

# Dietary Patterns and Risk of Incident Type 2 Diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA)

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**OBJECTIVE** — We characterized dietary patterns and their relation to incident type 2 diabetes in 5,011 participants from the Multi-Ethnic Study of Atherosclerosis (MESA).

**RESEARCH DESIGN AND METHODS** — White, black, Hispanic, and Chinese adults, aged 45–84 years and free of cardiovascular disease and diabetes, completed food frequency questionnaires at baseline (2000–2002). Incident type 2 diabetes was defined at three follow-up exams (2002–2003, 2004–2005, and 2005–2007) as fasting glucose >126 mg/dl, self-reported type 2 diabetes, or use of diabetes medication. Two types of dietary patterns were studied: four empirically derived (principal components analysis) and one author-defined (low-risk food pattern) as the weighted sum of whole grains, vegetables, nuts/seeds, low-fat dairy, coffee (positively weighted), red meat, processed meat, high-fat dairy, and soda (negatively weighted).

**RESULTS** — The empirically derived dietary pattern characterized by high intake of tomatoes, beans, refined grains, high-fat dairy, and red meat was associated with an 18% greater risk (hazard ratio per 1-score SD 1.18 [95% CI 1.06–1.32];  $P_{\text{trend}} = 0.004$ ), whereas the empirically derived dietary pattern characterized by high intake of whole grains, fruit, nuts/seeds, green leafy vegetables, and low-fat dairy was associated with a 15% lower diabetes risk (0.85 [0.76–0.95];  $P_{\text{trend}} = 0.005$ ). The low-risk food pattern was also inversely associated with diabetes risk (0.87 [0.81–0.99];  $P_{\text{trend}} = 0.04$ ). Individual component food groups were not independently associated with diabetes risk. Associations were not modified by sex or race/ethnicity.

**CONCLUSIONS** — Multiple food groups collectively influence type 2 diabetes risk beyond that of the individual food groups themselves.

*Diabetes Care* 31:1777–1782, 2008

Type 2 diabetes and obesity have reached epidemic proportions in the U.S. and the world. In addition to the role diet plays in preventing obesity and, consequently, type 2 diabetes, diet may also reduce risk of type 2 diabetes independent of changes in body weight. Whole grains (1), nuts/seeds (2), coffee (3), low-fat dairy (4,5), and vegetables (6, 7) have been inversely associated with incident type 2 diabetes or related metabolic traits independent of differences in body weight or other measures of

adiposity, whereas sugar-sweetened beverages (8,9), red meat (10), processed meats (11), and white potatoes (fried or baked/boiled) (12) have been positively associated.

In practice, each nutrient or food is part of a larger pattern consisting of many nutrients and foods, and, thus, characterization of multiple, concurrent dietary exposures has particular relevance to health. Using data-driven techniques, such as principal components analysis (PCA), several epidemiological studies have eval-

uated associations between dietary patterns and type 2 diabetes (10,11,13–15). Generally, studies show that dietary patterns characterized by high whole grain, fruit/vegetable, and low-fat dairy intake are inversely associated with type 2 diabetes risk. Analogously, dietary patterns characterized by high intake of red or processed meats, refined grains, fried foods, and foods containing high amounts of added sugars are associated with greater type 2 diabetes risk. Studies have been conducted in relatively homogenous populations (predominantly white cohorts) (10,11,13–15). Validation of these findings in racially/ethnically diverse samples is needed.

Empirical methods such as PCA do not necessarily maximize the disease-predictive value of each dietary pattern; rather, such methods maximize the amount of variation in dietary intake explained by each dietary pattern. Variation in dietary intake comprises dietary behaviors, taste, and convenience. For this reason, combinations of foods other than those identified by PCA might be more predictive of incident disease. Studies have shown that individual food groups, such as those listed above, are independently associated with incident type 2 diabetes. However, the collective contribution of these foods to type 2 diabetes risk has not been characterized.

Using data from the Multi-Ethnic Study of Atherosclerosis (MESA), we evaluated the relationship between type 2 diabetes risk and the following two trends: 1) PCA-derived dietary patterns and 2) a low-risk food pattern score based on the intake of foods previously associated with risk of type 2 diabetes (whole grains, vegetables, low-fat dairy foods, nuts/seeds, and coffee [positively weighted] and red meat, processed meat, high-fat dairy foods, white potatoes, and nondiet soda [negatively weighted]).

