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Gender May Modify the Effects of Macronutrient Intake on Metabolic Syndrome and Insulin Resistance in American Indians: The Strong Heart Study

Sigal Eilat-Adar, PhD, RD[1], **Jiaqiong Xu, PhD**[2], **Barbara V. Howard, PhD**[3], **Uri Goldbourt, PhD**[4], and **Helaine E. Resnick, PhD**[5]

1*Post-Doctoral Fellow; Medstar Research Institute, 6495 New Hampshire Avenue, Hyattsville, MD, USA.*

2*Assistant Professor of Research; Center for American Indian Health Research, University of Oklahoma Health Sciences Center, Oklahoma City, OK, USA.*

3*Senior Scientist, Medstar Research Institute, 6495 New Hampshire Avenue, Hyattsville, MD, USA.*

4*Professor of Epidemiology and Preventive Medicine, Department of Epidemiology and Preventive Medicine, Sackler Medical Faculty, Tel Aviv University, Tel Aviv, Israel; Head, Section of Epidemiology and Biostatistics, Henry N. Neufeld Cardiac Research Institute, Sheba, Medical Center, Tel-Hashomer, Israel*

5*Director of Research, American Association of Homes and Services for the Aging, 2519 Connecticut Avenue NW, Washington, DC, USA; Associate Professor of Medicine, Georgetown University, Washington, DC, USA.*

Abstract

Background—Diet has been related to several characteristics of metabolic syndrome (MS) and insulin resistance (IR), which carry an increased risk for diabetes and heart disease.

Objective—To examine the cross-sectional association between macronutrient intake, gender, and MS and IR in nondiabetic American Indians.

Design—Dietary intake, MS, and IR (estimated by homeostasis model assessment [HOMA]) were assessed.

Subjects/setting—Data were analyzed from participants with complete dietary data (n = 1,516 for MS, n = 1,458 for IR) from the 2nd examination (1993–95) of the Strong Heart Study, a longitudinal, population-based study of cardiovascular disease and its risk factors in American Indians.

Statistical analyses—Logistic regression and analysis of covariance were used to study associations among tertiles of macronutrient intake and MS and HOMA scores.

Results—Polyunsaturated fatty acid (PUFA) intake was associated with less MS and lower HOMA scores in women [OR and 95% CI for MS in the 3rd tertile: 0.69 (0.50–0.96)], but not men. Higher simple carbohydrate intake was associated with more MS in men [OR and 95% CI in the 3rd tertile: 1.72 (1.10–2.69)], but not women.

Conclusions—PUFA and simple carbohydrates may be associated with MS and IR in American Indians; gender may modify the association between dietary intake and MS and IR in this population. Further studies should focus on the longitudinal association between dietary intake and incidence of MS and IR and the role of gender in this relationship in American Indians and other populations.

Corresponding Author: Sigal Eilat-Adar, 16 Zeira St., Tel Aviv 69624 Israel. Phone: (+972) 3-647-6759; Fax: (+972) 3-648-6451; E-mail: eilatsi@017.net.il.

Keywords

macronutrient intake; metabolic syndrome; HOMA index; cross-sectional; gender

INTRODUCTION

Metabolic syndrome (MS) is a constellation of physical and metabolic characteristics common among insulin-resistant individuals (1). The presence of MS carries an increased risk of developing diabetes (2–5) as well as morbidity and mortality due to cardiovascular disease (CVD) (6–11). Diet has been related to several characteristics of MS (12–15), and some data suggest a link between diet and insulin resistance (IR) (16). Many aspects of the diet composition (carbohydrate, fat, fiber, vitamins, alcohol) have been considered to be important in the modulation of insulin resistance, but in the last few years, more attention has been given to the ability of the quality of dietary fat, independent of the total amount, to influence insulin sensitivity and, throughout this, the risk of type 2 diabetes. Certainly, the relevance of this specific topic arose especially from studies performed on animals, where diets rich in saturated fat clearly worsened insulin sensitivity, while those rich in unsaturated fat, particularly short and long chain ω-3 fatty acids, clearly improved insulin action. On the other hand, studies in humans in terms of this particular aspect are not many and, moreover, the results are not always in agreement and not as convincing as those in animals (17).

In the Strong Heart Study (SHS), a longitudinal, population-based study of CVD and CVD risk factors in American Indians (AI), MS prevalence was 43.6% and 56.7% in men and women, respectively (18), rates much higher than those of the general U.S. population. MS is prevalent even among non-diabetic AI. Because MS, IR, and nutrient intake differ in men and women in this population, our analyses were stratified by gender. Gender may not only make a difference in MS prevalence, but may also have a role in determining the association between diet and MS (19,20). This study examines the cross-sectional associations between dietary intake, gender, a proxy measure of IR, and the presence of MS in nondiabetic AI without established CVD.

