The Journal of Nutrition
Nutritional Epidemiology

Adherence to an (n-3) Fatty Acid/Fish Intake Pattern Is Inversely Associated with Metabolic Syndrome among Puerto Rican Adults in the Greater Boston Area[1–3]

Sabrina E. Noel,[4,5] P. K. Newby,[5,6] Jose M. Ordovas,[4] and Katherine L. Tucker[4,7]*

[4]USDA Human Nutrition Research Center on Aging, Tufts University, Boston, MA 02111; [5]Department of Pediatrics, Boston University School of Medicine, Boston Medical Center, Boston, MA 02118; [6]Department of Epidemiology, Boston University School of Public Health, Boston, MA 02118; and [7]Department of Health Sciences, Northeastern University, Boston, MA 02115

Abstract

Combinations of fatty acids may affect risk of metabolic syndrome. Puerto Ricans have a disproportionate number of chronic conditions compared with other Hispanic groups. We aimed to characterize fatty acid intake patterns of Puerto Rican adults aged 45–75 y and living in the Greater Boston area (n = 1207) and to examine associations between these patterns and metabolic syndrome. Dietary fatty acids, as a percentage of total fat, were entered into principle components analysis. Spearman correlation coefficients were used to examine associations between fatty acid intake patterns, nutrients, and food groups. Associations with metabolic syndrome were analyzed by using logistic regression and general linear models with quintiles of principal component scores. Four principal components (factors) emerged: factor 1, short- and medium-chain SFA/dairy; factor 2, (n-3) fatty acid/fish; factor 3, very long-chain (VLC) SFA and PUFA/oils; and factor 4, monounsaturated fatty acid/*trans* fat. The SFA/dairy factor was inversely associated with fasting serum glucose concentrations (P = 0.02) and the VLC SFA/oils factor was negatively related to waist circumference (P = 0.008). However, these associations were no longer significant after additional adjustment for BMI. The (n-3) fatty acid/fish factor was associated with a lower likelihood of metabolic syndrome (Q5 vs. Q1: odds ratio: 0.54, 95% CI: 0.34, 0.86). In summary, principal components analysis of fatty acid intakes revealed 4 dietary fatty acid patterns in this population. Identifying optimal combinations of fatty acids may be beneficial for understanding relationships with health outcomes given their diverse effects on metabolism. J. Nutr. 140: 1846–1854, 2010.

Introduction

The prevalence of metabolic syndrome in the US has been increasing (1) and is particularly high for older adults and certain ethnic groups. Hispanics have the highest reported prevalence of metabolic syndrome (2) and are more likely to be affected by type 2 diabetes than non-Hispanic whites (3–6). The term "Hispanic" encompasses several population subgroups that are diverse with respect to sociodemographics, health outcomes, and diet (6,7). Most health research on Hispanics has focused on Mexican Americans due to their majority as a Hispanic subgroup. Puerto Ricans, the second largest Hispanic subgroup in the US, are burdened by excess chronic health conditions compared with other Hispanic groups (8). We previously showed that Puerto Rican elders in Massachusetts were twice as likely to have type 2 diabetes compared with a representative neighborhood-based sample of non-Hispanic whites (4) and that up to 50% had metabolic syndrome (9).

Metabolic syndrome is characterized by alterations in anthropometrics, blood pressure, and lipoprotein and fasting glucose concentrations (10,11). Lifestyle behaviors such as diet play an important role in the development of metabolic syndrome (12). Recent research on dietary fat has shifted focus to quality of fat and fatty acids in relation to disease risk (13). Total fat and type of dietary fat consumed have been associated with metabolic syndrome (14) and its components (14,15), but results are inconsistent (16). Subtypes of fatty acids, particularly PUFA, have been shown to be associated with insulin sensitivity (15), blood lipoproteins (17), blood pressure (13,15), and body composition (18).

A combination of unsaturated fatty acids along with moderate total fat intake may offer the most benefit for several metabolic-related risk factors (13). In addition to type of dietary fat, quality can be represented as the composite of the individual fatty acids consumed. Warensjo et al. (19) recently examined the

[1] Supported by NIH grant number P01-AG023394 and by the USDA, Agriculture Research Institute agreement number 58-1950-7-707.
[2] Author disclosures: S. E. Noel, P. K. Newby, J. M. Ordovas, and K. L. Tucker, no conflicts of interest.
[3] Supplemental Table 1 is available with the online posting of this paper at jn.nutrition.org.
* To whom correspondence should be addressed. E-mail: kl.tucker@neu.edu.

Manuscript received March 18, 2010. Initial review completed April 19, 2010. Revision accepted July 12, 2010.
First published online August 11, 2010; doi:10.3945/jn.110.124297.

relationship between serum fatty acids and metabolic syndrome in a Swedish population-based cohort of men aged 50 y and older using principal components analysis to generate 3 fatty acid factors. A factor low in linoleic acid was associated with the development of metabolic syndrome in men, whereas an (n-3) PUFA factor was protective.

There is limited information on the dietary habits of Puerto Ricans living on the U.S. mainland (20–22). Puerto Ricans in Massachusetts reported diets lower in total and saturated fat and higher in PUFA than non-Hispanic whites (20). Modifying dietary intake may be most beneficial for reducing diet-related diseases. In this study, we aimed to characterize fatty acid patterns of Puerto Rican adults living in the Greater Boston area and examine associations between fatty acid patterns and metabolic syndrome in this unique population.

Materials and Methods

Study population. The Boston Puerto Rican Health Study is a longitudinal investigation of the relationship among physiological dysregulation, nutrition, and health outcomes in Puerto Rican adults living in the Greater Boston area (23). Participants between the ages of 45 and 75 y were recruited through door-to-door enumeration using year 2000 Census data to identify locations of high Hispanic density. Census blocks containing at least 10 Hispanic adults within the study age range were randomly selected for enumeration. Households from the blocks were visited up to 6 times at varying times of the day and on different days of the week, including weekend days. Although the primary method of recruitment was through block enumeration (80.1%), participants were also recruited from the community through random approach at local fairs and festivals (9.2%), responses to flyers distributed to the community (4.4%), and referrals from community members (6.3%). One person per household was randomly invited to participate in the study. Individuals were excluded if they were unable to answer questions due to a serious health condition, planned to move from the Boston area within 2 y, or had a Mini Mental State Examination score ≤ 10.

Of 2004 people eligible for the study, 280 individuals declined for various reasons, including not being interested, being too busy, and not wanting to participate in the blood draw. Those who declined had lived on the U.S. mainland longer than those who completed the baseline interview (32.7 vs. 28.7 y; $P < 0.001$). Of the remaining 1724 participants who agreed to participate, 309 did not complete the baseline interview due to difficulties in scheduling/frequent changes of address and phone numbers or to low Mini-Mental Sate Examination scores. At the time of this study, 1358 of the 1415 participants had complete and cleaned baseline data. Individuals with implausible (<2510 kJ/d or >20,083 kJ/d) or missing dietary data and/or missing metabolic syndrome data were not included in these analyses ($n = 151$). This cross-sectional analysis, therefore, included 1207 Puerto Rican adults aged 45–75 y with complete dietary and metabolic syndrome data.

