

Dietary Patterns Are Associated with Metabolic Risk Factors in South Asians Living in the United States^{1–4}

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Abstract

Background: South Asians are at high risk of metabolic syndrome, and dietary patterns may influence this risk.

Objectives: We aimed to determine prevalent dietary patterns for South Asians in the United States and their associations with risk factors for metabolic syndrome.

Methods: South Asians aged 40–84 y without known cardiovascular disease were enrolled in a community-based cohort called Mediators of Atherosclerosis in South Asians Living in America. A validated food frequency questionnaire and serum samples for fasting and 2-h glucose, insulin, glycated hemoglobin, triglycerides, and total and HDL cholesterol were collected cross-sectionally. We used principal component analysis with varimax rotation to determine dietary patterns, and sequential linear and logistic regression models for associations with metabolic factors.

Results: A total of 892 participants were included (47% women). We identified 3 major dietary patterns: animal protein; fried snacks, sweets, and high-fat dairy; and fruits, vegetables, nuts, and legumes. These were analyzed by tertile of factor score. The highest vs. the lowest tertile of the fried snacks, sweets, and high-fat dairy pattern was associated with higher homeostasis model assessment of insulin resistance (HOMA-IR) (β : 1.88 mmol/L · uIU/L) and lower HDL cholesterol (β : –4.48 mg/dL) in a model adjusted for age, sex, study site, and caloric intake ($P < 0.05$). The animal protein pattern was associated with higher body mass index (β : 0.73 m/kg²), waist circumference (β : 0.84 cm), total cholesterol (β : 8.16 mg/dL), and LDL cholesterol (β : 5.69 mg/dL) (all $P < 0.05$). The fruits, vegetables, nuts, and legumes pattern was associated with lower odds of hypertension (OR: 0.63) and metabolic syndrome (OR: 0.53), and lower HOMA-IR (β : 1.95 mmol/L · uIU/L) ($P < 0.05$).

Conclusions: The animal protein and the fried snacks, sweets, and high-fat dairy patterns were associated with adverse metabolic risk factors in South Asians in the United States, whereas the fruits, vegetables, nuts, and legumes pattern was linked with a decreased prevalence of hypertension and metabolic syndrome. *J Nutr* 2015;145:1211–7.

Keywords: dietary patterns, South Asian, metabolic syndrome, diabetes, atherosclerosis

Introduction

A dietary pattern provides a tangible, modifiable risk factor for cardiovascular disease (CVD) and type 2 diabetes (1). Patterns

and trends in dietary consumption may provide more informative investigations into healthful or harmful dietary habits than analyses of individual nutrients alone (2). Prior observational research has shown that a Western dietary pattern, with strong components of refined carbohydrates and red and processed meats, is associated with increased rates of CVD and diabetes (3–5). Conversely, patterns high in whole grain, nut, and vegetable intake such as the Mediterranean pattern appear to be protective (6–8). To date, there is a paucity of information on dietary patterns and health among different immigrant ethnic groups in the United States.

Individuals of South Asian (Bangladeshi, Indian, Nepali, Pakistani, and Sri Lankan) origins are at particular risk of severe early onset CVD and type 2 diabetes, even before the development of obesity (9–11). Metabolic risk factors such as waist circumference, dyslipidemia, and deregulation of glucose and

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⁴ Supplemental Tables 1–3 are available from the “Online Supporting Material” link in the online posting of the article and from the same link in the online table of contents at <http://jn.nutrition.org>.

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insulin often underlie oxidative stress and atherosclerotic changes in this population (12–14). In the United States, over 90% of South Asians are immigrants and may be undergoing changes in dietary patterns that influence metabolic risk (15, 16).

This investigation aimed to identify prevalent dietary patterns in South Asians in the United States and their associations with metabolic risk factors with the use of data from participants enrolled in the Mediators of Atherosclerosis in South Asians Living in America (MASALA) study. To our knowledge, this is one of the first studies to investigate the association between dietary patterns and cardiometabolic risk in US South Asians, a group with known disparities in CVD and diabetes.

Methods

We conducted a cross-sectional investigation of 906 South Asians who participated in the MASALA community-based cohort study. The detailed methods have been described elsewhere (17). Briefly, this was a prospective cohort study in which we enrolled community-dwelling individuals living in the San Francisco Bay and the greater Chicago areas from 2010–2013. Participants self-identified as having South Asian ancestry, were aged 40–84 y, and had no known CVD. Those on nitroglycerin, with active cancer, impaired cognitive ability, or a life expectancy <5 y, or who lived in a nursing home or had plans to relocate were excluded. The University of California, San Francisco and Northwestern University institutional review boards approved the study protocol and all study participants provided written informed consent. Over ~30 mo of recruitment, 906 participants were enrolled in the MASALA study (17).

Each participant underwent in-person interviews to determine age, sex, medical history, and smoking status. Food group intake was collected with the Study of Health Assessment and Risk in Ethnic groups South Asian FFQ, which was developed and validated in South Asians in Canada (18). The FFQ included 163 items, with 61 items unique to the South Asian diet, and assessed usual eating habits, frequency, and serving sizes over the past 12 mo (18). Individual food items from the Study of Health Assessment and Risk in Ethnic groups food FFQ were divided into 29 predefined subgroups reflecting likeness, underlying nutrient composition, and culinary usage in the South Asian diet. Several foods (e.g., coffee) were kept as individual categories, given their high reported intake (Supplemental Table 1). Participants recorded the serving size (small, medium, or large) and the frequency of consumption from 4 options (average per day, week, month, or year, or never). Items reported as serving size “small” were weighted by 0.5, and items reported as serving size “large” were weighted by 1.5. We excluded one individual with incomplete FFQ data and another 13 who did not meet a priori criteria of daily caloric ranges for men (800–4200 kcal/24 h) and women (500–3500 kcal/24 h). A total of 892 remaining participants were included in our analysis.