METHODS

Study population

The SHS was initiated in 1988 to investigate CVD and its risk factors in AI (21). The SHS design, recruitment, methods, and laboratory techniques have been reported (21–23). Briefly, the SHS cohort consisted of 4,549 participants ages 45–79 years undergoing baseline (1989–1992), second (1993–1995), and third (1997–1999) examinations. The age, body mass index (BMI), and self-reported diabetes and hypertension of non-participants were similar to those of participants (23). The Indian Health Service and Institutional Review Boards and participating tribes and the MedStar Research Institute approved the study. Written informed consent was obtained from each participant.

The current analysis was based on data from participants with complete dietary data from the second examination of the SHS (SHS2).

Measurements

Dietary data were collected via a single-24-hour dietary recall for all participants at the SHS2 examination. Interviewers were centrally trained and certified in data collection and form completion according to standardized methods (24). Use of dietary supplements was assessed as part of the medication inventory. Dietary intake was analyzed using the Minnesota Nutrition

Data System (NDS) (NDS Version 2.1) (25,26). Because calculation of trans fatty acids was not available in NDS Version 2.1, final calculations were conducted using Nutrition Coordinating Center Nutrient Database Version 36 (Nutrition Data System for Research (NDS-R) 2005 Minneapolis, MN). The NDS-R database updates analytic data while retaining nutrient profiles true to the version used for data collection (27).

Methods for measuring CVD risk factors (e.g., BMI, and blood pressure) have been described, and laboratory methods have been published (21–22). Briefly, height was measured with the participant standing erect in the Frankfort plane, using a stadiometer fixed to the wall. Weight was measured using a Tanita 627-a scale (Orange County Medical Sales, LLC, California), which was calibrated and adjusted daily. BMI was calculated as weight (kg)/height (m2). After the participant had been seated at rest for 5 minutes, three consecutive blood pressure measurements using the first and fifth Korotkoff sounds were made on the right arm with the appropriate size cuff and a Baum mercury sphygmamonometer (W.A. Baum Company, Copiaqhe, New York). Cholesterol, triglyceride, and fasting glucose (FG) levels were determined by enzymatic methods using a Hitachi chemistry analyzer and consistent, standardized reagents (Boehringer Mannheim Diagnostics, Indianapolis, IN) in the morning after at least a 12-hour overnight fast. Diabetes was defined according to American Diabetes Association criteria (28) (i.e., taking insulin or oral antidiabetic medication or having an FG concentration ≥ 126 mg/dl [6.993 mmol/l]). Cigarette smoking and alcohol consumption were determined by questionnaire.

Definition of metabolic syndrome

The National Cholesterol Education Program's Adult Treatment Panel III definition of MS includes the presence of three or more of the following characteristics: a) waist circumference > cm in men or > 88 cm in women; b) triglycerides > 1.69 mmol/L (150 mg/dl); c) high-density lipoprotein cholesterol < 1.04 mmol/L (40 mg/dl) in men and < 1.29 mmol/L (50 mg/dl) in women; d) blood pressure ≥ 130/85 mm Hg; and e) FG between 6.1 and 6.9 mmol/L (110–125 mg/dl). The characteristics that define MS are also associated with insulin resistance (IR) (1).

Measurement of baseline IR

The homeostasis model assessment (HOMA) estimates IR with the equation *[Fasting insulin (FI) (μU/ml) · FG (mmol/l)]/22.5* (29). HOMA score correlates with euglycemic clamp measures in men and women, younger and older adults, and obese and nonobese individuals (29–32). SHS data (33) show HOMA score correlates well with insulin sensitivity measured by the Minimal Model in individuals with FG < 126 mg/dl [6.993 mmol/l] (34).

Sample selection

The analysis was based on data from participants with complete dietary data from SHS2 (n = 3,450). Diabetic participants (n = 1,680) were excluded because the SHS data have demonstrated that the HOMA model, a key predictor in this report, is not an accurate reflection of IR at FG > 126 mg/dl [6.993 mmol/l], and because diabetic individuals are often advised to change their dietary intakes as part of routine management (35). Participants with established CVD (definite myocardial infarction, coronary heart disease, and stroke, n = 101) were also excluded because of possible confounding. Additional exclusion criteria included reported energy intake ≤ 600 kcal/day (n = 66); individuals with conditions affecting energy intake, such as dialysis, kidney transplant, or liver cirrhosis (n = 65); indeterminate diabetes status (n = 22); and missing data for FI (n = 58). The final sample for the MS and HOMA analyses consisted of 1,516 and 1,458 participants respectively, ages 47 to 80 years at SHS2. Additional dietary data were collected at the third SHS exam (SHS3; 1997–1999). Compared to SHS2, the SHS3 sample size was reduced by 30%.