Participants completed a home interview in the language of their preference (Spanish or English). The interview consisted of questionnaires to collect information such as socioeconomic status, health history and behaviors, acculturation, and dietary intake. Anthropometric and blood pressure measures were also obtained. During the interview, participants were provided instructions for collecting a 12-h urine sample and for fasting overnight for the following morning's blood draw. Biological samples including saliva, urine, and blood, were collected by the study phlebotomist. Written informed consent was obtained for all participants before enrolling in the study in accordance with the guidelines established by the Institutional Review Board at Tufts Medical Center.

Dietary pattern assessment. Dietary intake was assessed using a semiquantitative FFQ adapted for this population and validated in Hispanic adults aged 60 y and older (24–27). Participants were asked to report type, frequency, and portion size of foods consumed over the past 12 mo. Detailed questions on dietary fat intake, such as added spreads/oils and type of foods consumed (e.g., full fat vs. fat free), were included. The nutrient database in the Nutrition Data System for Research software, version 2007 (Nutrition Coordinating Center, University of Minnesota), was used to calculate nutrient content from reported food intakes. A total of 26 individual fatty acids were calculated as a percentage of total fat consumed and included fatty acids from supplements. We chose to use total fatty acids consumed (including fish oil supplements) rather than from food alone, because supplements comprise a large amount of certain fatty acids such as eicosapentaenoic acid (EPA)[8] and docosahexaenoic acid (DHA).

We performed exploratory dietary pattern analysis by entering 26 individual fatty acids into a principle components analysis using PROC FACTOR in SAS (SAS Institute). Resulting principal components were rotated with the Varimax option to maximize explanatory power and to theoretically result in noncorrelated principal components, or factors. Three to 8 principal components were specified; scree plots, Eigenvalues (derived from the correlation matrix) and the components themselves were examined to identify the most meaningful solution (28). A 4-factor solution was selected. Each participant received a factor score for each factor by summing intakes of fatty acids weighted by the loadings of each fatty acid. We performed principal component analysis separately for men and women; results were similar, and we therefore present the combined sample to maximize statistical power. We also repeated principal component analysis (using the methods described above) for dietary fatty acid intake excluding fatty acid contributions from supplements.

Metabolic syndrome ascertainment. Metabolic syndrome was defined using the 2001 National Cholesterol Education Program Adult Treatment Panel III definition, which was modified to reflect lower glucose concentrations by the American Diabetes Association (12). This definition requires that 3 of the 5 following criteria be present: *1*) waist circumference (WC) $\geq$ 102 cm for men, $\geq$ 88 cm for women; *2*) plasma triglycerides $\geq$ 1.7 mmol/L; *3*) plasma HDL-cholesterol $<$ 1.04 mmol/L for men, $<$ 1.3 mmol/L for women; *4*) blood pressure $\geq$ 130/ $\geq$85 mm Hg; and *5*) fasting serum glucose $\geq$ 5.6 mmol/L. The individual components of metabolic syndrome were considered as continuous variables and as categorical variables in secondary analyses.

Anthropometric and blood pressure measures. Weight was measured using a clinical scale (Toledo Weight Plate, Model I5S, Bay State and Systems). WC was obtained at the umbilicus using an anthropometric tape measure and standing height was measured using a standing stadiometer. All anthropometric measures were taken in duplicate to minimize recording and measurement errors. BMI was calculated as weight (kg) divided by height squared (m^2). Blood pressure measurements were obtained using an electronic sphygmomanometer (Dinamap Model 8260, Critikon) at 3 time points during the interview; the mean of the second and 3rd readings were used for systolic and diastolic blood pressure.

Sociodemographic and covariate assessment. Participants reported age, sex, education level attained, and household income. Poverty was calculated for each participant from annual household income and poverty guidelines by the Department of Health and Human Services while accounting for the year of the interview and the subject's family size. Acculturation was determined by reported preference of language used in various everyday activities to calculate an overall acculturation score. A modified Paffenbarger questionnaire of the Harvard Alumni Activity Survey (29,30) was used to create a score calculated by summing the amount of time spent in each activity multiplied by weighting factors that correspond with oxygen consumption by physical activity intensity for that activity. Physical activity scores were classified as sedentary (score $<$ 30), light (score $\geq$ 30 to $<$ 40), moderate (score $\geq$ 40 to $<$ 50)

[8] Abbreviations used: DHA, docosahexaenoic acid; DPA, docosapentaenoic acid; EPA, eicosapentaenoic acid; MUFA, monounsaturated fatty acid; OR, odds ratio; VLC, very long-chain; WC, waist circumference.

or heavy (score ≥ 50). Smoking and alcohol consumption were assessed through standard questionnaires as never, current, or past.

Biological samples. Fasting blood samples were drawn in the home by a certified phlebotomist on the morning following the interview or as soon as possible thereafter and brought to the Human Nutrition Research Center on Aging for analysis at the Nutrition Evaluation Laboratory. Blood samples were kept cooled at 4°C. Plasma was separated within 4 h and used for analysis of plasma lipid concentrations. Isolated serum was frozen (−80°C) for further analysis, which included serum glucose determinations. Serum glucose was measured by using an enzymatic kinetic reaction on the Olympus AU400 (Olympus America) with Olympus Glucose Reagents (OSCR6131). Analyses of plasma HDL cholesterol and triglyceride concentrations were completed using EDTA with enzymatic endpoint reaction on the Olympus AU400 and with Olympus HDL Reagents (OSR6195) and Olympus Triglyceride Reagents (OSR6033).

Statistical analyses. All statistical analyses were performed using SAS (version 9.2, SAS Institute). Hypothesis testing was 2-sided with a significance level of $P < 0.05$. All variables were assessed for normality. Plasma triglyceride and serum glucose concentrations were log-transformed to improve normality before inclusion in linear analyses. Age- and sex-adjusted means ± SE and frequencies were examined comparing the highest quintile (Q5) to the lowest quintile (Q1) of each component using ANCOVA. Partial Spearman correlation coefficients adjusted for age, sex, and total energy were calculated between each component and intakes of selected nutrients and food sources contributing to percent of total fat.

Two multivariable logistic regression models were used to test associations between factor scores and prevalence of metabolic syndrome and to estimate odds ratios (OR) and 95% CI. Model 1 adjusted for age, sex, education, smoking and alcohol use, acculturation, total energy, fish oil supplement use (yes/no), total fat and dietary fiber intake, and medication use. Model 2 included additional adjustment for BMI, which was included as a covariate to further isolate the independent effects of central adiposity, as measured by WC. Linear trend tests were performed using the median factor score for each component or median fat intake for each quintile as a continuous variable in the model. We evaluated significant differences between continuous metabolic syndrome components and factor quintiles using general linear models; a test for trend using the median score value was performed. Adjusted means (95% CI) were presented. All models were tested for interactions between sex and each of the factors and also for acculturation and each of the factors. There was a significant interaction between component 2 and acculturation for triglycerides ($P < 0.005$); however, when we stratified by acculturation, there were no significant associations with triglycerides, indicating that the interaction may have occurred by chance.