## RESEARCH DESIGN AND METHODS

MESA is a population-based study of 6,814 Caucasian, African American, Hispanic, and Chinese adults, aged 45–84 years, initiated to investigate the prevalence and progression of sub-

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Received 21 April 2008 and accepted 23 May 2008.

Published ahead of print at <http://care.diabetesjournals.org> on 10 June 2008. DOI: 10.2337/dc08-0760.

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clinical cardiovascular disease (CVD). Information on demographics, lifestyle characteristics, and clinical risk factors were obtained in six field centers: Baltimore City and County, Maryland; Chicago, Illinois; Forsyth County, North Carolina; New York, New York; Los Angeles County, California; and St. Paul, Minnesota (16). Each examination cycle spanned 2 years, with baseline (2000–2002) and three follow-up exams conducted from 2002–2003, 2004–2005, and 2005–2007. Institutional review board approval was obtained at all participating centers, and all participants gave informed consent. The longitudinal investigation presented here includes data from 5,011 participants, including 2,634 men and 2,377 women (2,177 white, 1,205 black, 1,016 Hispanic, and 613 Chinese), after excluding individuals with type 2 diabetes at the baseline examination ( $n = 859$ ), individuals for whom baseline diabetes status was unknown or for whom diabetes status was not updated over follow-up ( $n = 328$ ), and individuals who provided insufficient or implausible dietary information ( $n = 630$ ) (numbers not mutually exclusive).

### Assessment of type 2 diabetes

Fasting serum glucose was measured at each exam by rate reflectance spectrophotometry using thin film adaptation of the glucose oxidase method on the Vitros analyzer (Johnson & Johnson Clinical Diagnostics, Rochester, NY). Type 2 diabetes was defined as self-reported type 2 diabetes, fasting glucose  $\geq 126$  mg/dl at any exam, or use of antidiabetes medication. Incident cases comprise individuals without type 2 diabetes at baseline who met any one of the three criteria listed above at follow-up examinations. Consistency of the serum glucose assay over examinations was established by reanalyzing 200 samples from each of the four examinations over a short time period and then recalibrating the original observations. Event dates for incident type 2 diabetes were considered to be the examination dates (exam 2, 3, or 4) at which type 2 diabetes was first identified. Over the course of follow-up, 8.5% of those never diagnosed with type 2 diabetes did not attend the fourth exam, whereas 7.7% of those diagnosed with type 2 diabetes over follow-up did not attend the fourth exam.

### Diet assessment

Usual dietary intake over the preceding year was quantified by 120-item food fre-

quency questionnaire (FFQ) at baseline (17). The FFQ was developed in the validated block format, patterned after the FFQ used in the Insulin Resistance Atherosclerosis Study (IRAS), and validated in non-Hispanic white, African American, and Hispanic individuals (18). In order to accommodate the MESA subject population, the IRAS FFQ was modified to include unique Chinese foods and culinary practices. Participants recorded serving size (small, medium, or large) and frequency of consumption of specific beverage and food items. Nine frequency options were given, ranging from “rare or never” to a maximum of “2+ times per day” for foods and a maximum of “6+ times per day” for beverages. We calculated servings per day for each item as the product of the reported frequency and serving size (small weighted by 0.5, medium by 1.0, and large by 1.50).

### Dietary patterns

We created two types of dietary pattern scores: 1) a set of four PCA-derived dietary patterns each composed of 47 food groups (details previously described [17]) and 2) an a priori–defined low-risk food pattern score based on the intake of 10 food groups previously associated with risk of type 2 diabetes.

### Empirically derived dietary patterns

The four PCA dietary patterns were named according to the food groups loading highest on the respective dietary pattern (17): “fats and processed meats;” “vegetables and fish;” “beans, tomatoes, and refined grains;” and “whole grains and fruit” (supplementary Table 1, available in an online appendix at <http://dx.doi.org/10.2337/dc08-0760>). We calculated a dietary pattern score for each participant for each dietary pattern as food group servings/day  $\times$  food group factor loading, summed across all 47 food groups. Thus, a score for a particular dietary pattern represents a weighted sum of all 47 food groups, not just those with highest factor loads. A higher score indicated greater conformity with the pattern being calculated.