Statistical methodology

Intakes of macronutrients were considered the primary independent variables and HOMA score or MS the dependent variable. Intakes of nutrients were first compared by MS status in men and women using a t-test, Wilcoxon's rank-sum test, or χ^2 test as appropriate. Next, nutrient intakes were compared across tertiles of HOMA in men and women, using linear regression models. Odds of developing MS and HOMA means were then evaluated across tertiles of macronutrients. Logistic regression was used to study associations among tertiles of macronutrient intakes and MS, adjusted for age, study center, education, smoking, alcohol consumption, and energy intake. Adding BMI as a covariate did not change the results. Analysis of covariance (ANCOVA) was used to compare HOMA scores within tertiles of macronutrient intake, adjusted for the covariates listed in the logistic regression model plus BMI. To confirm gender differences, interactions were examined in the multivariate-adjusted models for tertile of macronutrients with gender. $P < .05$ was used to confirm evidence for gender differences.

The Hosmer-Lemeshow goodness-of-fit test was used to assess the fit of the binary response models; R-square was applied for the continuous response goodness-of-fit test. Tests for trend were conducted by modeling the median of each tertile-defined category as a continuous variable in the models. HOMA score was log-transformed before being entered into the models and was back transformed when presenting the results for means and confidence intervals among tertiles of macronutrient intake. Models were run expressing macronutrients in grams or in percentage of calories; these data are shown in the tables. All analyses were performed with SAS Version 9.0 (SAS Institute Inc, Cary, NC).

RESULTS

Of the 1,516 participants meeting selection criteria, 494 (54.1%) of 913 women and 218 (36.2%) of 603 men had MS (see Table 1). Compared to men, women were older, had a higher BMI and waist circumference, were more educated, smoked and consumed alcohol less frequently, had higher triglyceride and HDL-cholesterol levels and lower diastolic blood pressure and fasting glucose.

Energy intake, percentage of calories from total fat, saturated fatty acids, and monounsaturated fatty acids in those who had MS and those who did not were similar across gender (see Table 2). There was no difference between individuals with and without MS in percentage of calories derived from total, saturated fatty acid (SFA), or monounsaturated fatty acid (MUFA). However, women with MS consumed 0.5% of calories of polyunsaturated fatty acid (PUFA) and omega-6 less than those without MS, a statistically but not clinically significant difference that was not observed among men.

Simple carbohydrate intake, whether expressed as grams, as percentage of total carbohydrate intake, or of total calories, was higher among men with MS than among those without MS (see Table 2). Men with a higher percentage of calories derived from vegetable protein had less MS. None of these associations were observed in women.

Consistent with the data in Table 2, simple carbohydrate intakes, whether expressed as grams, as percentage of total carbohydrate intake, or of total calories, were higher with increasing HOMA score in men but not in women (see Table 3). With the exception of trans fatty acid intake, which increased along with HOMA score, no other trends were observed across HOMA tertiles for other macronutrients among men. Energy intake was higher in women across increasing tertiles of HOMA, but not for other macronutrients as percent of calories (see Table 3).

In a multivariate analysis, we observed that a higher PUFA intake was associated with lower MS prevalence in women but not men (p for trend = 0.03 across PUFA tertiles in women and 0.43 across tertiles in men) (see Table 4). The p-value for interaction between gender and PUFA was 0.04. A marginal association was observed in a multivariate analysis between PUFA and HOMA score (p for trend = 0.06) in women (see Table 5).

The relation between PUFA and MS was seen for omega-6 only in women (P for interaction = 0.04). No association was observed between omega-3 fatty acids and HOMA score in either gender (Table 5). There was no association between omega-6 and HOMA in either gender.

Simple carbohydrate intake as percentage of calories was positively and significantly associated with MS prevalence in men (p for trend = 0.03) but not women (see Table 4), once again suggesting that gender may modify the association between simple carbohydrates and MS prevalence (P for interaction = 0.03). Adding total carbohydrates as percentage of calories to the model did not change the results. Total carbohydrate intake as a percentage of total calories was negatively associated with HOMA score in women but not in men, p for trend = 0.04 (see Table 5).

In multivariate analyses, higher protein intake, especially that derived from animals, was associated with a higher MS prevalence and HOMA score in women, but not men (see Table 4 and Table 5). The interaction terms between gender and association of total protein intake or animal protein intake and MS or HOMA were not significant. There was a significant association between lower intakes of vegetable protein and a higher MS prevalence in men (P = 0.07).

In all logistic regression models, the p-values for the Hosmer-Lemeshow goodness-of-fit test were 0.11 to 0.78, and R-squares were 0.35 to 0.42 in all ANCOVA models. These findings suggest that binary response models fit the data well, and that the regression models explained a moderate level of variability in continuously distributed outcomes. The addition of AI blood quantum data and family history of diabetes did not change these results.