In secondary analysis, multivariable logistic regression was used to examine associations between fat subtypes (quintiles of intake as a percentage of total energy) and metabolic syndrome using the same models as described above. Analyses were performed comparing each quintile to the lowest quintile (reference) and OR and 95% CI were presented. Linear trend tests were performed using median fat intake for each quintile as a continuous variable in the model. Also, we assessed the relationships between factor scores and metabolic syndrome components treated as categorical outcomes by using multivariable logistic regression. We also examined factors derived using fatty acid intake without contributions from supplements included.

Results

Principal components analysis revealed 4 fatty acid patterns that were named based on the fatty acids that loaded more heavily on each of the factors; the individual fatty acids and factor loadings for each of the patterns are presented in **Table 1**. Factor 1, the SFA/dairy pattern, loaded heavily on capric, mystiric, lauric, and caproic acids. This component also loaded strongly on palmitic acid (long-chain SFA) and stearic acid and moderately on *trans* fats. An (n-3) fatty acid/fish factor (factor 2) was characterized primarily by DHA, docosapentaenoic acid (DPA), and EPA, but also by euric and gadoleic acid [monounsaturated fatty acids (MUFA)] and arachidonic acid [(n-6) PUFA]. Factor 3, very long-chain (VLC) SFA and PUFA/oils pattern, was defined by long-chain SFA (arachidic acid and behenic acid) and linolenic acid and moderately by MUFA (erucic, gadoleic, and oleic acids). A MUFA/*trans* factor (factor 4) was characterized by oleic and myristoleic acid (MUFA), *trans* fat, and long-chain SFA (stearic and margaric acids). The percentages of variation explained were 7.4 for factor 1, 4.1 for factor 2, 2.9 for factor 3, and 2.5 for factor 4.

TABLE 1 Factor loadings of 4 fatty acid patterns among Puerto Rican adults aged 45–75 y[1]

Factor 1: SFA/dairy		Factor 2: (n-3) fatty acid/fish		Factor 3: VLC SFA/ PUFA/oils		Factor 4: MUFA/*trans*	
Fatty acid	Factor loadings	Fatty acid	Factor loadings	Fatty acid	Factor loadings	Fatty acid	Factor loadings
Capric acid (SFA 10:0)	0.97	DHA (PUFA 22:6)	0.91	Arachidic acid (SFA 20:0)	0.68	Oleic acid (MUFA 18:1)	0.63
Myristic acid (SFA 14:0)	0.95	DPA (PUFA 22:5)	0.90	Behenic acid (SFA 22:0)	0.60	*Trans*-octadecenoic acid (*trans* isomer MUFA 18:1)	0.61
Caproic acid (SFA 6:0)	0.95	EPA (PUFA 20:5)	0.86	Linolenic acid (PUFA 18:3)	0.51	Myristoleic acid (MUFA 14:1)	0.54
Caprylic acid (SFA 8:0)	0.94	Erucic acid (MUFA 22:1)	0.68	Erucic acid (MUFA 22:1)	0.35	Stearic acid (SFA 18:0)	0.50
Butyric acid (SFA 4:0)	0.93	Gadoleic acid (MUFA 20:1)	0.59	Stearic acid (SFA 18:0)	−0.35	Margaric acid (SFA 17:0)	0.46
Palmitic acid (SFA 16:0)	0.77	Arachidonic acid (PUFA 20:4)	0.56	Arachidonic acid (PUFA 20:4)	−0.37	*Trans*-octadecadienoic (*trans* isomer PUFA 18:2)	0.44
Lauric acid (SFA 12:0)	0.76			Palmitic acid (SFA 16:0)	−0.53	Linoleic acid (PUFA 18:2)	−0.56
Stearic acid (SFA 18:0)	0.66			Palmitoleic acid (MUFA 16:1)	−0.76		
Trans-octadecadienoic (*trans* isomer PUFA 18:2)	0.36						
Oleic acid (MUFA 18:1)	−0.37						
Gadoleic acid (MUFA 20:1)	−0.40						
Arachidic acid (SFA 20:0)	−0.50						
Linoleic acid (PUFA 18:2)	−0.69						

[1] n = 1207, only factor loadings ≥ |0.35| were included for simplicity.

Participants were more acculturated in the highest quintile of factor 1 (SFA/dairy) (score of 30 vs. 23%), factor 2 [(n-3) fatty acid/fish] (score of 26 vs. 20%), and factor 4 (MUFA/*trans*) (score of 28 vs. 23%) compared with the lowest quintile, respectively (**Table 2**). Participants in the highest quintile of the (n-3) fatty acid/fish pattern were more likely to be older (59 vs. 56 y) and female (78 vs. 75%) compared with those in the lowest quintile. WC was lower for those in the highest quintile of the VLC SFA and PUFA/oils pattern compared with the lowest quintile (99.8 vs. 103.1), although there was no difference in BMI (30.6 vs. 31.5). The opposite was seen for the (n-3) fatty acid/fish pattern, where BMI was higher in Q5 (32) than Q1 (30), but WC did not differ (102.3 vs. 101.7). Participants in Q5 vs. Q1 of the MUFA/*trans* pattern (factor 4) were more likely to be men, more educated, a current smoker, and a consumer of alcohol. Not surprisingly, more participants in the highest quintile of factor 2 [(n-3) fatty acid/fish] reported taking fish oil supplements (15%) than those in the lowest quintile.

The SFA/dairy pattern was positively correlated with dairy foods, dairy desserts, and breakfast cereal and negatively correlated with oils, poultry, and meat (**Table 3**). The (n-3) fatty acid/fish pattern was correlated positively with fish, moderately with poultry and fruit, and negatively with French fries. The VLC SFA/PUFA oils pattern was moderately correlated with nuts, seeds, beans, and legumes and negatively correlated with processed meats and meat. This factor was also positively associated with canola oil ($r = 0.24$, $P < 0.001$) based on further analysis with more detailed foods and food groups (data not shown). The MUFA/*trans* pattern was correlated positively with processed meat, baked goods, and meat and negatively with beans and legumes, oils, and poultry.

The (n-3) fatty acid/fish pattern was negatively correlated whereas the MUFA/*trans* pattern was positively correlated with total energy intake adjusted for age and sex (**Table 4**). The (n-3) fatty acid/fish pattern was positively associated with protein intake; the VLC SFA/PUFA/oils pattern with carbohydrate and dietary fiber; and the SFA/dairy pattern with total sugar intake. The SFA/dairy, (n-3) fatty acid/fish, and VLC SFA/PUFA/oils patterns were each inversely associated with percent of energy from fat, whereas the MUFA/*trans* pattern was positively correlated with percent of energy from fat. The MUFA/*trans* pattern was also positively associated with percent of energy from monounsaturated and saturated fat intake.