### An a priori, low-risk food pattern

A low-risk food pattern score was calculated as the sum intake (each in servings per day) of whole grains, nuts/seeds, vegetables, low-fat dairy, and coffee (each weighted by +1.0) and high-fat dairy, red meat, processed meat, white potatoes,

and regular soda (each weighted  $-1.0$ ). Food groups were standardized to a mean of 0 and SD of 1 before weighting and summation. Standardization makes it possible to combine food groups with different quantitative intake so that the resulting summary score is not driven by those food groups eaten most commonly. A higher score indicated a healthier diet in terms of type 2 diabetes risk. Pearson correlations between the low-risk food pattern and the PCA dietary patterns were  $-0.52$ ,  $0.05$ ,  $-0.17$ , and  $0.63$  for correlations with factors 1–4, respectively ( $P < 0.001$  for all).

### Assessment of other relevant variables

At the baseline examination, a combination of self-administered and interviewer-administered questionnaires were used to collect information on demographics, education, medication use, smoking history, and physical activity. Total and HDL cholesterol, triglyceride, insulin, and glucose concentrations were measured directly with reagents from Roche Diagnostics, Indianapolis, Indiana (analyzed at the Collaborative Studies Clinical Laboratory, Fairview-University Medical Center; Minneapolis, MN), and LDL cholesterol was calculated with the Friedewald equation for specimens having a triglyceride value  $< 400$  mg/dl. Resting seated blood pressure was measured three times using a Dinamap model Pro 100 automated oscillometer (Critikon). The average of the last two measures was used in analyses. BMI (weight in kilograms divided by the square of height in meters) was calculated from weight measured to the nearest 0.45 kilogram and height measured to the nearest 0.1 centimeter. Waist circumference was measured at the umbilicus to the nearest centimeter. Three measurements were taken, and the average of the last two measurements was used in analyses.

### Statistical analyses

We conducted all statistical analyses with SAS version 9.1, SAS Institute, Cary, North Carolina. We calculated unadjusted participant characteristics and energy-adjusted nutrient and food group intakes for those participants who remained free of type 2 diabetes over follow-up compared with those who developed type 2 diabetes during follow-up.

For all dietary patterns and the low-risk food pattern score, we calculated hazard ratios (HRs) for type 2 diabetes across

score quintiles, with quintile 1 as the reference using Cox proportional hazards regression. We calculated *P* for trend by modeling the pattern score as a continuous variable. We also calculated risk of type 2 diabetes per 1 SD of score for each pattern (1.0 for PCA-derived dietary patterns and 3.8 for the low-risk food pattern).

We used three multivariable models in our analyses. Model 1 adjusted for energy intake (kilocalories per day), age (years), sex, race/ethnicity (white, black, Hispanic, or Chinese), and study center (California, Minnesota, Maryland, New York, Illinois, or North Carolina). Model 2 included the variables in model 1 plus education (less than a high school degree, a high school degree, and more than a high school degree), active leisure activities (walking, sport, and conditioning activities in MET minutes per week), inactive leisure activities (television, reading, and light sitting activities in MET minutes per week), smoking status, smoking pack-years, and nutritional supplement use (weekly users of vitamin, mineral, or other nutritional supplements vs. nonusers). Finally, because the association between dietary patterns and type 2 diabetes may be mediated by baseline differences in adiposity or changes in adiposity during follow-up, we estimated HRs with further adjustment for baseline waist circumference (model 3) and, also, change in body weight or change in waist circumference (most recent measurement minus baseline measurement).

To better understand the contribution of individual component food groups, we also estimated HRs for type 2 diabetes according to intake of each food group included in the a priori low-risk type 2 diabetes food pattern score. We tested interactions between food patterns and race, sex, waist circumference, and BMI by adding cross-product terms to model 2 with the pattern score modeled as a continuous variable. We also explored the role of race/ethnicity by conducting a PCA within each race/ethnic group and calculating HRs for type 2 diabetes.

## RESULTS

### Participant demographic, lifestyle, clinical, and dietary characteristics

After ~5 years of follow-up, 413 participants (8.2%) developed type 2 diabetes. Incidence was highest in Hispanic individuals (11.3%), followed by black (9.5%), Chinese (7.7%), and white (6.3%) individuals. Demographic, lifestyle, and select clinical

**Table 1—Demographic, lifestyle, clinical, and dietary characteristics of 5,011 participants in MESA stratified by type 2 diabetes status**