All visits were conducted by trained bilingual study staff, and all consent forms were translated into Hindi and Urdu. We gathered information on participant demographic data, tobacco use, alcohol consumption, and medication use. Intentional exercise in metabolic equivalent task-minutes per week was assessed with the use of the Typical Week’s Physical Activity Questionnaire (19). Weight was determined with the use of a digital scale, height with a stadiometer, and waist circumference with the use of a measuring tape halfway between the lower ribs and the anterior superior iliac spine, at the site of greatest circumference. Seated resting blood pressure was measured 3 times with the use of an automated blood pressure monitor (V100 Vital Signs Monitor, GE Healthcare) taking the mean of the last 2 readings for analysis. Hypertension was defined as self-reported treatment for hypertension or a systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg. Blood samples were obtained after a requested 12 h fast. Fasting plasma glucose was measured with the use of a glucose oxidase method (Ortho Clinical Diagnostics, Johnson & Johnson), and fasting serum insulin was measured by the sandwich immunoassay method (Roche Elecsys 2010, Roche Diagnostics). Diabetes

was classified if a participant was using a glucose-lowering medication or had a fasting plasma glucose of at least 126 mg/dL.

Plasma concentrations of total cholesterol, HDL cholesterol, and TGs were measured with the use of enzymatic methods (Quest), and LDL cholesterol was calculated with the use of the Friedewald formula (20). An oral glucose tolerance test was performed in which participants consumed a 75 g oral glucose solution, and blood samples for plasma glucose and insulin were taken after 120 min. HOMA-IR, an assessment of insulin resistance, was calculated as $[\text{Glucose (mmol/L)} \times \text{Insulin (uIU/mL)}] / 22.5$ (21). Values are presented in the text and tables as means and ORs (95% CIs).

Principal components analysis with varimax rotation was used to determine factor loadings for the 29 investigator-categorized food groups. Solutions containing 2–5 factors were considered. After evaluation of factor solutions with eigenvalues >1 , a 3-factor solution was chosen based on evaluation of the scree plot (1). After identifying 3 patterns that explained 23.2% of the variance, we named these patterns according to the food groupings loading highest for each pattern (Supplemental Table 2). Each participant was assigned a factor score for each dietary pattern based on the correlation of his or her FFQ data with the food groupings in the 3 prevalent patterns. We separately derived factor solutions for men and women, and ultimately combined the outcomes given their similarities.

Baseline characteristics of the MASALA participants were compared by dietary pattern with the use of the chi-square test and ANOVA, where appropriate. Logistic and linear regression analyses were used to determine associations of tertiles of dietary pattern with prespecified metabolic outcomes for each dietary pattern separately. Age, BMI, units of alcoholic drinks per week, and metabolic equivalent task-minutes of exercise per week were modeled as continuous covariates; sex, education, income, and smoking were categorical variables. Model 1 was adjusted for age, sex, study site, and total caloric intake. Model 2 additionally was adjusted for income, education, metabolic equivalent tasks of exercise per week, alcoholic drinks per week, and smoking. Model 3 was adjusted for BMI and waist circumference in those models with outcomes that did not include BMI or waist circumference. Linear trends were determined with the use of factor scores as continuous covariates. Outcomes were normally distributed. Testing for sex-specific interaction was completed by adding an interaction term to the regression analyses for each dietary pattern. Two-sided *P* values < 0.05 were considered statistically significant.

The analysis was completed with the use of STATA version 11.2, 2012.

Results

Data from 892 discrete participants with full dietary information participating in the MASALA study were included in our cross-sectional analysis. Approximately 47% of the participants were women, and the vast majority (84%) were of Indian origin. Principal component analysis identified the following 3 predominant dietary patterns: 1) animal protein; 2) fried snacks, sweets, and high-fat dairy; and 3) fruits, vegetables, nuts, and legumes (Table 1). Women were less likely to consume the animal protein and fried snacks, sweets, and high-fat dairy patterns. Those who ate outside of the home 2–3 times/wk, were younger, had a higher BMI, and were current smokers were more likely to have higher adherence to the animal protein pattern (Table 1). The animal protein pattern alone contained major nonvegetarian components.