In multivariable analyses, the (n-3) fatty acid/fish pattern was associated with lower likelihood of metabolic syndrome after adjustment for all covariates (OR: 0.54, 95% CI: 0.34, 0.86) (**Table 5**). There were no associations between dietary fat subtypes and metabolic syndrome for the highest compared with the lowest quintile of intakes ($P > 0.39$) (data not shown); however, there was a trend for increasing (n-3) fatty acid intake across quintiles after adjusting for covariates (P-trend = 0.04, for model 2) (**Supplemental Table 1**).

TABLE 2 Baseline sample characteristics by extreme quintile categories of fatty acid patterns among Puerto Rican adults[1]

Selected characteristics	Factor 1: SFA/dairy		Factor 2: (n-3) fatty acid/fish		Factor 3: VLC SFA/PUFA/oils		Factor 4: MUFA/*trans*	
	Q1, $n = 242$[2]	Q5, $n = 243$[2]	Q1, $n = 242$[2]	Q5, $n = 241$[2]	Q1, $n = 241$[2]	Q5, $n = 242$[2]	Q1, $n = 240$[2]	Q5, $n = 243$[2]
Age,[3,4] *y*	58.2 ± 0.5	58.7 ± 0.5	56.4 ± 0.5	58.5 ± 0.5**	57.3 ± 0.5	58.3 ± 0.5	59.5 ± 0.5	56.5 ± 0.5***
WC,[3,4] *cm*	101.4 ± 1.0	101.3 ± 1.0	101.7 ± 1.0	102.3 ± 1.0	103.1 ± 1.0	99.8 ± 1.0*	100.6 ± 1.0	102.6 ± 1.0
BMI,[3,4] *kg/m²*	30.8 ± 0.4	30.8 ± 0.4	30.1 ± 0.5	31.8 ± 0.5**	31.5 ± 0.4	30.6 ± 0.4	31.0 ± 0.5	31.2 ± 0.4
Fasting serum glucose,[3,4], *mmol/L*	6.7 ± 0.2	6.35 ± 0.2	6.88 ± 0.2	6.93 ± 0.2	6.68 ± 0.2	7.02 ± 0.2	6.65 ± 0.2	7.25 ± 0.2
Systolic blood pressure,[3,4] *mm Hg*	136.4 ± 1.2	135.7 ± 1.2	138.2 ± 1.2	135.7 ± 1.3	136.8 ± 1.2	136.4 ± 1.2	136.0 ± 1.3	136.8 ± 1.2
Diastolic blood pressure,[3,4] *mm Hg*	80.7 ± 0.7	81.0 ± 0.7	81.6 ± 0.7	80.7 ± 0.7	82.3 ± 0.7	80.5 ± 0.7	80.5 ± 0.7	81.5 ± 0.7
Plasma triglycerides,[3,4], *mmol/L*	1.94 ± 0.07	1.83 ± 0.07	1.77 ± 0.07	1.84 ± 0.07	1.86 ± 0.07	1.88 ± 0.07	1.89 ± 0.07	1.85 ± 0.07
Plasma HDL-C,[3,4], *mmol/L*	1.13 ± 0.02	1.14 ± 0.02	1.11 ± 0.02	1.13 ± 0.02	1.15 ± 0.02	1.13 ± 0.02	1.11 ± 0.02	1.09 ± 0.02
Acculturation score,[3,4] *%*	23.4 ± 1.4	30.1 ± 1.4***	20.3 ± 1.4	26.2 ± 1.4**	24.0 ± 1.4	27.0 ± 1.4	23.2 ± 1.4	28.3 ± 1.4**
Physical activity score[3,4]	31.4 ± 0.3	31.5 ± 0.3	31.3 ± 0.3	31.9 ± 0.3	31.4 ± 0.3	31.9 ± 0.3	32.1 ± 0.3	31.3 ± 0.3
Total energy intake,[3,4] *kJ/d*	9257.6 ± 240	9489.7 ± 241	9633.3 ± 241	8716.9 ± 243*	9435.4 ± 238	9397.7 ± 244	8808.5 ± 243	9999.6 ± 235**
Female,[5] *%*	71.5	77.0	75.2	78.0*	66.8	78.5**	74.6	64.6*
Less than 8th grade education level,[5] *%*	55.6	47.7*	52.3	45.2	48.6	45.6	56.9	44.0**
Below poverty,[5] *%*	59.2	59.2	63.5	57.3	56.0	60.0	65.2	56.8
Smoking status,[5] *%*								
Never smoked	41.3	47.0	45.2	49.6*	40.5	49.2	46.2	34.9**
Past smoker	31.1	28.0	22.4	32.5	32.1	29.0	30.3	31.5
Current smoker	27.7	25.1	32.4	17.9	27.4	21.9	23.5	33.6
Alcohol consumption,[5] *%*								
Never consumed	33.0	28.4	37.6	28.3	28.3	31.5	39.3	21.5***
Past consumer	25.4	34.2	30.2	30.8	28.8	29.9	23.9	31.0
Current consumer	41.7	37.5	32.2	40.8	42.9	38.6	36.8	47.5
Fish oil supplement use,[5] *%*	3.3	7.0*	0.4	15.0***	2.5	4.6*	5.0	4.1
Multivitamin use,[5] *%*	23.7	22.0*	16.7	24.5	18.3	21.6	18.1	15.3
Prevalence of metabolic syndrome,[5] *%*	69.0	66.5	69.0	63.6	63.5	64.1	68.9	66.8

[1] Values are means ± SE or percentage. Asterisks indicate different from Q1: *$P < 0.05$, **$P < 0.01$, and ***$P < 0.001$.
[2] Due to missing data for some covariates, sample sizes for each analysis vary around the reported sample size (*n*).
[3] Models were adjusted for age and sex.
[4] Q1 and Q5 were compared using ANCOVA.
[5] Q1 and Q5 were compared using chi-square tests.

TABLE 3 Spearman correlation coefficients between fatty acid intake factors and major food sources among Puerto Rican adults[1,2]

Factor 1: SFA/dairy		Factor 2: (n-3) fatty acid/ fish		Factor 3: VLC SFA/ PUFA/oils		Factor 4: MUFA/*trans*	
Food source	Correlation	Food source	Correlation	Food source	Correlation	Food source	Correlation
High-fat dairy	0.74	Fish	0.66	Nuts and seeds	0.29	Processed meat	0.31
Dairy desserts	0.29	Poultry	0.30	Bean and Legumes	0.20	Baked goods	0.26
Reduced-fat dairy	0.23	Fruit	0.24	Eggs	−0.21	Meat (beef, pork, lamb)	0.23
Breakfast cereal	0.23	French fries	−0.25	Meat (beef, pork, lamb)	−0.31	Poultry	−0.24
Candy/chocolate	0.21			Processed meat	−0.33	Beans and legumes	−0.28
Rice	−0.21					Oils	−0.50
Meat (beef, pork, lamb)	−0.22						
Poultry	−0.32						
Oils (corn)	−0.55						

[1] n = 1207. Only Spearman correlations ≥ |0.20| are shown.
[2] All correlation coefficients were significant, $P < 0.001$.