DISCUSSION

MS is thought to be determined by genetic (36) as well as environmental and behavioral factors (18). In this population with a high prevalence of MS and IR, several relations between these abnormalities and nutrient intake were found, and the relations differed by gender. The current data suggest that macronutrient intake is associated cross-sectionally with MS and IR. Of interest is the interaction of gender and specific macronutrients on the association with MS and IR and the possible gender differences in relations among dietary components and the risk of MS and IR.

Review of the literature revealed no earlier analyses examining the association between dietary intake and MS or HOMA score in AI or in other populations characterized by extremely high rates of obesity. In a cross-sectional study of middle-aged Irish men and women, significant differences were found in the gender, socio-economic status, and behavior profiles of participants in three dietary groups (37).

PUFA intake in the current study was inversely associated with both MS and HOMA. This observation is consistent with findings from animal and in vitro studies suggesting that intake of PUFA improves insulin sensitivity (16). A principal factor analysis was performed to define specific fatty acid factors in men participating in a population-based cohort study -- the Uppsala Longitudinal Study of Adult Men. In that study, relations between fatty acid factors and MS were investigated in cross-sectional and prospective analyses. The low-linoleic, omega-6 fatty acid, predicted MS development over 20 years, independent of smoking habits, physical

activity, and BMI (odds ratio: 1.51; 95% CI: 1.28, 1.79) (38). Possible mechanisms for this association are that PUFA may act as a potential anti-inflammatory agent (39) or as a nutrient sensor to determine whether fatty acids are to be stored or oxidized. In this way, PUFA may function as nutritional factor that reduces the risk of developing hepatic lipotoxicity and insulin resistance (40).

In the current study the relation was seen primarily with omega-6 fatty acids, although the analyses may have had limited power for omega-3, because overall consumption was low. A possible explanation for the interactions between gender and PUFA intake and the association with MS is the difference between genders in sources of PUFA. Men with and without MS consumed a higher percentage of their PUFA intake from traditional AI foods compared with women with and without MS (25.9%, 23% in men, respectively, vs. 20.2%, 19.4% in women). Women without MS consumed 19.4% of their PUFA intake from white potatoes and starchy vegetables, compared with 15.4% consumed by women with MS. Women with MS consumed a higher percentage of their PUFA intake from crackers and salty snacks made from grain products.

An association between higher animal protein intake and MS and HOMA score was observed in women. The mechanisms of this are not clear but may be related to the higher saturated fatty acids contained in animal protein.

A positive association was observed between simple carbohydrate intake and MS in men. Only a few observational studies have examined the intake of simple carbohydrates in relation to IR and no positive associations have been observed (41–42). A possible explanation for this finding in the SHS may be that in women the sources of simple carbohydrate also contained fiber (e.g., bananas). The effects of total fat and carbohydrate content on IR have been debated. Although animal studies show increases in IR accompanying high-fat diets, this increase has not been confirmed in humans (43). Also, most observational studies have not found significant associations between intake of total carbohydrate and estimates of IR (32,44–46). The current data support these findings, confirming that IR and MS are unlikely to be influenced by either total fat or carbohydrate, but instead may be related to specific fats or sources of carbohydrate. The current data leave open the possibility of gender differences in these associations.

This study's strengths include its large cohort with a wide range of IR, standardized measures, and quality dietary data. This study is limited by its cross-sectional design; associations between macronutrient intake and MS or HOMA must be interpreted with caution, given the possibility of reverse causation. Another important consideration is that this report uses dietary measurements performed over a dozen years ago. However, use of dietary data collected at the more recent exam would have reduced the sample size by 30% because of the new diabetes cases that developed between the second and third exams. Importantly, dietary patterns did not change appreciably across SHS Phases 2 and 3, but both genders reported consuming fewer calories and women had a lower mean BMI at Phase 3. It is important to consider some specific changes across these phases.

Compared with women in SHS2, those in SHS3 consumed a lower percentage of calories from saturated fatty acid (SFA) ($P = 0.03$), polyunsaturated fatty acid (PUFA) ($P = 0.008$) and simple carbohydrates ($P = 0.009$). Men in SHS3 consumed a higher percentage of calories from MUFA ($P = 0.002$), less PUFA ($P = 0.03$), and less vegetable protein ($P = 0.03$). The differences in macronutrient intake in SHS3 compared with SHS2 may reflect women's changes toward a healthier diet, which is also reflected in a lower BMI. Alternatively, these changes may arise from less favorable health status and/or the well-described loss of weight and nutritional changes that occur with aging. Despite the reduced power associated with the smaller sample size in SHS3, results were generally consistent with those observed from SHS2.

