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Lunch energy density and the metabolic syndrome in patients with type 2 diabetes mellitus

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Abstract

The aim of the present study was to investigate the possible associations between dietary energy density (ED) and the metabolic syndrome (MetS) in patients with type 2 diabetes. In the present case–control study, the dietary ED of 125 patients with type 2 diabetes (seventy-eight with (cases) the MetS and forty-seven without (controls) the MetS; mean age 62·0 (SD 9·4) years, mean diabetes duration 12·5 (SD 8·4) years and mean glycated Hb 7·2 (SD 1·3) %) was assessed by weighed diet records. The MetS was defined according to the 2009 Joint Interim Statement and ED by the amount of energy (kJ) in a given weight of food. Data are expressed as means (standard deviations) or medians (interquartile ranges). Patients with the MetS reported lower intakes of total energy and fibre, and a higher total food amount than the controls; the total ED did not differ, but the cases had a higher ED at lunch (mean 6·3 (SD 1·3) *v.* 5·9 (SD 0·8) kJ/g; $P=0·017$). In this meal, patients with the MetS had lower intakes of beans (median 0·7 (interquartile range 0·4–1·1) *v.* 1·1 (interquartile range 0·6–1·6) g/kg; $P=0·020$), vegetables (median 1·2 (interquartile range 0·6–1·7) *v.* 1·4 (interquartile range 1·0–2·0) g/kg; $P=0·046$) and total meat (median 1·3 (interquartile range 1·0–1·6) *v.* 1·4 (interquartile range 1·2–1·8) g/kg; $P=0·034$) than patients without the MetS. The associations between lunch ED (kJ/g) and food groups (g/kg) were confirmed for vegetables (r −0·584; $P<0·001$), fruits (r −0·233; $P=0·070$), beans (r −0·189; $P=0·037$) and oils (r 0·323; $P<0·001$). In a multivariate logistic regression model, a high lunch ED was associated with the MetS (OR 6·89, 95% CI 1·35, 35·15; $P=0·020$) after adjusting for confounders. In conclusion, a high ED at lunch increased the odds of the presence of the MetS in patients with type 2 diabetes. Beans and vegetables may be the major contributors to this association and their consumption might be considered to decrease ED.

Key words: Diets: Energy density: Metabolic syndrome: Type 2 diabetes

The metabolic syndrome (MetS) comprises a set of inter-related risk factors for CVD and diabetes mellitus[1]. Indeed, the MetS is associated with a 2-fold increase in cardiovascular outcomes and a 1·5-fold increase in all-cause mortality in the general population[2]. The prevalence of the MetS in patients with type 2 diabetes ranges from 75 to 85 %[3–5], and the aggregation of MetS components was significantly associated with macro-[4] and microvascular complications in these patients[4,6].

Coronary artery disease is the leading cause of death in patients with type 2 diabetes. However, it is widely recognised that the absolute risk of CVD varies among patients with diabetes, and an accurate assessment of risk clearly depends on individual characteristics[7]. In this context, MetS detection can contribute to the stratification of cardiovascular risk in these patients.

Lifestyle changes, including sustained 5–10 % weight loss, a moderate increase in physical activities and changes in dietary intake are the first-line strategies for treatment of the MetS[8]. In this context, dietary composition has been associated with the presence of the MetS in the general population[9–14] and in patients with diabetes[15,16]. Regarding the potential relationship between particular meals and the MetS, our group has demonstrated that patients with diabetes and the MetS have a higher-glycaemic index breakfast than diabetic

Abbreviations: ED, energy density; MetS, metabolic syndrome.

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patients without the MetS[16]. In another observational study, an inverse association between regular eating meals and the presence of the MetS has been demonstrated[17]. The meals taken during the 24 h of the day are part of one's individual eating pattern, and further exploration of meal composition is warranted. Randomised clinical trials have demonstrated the beneficial effects of particular dietary patterns such as the Mediterranean diet[18] and the Dietary Approaches to Stop Hypertension model[19] on the components of the MetS. The Dietary Approaches to Stop Hypertension diet emphasises the consumption of fruits, vegetables and low-fat dairy foods, and includes whole grains, poultry, fish and small amounts of red meat, sweets and sugar-containing beverages. The Mediterranean diet includes fruits, vegetables and whole grains, encourages the intake of nuts and fish and advocates olive oil as the main source of dietary fat[20]. Interestingly, most foods common to both diets have low energy density (ED).