After adjustment for potential confounding variables, the SFA/dairy pattern was inversely associated with fasting serum glucose concentration ($P = 0.02$) (**Table 6**). The VLC SFA/PUFA/oils pattern was inversely associated with WC after adjustment for covariates ($P = 0.008$). These associations were attenuated after further adjustment for BMI. The (n-3) fatty acid/fish pattern tended to be associated with larger WC (Q5: 101.4 cm vs. Q1: 98.7 cm; $P = 0.06$). The MUFA/*trans* pattern was not significantly associated with any of the metabolic syndrome risk factors.

We investigated the relationship between the factors and metabolic syndrome measures as individual dichotomous outcomes. Results were similar to those observed for continuous metabolic syndrome measures as described above (data not shown). The SFA/dairy pattern was associated with higher likelihood of elevated serum glucose concentration after adjustment for covariates (OR: 0.86, 95% CI: 0.75, 0.99) and in this model, the association remained significant after additional adjustment for BMI (OR: 0.85, 95% CI: 0.74, 0.97). There tended to be an inverse association between the (n-3) fatty acid/fish pattern and elevated blood pressure after adjustment for covariates and BMI (OR: 0.88, 95% CI: 0.77, 1.005). The VLC SFA/PUFA/oils pattern was, again, associated with WC after covariate adjustment (OR: 0.83, 95% CI: 0.71, 0.96) but was no longer significant after additional adjustment for BMI ($P = 0.54$.)

In additional analyses, we examined dietary fatty acid factors excluding fish oil supplement use in the derivation of the principal components. The fatty acid factors looked similar to those that emerged when fatty acids from fish oil supplements were included and associations between the principal components and metabolic syndrome measures were similar to results using the principal components including fish oil supplements (data not shown). After adjusting for potential confounders, the SFA/dairy pattern was inversely associated with fasting serum glucose ($P = 0.01$), although this association was no longer significant after additional adjustment for BMI ($P = 0.09$). However, the VLC SFA/PUFA/oils pattern remained significantly associated with WC after adjustment ($P = 0.009$).

Discussion

Quality of dietary fat is often described based on saturation (e.g., saturated fat). However, individual fatty acids can have differential effects on metabolism, such as influencing insulin action and insulin sensitivity (31). In this study, we used principal components analysis to derive fatty acid patterns and examined the major food sources contributing to these patterns and associations with metabolic syndrome and its components in a sample of Puerto Ricans living in the Greater Boston area. Although 1 study (19) used factor analysis to examine patterns

TABLE 4 Partial Spearman correlations among fatty acid patterns, energy, and selected nutrient intakes among Puerto Rican adults[1]

Energy and nutrients	Factor 1: SFA/dairy	Factor 2: (n-3) fatty acid/fish	Factor 3: VLC SFA/ PUFA/oils	Factor 4: MUFA/*trans*
Energy,[2] *kJ*	0.05	−0.09*	−0.02	0.11*
Carbohydrate,[3] *% of energy*	0.14 *	−0.16*	0.34*	−0.07*
Protein,[3] *% of energy*	−0.09*	0.47*	−0.39*	0.03
Fat,[3] *% of energy*	−0.04	−0.07*	−0.19*	0.10*
Saturated fat,[3] *% of energy*	0.51*	−0.09*	−0.32*	0.22*
Monounsaturated fat,[3] *% of energy*	−0.14*	−0.01	−0.13*	0.32*
Polyunsaturated fat,[3] *% of energy*	−0.54*	−0.09*	0.07*	−0.33*
Fiber,[3] *g*	−0.16*	0.08*	0.26*	−0.11*
Total sugar,[3] *g*	0.37*	−0.04	0.09*	0.05
Alcohol,[3] *g*	0.01	0.04	−0.0005	0.09*

[1] n = 1207; correlation coefficients > 0.06 or ≤ −0.07 are significant, *$P < 0.05$.
[2] Adjusted for age and sex.
[3] Adjusted for age, sex, and total energy.

TABLE 5 OR and 95% CI of metabolic syndrome across extreme quintile categories of fatty acid factors in Puerto Rican adults[1–3]

	Factor 1: SFA/dairy		Factor 2: (n-3) fatty acid/fish		Factor 3: VLC SFA/ PUFA/oils		Factor 4: MUFA/*trans*	
	Q1	Q5	Q1	Q5	Q1	Q5	Q1	Q5
n[2]	242	243	242	241	241	242	240	243
Model 1: Multivariable adjusted[1]	1.0	0.92 (0.61, 1.4)	1.0	0.72 (0.47, 1.1)	1.0	0.83 (0.55, 1.3)	1.0	1.01 (0.66, 1.5)
Model 2: Multivariable adjusted + BMI	1.0	0.94 (0.60, 1.5)	1.0	0.54 (0.34, 0.86)	1.0	0.87 (0.56,1.4)	1.0	1.03 (0.66, 1.6)

[1] Logistic regression models were used to compare the highest quintile (Q5) of each factor to the reference group (Q1) and to estimate OR and 95% CI.
[2] Due to missing data for some covariates, sample sizes for each analysis vary around the reported sample size (*n*).
[3] Adjusted for age, sex, smoking and alcohol use, physical activity, education, fish oil supplement use, acculturation, total energy, total fat, dietary fiber, and lipid-lowering medication use.

of serum fatty acids in relation to metabolic syndrome, no studies to our knowledge have used this method to study the relationship between dietary fatty acids and metabolic syndrome.

In general, the food sources from the patterns we observed were similar to those reported by other studies of Caribbean Latinos, with major dietary fat sources coming from oils, particularly corn oil, chicken, meat and processed meat, cheese, and milk (32). The SFA/dairy pattern was inversely associated with fasting serum glucose concentrations after adjustment, although this was no longer significant after further adjustment for BMI. A few studies have reported associations between SFA intake and impaired insulin sensitivity and fasting glucose concentrations (15,33). Medium-chain fatty acids may improve insulin sensitivity and glucose concentrations through their direct transport to the liver without requiring fatty acid-binding proteins and their preferential use for β-oxidation (34,35). However, a recent study of Japanese patients with type 2 diabetes examined serum concentrations of fatty acids and found that some short- and medium-chain fatty acids were positively correlated with the homeostasis model insulin resistance index (36). Dairy products, which contain oleic acid and short- and medium-chain fatty acids (34,35), were among the top major food sources contributing to this pattern. Epidemiological studies have demonstrated protective effects of dairy products for cardiovascular disease risk factors such as WC, hyperinsulinemia, dyslipidemia, and blood pressure (37–39) and for metabolic syndrome (38,40). In the current study, the SFA/dairy pattern was associated with only serum glucose, which may be due to the influence of other fatty acids that loaded on this factor (stearic and *trans* fats) or other nutrients that were correlated with this factor. For example, higher consumption of saturated and *trans* fats can negatively influence blood lipoprotein concentrations (41) and blood pressure (42).

