British Journal of Nutrition (2006), **96**, 532–538
DOI: 10.1079/BJN20061876

Dietary intakes associated with metabolic syndrome in a cohort of Japanese ancestry

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(Received 21 September 2005 – Revised 3 May 2006 – Accepted 9 May 2006)

The present study examined the association between dietary factors and metabolic syndrome in a 7-year follow-up of Japanese–Brazilians. In 1993, a survey estimated the prevalence of diabetes in a Japanese–Brazilian population aged 40–79 years. From 647 subjects studied at baseline, 394 (60·9 %) participated in a second survey (2000); after exclusion of subjects with self-reported diseases, 23·4 % (*n* 151) were included in the present analysis. Metabolic syndrome diagnosis was based on the National Cholesterol Education Program criteria modified for Asians. Food intake was assessed by a validated food-frequency questionnaire. At baseline, mean ages were 56·1 (SD 9·7) and 54·7 (SD 10·6) years for men and women, respectively. Similar cumulative incidence rates of the metabolic syndrome were found in both sexes (36·9 (95 % CI 26·6, 48·1)% for men and 38·8 (95 % CI 27·1, 51·4) % for women). In 1993, mean values of blood pressure, waist circumference for men and 2 h plasma glucose for both sexes were higher among subjects who developed metabolic syndrome when compared with those who did not. Comparisons of nutrient intakes between subsets of subjects who developed or did not develop metabolic syndrome showed an association of protein intake with the syndrome only in men (96·4 (SD 56·8) *v.* 74·3 (SD 26·0) g/d; $P<0·05$). Odds ratios of the metabolic syndrome across tertiles of food groups intake showed that among men the highest tertile of red meat consumption was associated with a 4·7-fold increase in risk of developing the syndrome, after adjustments for confounders. However, the statistical significance of this model disappeared when saturated fatty acids were added. The data raised the possibility of a role of red meat consumption for the occurrence of metabolic syndrome in Japanese–Brazilian men. The present findings may have implications for the prevention of metabolic syndrome in this high-risk population.

Nutrition surveys: Dietary patterns: Abdominal obesity: Metabolic syndrome: Japanese–Brazilians

Metabolic syndrome represents a cluster of abnormalities linked by insulin resistance, which is associated with increased cardiovascular risk. In addition to insulin resistance and glucose intolerance, classical components of metabolic syndrome are high triacylglycerols, low HDL-cholesterol levels and elevated body adiposity (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). In Asians, the increased risks associated with obesity occur at lower BMI and waist circumference values than in Caucasians (Tan *et al.* 2004).

Insulin sensitivity is affected by body adiposity as well as the diet composition, which may influence several factors clustering in metabolic syndrome. Glucose and lipid metabolism are strongly related and derangements of glucose metabolism induced by a high-carbohydrate diet will also increase plasma triacylglycerols and decrease plasma HDL concentrations; this was clearly shown in a meta-analysis comparing a high-monounsaturated-fat diet with a high-carbohydrate diet (Garg, 1998). The effects of the quality of dietary fat are even of greater interest. In animals, saturated fat leads to insulin resistance (Storlien *et al.* 1991; Alstrup *et al.* 1999); in man, indirect evidence is supportive of high saturated fat intake being associated with impaired insulin action (Zhou & Grill, 1995; Storlien *et al.* 1996). The fatty acid component of dietary lipid not only influences hormonal signalling events by modifying membrane lipid composition, but fatty acids have a strong direct influence on molecular events governing gene expression (Clarke, 2000).

Since 1993, our group has conducted a population-based study in Japanese migrants and their descendants in Brazil, who have one of the highest prevalence of glucose metabolism disturbances worldwide (Costa *et al.* 2000; Gimeno *et al.*

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2002). High prevalence rates of type 2 diabetes in Japanese migrants living in the Americas (Fujimoto *et al.* 1987; Ferreira *et al.* 1996) suggest that the exposure to a different lifestyle exacerbates an inherent tendency to accumulate fat and develop diabetes (Gimeno *et al.* 2002). Dietary changes imposed by immigration such as increase in animal fat and reduction in complex carbohydrate intake have been reported (Costa *et al.* 2000; Freire *et al.* 2003). Little is available regarding the association of dietary factors with clusters of diseases, which have in common a nutritional basis such as metabolic syndrome. The objective of the present study was to examine the association between dietary factors and the metabolic syndrome in a 7-year follow-up of a cohort of Japanese–Brazilians.