ED refers to the amount of energy (kJ) in a given weight of food. Foods with a high ED provide more energy per g than do foods with a low ED[21]. Many studies have demonstrated the association between ED and obesity[22–25]. However, data on the possible association between ED and the incidence of diabetes[26], cardiometabolic risk factors[27] and, especially, the MetS[13,14] are scarce. Furthermore, no studies have investigated the potential relationship between dietary ED and the MetS in patients with diabetes. Therefore, the aim of the present study was to evaluate the possible association between ED, both of the total daily diet and of main meals, and the presence of the MetS in patients with type 2 diabetes.

Methods

Patients

The present case–control study was carried out in consecutive outpatients with type 2 diabetes from the Endocrinology Division of Hospital de Clínicas de Porto Alegre between June 2008 and December 2009. Patients were selected according to the following inclusion criteria: absence of dietary counselling by a registered nutritionist in the previous 6 months and age <80 years. Patients with BMI <40 kg/m^2, urinary albumin excretion <300 mg/24 h, serum creatinine <152·52 μmol/l and malabsorption were excluded because they usually receive specific dietary advice. Type 2 diabetes was defined as patients over 30 years of age at the onset of diabetes, no previous episodes of ketoacidosis or documented ketonuria and, if insulin users, the treatment with insulin began only 5 years after diagnosis[28]. The study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving patients were approved by the Hospital Ethics Committee. Written informed consent was obtained from all patients.

Clinical and laboratory evaluation

Blood pressure measurements were obtained in duplicate, with the patient in a seated position after 10 min rest, using an Omron HEM-705CP sphygmomanometer (Omron Healthcare, Inc.). Patients were classified as normoalbuminuric (urinary albumin excretion <30 mg/24 h or <17 mg/l in a spot urine sample) or microalbuminuric (urinary albumin excretion 30–299 mg/24 h or 18–174 mg/l in a spot urine sample). Microalbuminuria was always confirmed[29]. Physical activity was graded in levels according to activities during a typical day based on a standardised questionnaire[30] adapted to local habits. A total of four levels were defined, ranging from sedentary lifestyle to high physical activity. Alcohol intake was taken into account when patients mentioned the current consumption of alcoholic beverages. Patients were classified as current smokers or not and self-identified as white or non-white.

The MetS was defined in this sample as the presence of two or more of the following risk factors, in addition to diabetes[1]: waist circumference ≥94 cm in men or ≥80 cm in women; serum TAG ≥1·70 mmol/l (or drug treatment of elevated TAG levels); serum HDL-cholesterol <1·04 mmol/l in men or <1·30 mmol/l in women (or drug treatment of low HDL-cholesterol levels); blood pressure ≥130/85 mmHg or current treatment with antihypertensive drugs[31]. Patients with the MetS were allocated to the case group and the remainder as control patients.

Laboratory measurements

Blood samples were collected after a 12 h fast. Plasma glucose was measured by the glucose oxidase method, glycated Hb by an ion-exchange HPLC procedure using a reference range of 4·8–6 %[32], total cholesterol and TAG by specific enzymatic colorimetric methods and HDL-cholesterol by the homogeneous direct method. LDL-cholesterol was estimated using the Friedewald formula (total cholesterol − HDL-cholesterol − (TAG/2·2))[33] for patients with serum TAG levels <4·52 mmol/l. Urinary creatinine was measured by Jaffe's reaction, urinary albumin excretion was measured by immunoturbidimetry (Ames-Bayer) and urinary urea N by the UV kinetic method. All tests were performed at the Hospital de Clínicas de Porto Alegre Clinical Pathology Laboratory.