The (n-3) fatty acid/fish pattern was associated with lower odds of metabolic syndrome, which is similar to findings from a

TABLE 6 Adjusted means and 95% CI of metabolic syndrome components across extreme quintile categories in Puerto Rican adults[1–3]

	Factor 1: SFA/dairy			Factor 2: (n-3) fatty acid/fish			Factor 3: VLC SFA/PUFA/oils			Factor 4: MUFA/*trans*		
	Q1	Q5	*P*-trend	Q1	Q5	*P*-trend	Q1	Q5	*P*-trend	Q1	Q5	*P*-trend
n[4]	242	243		242	241		241	242		240	243	
WC, *cm*												
Model 1	100.1 (97, 103)	100.5 (98, 103)	0.82	98.7 (96, 102)	101.4 (99, 104)	0.06	101.9 (99, 105)	98.9 (96, 102)	0.008	100.5 (98, 103)	100.9 (98, 104)	0.62
Model 2	104.0 (102, 106)	103.8 (102, 105)	0.50	104.0 (102, 106)	103.0 (102, 104)	0.24	104.1 (103, 106)	102.8 (101, 104)	0.12	103.6 (102, 105)	103.9 (102, 105)	0.47
Plasma triglycerides, *mmol/L*												
Model 1	1.65 (1.5, 1.8)	1.62 (1.5, 1.8)	0.62	1.55 (1.4, 1.7)	1.60 (1.4, 1.8)	0.22	1.63 (1.5, 1.8)	1.64 (1.5, 1.8)	0.81	1.62 (1.5, 1.8)	1.57 (1.4, 1.7)	0.66
Model 2	1.69 (1.5, 1.9)	1.7 (1.5, 1.8)	0.55	1.60 (1.4, 1.8)	1.63 (1.5, 1.8)	0.32	1.64 (1.5, 1.8)	1.65 (1.5, 1.8)	0.96	1.61 (1.5, 1.8)	1.62 (1.5, 1.8)	0.94
Plasma HDL-cholesterol, *mmol/L*												
Model 1	1.13 (1.1, 1.2)	1.12 (1.1, 1.2)	0.69	1.11 (1.05, 1.2)	1.11 (1.05, 1.2)	0.84	1.14 (1.1, 1.2)	1.12 (1.1, 1.2)	0.73	1.09 (1.03, 1.1)	1.08 (1.02, 1.1)	0.41
Model 2	1.12 (1.1, 1.2)	1.12 (1.1, 1.2)	0.72	1.09 (1.02, 1.2)	1.11 (1.05, 1.2)	0.81	1.13 (1.1, 1.2)	1.12 (1.1, 1.2)	0.67	1.09 (1.03,1.1)	1.08 (1.02, 1.1)	0.68
Systolic blood pressure, *mm Hg*												
Model 1	133.5 (130, 137)	133.7 (130, 137)	0.81	136.1 (132, 140)	133.9 (131, 137)	0.36	133.6 (130, 137)	134.4 (131, 138)	0.64	134.0 (130, 138)	133.9 (130, 138)	0.85
Model 2	134.1 (133, 138)	133.6 (130, 137)	0.90	136.8 (133, 141)	134.3 (131, 138)	0.42	133.7 (130, 137)	134.5 (131, 138)	0.69	134.4 (131, 138)	134.1 (131, 138)	0.92
Diastolic blood pressure, *mm Hg*												
Model 1	78.6 (77, 81)	78.9 (77, 81)	0.86	79.7 (78, 82)	79.1 (77, 81)	0.55	79.7 (78, 82)	78.6 (77, 81)	0.35	78.6 (77, 81)	79.2 (77, 81)	0.52
Model 2	79.1 (77, 82)	79.2 (77, 81)	0.98	80.6 (79, 83)	79.5 (78, 82)	0.41	79.7 (78, 82)	79.0 (77, 81)	0.56	79.1 (77, 81)	79.6 (78, 82)	0.64
Fasting serum glucose, *mmol/L*												
Model 1	6.82 (6.4, 7.2)	6.55 (6.2, 6.9)	0.02	6.82 (6.4, 7.3)	6.89 (6.5, 7.3)	0.68	6.82 (6.4, 7.2)	6.96 (6.6, 7.4)	0.97	6.89 (6.5, 7.3)	6.96 (6.6, 7.4)	0.95
Model 2	6.82 (6.4, 7.2)	6.7 (6.3, 7.1)	0.11	6.82 (6.4, 7.2)	6.96 (6.6, 7.3)	0.42	6.52 (6.4, 7.2)	7.03 (6.6, 7.5)	0.70	6.82 (6.5, 7.3)	7.03 (6.6, 7.4)	0.53

[1] General linear models were used to estimate adjusted means (95% CI) for quintiles of each factor. For simplicity, the adjusted means (95% CI) are presented for the highest and lowest quintiles only; however, linear trend tests were performed across quintiles.
[2] Model 1 was adjusted for age, sex, smoking and alcohol use, physical activity, education, total energy, acculturation, fish oil supplement use, total fat and dietary fiber, and medication use.
[3] Model 2 was adjusted for all covariates in model 1 and BMI.
[4] Due to missing data for some covariates, sample sizes for each analysis vary around the reported sample size (*n*).

population-based cohort study of men aged 50 y and older that generated fatty acid factors from serum fatty acids (19). In that study, an (n-3) PUFA factor was associated with lower odds of metabolic syndrome at age 50 y (unadjusted OR: 0.56, 95% CI: 0.48, 0.64) and inversely associated with the development of metabolic syndrome over 20 y, after adjusting for confounding (OR: 0.74, 95% CI: 0.62, 0.89). Both the (n-3) fatty acid/fish pattern and (n-3) fatty acid intake alone were associated with lower odds of metabolic syndrome for the highest compared with the lowest quintile. However, the (n-3) fatty acid/fish pattern resulted in a lower OR of 0.54 [compared with 0.80 for (n-3) fatty acid intake alone] and was significant (95% CI: 0.34, 0.86). There was also a significant trend with increasing quintiles of (n-3) fatty acid intakes. These findings suggest that considering combinations of fatty acids may provide additional information on fat quality relative to actual exposure than traditional fat subtypes alone.

Dietary (n-3) fatty acids have been reported to improve several metabolic syndrome components, as reviewed by Carpentier et al. (43). In a study of Inupiat Eskimos, protective associations between (n-3) fatty acid intakes and blood pressure, serum triglycerides, 2-h glucose, HDL-cholesterol (DHA only), fasting insulin, and homeostasis model assessment were observed (42). The population-based INTERMAP study reported inverse associations between dietary sources of (n-3) fatty acids [total (n-3) PUFA and linolenic acid] and blood pressure (44). Additionally, higher MUFA intake and/or replacing SFA with MUFA have been associated with improvements in blood pressure (45). In our study, the (n-3) fatty acid/fish pattern was not associated with lower blood pressure, which may be due to the fact that consumption of marine sources of (n-3) fatty acids may not have been high enough to detect associations with blood pressure. In this population, fish oil supplements contributed greatly to intakes of EPA and DHA. However, only 15% of participants in the highest quintile of this factor reported consuming a fish oil supplement (n = 32). In our study, median intakes of EPA, DPA, and DHA combined were 0.20 g/d for men and 0.15 g/d for women, which is slightly higher than national estimates based on NHANES data for U.S. adults aged 20–59 y (0.17 and 0.11 g/d for men and women, respectively) (46).