Research design and methods

In 1993, a survey was designed to estimate the prevalence of diabetes and associated diseases in a Japanese–Brazilian population living in Bauru, Sao Paulo, Brazil (Gimeno *et al.* 2002). At that time, 647 subjects of first-generation (Japan-born, 37·3 %) and second-generation (Brazil-born, 62·7 %) Japanese–Brazilians of both sexes aged 40–79 years were included. A total of 394 subjects studied in 1993 (60·9 %) participated in a second phase in 2000. From the original study sample, sixty-nine subjects (10·6 %) had died, fifty-seven (8·7 %) had moved, 127 (19·7 %) refused to participate and for sixty-seven (10·3 %) dietary or laboratory data were missing. One hundred and seventy-six (27·2 %) subjects with self-reported diabetes or hypertension or dyslipidaemia were excluded from the present analysis because of the possibility of modifications in their diet following diagnosis. Therefore, the results refer to 151 (23·4 %) subjects without metabolic syndrome in 1993. This was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants. The protocol has been described in detail elsewhere (Gimeno *et al.* 2002). The subjects were interviewed by trained interviewers using standardised questionnaires and scheduled for physical examination and laboratory procedures.

Dietary data were obtained by a quantitative food-frequency questionnaire developed and validated for the Japanese–Brazilian population (Cardoso *et al.* 2001). Subjects were asked about their usual consumption of foods and food groups (122 items) during the previous year. Participants reported their habitual frequency of consumption of each item, the respective unit of time (if daily, weekly, monthly or annually) and size of the usual individual portion (if small, average, large or extra large in relation to the reference portion for each item of the food-frequency questionnaire). Questions concerning use of sauce, frequency of intake of visible fat and type of fat used in cooking procedures were also included. Complete rechecking of the coding as well as double keying were performed on every questionnaire. In addition to standard range edits, internal consistency edits and nutrient calculations for the diets were performed using the Dietsys 4.01 software (Block *et al.* 1994). The nutrient database used was based primarily on US Department of Agriculture publications supplied by Dietsys, supplemented by recent editions of standard food composition tables of Brazil (Fundação Instituto Brasileiro de Geografia e Estatística, 1996) and Japan (Resources Council, Science and Technology Agency, 1993).

Physical activity was assessed by questionnaire including type and duration of physical activities performed during work and leisure periods. A score was attributed to each subject and two categories were created: sedentary/light or moderate/heavy activity. Smoking habit was classified into two categories: current smoker or non-smoker. Education level was classified into three categories (<1, $1-8$ and >8 years). Body weight and height were measured while subjects wore light clothing and no shoes using calibrated electronic scales and a fixed stadiometer. BMI was calculated as weight divided by height squared and waist circumference measured at the umbilicus. Blood pressure was taken three times by an automatic device (Omron model HEM-712C; Omron Health Care, Inc., Bannockburn, IL, USA); mean values of the two last measurements were used to express systolic and diastolic blood pressure.

Blood samples were obtained after overnight fasting. Plasma glucose was determined by the glucose-oxidase method, and cholesterol, its fractions, triacylglycerol and uric acid measured enzymically by automatic analyser. Insulin concentrations were determined by monoclonal antibody-based immunofluorimetric assay (Vieira *et al.* 1995) and insulin resistance was assessed by the homeostasis model assessment of relative insulin resistance (Matthews *et al.* 1985).