Nutritional assessment

Anthropometric parameters evaluated for the assessment of nutritional status were body mass, height and waist circumference measured midway between the lowest rib margin and the iliac crest. Measurements were obtained with medical scales and a non-stretch fibreglass tape measure. BMI was calculated.

The patient's usual diet was assessed by means of the 3 d weighed diet record technique (two non-consecutive weekdays and one weekend day) as standardised previously[34]. Compliance with the weight record technique was confirmed by comparison between the protein intake estimate from weighed diet records and the 24 h urinary N output. To be included in the present study, patients had to have an acceptable ratio, between the two protein intake estimates, from 0·79 to 1·26, and values out of this range were considered

'under- or over-reporting'[35]. Therefore, fifty patients were not included in the present study. Nutritional composition from diet records was calculated with Nutribase 2007 Clinical Nutritional Manager software version 7.14 (Cybersoft) and updated[36].

ED was estimated taking into account only solid foods, excluding all fluids from analyses: ED = energy from the solid food consumed divided by the weight of the solid food consumed[21]. Foods were grouped for analyses: 'cereals, tubers and roots', 'fruit', 'vegetables', 'beans', 'meat and eggs', 'oils and fats' and 'sweets'.

Statistical analysis

The 85 % prevalence of the MetS in patients with type 2 diabetes[5] and a 1·05 kJ/g difference in ED between the cases (mean 7·49 (SD 1·67) kJ/g) and controls (mean 6·44 (SD 1·26) kJ/g) obtained after a preliminary pilot study of the usual diet in patients with type 2 diabetes (fifteen patients with and fifteen patients without the MetS) were considered to calculate the sample size. The minimum sample size was sixty cases and thirty controls, with 80 % power and an α of 0·05.

The Gaussian distribution of variables was tested by one-sample Kolmogorov–Smirnov and Shapiro–Wilk tests. Variables are expressed as mean (standard deviation), median (interquartile range) or number (percentage) of patients with the analysed characteristic. Patient characteristics (cases *v.* controls) were compared using Student's *t* test, the Mann–Whitney *U* test, and Pearson's χ^2 or Fisher's exact test (adjusted standardised residual was adopted as appropriate). Fibre intakes (total, soluble and insoluble) were adjusted for total energy intake according to the residual method[37]. Multiple logistic regression models were constructed to assess the possible associations of dietary ED with the presence of the MetS (dependent variable). Analyses were adjusted for confounding variables selected according to clinical relevance or their significance ($P<0·10$) on univariate analyses: sex; duration of diabetes; glycated Hb; fibre intake. *P* values $<0·05$ (two-sided) were considered as statistically significant. All analyses were performed using PASW 18.0 (SPSS, Inc.).

Results

A total of seventy-eight patients with the MetS (62·4 %; cases) and forty-seven without the MetS (controls) were studied. Their main clinical and laboratory characteristics according to the presence of the MetS are described in Table 1. A higher proportion of women was observed in the MetS group. Patients with the MetS also had a shorter duration of diabetes, lower glycated Hb values and were more overweight when compared with the controls. Differences in the prevalence of MetS components were observed, as expected. There were no differences in age, lifestyle characteristics, microalbuminuria, and fasting blood glucose, total cholesterol or LDL-cholesterol between the two groups. In this sample of patients with type 2 diabetes (*n* 125), the most prevalent component of the MetS was high blood pressure (76·0 %), followed by abnormal waist circumference (64·0 %), hypertriacylglycerolaemia (33·6 %) and low HDL-cholesterol values (30·4 %).

The characteristics of the usual diet of the studied patients are described in Table 2. Patients with the MetS had a higher total energy intake and a lower intake of total dietary fibre, soluble and insoluble fibre (crude and energy-adjusted values) than the controls. The distribution of the macronutrients was as follows: 46·2 (SD 8·5) % energy from carbohydrates, 18·8 (SD 3·4) % energy from protein and 33·5 (SD 7·7) % energy from fat, with no significant differences between the patients with and without the MetS. Furthermore, there were no differences in the proportion of SFA, MUFA, PUFA and *trans*-unsaturated fatty acid intakes between the two groups.