This study represents one of the largest sources of data on AI dietary patterns that is currently available, and is, therefore, of considerable interest. This study is limited by its use of a single 24-hour recall (47), because diet was measured only once. The 24-hour recall, however, can provide detailed information on specific foods (48) and is considered ideal for intercultural comparisons of mean dietary intake levels, because it allows for detailed reporting of heterogeneous types of food (47).

CONCLUSIONS

This cross sectional study suggests that in addition to simple carbohydrates, PUFA intakes may be associated with MS and IR in AI, and also that gender may modify the association between dietary intake and MS and IR in AI. Future studies should focus on the longitudinal association between dietary intake and incidence of MS and IR in AI and other populations.

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Table 1

Baseline characteristics of Strong Heart Study 2 (1993–1995) participants with complete dietary data, by gender

	Men (n = 603)	Women (n = 913)	P-value
Age (years)	58.8 ± 7.9	59.9 ± 8.2	<0.01
Body mass index (kg/m^2)	28.9 ± 5.0	31.1 ± 6.7	<0.001
Education (years)	11.4 ± 3.1	11.7 ± 3.0	0.05
Smoking			<0.001
Nonsmoker	92 (15.6)	344 (38.4)	
Former smoker	239 (40.5)	279 (31.2)	
Current smoker	259 (43.9)	272 (30.4)	
Reported alcohol consumption			<0.001
Nondrinker	36 (6.0)	246 (27.3)	
Former drinker	254 (42.4)	401 (44.5)	
Current drinker[a]	309 (51.6)	255 (28.2)	
Waist circumference (cm)	102 ± 12	105 ± 16	<0.001
Triglycerides (mg/dl)	130 ± 79	140 ± 81	0.02
HDL-C (mg/dl)	40 ± 14	46 ± 14	<0.001
Systolic blood pressure (mm Hg)	126 ± 18	126 ± 19	0.99
Diastolic blood pressure (mm Hg)	77 ± 11	73 ± 9	<0.001
Fasting glucose (mg/dl)	103 ± 10	102 ± 11	0.04
Metabolic syndrome (%)	218 (36.2)	494 (54.1)	<0.001
HOMA index	3.9 ± 3.2	4.5 ± 4.1	<0.001

Note: HDL-C = high-density lipoprotein cholesterol; HOMA = homeostasis model assessment, an estimate of insulin resistance. Conversion factors to SI units are as follows: triglycerides, multiply by 0.0113 to calculate mmol/L; HDL-C, multiply by 0.0259 to calculate mmol/L; fasting glucose, multiply by 0.0555 to calculate mmol/L.

[a]The current drinkers are defined as drinking at least one drink of any kind of alcoholic beverage 442 in the past and drinking today.

Table 2

Macronutrient intake by metabolic syndrome status in men and women, Strong Heart Study (2nd examination, 1993–1995), (N = 1516)

	Men		P value	Women		P value
	Metabolic syndrome			Metabolic syndrome		
	Yes	No		Yes	No	
Energy (kcal)	2093 ± 882	2074 ± 942	0.81	1739 ± 677	1739 ± 632	0.99
Total fat						
(g)	84.6 ± 44.8	83.1 ± 46.6	0.69	67.0 ± 33.6	67.8 ± 31.1	0.72
% of calories	35.8 ± 9.0	35.5 ± 10.3	0.66	34.2 ± 9.3	35.0 ± 8.9	0.22
Saturated fatty acids						
(g)	28.8 ± 15.2	28.7 ± 17.0	0.91	22.8 ± 12.1	22.9 ± 11.7	0.86
% of calories	12.3 ± 3.7	12.2 ± 4.1	0.76	11.7 ± 3.9	11.8 ± 3.8	0.70
Monounsaturated fatty acids						
(g)	32.8 ± 18.4	32.0 ± 19.1	0.63	25.5 ± 13.7	25.5 ± 12.7	0.99
% of calories	13.8 ± 4.1	13.6 ± 4.5	0.49	13.0 ± 4.1	13.1 ± 3.9	0.61
Polyunsaturated fatty acids						
(g)	15.5 ± 11.5	14.6 ± 9.6	0.33	12.6 ± 8.1	13.3 ± 7.8	0.16
% of calories	6.5 ± 3.1	6.3 ± 3.1	0.61	6.4 ± 3.0	6.9 ± 3.3	0.008
Omega 3						
(g)	1.5 ± 1.2	1.4 ± 1.1	0.56	1.2 ± 0.8	1.3 ± 0.9	0.24
% of calories	0.6 ± 0.3	0.6 ± 0.4	0.96	0.6 ± 0.4	0.7 ± 0.4	0.08
Omega 6						
(g)	13.9 ± 10.4	13.1 ± 8.7	0.31	11.3 ± 7.3	12.0 ± 7.1	0.16
% of calories	5.8 ± 2.8	5.7 ± 2.8	0.62	5.7 ± 2.7	6.2 ± 3.0	0.008
Trans fatty acids						
(g)	5.8 ± 4.2	5.5 ± 4.2	0.39	4.8 ± 3.6	4.7 ± 3.3	0.73
% of calories	2.4 ± 1.4	2.3 ± 1.4	0.44	2.4 ± 1.4	2.4 ± 1.3	0.90
Carbohydrates						
(g)	255 ± 119	246 ± 127	0.41	222 ± 98	222 ± 97	0.92
% of calories	48.9 ± 11.3	47.7 ± 12.3	0.25	51.3 ± 11.8	51.0 ± 11.2	0.66
Simple carbohydrates						
(g)	105 ± 72	92 ± 73	0.04	97 ± 64	99 ± 65	0.66