The VLC SFA/PUFA/oils pattern was related to smaller WC. This relationship was attenuated after adjustment for BMI, suggesting that the association with central adiposity was partially explained by total body fat. We examined associations with and without adjustment for BMI as a way to account for overall adiposity and to isolate the effects of central adiposity, one of the components of metabolic syndrome. Results from metabolic studies suggest that medium-chain fatty acids may lead to a decrease in body weight through increased energy expenditure, increased rate of oxidation, and increased thermogenesis (18). An inverse relationship between central adiposity and fish intake (47) and between BMI and a Mediterranean diet have been reported (48,49). The relationship between individual fatty acids and body weight is complex and inconsistent, according to a recent review (18), and more research is needed.

Overall adiposity, as measured by BMI, is correlated with WC and is also an independent risk factor for metabolic syndrome (50). We adjusted our models for BMI to isolate the effect of our patterns on metabolic syndrome independent of total adiposity. Although adjustment for BMI attenuated associations between most of the fatty acid patterns and metabolic syndrome components, associations observed for the (n-3) fatty acid/fish pattern become stronger. In another study of dietary patterns and metabolic syndrome, Esmaillzadeh et al. (51) also adjusted their models for BMI and found that the inverse associations between a healthy dietary pattern and metabolic syndrome and the positive association with a Western pattern remained significant after additional adjustment for BMI. This suggests that abdominal adiposity rather than overall obesity may be responsible for some associations between diet and metabolic syndrome. Another study also found that the addition of WC explained a higher proportion of the variance of several metabolic syndrome risk factors compared with models including just percent total body fat (52).

Our study has several limitations. This study was cross-sectional and therefore the results cannot be used to make statements about causation. Principle components analysis involves making several subjective decisions such as how to treat the dietary variables. We chose to use individual fatty acids as percent of total fat intake to represent the combination of fatty acids adjusted for total energy intake in all models. We performed a number of statistical tests between the factors and metabolic syndrome components, which could lead to significant findings due to chance alone. However, all tests were specified a priori and were adjusted for potential confounding. Finally, our sample included Puerto Ricans living in an urban area and may not be representative of all Puerto Ricans living in the US. However, results from this study are likely to represent the majority of Puerto Ricans living in low-income urban communities in the US and we do not expect that biological associations between fatty acids and metabolic syndrome would be substantially different in other populations, although intakes may differ.

In conclusion, there is limited information on fatty acid patterns consumed by Puerto Ricans living in the US and whether these patterns are associated with metabolic syndrome. Only the (n-3) fatty acid/fish pattern showed significant inverse associations with metabolic syndrome. Our results are consistent with other studies of fatty acids and metabolic risk factors, but our study is unique in its use of factor analysis to derive fat patterns using individual fatty acid intakes rather than foods or nutrients. Our findings suggest that a combination of the fatty acids in several of the patterns may be most advantageous for metabolic risk and may be a useful complementary method to understanding diet-disease associations in addition to the focus on traditional fat subtypes. More research is needed to identify the optimal combination of fatty acids from saturated, polyunsaturated, and monounsaturated fat on health outcomes in light of their diverse effects on metabolism and disease risk factors.

Acknowledgments

We thank Dr. Frank Hu for comments on an earlier version of this manuscript. S.E.N. designed the research, analyzed the data, and wrote the manuscript; P.K.N. designed the research and helped in the interpretation of the results and in writing the manuscript; J.M.O. helped in writing the manuscript; K.L.T. designed the research, provided essential materials, and helped in the interpretation and writing of the manuscript. All authors have critically reviewed and approved the final manuscript.

Literature Cited

1. Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among U.S. adults. Diabetes Care. 2004;27: 2444–9.
2. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. JAMA. 2002;287:356–9.