All patients reported eating the three main meals of the day (breakfast, lunch and dinner), without differences between the patients with and without the MetS. It was observed that one-quarter of patients did not have a mid-morning snack (23 % of cases *v.* 21 % of controls; $P=0·499$), 4 % of cases did not have an afternoon snack, whereas all controls did ($P=0·239$), and one-third of patients did not have supper (32 % in both groups; $P=0·574$). We analysed ED values for the overall diet and the ED of each meal (Table 3). Patients with the MetS had a higher lunch ED when compared with patients without the MetS. There were no significant differences in total dietary ED or the ED of the other meals.

The amount of food eaten was evaluated in patients with and without the MetS. Patients with the MetS had a higher total food intake during the day (mean 1145·0 (SD 260·7) *v.* 1041·1 (SD 289·2) g; $P=0·041$), but ate less at lunch (median 411·4 (interquartile range 346·9–506·4) *v.* 485·8 (interquartile range 374·6–562·7) g; $P=0·028$) and dinner (median 245·2 (interquartile range 167·1–328·1) *v.* 306·0 (interquartile range 227·5–388·7) g; $P=0·032$) than the controls. There were no differences between the patients with and without the MetS in the amount of daily energy fluids consumed (median 311·5 (interquartile range 190·4–470·8) *v.* 371·7 (interquartile range 170·0–608·3) ml; $P=0·324$) or total energy intake from fluids (median 585·8 (interquartile range 322·2–866·1) *v.* 765·5 (interquartile range 343·1–1167·3) kJ; $P=0·190$). Regarding energy intake, patients with the MetS consumed less energy at dinner (median 1815·9 (interquartile range 1493·7–2397·4) *v.* 2305·4 (interquartile range 1623·4–2702·7) kJ; $P=0·027$) than the controls, without any difference at the other meals (data not shown). ED was evaluated, per 24 h period and at each meal, according to the groups divided by the presence of normal or abnormal individual components of the MetS. These analyses failed to identify any between-group differences (Table S1, available online).

We also conducted analyses according to the intake of food groups in order to better understand the observed differences at lunch (ED and the amount of food consumed) in patients with and without the MetS. The amount of food from each group eaten by the cases and the controls is reported in Table 4. Patients with the MetS ate less (g/kg body weight) beans, vegetables and meat than the controls. In a sub-analysis

Table 1. Clinical and laboratory characteristics of patients with type 2 diabetes according to the presence or absence of the metabolic syndrome (MetS)

(Mean values and standard deviations; medians and 25th–75th percentiles; percentages)

	Patients with the MetS		Patients without the MetS		
	Mean	SD	Mean	SD	*P*
n	78		47		–
Age (years)	62·1	9·1	61·8	9·9	0·887*
Females (%)	56·4		31·9		0·010†
Whites (%)	88·5		87·0		0·784†
Education (years)					
Median	8·0		8·0		0·313‡
25th–75th percentiles	5·0–11·0		5·0–11·0		
Diabetes duration (years)	11·4	8·2	14·5	8·5	0·045*
Current smokers (%)	20·5		19·1		1·000†
Current drinkers (%)	58·1		42·5		0·157†
Sedentary lifestyle (%)	59·2		62·2		0·848†
Microalbuminuria (%)	25·6		19·1		0·513†
Hypertension (%)	89·7		53·2		<0·001†
Systolic blood pressure (mmHg)	140·8	21·9	127·6	15·8	0·001*
Diastolic blood pressure (mmHg)	80·9	10·9	74·7	9·1	<0·001*
BMI (kg/m^2)	28·3	4·5	25·1	3·3	<0·001*
Overweight (%)	71·8		44·7		0·004*
Waist circumference (cm)					
Males (*n* 66)	99·3	9·5	90·0	6·2	<0·001*
Females (*n* 59)	98·4	10·7	89·8	11·6	0·011*
Fasting blood glucose (mmol/l)	8·3	3·2	7·8	2·5	0·363*
HbA1c (%)	7·0	1·1	7·5	1·5	0·036*
TAG (mmol/l)					
Median	1·7		1·0		<0·001‡
25th–75th percentiles	1·2–2·4		0·8–1·3		
Total cholesterol (mmol/l)	5·2	1·2	4·9	1·1	0·136*
LDL-cholesterol (mmol/l)	3·2	1·1	3·0	1·0	0·184*
HDL-cholesterol (mmol/l)					
Males (*n* 66)	1·1	0·2	1·4	0·3	<0·001*
Females (*n* 59)	1·3	0·3	1·6	0·3	0·001*
Serum creatinine (μmol/l)	79·6	17·7	79·6	17·7	0·989*