	Men		P value	Women		P value
	Metabolic syndrome			Metabolic syndrome		
	Yes	No		Yes	No	
% of calories	19.8 ± 10.6	17.4 ± 11.1	0.01	22.2 ± 12.0	22.2 ± 10.9	0.96
% of carbohydrates	39.1 ± 17.1	35.2 ± 17.8	0.009	41.8 ± 17.3	42.6 ± 16.3	0.48
Protein						
(g)	77.5 ± 34.9	80.6 ± 40.3	0.32	65.9 ± 29.7	63.9 ± 27.0	0.30
% of calories	15.3 ± 4.3	15.8 ± 4.4	0.13	15.3 ± 4.3	15.0 ± 4.4	0.26
Animal protein						
(g)	49.5 ± 29.4	50.5 ± 33.4	0.72	42.3 ± 25.4	40.8 ± 23.6	0.36
% of calories	9.8 ± 4.8	9.9 ± 5.0	0.91	9.7 ± 4.6	9.5 ± 4.8	0.57
Vegetable protein						
(g)	27.7 ± 15.8	29.8 ± 19.1	0.15	23.4 ± 12.2	22.9 ± 12.0	0.56
% of calories	5.4 ± 2.4	5.9 ± 2.9	0.02	5.5 ± 2.3	5.4 ± 2.2	0.36

Table 3

Macronutrient intake by HOMA tertiles in men and women, Strong Heart Study (2nd examination, 1993–1995)

	Men (n = 577)				Women (n = 881)			
	1st tertile	2nd tertile	3rd tertile	P for trend	1st tertile	2nd tertile	3rd tertile	P for trend
Energy (kcal)	2105 ± 887	2004 ± 902	2124 ± 976	0.44	1650 ± 589	1771 ± 667	1789 ± 699	0.01
Total fat								
(g)	83.0 ± 43.5	82.8 ± 45.6	84.4 ± 47.0	0.31	65.1 ± 29.5	66.5 ± 32.5	69.7 ± 34.5	0.14
% of calories	35.1 ± 10.7	36.5 ± 9.2	35.3 ± 9.2	0.61	35.5 ± 9.5	33.5 ± 9.0	34.6± 8.7	0.26
Saturated fatty acids								
(g)	29.1 ± 16.4	27.9 ± 15.7	28.8 ± 16.4	0.03	22.0 ± 11.0	22.8 ± 11.8	23.6 ± 12.5	0.19
% of calories	12.3 ± 4.2	12.4 ± 3.8	12.0 ± 3.7	0.90	11.9 ± 4.0	11.5 ± 3.7	11.7 ± 3.7	0.28
Monounsaturated fatty acids								
(g)	31.7 ± 17.7	32.4 ± 18.7	32.5 ± 19.5	0.19	24.5 ± 12.1	25.4 ± 13.3	26.4 ± 14.0	0.14
% of calories	13.4 ± 4.6	14.2 ± 4.1	13.5 ± 4.1	0.33	13.3 ± 4.1	12.7 ± 4.0	13.1 ± 3.9	0.45
Polyunsaturated fatty acids								
(g)	14.5 ± 9.3	14.9 ± 10.5	15.2 ± 10.7	0.41	12.9 ± 7.7	12.4 ± 8.1	13.4 ± 8.1	0.49
% of calories	6.2 ± 3.1	6.5 ± 3.0	6.4 ± 3.2	0.96	7.1 ± 3.4	6.2 ± 3.0	6.6 ± 2.9	0.17
Omega 3								
(g)	1.5 ± 1.2	1.3 ± 1.0	1.5 ± 1.2	0.18	1.3 ± 1.0	1.2 ± 0.8	1.3 ± 0.8	0.56
% of calories	0.6 ± 0.4	0.6 ± 0.3	0.6 ± 0.4	0.26	0.7 ± 0.5	0.6 ± 0.4	0.6 ± 0.4	0.10
Omega 6								
(g)	13.0 ± 8.4	13.5 ± 9.6	13.7 ± 9.7	0.39	11.6 ± 6.9	11.1 ± 7.3	12.1 ± 7.4	0.43
% of calories	5.5 ± 2.8	5.9 ± 2.7	5.7 ± 3.0	0.89	6.4 ± 3.1	5.6 ± 2.8	6.0 ± 2.6	0.24
Trans fatty acids								
(g)	5.1 ± 3.7	5.9 ± 4.7	5.7 ± 4.2	0.002	4.6 ± 3.2	4.6 ± 3.6	5.0 ± 3.5	0.51
% of calories	2.2 ± 1.3	2.6 ± 1.5	2.3 ± 1.3	0.05	2.5 ± 1.4	2.3 ± 1.4	2.5 ± 1.3	0.38
Carbohydrates								
(g)	252± 124	237 ± 118	260 ± 133	0.23	208 ± 91	233 ± 101	226 ± 99	0.01
% of calories	48.0 ± 12.6	47.4 ± 11.6	49.0 ± 11.4	0.33	50.2 ± 12.3	52.7 ± 11.0	50.8 ± 11.2	0.540
Simple carbohydrates								
(g)	93.6 ± 70.0	91.9 ± 70.1	103.5 ± 79.1	0.05	90.9 ± 61.7	103.6 ± 66.8	98.4 ± 64.7	0.14
% of calories	17.7 ± 11.3	17.8 ± 11.0	19.2 ± 10.8	0.04	21.7 ± 11.4	23.2 ± 11.3	21.9 ± 11.9	0.74