3. National Institute of Diabetes and Digestive and Kidney Diseases. National diabetes statistics 2007. NIH Publication No. 08–3892. NIDDK, NDIC 2008 [cited 2009 May]. Available from: http://diabetes.niddk.nih.gov/dm/pubs/statistics/DM_Statistics.pdf.
4. Tucker KL, Bermudez OI, Castañeda C. Type 2 diabetes is prevalent and poorly controlled among Hispanic elders of Caribbean origin. Am J Public Health. 2000;90:1288–93.
5. Kasim-Karakas SE. Ethnic differences in the insulin resistance syndrome. Am J Clin Nutr. 2000;71:670–1.
6. Escarce JJ, Morales LS, Rumbaut RG. The health status and health behaviors of Hispanics. In: Tienda M, Mitchell F, editors. Hispanics and the future of America. Washington, DC: The National Academies Press; 2006. p. 362–409.
7. US Census Bureau. Race and Hispanic origin in 2005. Population profile of the United States: dynamic version. U.S Government Printing Office, Washington (DC): 2005.
8. Council on Scientific Affairs. Hispanic health in the United States. JAMA. 1991;265:248–52.
9. Gao X, Nelson ME, Tucker KL. Television viewing is associated with prevalence of metabolic syndrome in Hispanic elders. Diabetes Care. 2007;30:694–700.
10. Expert Panel on Detection Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.
11. Grundy SM. Metabolic syndrome scientific statement by the American Heart Association and the National Heart, Lung and Blood Institute. Arterioscler Thromb Vasc Biol. 2005;25:2243–4.
12. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005;112:2735–52.
13. Kris-Etherton P, Daniels SR, Eckel RH, Engler M, Howard BV, Krauss RM, Lichtenstein AH, Sacks F, St Jeor S, et al. Summary of the scientific conference on dietary fatty acids and cardiovascular health: conference summary from the nutrition committee of the American Heart Association. Circulation. 2001;103:1034–9.
14. Freire RD, Cardoso MA, Gimeno SG, Ferreira SR. Dietary fat is associated with metabolic syndrome in Japanese Brazilians. Diabetes Care. 2005;28:1779–85.
15. Riccardi G, Giacco R, Rivellese AA. Dietary fat, insulin sensitivity and the metabolic syndrome. Clin Nutr. 2004;23:447–56.
16. Wannamethee SG, Shaper AG, Whincup PH. Modifiable lifestyle factors and the metabolic syndrome in older men: effects of lifestyle changes. J Am Geriatr Soc. 2006;54:1909–14.
17. Denke MA. Dietary fats, fatty acids, and their effects on lipoproteins. Curr Atheroscler Rep. 2006;8:466–71.
18. Moussavi N, Gavino V, Receveur O. Could the quality of dietary fat, and not just its quantity, be related to risk of obesity? Obesity (Silver Spring). 2008;16:7–15.
19. Warensjo E, Sundstrom J, Lind L, Vessby B. Factor analysis of fatty acids in serum lipids as a measure of dietary fat quality in relation to the metabolic syndrome in men. Am J Clin Nutr. 2006;84:442–8.
20. Bermudez OI, Falcon LM, Tucker KL. Intake and food sources of macronutrients among older Hispanic adults: association with ethnicity, acculturation, and length of residence in the United States. J Am Diet Assoc. 2000;100:665–73.
21. Gans KM, Burkholder GJ, Upegui DI, Risica PM, Lasater TM, Fortunet R. Comparison of baseline fat-related eating behaviors of Puerto Rican, Dominican, Colombian, and Guatemalan participants who joined a cholesterol education project. J Nutr Educ Behav. 2002;34:202–10.
22. Polednak AP. Use of selected high-fat foods by Hispanic adults in the northeastern US. Ethn Health. 1997;2:71–6.
23. Tucker KL. Stress and nutrition in relation to excess development of chronic disease in Puerto Rican adults living in the Northeastern USA. J Med Invest. 2005;52 Suppl:252–8.
24. Bermudez OI, Ribaya-Mercado JD, Talegawkar SA, Tucker KL. Hispanic and non-Hispanic white elders from Massachusetts have different patterns of carotenoid intake and plasma concentrations. J Nutr. 2005;135:1496–502.
25. Gao X, Martin A, Lin H, Bermudez OI, Tucker KL. α-Tocopherol intake and plasma concentration of Hispanic and non-Hispanic white elders is associated with dietary intake pattern. J Nutr. 2006;136:2574–9.
26. Kwan LL, Bermudez OI, Tucker KL. Low vitamin B-12 intake and status are more prevalent in Hispanic older adults of Caribbean origin than in neighborhood-matched non-Hispanic whites. J Nutr. 2002;132:2059–64.
27. Tucker KL, Bianchi LA, Maras J, Bermudez OI. Adaptation of a food frequency questionnaire to assess diets of Puerto Rican and non-Hispanic adults. Am J Epidemiol. 1998;148:507–18.
28. Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. Nutr Rev. 2004;62:177–203.
29. Paffenbarger RS Jr, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. N Engl J Med. 1993;328:538–45.
30. Paffenbarger RS Jr, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol. 1978;108:161–75.
31. Haag M, Dippenaar NG. Dietary fats, fatty acids and insulin resistance: short review of a multifaceted connection. Med Sci Monit. 2005;11:RA359–67.
32. Brunt MJ, Milbauer MJ, Ebner SA, Levenson SM, Millen BE, Quatromoni P, Chipkin SR. Health status and practices of urban Caribbean Latinos with diabetes mellitus. Ethn Dis. 1998;8:158–66.
33. Vessby B, Unsitupa M, Hermansen K, Riccardi G, Rivellese AA, Tapsell LC, Nalsen C, Berglund L, Louheranta A, et al. Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. Diabetologia. 2001;44:312–9.
34. Pfeuffer M, Schrezenmeir J. Milk and the metabolic syndrome. Obes Rev. 2007;8:109–18.
35. Marten B, Pfeuffer M, Schrezenmeir J. Medium chain triglycerides. Int Dairy J. 2006;16:1374–82.
36. Kusunoki M, Tsutsumi K, Nakayama M, Kurokawa T, Nakamura T, Ogawa H, Fukuzawa Y, Morishita M, Koide T, et al. Relationship between serum concentrations of saturated fatty acids and unsaturated fatty acids and the homeostasis model insulin resistance index in Japanese patients with type 2 diabetes mellitus. J Med Invest. 2007;54:243–7.
37. Mennen LI, Lafay L, Feskens EJ, Novak M, Lepinay P, Balkau B. Possible protective effect of bread and dairy products on the risk of the metabolic syndrome. Nutr Res. 2000;20:335–47.
38. Pereira MA, Jacobs DR Jr, Van Horn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. JAMA. 2002;287:2081–9.
39. Wirfalt E, Hedblad B, Gullberg B, Mattisson I, Andren C, Rosander U, Janzon L, Berglund G. Food patterns and components of the metabolic syndrome in men and women: a cross-sectional study within the Malmo Diet and Cancer cohort. Am J Epidemiol. 2001;154:1150–9.
40. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi F. Dairy consumption is inversely associated with the prevalence of the metabolic syndrome in Tehranian adults. Am J Clin Nutr. 2005;82:523–30.
41. Hu FB, Manson JE, Willett WC. Types of dietary fat and risk of coronary heart disease: a critical review. J Am Coll Nutr. 2001;20:5–19.
42. Ebbesson SO, Tejero ME, Nobmann ED, Lopez-Alvarenga JC, Ebbesson L, Romenesko T, Carter EA, Resnick HE, Devereux RB, et al. Fatty acid consumption and metabolic syndrome components: the GOCADAN study. J Cardiometab Syndr. 2007;2:244–9.
43. Carpentier YA, Portois L, Malaisse WJ. n-3 Fatty acids and the metabolic syndrome. Am J Clin Nutr. 2006;83:S1499–504.
44. Ueshima H, Stamler J, Elliott P, Chan Q, Brown IJ, Carnethon MR, Daviglus ML, He K, Moag-Stahlberg A, et al. Food omega-3 fatty acid intake of individuals (total, linolenic acid, long-chain) and their blood pressure: INTERMAP study. Hypertension. 2007;50:313–9.
45. Rasmussen BM, Vessby B, Uusitupa M, Berglund L, Pedersen E, Riccardi G, Rivellese AA, Tapsell L, Hermansen K. Effects of dietary saturated, monounsaturated, and n-3 fatty acids on blood pressure in healthy subjects. Am J Clin Nutr. 2006;83:221–6.

46. Ervin RB, Wright JD, Wang CY, Kennedy-Stephenson J. Dietary intake of fats and fatty acids for the United States population: 1999–2000. Adv Data. 2004;1–6.
47. Williams DE, Prevost AT, Whichelow MJ, Cox BD, Day NE, Wareham NJ. A cross-sectional study of dietary patterns with glucose intolerance and other features of the metabolic syndrome. Br J Nutr. 2000;83: 257–66.
48. Panagiotakos DB, Pitsavos C, Arvaniti F, Stefanadis C. Adherence to the Mediterranean food pattern predicts the prevalence of hypertension, hypercholesterolemia, diabetes and obesity, among healthy adults; the accuracy of the MedDietScore. Prev Med. 2007;44:335–40.
49. Shubair MM, McColl RS, Hanning RM. Mediterranean dietary components and body mass index in adults: the peel nutrition and heart health survey. Chronic Dis Can. 2005;26:43–51.
50. Grundy SM. Metabolic syndrome pandemic. Arterioscler Thromb Vasc Biol. 2008;28:629–36.
51. Esmaillzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. Am J Clin Nutr. 2007;85:910–8.
52. Vega GL, Adams-Huet B, Peshock R, Willett D, Shah B, Grundy SM. Influence of body fat content and distribution on variation in metabolic risk. J Clin Endocrinol Metab. 2006;91:4459–66.