We examined within-pattern trends (Table 2). Comparing the highest to the lowest tertiles of each dietary pattern, an increase in factor score of the animal protein dietary pattern was associated with a higher BMI (β : 0.73 kg/m²; 95% CI: 0.01, 1.45), waist circumference (β : 1.15 cm; 95% CI: –0.49, 2.79), and total cholesterol (β : 8.16 mg/dL; 95% CI: 2.15, 14.17) and LDL cholesterol (β : 5.69 mg/dL; 95% CI: 0.41, 10.96) concentrations

TABLE 1 Baseline characteristics of the MASALA study population by dietary pattern in tertiles, 2010–2013¹

	Animal protein			Fried snacks, sweets, and high-fat dairy			Fruits, vegetables, nuts, and legumes					
	Tertile 1 (n = 297)	Tertile 2 (n = 297)	Tertile 3 (n = 298)	P-trend	Tertile 1 (n = 297)	Tertile 2 (n = 297)	Tertile 3 (n = 298)	P-trend	Tertile 1 (n = 297)	Tertile 2 (n = 297)	Tertile 3 (n = 298)	P-trend
Women, n (%)	176 (59)	138 (46)	106 (36)	<0.001	164 (55)	140 (47)	116 (39)	<0.001	129 (43)	152 (51)	139 (47)	0.59
Age, y	56.2 ± 9.29	55.9 ± 9.36	53.8 ± 9.35	0.001	56.1 ± 9.22	55.5 ± 9.30	54.4 ± 9.57	0.12	54.9 ± 9.56	54.9 ± 9.27	56.1 ± 9.29	0.09
BMI, kg/m ²	25.7 ± 3.76	26.0 ± 4.02	26.4 ± 5.04	0.003	26.0 ± 4.68	25.7 ± 4.13	26.4 ± 4.10	0.41	26.3 ± 4.49	26.0 ± 3.64	25.8 ± 4.73	0.40
Birth country, %				<0.001				0.90				0.95
Bangladesh	0	1	1		1	1	0		1	0	0	
India	93	90	69		81	87	83		82	85	84	
Pakistan	0	3	10		3	4	6		5	4	4	
Sri Lanka	0	0	3		2	1	1		2	0	1	
United States	1	0	5		3	1	1		3	2	1	
Subsaharan Africa	3	3	3		5	2	2		3	2	4	
Other	3	4	9		5	4	7		4	5	6	
Religious affiliation, %				0.002				0.97				0.38
Hinduism	84	70	48		68	69	65		65	69	68	
Christianity	0	2	7		3	4	3		4	2	3	
Islam	0	6	15		5	5	10		9	7	5	
Jainism	11	7	1		7	7	5		4	8	6	
Sikhism	3	8	12		5	8	10		7	5	10	
Other	2	7	17		11	8	7		10	8	7	
Family income >\$75K, %	70	78	72	0.96	80	74	66	<0.001	69	76	75	0.06
Smoker, %				<0.001				0.221				0.04
Never	93	84	73		85	84	81		80	85	85	
Former	6	15	20		12	13	15		14	14	13	
Current	1	2	7		3	3	3		6	1	3	
Exercise, log MET-min/wk	6.97 ± 0.89	7.01 ± 0.88	6.95 ± 1.02	0.32	7.10 ± 0.86	7.05 ± 0.88	6.75 ± 1.02	<0.001	6.79 ± 0.98	6.93 ± 0.94	7.17 ± 0.83	<0.001
Eating out, times/wk, %				<0.001				0.18				0.89
2 or 3	4	9	20		14	10	9		10	13	10	
1 time	28	37	34		32	31	37		33	33	33	
<1 time	68	54	46		55	59	54		57	55	57	
Energy intake, kcal/d	1620 ± 467	1590 ± 435	1840 ± 566	<0.001	1370 ± 371	1650 ± 398	2030 ± 502	<0.001	1370 ± 383	1640 ± 384	2040 ± 489	<0.001

¹ Values are means ± SDs unless otherwise indicated. MET, metabolic equivalent task.

TABLE 2 Associations between metabolic outcomes and dietary pattern for 892 MASALA study participants¹