	Men (n = 577)				Women (n = 881)			
% of carbohydrates	35.6 ± 18.0	36.2 ± 17.9	37.8 ± 17.3	0.05	41.8 ± 17.0	42.9 ± 16.5	41.5 ± 17.1	0.97
Protein								
(g)	80.5 ± 39.4	77.0 ± 36.3	80.5 ± 38.2	0.25	62.3 ± 26.6	64.5 ± 28.5	67.4 ± 29.9	0.02
% of calories	15.5 ± 4.5	15.8 ± 4.1	15.7 ± 4.5	0.96	15.3 ± 4.7	14.7 ± 4.0	15.3 ± 4.2	0.94
Animal protein								
(g)	50.0 ± 33.7	49.4 ± 29.4	50.5 ± 30.2	0.09	40.1 ± 22.7	40.6 ± 24.6	43.4 ± 25.8	0.11
% of calories	9.5 ± 4.9	10.2 ± 4.6	9.9 ± 5.0	0.39	9.9 ± 5.2	9.2 ± 4.4	9.7 ± 4.4	0.62
Vegetable protein								
(g)	30.2 ± 20.0	27.4 ± 16.6	29.7 ± 17.8	0.95	22.0 ± 11.4	23.7 ± 12.7	23.8 ± 12.2	0.03
% of calories	5.9 ± 3.1	5.6 ± 2.3	5.7 ± 2.7	0.98	5.4 ± 2.1	5.5 ± 2.2	5.5 ± 2.4	0.24

Note: HOMA = homeostasis model assessment, an estimate of insulin resistance.

Table 4

Odds ratio (95% CI) of having MS, by tertile of dietary intake,[a], Strong Heart Study (2nd examination, 1993–1995)

	Men (n = 603)				Women (n = 913)			
	1st tertile	2nd tertile	3rd tertile	P for trend	1st tertile	2nd tertile	3rd tertile	P for trend
Total fat	1	1.51 (0.97–2.33)	1.13 (0.74–1.74)	0.58	1	0.80 (0.57–1.11)	0.98 (0.71–1.37)	0.93
SFA	1	1.41 (0.92–2.17)	1.13 (0.73–1.73)	0.64	1	0.82 (0.58–1.14)	0.99 (0.71–1.37)	0.99
MUFA	1	1.07 (0.70–1.66)	1.15 (0.76–1.76)	0.51	1	0.79 (0.56–1.10)	0.99 (0.71–1.38)	0.97
PUFA	1	1.12 (0.73–1.71)	1.19 (0.78–1.82)	0.43	1	0.80 (0.57–1.12)	0.69 (0.49–0.95)	0.03
Omega 3	1	1.21 (0.80–1.85)	1.14 (0.74–1.75)	0.61	1	0.94 (0.67–1.31)	0.76 (0.55–1.05)	0.09
Omega 6	1	1.27 (0.83–1.94)	1.25 (0.82–1.92)	0.35	1	0.89 (0.64–1.25)	0.70 (0.50–0.97)	0.03
TFA	1	1.49 (0.97–2.30)	1.21 (0.79–1.86)	0.51	1	0.90 (0.64–1.25)	0.95 (0.68–1.32)	0.79
Carbohydrates	1	1.26 (0.82–1.93)	1.00 (0.65–1.54)	0.99	1	1.06 (0.76–1.47)	1.04 (0.74–1.44)	0.83
Simple carbohydrates	1	1.27 (0.82–1.97)	1.63 (1.06–2.52)	0.03	1	0.78 (0.56–1.09)	1.02 (0.73–1.42)	0.86
Protein	1	0.83 (0.55–1.27)	0.77 (0.50–1.19)	0.24	1	1.22 (0.88–1.70)	1.36 (0.98–1.90)	0.07
Animal protein	1	1.03 (0.67–1.56)	0.91 (0.59–1.39)	0.64	1	0.96 (0.69–1.34)	1.36 (0.98–1.90)	0.05
Vegetable protein	1	0.93 (0.61–1.41)	0.68 (0.44–1.05)	0.07	1	1.06 (0.76–1.47)	1.06 (0.75–1.48)	0.76