HbA1c, glycated Hb.
* Student's *t* test.
† Pearson's χ^2.
‡ Mann–Whitney *U* test.

regarding the type of meats, a greater proportion of patients with the MetS ate red meat than patients without the MetS (94·7 *v.* 82·2 %; *P*=0·028), without differences in the proportion of patients who ate white meat (64·0 *v.* 68·9 %; *P*=0·585). No differences were observed between the patients with and without the MetS regarding the amount of consumed red (median 65·0 (interquartile range 37·0–81·7) *v.* 60·0 (interquartile range 18·3–97·5) g; *P*=0·651) or white meats (median 26·7 (interquartile range 0–43·3) *v.* 36·7 (interquartile range 0–62·7) g; *P*=0·379), respectively.

Considering data from all patients (*n* 125), correlation coefficients between lunch ED and food groups (g/kg body weight) consumed at lunch were significant for vegetables (r − 0·584; $P<0·001$), fruits (r − 0·233; P=0·070), beans (r − 0·189; P=0·037) and oils (r 0·323; $P<0·001$). We did not observe correlations of lunch ED with meats (white or red), cereals and sweets (data not shown).

In the multiple logistic regression model, lunch ED was associated with the presence of the MetS: an increase of 4·184 kJ/g in lunch ED was associated with a greater than 6-fold increase in the odds of the MetS (OR 6·89, 95 % CI 1·35, 35·15; P = 0·020), after adjusting for sex (OR 3·04, 95 % CI 1·33, 6·96; P = 0·008), diabetes duration (OR 0·95, 95 % CI 0·91, 1·00; P=0·058), glycated Hb (OR 0·69, 95 % CI 0·49, 0·97; P=0·032) and total energy-adjusted fibre intake at lunch (OR 0·94, 95 % CI 0·84, 1·07; P=0·349).

Discussion

In the present case–control study, lunch ED was independently associated with the presence of the MetS, with the odds of the MetS increasing 6·30 times for each 4·184 kJ/g increment in lunch ED. This was the first study to explore the relationship between meal ED and a co-morbidity outcome in patients with type 2 diabetes.

Other authors[13,14] have reported weaker associations between ED and the MetS in patients without diabetes when compared with the present results, possibly due to the variability in the prevalence of the MetS in different populations. In fact, approximately 85 % of patients with type 2 diabetes have the MetS[5], whereas the prevalence is lower (20–25 %) in the general population[8]. Mendoza *et al.*[13] reported that

Table 2. Characteristics of the usual diet of patients with type 2 diabetes according to the presence or absence of the metabolic syndrome (MetS)

(Mean values and standard deviations; medians and 25th–75th percentiles)