	Animal protein			Fried snacks, sweets, and high-fat dairy			Fruits, vegetables, nuts, and legumes		
	Tertile 2	Tertile 3	P-trend ²	Tertile 2	Tertile 3	P-trend ²	Tertile 2	Tertile 3	P-trend ²
Hypertension (mm Hg)									
Model 1	1.04 (0.73, 1.49)	1.18 (0.82, 1.71)	0.17	1.23 (0.86, 1.78)	0.86 (0.56, 1.31)	0.60	0.95 (0.66, 1.37)	0.63 (0.41, 0.97)	0.03
Model 2	1.11 (0.74, 1.66)	1.06 (0.68, 1.67)	0.45	1.40 (0.94, 2.10)	0.73 (0.45, 1.21)	0.12	1.04 (0.68, 1.59)	1.02 (0.47, 1.28)	0.25
Model 3	1.06 (0.70, 1.60)	1.03 (0.65, 1.62)	0.80	1.53 (1.01, 2.31)	0.80 (0.95, 2.23)	0.19	1.03 (0.67, 1.59)	0.80 (0.62, 1.51)	0.24
Metabolic syndrome									
Model 1	0.76 (0.54, 1.07)	0.90 (0.63, 1.28)	0.14	1.24 (0.87, 1.77)	1.16 (0.77, 1.73)	0.56	0.91 (0.64, 1.29)	0.53 (0.35, 0.82)	0.007
Model 2	0.71 (0.48, 1.04)	0.80 (0.52, 1.24)	0.57	1.25 (0.85, 1.85)	0.87 (0.54, 1.39)	0.10	1.02 (0.68, 1.53)	0.62 (0.38, 1.02)	0.08
Model 3	0.61 (0.40, 1.11)	0.69 (0.43, 1.10)	0.73	1.46 (0.95, 2.23)	0.95 (0.56, 1.59)	0.18	0.91 (0.62, 1.51)	0.65 (0.38, 1.11)	0.08
Diabetes									
Model 1	0.94 (0.69, 1.29)	1.09 (0.80, 1.50)	0.54	1.39 (1.01, 1.90)	1.17 (0.81, 1.68)	0.55	0.94 (0.68, 1.29)	0.70 (0.48, 1.01)	0.15
Model 2	0.92 (0.65, 1.30)	1.10 (0.75, 1.62)	0.71	1.34 (0.95, 1.89)	0.88 (0.58, 1.34)	0.19	1.01 (0.71, 1.45)	0.83 (0.54, 1.27)	0.46
Model 3	0.89 (0.63, 1.25)	1.05 (0.71, 1.54)	0.91	1.42 (1.01, 2.02)	0.94 (0.62, 1.44)	0.31	0.98 (0.68, 1.40)	0.84 (0.54, 1.29)	0.40
BMI (kg/m²)									
Model 1	0.38 (-0.33, 1.08)	0.73 (0.01, 1.45)	0.004	-0.29 (-1.01, 0.42)	0.30 (-0.52, 1.12)	0.60	-0.56 (-1.28, 0.17)	-0.88 (-1.72, -0.04)	0.14
Model 2	0.30 (-0.48, 1.07)	0.50 (-0.36, 1.36)	0.06	-0.60 (-1.37, 0.18)	-0.28 (-1.20, 0.65)	0.29	-0.30 (-1.13, 0.52)	-0.61 (-1.57, 0.36)	0.08
Waist circumference (cm)									
Model 1	0.60 (-1.00, 2.20)	1.15 (-0.49, 2.79)	0.004	-0.45 (-2.09, 1.19)	0.53 (-1.33, 2.40)	0.13	0.30 (-1.35, 1.94)	-1.54 (-3.45, 0.38)	0.21
Model 2	0.82 (-0.89, 2.53)	0.66 (-1.24, 2.57)	0.06	-1.34 (-3.06, 0.38)	-1.53 (-3.59, 0.52)	0.37	1.21 (-0.60, 3.02)	-0.61 (-2.74, 1.51)	0.90
HOMA-IR (mmol/L · uIU/L)									
Model 1	0.93 (-0.10, 1.95)	0.78 (-0.28, 1.84)	0.16	0.54 (-0.51, 1.59)	1.88 (0.67, 3.08)	0.001	-1.16 (-2.21, -0.10)	-1.95 (-3.18, -0.72)	0.001
Model 2	0.96 (-0.12, 2.03)	1.25 (0.05, 2.45)	0.05	-0.09 (-1.18, 1.00)	0.40 (-0.91, 1.72)	0.27	-0.93 (-2.07, 0.21)	-1.75 (-3.08, -0.41)	0.03
Model 3	0.87 (-0.19, 1.93)	1.13 (-0.05, 2.32)	0.12	0.10 (-0.98, 1.17)	0.54 (-0.76, 1.83)	0.19	-0.97 (-2.09, 0.16)	-1.64 (-2.96, -0.32)	0.03
Total cholesterol (mg/dL)									
Model 1	4.20 (-1.66, 10.1)	8.16 (2.15, 14.2)	0.009	-1.92 (-9.10, 4.63)	-2.25 (-9.10, 4.63)	0.63	-4.55 (-10.6, 1.50)	-1.51 (-8.56, 5.54)	0.85
Model 2	3.84 (-2.63, 10.3)	8.81 (1.60, 16.0)	0.02	-3.76 (-10.3, 2.77)	-5.89 (-13.7, 1.90)	0.27	-7.17 (-14.1, -0.31)	-1.66 (-9.72, 6.41)	0.34
Model 3	3.84 (-2.64, 10.3)	8.36 (1.14, 15.6)	0.02	-3.84 (-10.4, 2.69)	-6.45 (-14.2, 1.35)	0.26	-7.21 (-14.1, -0.32)	-1.67 (-9.74, 6.39)	0.34
TGs (mg/dL)									
Model 1	-5.74 (-17.2, 5.72)	-0.02 (-11.8, 11.7)	0.288	1.41 (-10.3, 13.1)	6.22 (-7.17, 19.6)	0.21	-6.96 (-18.8, 4.84)	-10.7 (-24.4, 3.08)	0.30
Model 2	-6.19 (-18.9, 6.47)	0.28 (-13.8, 14.4)	0.43	0.51 (-12.2, 13.2)	1.49 (-13.7, 16.7)	0.81	-6.17 (-19.6, 7.3)	-8.62 (-24.4, 7.15)	0.79
Model 3	-7.12 (-19.7, 5.42)	-0.92 (-14.9, 13.1)	0.64	2.09 (-10.6, 14.7)	3.02 (-12.1, 18.1)	0.71	-7.33 (-20.7, 6.04)	-7.73 (-23.4, 7.90)	0.78
LDL-C (mg/dL)									
Model 1	3.41 (-1.73, 8.55)	5.69 (0.41, 11.0)	0.04	1.43 (-3.83, 6.69)	2.50 (-3.52, 8.52)	0.55	-4.65 (-9.96, 0.66)	-2.25 (-8.42, 3.93)	0.97
Model 2	3.18 (-2.49, 8.84)	7.11 (0.80, 13.4)	0.02	-0.67 (-6.39, 5.04)	-1.11 (-7.93, 5.70)	0.72	-7.10 (-13.1, -1.09)	-2.47 (-9.52, 4.58)	0.41
Model 3	3.14 (-2.52, 8.79)	6.52 (0.21, 12.8)	0.03	-0.63 (-6.33, 5.08)	-1.65 (-8.45, 5.16)	0.72	-6.94 (-13.0, -0.91)	-2.31 (-9.33, 4.73)	0.39
HDL-C (mg/dL)									
Model 1	1.52 (-0.46, 3.50)	2.44 (0.41, 4.47)	0.12	-3.21 (-5.21, -1.20)	-4.48 (-6.77, -2.19)	<0.001	0.70 (-1.33, 2.74)	1.78 (-0.59, 4.16)	0.11
Model 2	1.66 (-0.51, 3.83)	1.77 (-0.64, 4.18)	0.41	-2.64 (-4.82, -0.47)	-3.17 (-5.76, -0.58)	0.07	-0.17 (-2.48, 2.13)	1.14 (-1.56, 3.84)	0.48
Model 3	1.86 (-0.26, 3.98)	2.12 (-0.25, 4.49)	0.19	-3.04 (-5.17, 0.92)	-3.46 (-6.00, -0.92)	0.04	-0.12 (-2.39, 2.15)	0.84 (-1.81, 3.49)	0.50