Diagnosis of the metabolic syndrome was based on the Third Report of the National Cholesterol Education Program–Adult Treatment Panel III (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001), replacing the anthropometric cut-offs by those previously proposed by WHO for Asians (Inoue *et al.* 2000). Three or more of the following components defined metabolic syndrome: fasting plasma glucose $\geq 6{\cdot}1$ mmol/l; systolic or diastolic blood pressure ≥ 130 or ≥ 85 mmHg; HDL-cholesterol $<1{\cdot}29$ mmol/l for women or $<1{\cdot}04$ mmol/l for men; triacylglycerol $\geq 1{\cdot}79$ mmol/l; waist circumference ≥ 80 cm for women or ≥ 90 cm for men.

Statistical analysis was performed using SPSS version 10.0 (SPSS Inc., Chicago, IL, USA). Rates of metabolic syndrome were calculated by point and interval. Clinical, laboratory and nutritional data of the participants with and without the syndrome were compared by Student's t test; the Mantel–Haenszel χ^2 test for trend was applied for categorical variables. The paired Student's t test was used to compare nutrient intake data at baseline and after 7 years of follow-up. For positively skewed biological parameters (insulin, homeostasis model assessment and triacylglycerol), nutrients and food group variables, logarithmic transformations were used before analysis. The selection of nutrients and food groups for multivariate analysis was based on previous findings from literature and statistical criteria. Nutrients (carbohydrate, protein, total fat, saturated fatty acids, oleic acid, linoleic acid, total cholesterol, fibre and alcohol) or food groups (chicken and beef liver, red meat, ham, lunch meats, sausage, poultry, fish and shellfish, nuts, eggs, soya, dairy milk, oils and food fries, bread, cereal, rice, pasta, fruits and fruit juice, vegetables, sweets and dessert), compared according to the presence of metabolic syndrome in the crude analysis, showing $P<0{\cdot}20$, entered the multivariate models. Adjustments for

non-dietary variables, such as lifestyle characteristics, were also included. Energy-adjusted nutrient intakes were calculated as the residuals from the regression model, with absolute nutrient intake as the dependent variable and total energy intake the independent variable by sex (Willett & Stampfer, 1986). Logistic regression was used to calculate the odds ratios and their 95 % CI for metabolic syndrome. The lowest tertile of macronutrient or food group intake was considered the reference category. The initial model was adjusted for age, sex, physical activity, smoking habit, education level, alcohol and total energy intake. Other models of food groups included additional adjustments for total fat intake, saturated fatty acids and protein. To assess trends across tertile categories, we assigned the median intake of each tertile category to individuals with intakes in the category and then included this tertile median variable as a continuous factor in the logistic regression models. The *P* for trend was the resulting *P* value for the associated logistic regression coefficient (Kleinbaum *et al.* 1988). Statistical significance was defined as a two-tailed $P<0{\cdot}05$.

Results

Of the 218 subjects enrolled in the study, nutritional data were not available for sixty-seven subjects. No difference was observed between individuals who participated or not in this analysis considering age, sex and generation. Among the 151 Japanese–Brazilians, 56·0 % (*n* 84) were men and 44 % (*n* 67) women. In 1993, mean ages of men and women were 56·1 (SD 9·7) years and 54·7 (SD 10·6) years, respectively. Similar 7-year risks of metabolic syndrome were found in men (36·9 (95 % CI 26·6, 48·1) %) and women (38·8 (95 % CI 27·1, 51·4) %).

In 1993, mean systolic and diastolic blood pressure, waist circumference for men and 2 h glucose for both sexes were higher among subjects who developed metabolic syndrome in 2000 as compared with those who did not (Table 1). No weight gain was detected in any subset of subjects (data not shown).

Over time, men and women decreased their protein (from 76·0 (SD 24·6) to 68·7 (SD 13·5) g/d) and total cholesterol (from 244·1 (SD 114·6) to 184·6 (SD 67·1) mg/d) intakes, and increased their total fat intake (from 56·8 (SD 14·9) to 69·5 (SD 16·5) g/d), particularly of monounsaturated fat and polyunsaturated fat. The consumption of red meat and dairy milk decreased and the consumption of dairy skimmed milk increased over the follow-up period (data not shown).