	Patients with the MetS		Patients without the MetS		
Daily dietary intake	Mean	SD	Mean	SD	*P*
n	78		47		–
Total energy intake (kJ)	7606·5	2066·9	8568·8	1953·9	0·010*
Total energy intake (kJ/kg)	104·6	29·3	129·7	29·3	<0·001*
Carbohydrates (% of TEI)	45·7	9·2	47·2	7·2	0·338*
Protein (% of TEI)	19·0	3·1	18·5	3·9	0·385*
Fats (% of TEI)	33·3	7·5	34·0	8·2	0·631*
MUFA (% of TEI)	11·6	3·9	11·3	3·5	0·579*
PUFA (% of TEI)	10·1	3·9	9·4	3·3	0·339*
SFA (% of TEI)	9·9	3·6	10·0	2·6	0·857*
TFA (% of TEI)					
Median	0·9		0·9		0·290†
25th–75th percentiles	0·6–1·5		0·5–1·4		
Dietary fibre (g)					
Crude (g)	16·7	6·8	21·6	7·3	<0·001*
Energy adjusted (g)‡	17·3	5·8	20·5	7·1	0·005*
Soluble fibre					
Crude (g)	5·2	2·1	6·9	3·3	0·001*
Energy adjusted (g)‡	5·4	1·8	6·6	3·3	0·011*
Insoluble fibre (g)					
Crude (g)	11·4	4·9	14·7	5·1	<0·001*
Energy adjusted (g)‡	11·8	4·3	14·1	5·0	0·008*

TEI, total energy intake; TFA, *trans*-fatty acids.
* Student's *t* test.
† Mann–Whitney *U* test.
‡ Fibre intake values (total, soluble and insoluble) were adjusted for total energy intake according to the residual method.

a 10% increase in the prevalence of the MetS could be attributed to a 4·10 kJ/g increase in the dietary ED of US adults. In Iranian women, high-ED diets were associated with 54% higher odds for the MetS compared with women who consumed a lower-ED diet (a difference of −2·0 kJ/g)[14]. Finally, the impact of ED may be greater in individuals with a greater predisposition for the MetS as occurs with type 2 diabetic patients.

In view of the significant difference in lunch ED between the patients with and those without the MetS, and considering that this difference may have been due to a greater intake of high-ED foods, we analysed the intake amount according to food groups consumed. Patients with the MetS ate less beans, vegetables and meat, which may at least partly explain (especially the reduced intake of vegetables and beans) the higher lunch ED. Beans and vegetables are low-ED foods, mostly due to their extremely high water content (approximately 80 and >90%, respectively), are relevant sources of dietary fibre and have a low glycaemic index – all characteristics associated with a low MetS prevalence in patients with diabetes[15,16]. Furthermore, lunch accounted for 37·4 (SD 9·3)% of the total energy intake of this sample of patients with diabetes and could partially explain the results observed, considering that this meal was the major contributor to daily energy intake, with a significant difference (general linear model analysis; $P<0·001$) on comparison with the other five meals.

The links between ED and the MetS are poorly understood. The high glycaemic index and the amount of saturated fat observed in high-ED diets may contribute to the development of insulin resistance[38,39]. The relationship between ED and insulin resistance/sensitivity could help explain its links with the MetS, especially due to the importance of insulin resistance to the pathophysiology of the MetS[40].

Table 3. Energy density values in the usual diet of patients with type 2 diabetes according to the presence or absence of the metabolic syndrome (MetS)

(Mean values and standard deviations; medians and 25th–75th percentiles)

	Patients with the MetS		Patients without the MetS		
Energy density (kJ/g)	Mean	SD	Mean	SD	*P*
n	78		47		–
Overall diet	7·5	1·7	7·5	1·7	0·687*
Breakfast	10·9	5·0	10·5	3·8	0·686*
Mid-morning snack					
Median	2·1		2·5		0·304†
25th–75th percentiles	0·8–3·8		2·1–3·8		
Lunch	6·3	1·3	5·9	0·8	0·017*
Afternoon snack					
Median	7·5		8·8		0·250†
25th–75th percentiles	4·2 – 11·3		5·4 – 11·3		
Dinner	8·4	2·9	8·0	2·5	0·597*
Supper					
Median	2·1		2·5		0·584†
25th–75th percentiles	0·0–5·9		0·0–8·0		

* Student's *t* test.
† Mann–Whitney *U* test.