(Continued)

TABLE 2 Continued

	Animal protein			Fried snacks, sweets, and high-fat dairy			Fruits, vegetables, nuts, and legumes		
	Tertile 2	Tertile 3	P-trend ^z	Tertile 2	Tertile 3	P-trend ^z	Tertile 2	Tertile 3	P-trend ^z
Fasting glucose (mg/dL)									
Model 1	1.50 (-2.45, 5.45)	3.28 (-0.79, 7.35)	0.03	4.86 (0.83, 8.90)	0.94 (-3.66, 5.54)	0.43	-0.55 (-4.62, 3.51)	-4.11 (-8.87, 0.64)	0.48
Model 2	1.31 (-3.03, 5.65)	4.45 (-0.40, 9.30)	0.02	3.16 (-1.21, 7.52)	-2.66 (-7.87, 2.55)	0.05	-0.93 (-5.54, 3.68)	-3.94 (-9.36, 1.48)	0.52
Model 3	1.03 (-3.28, 5.33)	4.26 (-0.56, 9.08)	0.04	3.69 (-0.64, 8.02)	-2.11 (-7.28, 3.06)	0.07	-1.16 (-5.75, 3.43)	-3.61 (-8.98, 1.76)	0.52
Hb A _{1c} (%)									
Model 1	0.06 (-0.08, 0.20)	0.09 (-0.05, 0.24)	0.15	0.17 (0.03, 0.31)	0.05 (-0.11, 0.21)	1.0	0.01 (-0.13, 0.15)	-0.14 (-0.31, 0.02)	0.43
Model 2	0.08 (-0.07, 0.23)	0.15 (-0.01, 0.32)	0.12	0.10 (-0.05, 0.25)	-0.10 (-0.28, 0.08)	0.07	0.03 (-0.13, 0.19)	-0.09 (-0.27, 0.10)	0.87
Model 3	0.07 (-0.08, 0.22)	0.13 (-0.03, 0.30)	0.23	0.12 (-0.03, 0.27)	-0.09 (-0.26, 0.09)	0.08	0.14 (-0.14, 0.17)	-0.08 (-0.26, 0.11)	0.85

¹ Values are ORs (95% CIs) for hypertension, metabolic syndrome, and diabetes categories, and βs (95% CIs) for all other categories. Associations are compared with lowest tertile of dietary pattern factor score. Model 1 is adjusted for kcal/24 h, study site, age, and sex. Model 2 is additionally adjusted for income, education, smoking, alcohol intake, and exercise. Model 3 is additionally adjusted for BMI and waist circumference. Hb A_{1c}, glycated hemoglobin; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; MASALA, Mediators of Atherosclerosis in South Asians Living in America.

² P-trend using dietary pattern factor score as continuous covariate.

(*P*-trend < 0.05) in a model adjusted for age, sex, study site, and caloric intake (Model 1). When additionally adjusted for income, education, metabolic equivalent tasks of exercise per week, alcoholic drinks per week, and smoking, we found a higher total cholesterol (β: 8.81 mg/dL; 95% CI: 1.60, 16.01) and LDL cholesterol (β: 7.11 mg/dL; 95% CI: 0.80, 13.42) concentration (Model 2, *P* = 0.05). The addition of BMI and waist circumference to the covariates (Model 3) did not change the significance of total and LDL cholesterol concentration outcomes (*P* < 0.05) (Table 2). An increase in factor score of the fried snacks, sweets, and high-fat dairy pattern was associated with a significantly higher HOMA-IR (β: 1.88 mmol/L · uIU/L; 95% CI: 0.67, 3.08; *P*-trend = 0.001) and lower HDL cholesterol (β: -4.48 mg/dL; 95% CI: -6.77, -2.19; *P* < 0.001) in Model 1. Greater consumption of the fruits, vegetables, nuts, and legumes pattern was associated with lower odds of hypertension (OR: 0.63; 95% CI: 0.41, 0.97) and metabolic syndrome (OR: 0.53; 95% CI: 0.35, 0.82) (*P* < 0.05) in Model 1 and a lower HOMA-IR (β: -1.95; 95% CI: -3.18, -0.72; *P* = 0.001) (β: -1.75; 95% CI: -3.08, -0.41; *P* = 0.03) in Models 1 and 2, respectively.