In the crude analysis, a significant difference in nutrient intake was observed only in men, when comparing those with and without metabolic syndrome. At baseline, men with the syndrome in 2000 presented a higher mean protein intake (96·4 (SD 56·8) *v.* 74·3 (SD 26·0) g/d; $P<0{\cdot}05$) than those without metabolic syndrome (Table 2). Considering a number of adjustments (age, sex, education level, smoking habit, physical activity, alcohol and total energy), the odds ratios of metabolic syndrome across tertiles of nutrient intakes did not indicate any association with the occurrence of the syndrome in the Japanese–Brazilians studied.

Table 1. Main characteristics of Japanese–Brazilians in 1993 (baseline) according to sex and occurrence of metabolic syndrome in 2000

(Mean values and standard deviations)

	Men				Women			
	Without metabolic syndrome (*n* 53)		With metabolic syndrome (*n* 31)		Without metabolic syndrome (*n* 41)		With metabolic syndrome (*n* 26)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (years)	57·7	9·9	53·5*	8·8	55·2	11·3	53·8	9·7
Generation (%)								
Japan-born	23	43·4	7*	22·6	13	31·7	6	23·1
Brazil-born	30	56·6	24	77·4	28	68·3	20	76·9
Physical activity (%)								
Sedentary/light	43	81·1	23	74·2	31	75·6	22	84·6
Moderate/heavy	10	18·9	8	25·8	10	24·4	4	15·4
BMI (kg/m^2)	24·8	3·9	24·3	4·0	24·4	3·0	25·3	3·2
Waist (cm)	81·7	6·1	89·0**	7·2	78·3	10·1	80·2	8·3
Systolic blood pressure (mmHg)	118·6	13·8	129·2**	19·9	118·9	15·4	125·3	16·8
Diastolic blood pressure (mmHg)	73·1	9·3	80·5**	8·7	74·2	9·0	76·7	9·5
Fasting glucose (mmol/l)	5·3	1·3	5·3	0·6	4·8	0·4	5·5*	2·2
2 h Glucose (mmol/l)	5·3	1·4	6·6*	3·0	5·6	1·5	6·7*	2·8
Insulin (pmol/l)†	26·7	36·4	23·3	27·9	18·1	21·6	29·8	23·1
HOMA-IR†	1·0	1·3	0·9	1·1	0·6	0·8	1·2*	1·0
Total cholesterol (mmol/l)	5·3	1·0	5·5	1·2	5·7	1·1	5·4	0·8
LDL (mmol/l)	3·5	0·8	3·9	1·0	3·7	1·0	3·7	0·7
HDL (mmol/l)	1·1	0·2	1·0	0·2	1·4	0·9	1·2	0·2
Triacylglycerol (mmol/l)†	1·8	1·6	1·6	0·7	1·0	0·4	1·2	0·3

HOMA-IR, homeostasis model assessment of relative insulin resistance.
Mean values were significantly different from those without metabolic syndrome: * $P=0{\cdot}05$, ** $P<0{\cdot}01$.
† Log-transformed values were used for statistical analysis.
For details of subjects and procedures, see p. 533.

Table 2. Daily intake of energy and nutrients in Japanese–Brazilians in 1993 according to sex and occurrence of metabolic syndrome in 2000†

(Mean values and standard deviations)