Note: CI = confidence interval; MS = metabolic syndrome; SFA = saturated fatty acid; MUFA = monounsaturated fatty acid; PUFA = polyunsaturated fatty acid; TFA = trans fatty acid.

[a]Intakes are included in the models as percent of calories.

All models were adjusted for age, study center, education, smoking, drinking, and energy intake.

Table 5

Adjusted mean (95% CI) of HOMA scores, by tertile of dietary intake,[a] Strong Heart Study (2nd examination, 1993–1995)

	Men (n = 577)				Women (n = 881)			
	1st tertile	2nd tertile	3rd tertile	P for trend	1st tertile	2nd tertile	3rd tertile	P for trend
Total fat	2.88 (2.58–3.21)	3.04 (2.79–3.32)	2.90 (2.60–3.23)	0.93	3.35 (3.01–3.72)	3.47 (3.23–3.74)	3.26 (2.94–3.62)	0.78
SFA	2.91 (2.64–3.20)	3.06 (2.81–3.34)	2.85 (2.59–3.14)	0.79	3.40 (3.11–3.73)	3.37 (3.13–3.63)	3.30 (3.02–3.60)	0.67
MUFA	2.75 (2.48–3.06)	2.90 (2.66–3.16)	3.17 (2.86–3.51)	0.11	3.54 (3.21–3.90)	3.31 (3.08–3.56)	3.24 (2.94–3.56)	0.28
PUFA	3.00 (2.74–3.28)	2.85 (2.62–3.10)	2.97 (2.73–3.24)	0.88	3.45 (3.20–3.72)	3.53 (3.28–3.80)	3.12 (2.89–3.35)	0.06
Omega 3	2.92 (2.68–3.18)	2.93 (2.69–3.20)	2.96 (2.71–3.24)	0.81	3.33 (3.08–3.59)	3.62 (3.36–3.90)	3.15 (2.92–3.39)	0.32
Omega 6	3.00 (2.75–3.28)	2.88 (2.64–3.14)	2.93 (2.69–3.20)	0.73	3.42 (3.18–3.69)	3.56 (3.31–3.83)	3.11 (2.89–3.35)	0.09
TFA	2.83 (2.59–3.09)	2.96 (2.72–3.23)	3.03 (2.78–3.30)	0.28	3.36 (3.12–3.63)	3.34 (3.10–3.59)	3.37 (3.13–3.63)	0.96
Carbohydrates	2.79 (2.42–3.22)	3.00 (2.75–3.27)	3.03 (2.62–3.49)	0.54	3.82 (3.37–4.34)	3.31 (3.08–3.56)	3.00 (2.65–3.39)	0.04
Simple carbohydrates	2.83 (2.58–3.11)	2.84 (2.61–3.09)	3.15 (2.86–3.47)	0.16	3.37 (3.10–3.66)	3.42 (3.18–3.67)	3.29 (3.02–3.57)	0.70
Protein	2.88 (2.63–3.16)	2.90 (2.66–3.16)	3.03 (2.77–3.32)	0.47	3.15 (2.91–3.42)	3.28 (3.05–3.53)	3.64 (3.37–3.94)	0.02
Animal protein	2.81 (2.55–3.10)	2.97 (2.73–3.23)	3.04 (2.75–3.36)	0.33	3.05 (2.80–3.31)	3.41 (3.17–3.67)	3.64 (3.35–3.94)	0.007
Vegetable protein	2.87 (2.62–3.13)	2.92 (2.68–3.18)	3.02 (2.77–3.31)	0.43	3.49 (3.24–3.77)	3.21 (2.99–3.46)	3.37 (3.13–3.63)	0.53

Note: CI = confidence interval; HOMA = homeostasis model assessment, an estimate of insulin resistance; SFA = saturated fatty acid; MUFA = monounsaturated fatty acid; PUFA = polyunsaturated fatty acid; TFA = trans fatty acid.

[a] Intakes were included as percentage of total calories. All models were adjusted for age, study center, education, smoking, drinking, body mass index, and energy intake.