Testing for interaction between sex and dietary pattern yielded a significant (*P* = 0.03) result for the fried snacks, sweets, and high-fat dairy pattern for the HDL cholesterol outcome. In Model 1 and Model 2, only women had a significantly lower HDL cholesterol with an increasing tertile for the fried snacks, sweets, and high-fat dairy factor score (*P*-trend: 0.001 and 0.008, respectively) (Supplemental Table 3).

Discussion

Our investigation identified 3 prevalent dietary patterns in South Asians living in the United States that we termed animal protein; fried snacks, sweets, and high-fat dairy; and fruits, vegetables, nuts, and legumes. The animal protein dietary pattern was associated with higher BMI and waist-to-hip ratio measurements. The fried snacks, sweets, and high-fat dairy pattern, a vegetarian pattern, was similarly associated with greater insulin resistance, as measured by HOMA-IR, and lower HDL cholesterol. Both the animal protein and fried snacks, sweets, and high-fat dairy dietary patterns showed evidence of association with adverse metabolic outcomes, suggesting that modification of major components of these dietary patterns may ameliorate metabolic risk factors.

Diet is a major modifiable risk factor for diabetes and heart disease. Long-term programs that impose intensive diet and lifestyle changes have been shown to reduce progression to diabetes from a prediabetes state (22, 23). Certain food patterns high in refined carbohydrates, such as the fried snacks, sweets, and high-fat dairy pattern, and red and processed meats, such as the animal protein pattern in this study, have been identified as particular contributors to adiposity, β cell dysfunction and overall weight gain (5, 6, 24, 25). Alterations in dietary patterns toward the fruit, vegetables, nuts, and legumes pattern is one potential public health tactic to elicit population-based decreases in diabetes and CVD risk.

South Asians are at markedly elevated risk of the development of diabetes and CVD compared with many other racial or ethnic groups. It is estimated that India alone will have over 100 million diabetic individuals by the year 2030 (26), the vast majority suffering from type 2 diabetes. This epidemic has occurred side by side with the greater availability of calorie-dense and nutritionally-poor foods, sedentary lives, and a rise in

obesity and tobacco use (15, 27). In the United States, there are no comprehensive programs to combat these disparities in health risk for the rapidly growing South Asian population. In our findings, the attenuation of associations between Models 1 and 3 suggests that BMI, physical exercise, and social determinants may lie on the causal pathway between diet and the development of diabetes. Changing dietary habits, in concert with lifestyle factors, is an area of major public health relevance and concern for this population, because it represents a potential avenue for future risk mitigation.

Insulin resistance is an underlying deregulation of the physiologic mechanisms required to process sugars and starches, and precedes the development of diabetes (28). We measured insulin resistance by the surrogate marker, HOMA-IR (21), and observed a strong association between elevated HOMA-IR and increased consumption of the fried snacks, sweets, and high-fat dairy pattern. Added sugars, refined carbohydrates, and white rice (29) are all major components of the fried snacks, sweets, and high-fat dairy dietary pattern, and have been associated with an increased risk of insulin resistance and diabetes. The dairy products and sweets abundant in this dietary pattern contain high amounts of added sugars and saturated fat, which, in a recent study of high saturated fat intake in South Asians and Caucasians, was shown to impair insulin regulation specifically in the South Asian individuals (14, 30). In this investigation, young, lean, and healthy South Asian and Caucasian men were given a high saturated fat diet for 5 d, and their insulin resistance was measured dynamically with a hyperinsulinemic-euglycemic clamp before and after the intervention. The mechanism of the difference is as yet unclear, but may involve derailment of mitochondrial FA oxidation by a high saturated fat diet. Elucidation of this mechanism may hold clues to why the adoption by South Asians of a fried snacks, sweets, and high-fat dairy dietary pattern such as that described in our study may be a major factor in the development of metabolic syndrome in this population.

The animal protein pattern was associated with higher fasting glucose. These findings are supported by similar results in established longitudinal cohorts that show a higher incidence of diabetes with greater long-term consumption of animal protein in several populations (3, 5, 25). In comparison, the risk of metabolic syndrome was lower with increasing intake of the fruits, vegetables, nuts, and legumes pattern, suggesting that a plant-based pattern may successfully reduce risk of this disease.

A second major metabolic risk factor is low HDL cholesterol, which has previously been associated with poor cardiovascular outcomes and is part of the South Asian dyslipidemia pattern (9, 31). The fried snacks, sweets, and high-fat dairy pattern includes white flour-based snacks, typically fried in vegetable oil, and high-fat dairy products that are often sweetened, such as ice cream and dairy-based South Asian desserts. The consumption of these foods over time has been associated with lower HDL cholesterol, and, in turn, with the development of atherosclerosis (32, 33). Anchored by fried foods and high amounts of saturated fat intake, the fried snacks, sweets, and high-fat dairy pattern is a major example of an unhealthy predominantly vegetarian style of eating. In our pilot study, a dietary pattern we termed the “vegetarian” diet, containing major components of sugar-sweetened beverages and refined grains, was also associated with significantly lower HDL cholesterol (34). Taken together, these findings strongly imply that a dietary pattern with added sugars and refined grains may negatively affect HDL cholesterol concentrations.