	Men				Women			
	Without metabolic syndrome (*n* 53)		With metabolic syndrome (*n* 31)		Without metabolic syndrome (*n* 41)		With metabolic syndrome (*n* 26)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Energy (MJ)	8·40	2·3	9·1	2·9	6·7	2·0	7·3	2·8
Carbohydrate (g)	287·7	92·4	292·8	93·1	220·4	61·3	248·5	108·1
% total energy	57·4	10·4	54·7	11·6	55·3	7·0	56·0	9·3
Protein (g)	74·3	26·0	96·4*	56·8	65·5	30·1	71·8	29·4
% total energy	14·8	2·9	17·1*	5·8	16·0	3·9	16·5	3·9
Total fat (g)	57·3	23·6	62·6	28·3	53·6	24·5	54·0	21·6
% total energy	25·7	6·7	25·4	6·2	29·4	5·9	27·8	5·3
Oleic acid (g)	18·8	9·6	21·8	10·9	18·3	10·4	18·1	7·7
Linoleic acid (g)	9·1	6·6	7·8	3·7	8·3	4·8	7·9	4·1
Saturated fatty acids (g)	16·4	6·6	19·4	9·2	16·2	8·1	16·1	6·1
Cholesterol (mg)	243·6	149·9	308·9	177·3	209·3	114·4	222·4	110·2
Fibre (g)	18·2	7·6	18·4	8·3	15·2	5·1	16·6	11·2
Alcoholic beverage (g)	136·7	285·5	254·3	505·7	5·1	23·7	13·6	56·2

* Mean values were significantly different from those without metabolic syndrome ($P<0·05$).
† Log-transformed values were used for statistical analysis.
For details of subjects and procedures, see p. 533.

As far as food groups are concerned (Table 3), the comparison of intakes between subjects who developed or did not develop metabolic syndrome showed that the subgroup of men with the syndrome had a significantly higher consumption of red meat in 1993 than those without the syndrome (137·3 (SD 153·1) *v.* 67·6 (SD 61·6) g/d; $P<0·05$). This difference was not detected between the subgroups of women. Concordantly, only in men, the odds ratios across tertiles of food group intakes (Table 4) showed that the highest tertile of red meat consumption was associated with a 4·7-fold increase in the risk of the syndrome after adjustment for age, physical activity, education level, smoking habit, alcohol, total energy intake and total fat, but not in women (model 3). This association of red meat and metabolic syndrome was weakened by additional adjustments for other food groups; the inclusion of fried foods (model 4) led to a borderline

Table 3. Daily intake of food groups of Japanese–Brazilians in 1993 according to sex and occurrence of metabolic syndrome in 2000†

(Mean values and standard deviations)

	Men				Women			
	Without metabolic syndrome (*n* 53)		With metabolic syndrome (*n* 31)		Without metabolic syndrome (*n* 41)		With metabolic syndrome (*n* 26)	
Food groups (g/d)	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Chicken liver, beef liver	2·2	4·8	1·6	3·4	1·5	2·7	2·7	5·2
Red meat	67·6	61·6	137·3*	153·1	76·2	88·2	81·3	62·8
Ham, lunch meats, sausage	12·8	11·5	13·9	13·7	7·8	6·4	12·1	13·8
Poultry	21·3	32·8	18·7	18·1	15·3	15·6	21·8	19·5
Fish and shellfish	23·9	30·1	30·5	55·8	19·0	15·6	30·4	61·7
Nuts	4·7	16·1	6·1	11·7	5·1	20·1	4·0	7·2
Eggs	18·4	25·0	18·3	14·9	10·9	12·7	10·8	12·4
Soya	60·0	100·7	58·5	94·4	57·3	65·5	46·5	58·2
Dairy milk	130·0	97·6	142·9	112·2	131·3	105·3	114·5	104·4
Dairy skimmed milk	11·0	36·5	3·3	8·2	18·8	43·2	9·8	33·1
Oils, food fries	17·9	42·1	11·0	10·6	12·8	11·3	14·5	15·3
Bread, cereal, rice, pasta	514·2	230·1	528·5	267·7	335·6	168·9	397·1	174·1
Fruits, fruit juice	362·9	407·3	343·2	227·4	319·5	377·3	352·0	325·0
Vegetables	227·7	126·7	253·0	185·3	227·9	120·3	281·5	172·2
Sweets, desserts	159·2	134·2	180·6	172·6	152·8	112·2	153·3	162·8

† Log-transformed values were used for statistical analysis.
* Mean values were significantly different from those without metabolic syndrome ($P<0·01$).
For details of subjects and procedures, see p. 533.