Table 4. Food groups at lunch of patients with type 2 diabetes according to the presence or absence of the metabolic syndrome (MetS)

(Medians and interquartile ranges)

	Patients with the MetS		Patients without the MetS		
Daily dietary intake	Median	Interquartile range	Median	Interquartile range	*P*
Cereals, roots and tubers					
Total (g)	143·3	98·3–190·8	152·3	103·3–200·4	0·553*
Body weight (g/kg)	1·9	1·4–2·6	2·2	1·5–3·0	0·111*
Meat and eggs					
Total (g)	89·6	77·0–113·7	96·2	81·7–117·5	0·396*
Body weight (g/kg)	1·3	1·1–1·6	1·4	1·2–1·8	0·034*
Beans					
Total (g)	50·0	32·0–83·3	65·8	33·5–104·6	0·080*
Body weight (g/kg)	0·7	0·4–1·1	1·0	0·6–1·6	0·020*
Vegetables					
Total (g)	98·3	43·2–123·5	101·7	70·9–128·6	0·237*
Body weight (g/kg)	1·2	0·6–1·7	1·4	1·0–2·1	0·046*
Fruit					
Total (g)	0·0	0·0–40·8	0·0	0·0–32·9	0·946*
Body weight (g/kg)	0·0	0·0–0·5	0·0	0·0–0·4	0·958*
Oils and fats					
Total (g)	15·4	10·0–20·2	14·9	9·3–21·4	0·728*
Body weight (g/kg)	0·2	0·1–0·3	0·2	0·1–0·3	0·707*
Sugars and sweets					
Total (g)	0·0	0·0–0·0	0·0	0·0–0·0	0·317*
Body weight (g/kg)	0·0	0·0–0·5	0·0	0·0–0·0	0·314*

*Mann–Whitney *U* test.

We took particular care with some methodological aspects of the present study, such as the choice of dietary records and the formula to estimate the ED of foods. Information on habitual dietary intake was obtained by means of a 3 d weighed diet record, a method previously validated by using a biomarker (protein intake) in patients with type 2 diabetes[34]. Furthermore, only patients whose dietary records were not considered implausible were included in the study[35]. Regarding dietary ED, although a variety of methods are available[21,41], we chose to conduct the present analyses on the basis of solid foods alone, due to the lower within-person variability of solid-food ED[21]. This estimation method has been recommended[23] because fluid intake generally has no impact on overall energy intake but increases the volume of food ingested over the course of a day, thus reducing the overall ED. Moreover, the contribution of fluids towards daily energy intake in our sample was lower than 10 %.

One possible limitation of the present study includes the greater proportion of women in the MetS group when compared with patients without the MetS. This weakness could preclude the generalisation of the present findings. However, the prevalence of the MetS can differ between sexes (greater in males than in females), as reported elsewhere[3,4]. Finally, we included sex as an independent variable in the multivariate analysis and the association of ED with the MetS was maintained. Although we have presented a power calculation to justify the sample size, the significance of the reported results should be interpreted with caution, because many statistical tests were performed in the present study, which increases the chance of type I error.

The present results showed that, in patients with type 2 diabetes, lunch food choices that reduce ED are inversely associated with the presence of the MetS. An increase in the intake of beans and vegetables should be the focus of patients' dietary changes at lunch in order to achieve reductions in lunch ED. Beans are good sources of vegetable protein and fibre and their daily intake has been encouraged for the general population[42–44], as well as the consumption of at least five daily servings of fruit and vegetables (approximately 400 g/d)[45]. The addition of two servings of vegetables (e.g. four tomato slices (80 g) and one serving spoonful of raw carrots (40 g), or three slices of boiled beet (40 g) and three tablespoonfuls of cooked spinach (70 g)) in a lunch consisting of 125 g rice, 100 g beans and one 100 g serving of white meat would lead to a reduction in ED of the order of 1·05 kJ/g, and should therefore be encouraged.

In conclusion, eating a high-ED lunch is associated with a 6-fold increase in the presence of the MetS in patients with type 2 diabetes. Beans and vegetables appear to be the key foods for reducing lunch ED, and should be considered in further research on dietary advice in these patients. In this regard, randomised clinical trials are required to evaluate the impact of these strategies on the prevention and treatment of the MetS in these patients.

Supplementary material

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