A third major metabolic risk factor is abdominal obesity, reflected in our investigation through waist circumference. We found both a higher BMI and waist circumference associated with increased consumption of the animal protein pattern, a result supported by several long-term observational studies (15, 35). Participants who have higher adherence to this pattern are most likely to eat outside of the home at least 2–3 times/wk, which suggests that they may consume more processed or externally prepared foods that contain fewer plant-based ingredients. A body of observational and controlled trial-based evidence has also developed showing that increased consumption of red and processed meats may also increase the risk of insulin resistance and diabetes (5, 25, 33, 36). Because obesity in both native South Asians and South Asian diaspora is increasing, and is closely associated with diabetes and CVD risk factors (37–39), shifting of dietary patterns to control this trend is paramount.

The limitations of our investigation include the cross-sectional nature of our analysis of dietary patterns and metabolic risk. In the future, we plan to investigate longitudinal effects of these dietary patterns with the use of the ongoing MASALA cohort. Dietary data were self-reported and may lend some biases to the evaluation of individual food groups; however, we used an FFQ validated in and tailored to South Asians, and overall patterns should still reflect trends in consumption. The major strength remains that this is the first multicenter evaluation of dietary patterns and metabolic disease in a cohort of South Asians in the United States.

Dietary patterns are collective habits of nutrient intake that have associations with metabolic disease. South Asians in the United States have particular constellations of dietary preferences associated with components of metabolic syndrome, including an animal protein pattern, here associated with higher waist circumference, and a fried snacks, sweets, and high-fat dairy dietary pattern, associated with lower HDL cholesterol. Culturally appropriate interventions to alter deleterious dietary patterns can be cornerstones to improving metabolic risk factors in the rapidly growing and high-risk US South Asian population.

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MDG conceived of the analytic design, performed the analysis, and wrote the manuscript. CAMA and NRK contributed to the interpretation of the results and reviewed and edited the manuscript. AMK conceived of the project idea, contributed to the interpretation of the results, and reviewed and edited the manuscript. MDG had primary responsibility for the final content. All authors read and approved the final manuscript.

References

1. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol* 2002;13:3–9.
2. Newby PK, Muller D, Hallfrisch J, Andres R, Tucker KL. Food patterns measured by factor analysis and anthropometric changes in adults. *Am J Clin Nutr* 2004;80:504–13.
3. Bauer F, Beulens JW, van der ADL, Wijmenga C, Grobbee DE, Spijkerman AM, van der Schouw YT, Onland-Moret NC. Dietary patterns and the risk of type 2 diabetes in overweight and obese individuals. *Eur J Nutr* 2013;52:1127–34.
4. Fagherazzi G, Vilier A, Saes Sartorelli D, Lajou M, Balkau B, Clavel-Chapelon F. Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidemiologique aupres des femmes de la Mutuelle Generale de l'Education Nationale-European Prospective Investigation into Cancer and Nutrition cohort. *Am J Clin Nutr* 2013;97:517–23.