Table 4. Risk of the metabolic syndrome in Japanese–Brazilians across tertiles of food groups intake, adjusted for several variables (Odds ratios (OR) and 95 % CI)

	Tertiles of intake					
	1 (lowest)	2		3 (highest)		
	OR	OR	95 % CI	OR	95 % CI	*P* for trend
Red meat						
Median intake (g/d)	19·5	73·5		144·2		
Multivariate model 1*	1·00	1·59	0·67, 3·75	1·73	0·73, 4·08	0·21
Multivariate model 2†	1·00	1·75	0·72, 4·22	1·98	0·81, 4·84	0·13
Multivariate model 3‡	1·00	3·25	0·73, 14·48	5·38	1·18, 24·44	0·03
Multivariate model 4§	1·00	1·84	0·51, 6·67	3·18	0·87, 11·5	0·07
Poultry						
Median intake (g/d)	4·6	13		28·7		
Multivariate model 1*	1·00	1·49	0·62, 3·58	1·87	0·79, 4·39	0·15
Multivariate model 2†	1·00	1·53	0·63, 3·69	2·07	0·86, 4·98	0·10
Multivariate model 3‡	1·00	2·39	0·71, 8·07	1·35	0·37, 4·88	0·6
Multivariate model 4§	1·00	2·57	0·75, 8·83	1·36	0·38, 4·78	0·62
Dairy milk						
Median intake (g/d)	12·4	141·7		223·7		
Multivariate model 1*	1·00	1·05	0·44, 2·48	0·72	0·30, 1·71	0·46
Multivariate model 2†	1·00	1·04	0·43, 2·46	0·76	0·31, 1·83	0·55
Multivariate model 3‡	1·00	0·92	0·26, 3·21	1·02	0·30, 3·47	0·96
Multivariate model 4§	1·00	0·92	2·26, 3·27	0·93	0·28, 3·00	0·90

* Model 1, adjusted for age (continuous), sex, physical activity (sedentary + light, moderate + heavy), smoking (yes/no), education level (< 1 year, 1–8 years and > 8 years), alcohol (continuous), total energy intake (continuous).
† Model 2, model 1 with additional adjustment for total fat intake (continuous).
‡ Model 3, model 2 for men only.
§ Model 4, model 3 with additional adjustment for fried foods (continuous).

significance (*P* for trend = 0·07). When saturated fatty acids or protein – instead of other food groups – were added to model 3, the statistical significance of red meat disappeared (data not shown).

Discussion

Metabolic syndrome is becoming highly prevalent in many populations. Whereas 24 % of the US adult population are affected (Ford *et al.* 2002), a prevalence twice this was recently reported in the Japanese–Brazilian population (Rosenbaum *et al.* 2005). Therefore, we had a unique opportunity to investigate possible factors involved in the aetiopathogenesis of the syndrome. Presumably, this involves a complex interaction between genetic and environmental factors, including diet (Costa *et al.* 2000; Groop, 2000; Lidfeldt *et al.* 2003), and knowledge of the syndrome's underlying mechanisms is essential to promote prevention behaviours.

In this cohort of Japanese–Brazilians followed for 7 years, their usual intake of red meat was positively associated with metabolic syndrome, particularly among men. It is well known that a major characteristic of the Western diet is a high intake of meat, which is a source of animal protein and saturated fat. In a cross-sectional analysis of subjects from the same population, our group recently showed total fat intake was associated with the syndrome as well as with fried food consumption (Freire *et al.* 2005). The present study adds that red meat consumption may predict metabolic syndrome in Japanese–Brazilians, even after adjustments for dietary and other non-dietary risk factors. The present findings are, at least in part, in agreement with the Health Professionals' Follow-up Study, in which processed meat appeared entirely responsible for the elevated diabetes risk associated with total red meat in men (van Dam *et al.* 2002). The findings are also consistent with evidence from a large prospective study in which a higher consumption of red meat – especially total processed meat – was associated with an increased risk of developing type 2 diabetes independent of known diabetes risk factors; however, such data were found in middle-aged and older US women (Fung *et al.* 2004; Song *et al.* 2004). In Japanese–Brazilian men, as seen in the Nurses' Health Study II, total red meat was implicated in the risk of diabetes after adjustments for confounders (Schulze *et al.* 2003). Bearing in mind the results of our cross-sectional study (Freire *et al.* 2005) and the present cohort, a role for the fat content in red meat contributing to metabolic syndrome in Japanese–Brazilians cannot be definitively excluded.

