inactive	1235	(28.2)	1279	(29.2)	1272	(29.0)	1272	(29.0)	1276	(29.1)	
moderately inactive	1313	(30.0)	1353	(30.9)	1290	(29.4)	1246	(28.4)	1142	(26.1)	
moderately active	1026	(23.4)	981	(22.4)	1032	(23.5)	1005	(22.9)	1029	(23.5)	
active	810	(18.5)	771	(17.6)	790	(18.0)	861	(19.6)	935	(21.3)	
Family history of diabetes, yes (%)	552	(12.6)	535	(12.2)	566	(12.9)	570	(13.0)	522	(11.9)	0.487
Obesity status, N (%)											<0.001
normal	1561	(35.7)	1708	(39.1)	1711	(39.1)	1855	(42.4)	1867	(42.7)	
overweight	2012	(46)	1946	(44.5)	2027	(46.3)	1922	(43.9)	1964	(44.9)	
obesity	787	(18.0)	706	(16.2)	628	(14.3)	571	(13.1)	510	(11.7)	

Data are means (SD) or frequency (%). P values are from ANOVA for continuous variables and χ^2 test for categorical variables.

Table 2 Association between dietary energy density (continuous and quintiles) and the risk of type 2 diabetes: EPIC Norfolk study

	DED	P value	Dietary energy density quintiles					P for trend
			1	2	3	4	5	
Dietary energy density (kJ/g)	1.04-7.97		1.04-2.43	2.43-2.78	2.78-3.12	3.12-3.55	3.55-7.97	
Men	1.30-7.53		1.30-2.55	2.55-2.92	2.92-3.26	3.26-3.70	3.70-7.53	
Women	1.04-7.97		1.04-2.35	2.35-2.67	2.67-3.00	3.00-3.42	3.42-7.97	
Incident cases	725		135	140	138	143	169	
Model 1	1.12 (1.01-1.25)	0.032	1.00	1.07 (0.83-1.37)	1.05 (0.82-1.35)	1.11 (0.87-1.43)	1.34 (1.05-1.70)	0.022
Model 2	1.13 (1.01-1.26)	0.028	1.00	1.04 (0.80-1.34)	1.06 (0.82-1.36)	1.10 (0.86-1.42)	1.35 (1.06-1.73)	0.016
Model 3	1.20 (1.05-1.37)	0.007	1.00	1.10 (0.85-1.42)	1.15 (0.88-1.49)	1.23 (0.93-1.61)	1.58 (1.18-2.12)	0.003

Data are Odds Ratio (95% Confidence Interval). Adjustments of covariates were performed using multiple regression analyses by cumulatively adding the following covariates into the model. Model 1, age, sex and baseline BMI; model 2, model 1 + occupational status, smoking, physical activity, and family history of type 2 diabetes; model 3, model 2 + alcohol consumption and total energy intake.

Table 3 Intake pattern of food groups by quintiles of dietary energy density at baseline: EPIC Norfolk study

Food groups	Dietary energy density quintiles									
	1	2	3	4	5	P for trend				
Fresh fruit, g/day	279.6 (226.2)	256.3 (195.4)	232.2 (171.2)	209.2 (154.0)	186.5 (140.9)	<0.001				
Dried fruit, g/day	2.9 (8.1)	3.2 (7.6)	3.1 (7.4)	3.3 (7.9)	3.3 (9.6)	0.171				
Vegetables, g/day	282.5 (151.7)	283.2 (138.2)	274.7 (125.7)	262.6 (121.0)	251.0 (121.1)	<0.001				
Meat, g/day	59.3 (34.7)	66.0 (36.9)	68.9 (38.4)	71.4 (42.2)	74.8 (48.1)	<0.001				
Processed meat, g/day	18.6 (15.8)	22.2 (16.9)	25.0 (19.7)	27.7 (21.7)	31.4 (26.6)	<0.001				
Vitamin C (FFQ), mg/day	132.9 (69.3)	131.0 (63.1)	124.9 (56.4)	119.6 (53.2)	112.9 (50.7)	<0.001				
Non energy containing beverages, g/day	1343.5 (394.6)	1172.9 (336.3)	1081.4 (328.6)	1003.7 (333.5)	833.5 (380.7)	<0.001				
Energy containing beverages, g/day	620.6 (371.2)	620.5 (330.4)	611.5 (300.1)	593.5 (268.8)	557.2 (245.6)	<0.001				
Soft drinks*, g/day	25.5 (79.6)	31.9 (79.4)	35.6 (79.3)	38.6 (79.5)	48.2 (82.0)	<0.001				
Alcoholic drinks, g/day	162.0 (321.8)	148.8 (267.5)	128.5 (227.0)	112.7 (186.4)	94.3 (148.9)	<0.001				
Total energy intake (kcal)	1539.0 (372.7)	1830.2 (408.9)	2024.2 (436.3)	2234.1 (499.0)	2592.1 (643.5)	<0.001				
Energy from carbohydrate, %	50.9 (7.5)	50.2 (6.5)	50.0 (6.4)	49.9 (6.2)	50.0 (6.3)	<0.001				
Energy from fat, %	29.0 (5.8)	31.8 (5.2)	33.5 (5.2)	34.9 (5.0)	36.6 (5.2)	<0.001				
Energy from protein, %	18.7 (3.2)	17.3 (2.8)	16.5 (2.6)	15.7 (2.5)	14.4 (2.6)	<0.001				

Data are means (SD).

*Soft drinks were defined as fizzy soft drinks plus fruit squash or cordial.