5. Bendinelli B, Palli D, Masala G, Sharp SJ, Schulz MB, Guevara M, van der AD, Sera F, Amiano P, Balkau B, et al. Association between dietary meat consumption and incident type 2 diabetes: the EPIC-InterAct study. *Diabetologia* 2013;56:47–59.
6. Nettleton JA, Polak JF, Tracy R, Burke GL, Jacobs DR, Jr. Dietary patterns and incident cardiovascular disease in the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr* 2009;90:647–54.
7. Newby PK. Plant foods and plant-based diets: protective against childhood obesity? *Am J Clin Nutr* 2009;89:1572S–87S.
8. Liu E, McKeown NM, Newby PK, Meigs JB, Vasan RS, Quatromoni PA, D'Agostino RB, Jacques PF. Cross-sectional association of dietary patterns with insulin-resistant phenotypes among adults without diabetes in the Framingham Offspring Study. *Br J Nutr* 2009;102:576–83.
9. Anand SS, Yusuf S, Vuksan V, Devarnesen S, Teo KK, Montague PA, Kelemen L, Yi C, Lonn E, Gerstein H, et al. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). *Lancet* 2000;356:279–84.
10. Bakker LE, Sleddering MA, Schoones JW, Meinders AE, Jazet IM. Pathogenesis of type 2 diabetes in South Asians. *Eur J Endocrinol* 2013;169:R99–114.
11. Staimez LR, Weber MB, Narayan KM, Oza-Frank R. A systematic review of overweight, obesity, and type 2 diabetes among Asian American subgroups. *Curr Diabetes Rev* 2013;9:312–31.
12. Razak F, Anand S, Vuksan V, Davis B, Jacobs R, Teo KK, Yusuf S. SHARE Investigators. Ethnic differences in the relationships between obesity and glucose-metabolic abnormalities: a cross-sectional population-based study. *Int J Obes (Lond)* 2005;29:656–67.
13. Sleddering MA, Bakker LE, Janssen LG, Meinders AE, Jazet IM. Higher insulin and glucagon-like peptide-1 (GLP-1) levels in healthy, young South Asians as compared to Caucasians during an oral glucose tolerance test. *Metabolism* 2014;63:226–32.
14. Bakker LE, van Schinkel LD, Guigas B, Streefland TC, Jonker JT, van Klinken JB, van der Zon GC, Lamb HJ, Smit JW, Pijl H, et al. A 5-day high-fat, high-calorie diet impairs insulin sensitivity in healthy, young South Asian men but not in Caucasian men. *Diabetes* 2014;63:248–58.
15. Skar M, Villumsen AB, Christensen DL, Petersen JH, Deepa M, Anjana RM, Pradeepa R, Mohan V. Increased risk of type 2 diabetes with ascending social class in urban South Indians is explained by obesity: The Chennai urban rural epidemiology study (CURES-116). *Indian J Endocrinol Metab* 2013;17:1084–9.
16. Novotny R, Chen C, Williams AE, Albright CL, Nigg CR, Oshiro CE, Steves VJ. US acculturation is associated with health behaviors and obesity, but not their change, with a hotel-based intervention among Asian-Pacific Islanders. *J Acad Nutr Diet* 2012;112:649–56.
17. Kanaya AM, Kandula N, Herrington D, Budoff MJ, Hulley S, Vittinghoff E, Liu K. Mediators of Atherosclerosis in South Asians Living in America (MASALA) study: objectives, methods, and cohort description. *Clin Cardiol* 2013;36:713–20.
18. Kelemen LE, Anand SS, Vuksan V, Yi Q, Teo KK, Devarnesen S, Yusuf S. SHARE investigators. development and evaluation of cultural food frequency questionnaires for South Asians, Chinese, and Europeans in North America. *J Am Diet Assoc* 2003;103:1178–84.
19. Ainsworth BE, Irwin ML, Addy CL, Whitt MC, Stolarczyk LM. Moderate physical activity patterns of minority women: the Cross-Cultural Activity Participation Study. *J Womens Health Gend Based Med* 1999;8:805–13.
20. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972;18:499–502.
21. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412–9.
22. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM., Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393–403.
23. Ramachandran A, Arun N, Shetty AS, Snehalatha C. Efficacy of primary prevention interventions when fasting and postglucose dysglycemia coexist: analysis of the Indian Diabetes Prevention Programmes (IDPP-1 and IDPP-2). *Diabetes Care* 2010;33:2164–8.
24. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* 2014;174:516–24.
25. van Woudenberg GJ, Kuijsten A, Tigcheler B, Sijbrands EJ, van Rooij FJ, Hofman A, Witteman JC, Feskens EJ. Meat consumption and its association with C-reactive protein and incident type 2 diabetes: the Rotterdam Study. *Diabetes Care* 2012;35:1499–505.
26. International Diabetes Federation. IDF diabetes atlas, 6th edn [Internet]. Brussels: International Diabetes Federation. 2013 [cited 2014 Feb 21]. Available from: <http://www.idf.org/diabetesatlas>.
27. Mohan V, Radhika G, Sathya RM, Tamil SR, Ganesan A, Sudha V. Dietary carbohydrates, glycaemic load, food groups and newly detected type 2 diabetes among urban Asian Indian population in Chennai, India (Chennai Urban Rural Epidemiology Study 59). *Br J Nutr* 2009;102:1498–506.
28. National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). 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) final report. *Circulation* 2002;106:3143–421.
29. Sun Q, Spiegelman D, van Dam RM, Holmes MD, Malik VS, Willett WC, Hu FB. White rice, brown rice, and risk of type 2 diabetes in US men and women. *Arch Intern Med* 2010;170:961–9.
30. Merchant AT, Kelemen LE, de Koning L, Lonn E, Vuksan V, Jacobs R, Davis B, Teo KK, Yusuf S, Anand SS, et al. Interrelation of saturated fat, trans fat, alcohol intake, and subclinical atherosclerosis. *Am J Clin Nutr* 2008;87:168–74.
31. Flowers E, Molina C, Mathur A, Reaven GM. Adiposity and cardiovascular risk clustering in South Asians. *Metab Syndr Relat Disord* 2013;11:434–40.
32. Merchant AT, Anand SS, Kelemen LE, Vuksan V, Jacobs R, Davis B, Teo K, Yusuf S., SHARE and SHARE-AP Investigators. Carbohydrate intake and HDL in a multiethnic population. *Am J Clin Nutr* 2007;85:225–30.
33. Lopez-Garcia E, Schulze MB, Fung TT, Meigs JB, Rifai N, Manson JE, Hu FB. Major dietary patterns are related to plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr* 2004;80:1029–35.
34. Gadgil MD, Anderson CA, Kandula NR, Kanaya AM. Dietary patterns in Asian Indians in the United States: an analysis of the metabolic syndrome and atherosclerosis in South Asians living in America study. *J Acad Nutr Diet* 2014;114:238–43.
35. Newby PK, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and changes in body mass index and waist circumference in adults. *Am J Clin Nutr* 2003;77:1417–25.
36. Nettleton JA, Steffen LM, Mayer-Davis EJ, Jenny NS, Jiang R, Herrington DM, Jacobs DR, Jr. Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr* 2006;83:1369–79.
37. Chopra SM, Misra A, Gulati S, Gupta R. Overweight, obesity and related non-communicable diseases in Asian Indian girls and women. *Eur J Clin Nutr* 2013;67:688–96.
38. Prasad DS, Kabir Z, Dash AK, Das BC. Effect of obesity on cardiometabolic risk factors in Asian Indians. *J Cardiovasc Dis Res* 2013;4:116–22.
39. Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. *Nutrients* 2013;5:2708–33.