Studies examining the association of nutrients and food groups particularly with the cluster of abnormalities integrating metabolic syndrome and in non-Caucasian populations are rare. Our prospective design provides an opportunity to investigate the effect of this cluster of lifestyle-related diseases linked by insulin resistance in a pure Japanese population. Considering only macronutrients, they were not predictive of metabolic syndrome. These findings contrasted with previous evidence that polyunsaturated fat-rich diets increased insulin receptor number but decreased receptor affinity (Tsunehara *et al.* 1991; Vessby, 2000), with benefits for higher intake levels of polyunsaturated fat and detrimental effects for saturated fat (Feskens *et al.* 1995; Mayer-Davis *et al.* 1997; Fields, 1998) and with our cross-sectional analysis (Freire *et al.* 2005). An increase of odds ratio of metabolic syndrome with quintiles of total fat intake was observed in

a subset of Japanese–Brazilians. Inconsistencies in these findings may be attributed to different study designs and adjustments for confounding factors.

The lack of association of carbohydrate consumption and metabolic syndrome is somehow in agreement with other reports in which total carbohydrate intake was not related to fasting insulin (Ludwing *et al.* 1999) or to the risk of developing type 2 diabetes (Salmeron *et al.* 1997*a*,*b*; Meyer *et al.* 2000). The relationship between dietary carbohydrates and insulin sensitivity is a matter of controversy. Simple sugars may have a detrimental effect (Daly *et al.* 1997) while starches and polysaccharides may have benefits on glucose metabolism (Meyer *et al.* 2000; McKeown *et al.* 2004). Observational studies have found that fasting insulin levels were lower among individuals reporting high dietary fibre (Marshall *et al.* 1997; Ludwing *et al.* 1999) or whole-grain intakes (McKeown *et al.* 2002). Therefore, source and quality of dietary carbohydrates may differentially optimise insulin action and thereby affect the degree of insulin resistance, a key underlying feature of the syndrome.

The present study has limitations. The major limitation refers to the small number of subjects followed during this 7-year period. As a consequence of the sample size, the number of adjustments in the multivariate analysis needed to be restricted. The resulting possibility is that some true associations of nutrients or food groups with metabolic syndrome might have been missed. A detailed analysis of our data attempted to avoid an excessive number of variables, although considering background information from the literature. Several multivariate models, with different combinations of dietary factors, adjusted for lifestyle characteristics, were tested, in which persistent associations of red meat intake with metabolic syndrome were found in men. A certain degree of collinearity could have reduced our ability to reliably distinguish the effect of red meat from intakes of its major components. This possibility is suggested by the finding that the statistical significance of multivariate model 3 disappears if saturated fatty acids or protein were added (data not shown). Relatively low consumption of other subtypes of meat, rather than red meat, and restricted variation of their intakes in our cohort could have limited the detection of associations with other meats. A final limitation is a well-known phenomenon in nutritional epidemiology, that is, the possibility of underreporting food intake (Lafay *et al.* 2000).

In conclusion, the data raised the possibility of a role of red meat consumption for the occurrence of metabolic syndrome in Japanese–Brazilian men, although a role for the fat contained in meats cannot be excluded. Major sources of protein and fat other than meat do not appear to have this deleterious effect. Underlying mechanisms, by which consumption of red meat would influence the risk of the syndrome, require further investigation. The present findings may have implications for the prevention of metabolic syndrome in this high-risk population and support the existing dietary recommendations (National Cholesterol Education Program, 2002; World Health Organization & Food and Agriculture Organization, 2002) to limit red meat intake and saturated fat in subjects at risk for metabolic syndrome.

Acknowledgements

The present research was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP) (98/04 178-7).

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