	Free of type 2 diabetes through follow-up	Type 2 diabetes diagnosed at one of three follow-up exams	<i>P</i>
<i>n</i>	4,598	413	
Age (years)	61.7 ± 0.2	61.5 ± 0.5	0.69
Sex (% male)	47.3	49.4	0.41
Race/ethnicity (%)			<0.001
White	44.4	33.2	
Black	19.6	27.6	
Hispanic	23.7	27.9	
Chinese	12.3	11.4	
High school degree (%)	85.0	79.9	0.006
Active leisure activity (MET min/week)	2,522 ± 45	2,345 ± 151	0.26
Inactive leisure activity (MET min/week)	1,664 ± 16	1,815 ± 55	0.008
Current smokers (%)	14.3	13.8	0.79
Weekly supplement use (%)	58.3	56.4	0.54
BMI (kg/m <sup>2</sup> )	27.6 ± 0.1	31.2 ± 0.3	<0.001
Waist circumference (cm)	96.1 ± 0.2	106 ± 0.7	<0.001
Systolic blood pressure (mmHg)	125 ± 0.3	130 ± 1	<0.001
LDL cholesterol	118 ± 0.5	117 ± 2	0.52
HDL cholesterol	52.1 ± 0.2	46.6 ± 0.7	<0.001
Triglycerides (mg/dl)	125 ± 1	149 ± 4	<0.001
Fasting insulin (mg/dl)	6.1 ± 0.1	9.7 ± 0.2	<0.001
Fasting glucose (mg/dl)	88.3 ± 0.1	103 ± 0.5	<0.001
Total energy intake (kcal)	1,676 ± 11	1,802 ± 38	0.001
Protein (% of total kcal)	16 ± 0.1	16 ± 0.2	0.49
Total fat (% of total kcal)	34 ± 0.1	35 ± 0.3	0.01
Saturated fat (% of total kcal)	11 ± 0.04	11 ± 0.1	0.44
Monounsaturated fat (% of total kcal)	12 ± 0.04	13 ± 0.1	0.01
Polyunsaturated fat (% of total kcal)	7.7 ± 0.03	8.0 ± 0.1	0.07
Carbohydrate (% of total kcal)	50 ± 0.1	49 ± 0.4	0.06
Fiber (g/day)	18 ± 0.1	18 ± 0.3	0.36
Whole grains (servings/day)	0.60 ± 0.01	0.54 ± 0.03	0.02
Vegetables (servings/day)*	2.3 ± 0.02	2.2 ± 0.07	0.25
Nuts/seeds (servings/day)	0.29 ± 0.01	0.25 ± 0.02	0.04
Low-fat dairy (servings/day)	0.79 ± 0.02	0.68 ± 0.05	0.07
Coffee (servings/day)	1.2 ± 0.02	1.1 ± 0.1	0.17
Red meat (servings/day)	0.38 ± 0.004	0.39 ± 0.01	0.22
Processed meat (servings/day)	0.17 ± 0.004	0.18 ± 0.01	0.34
High-fat dairy (servings/day)†	0.50 ± 0.01	0.57 ± 0.03	0.05
White potatoes (servings/day)‡	0.20 ± 0.003	0.20 ± 0.01	0.84
Regular soda (servings/day)§	0.40 ± 0.01	0.45 ± 0.04	0.22

Data are means ± SE. Except for total energy intake, dietary variables are adjusted for energy intake (kcal/day). *P* for difference by *F* test from linear regression (continuous variables) or  $\chi^2$  (categorical variables). \*Vegetables include green leafy vegetables, cruciferous vegetables, dark-yellow vegetables, other vegetables, and tomatoes (food groups used in the principal components analyses were combined). †High-fat dairy combines whole milk and high-fat cheese/cream sauces (food groups used in the principal components analyses were combined). ‡White potatoes include white potatoes (baked, boiled, or mashed) and fried potatoes (food groups used in the principal components analyses were combined). §Regular soda includes nondiet soda, sweetened mineral water, and nonalcoholic beer (participant response to single question listing these three beverages).

characteristics and energy-adjusted dietary intake of participants who developed type 2 diabetes over follow-up are compared with those participants remaining free of disease in Table 1.

### Empirically derived dietary pattern scores and risk of type 2 diabetes

Scores on the “beans, tomatoes, and refined grains” dietary pattern were associated with greater risk of type 2 diabetes ( $P_{\text{trend}} =$

Table 2—Risk of type 2 diabetes according to two dietary patterns derived by principal components analysis and one a priori–defined low-risk food pattern in 5,011 men and women from MESA

Incident diabetes per person-years of follow-up	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	$P_{\text{trend}}$	HR (95% CI) per 1-score SD*
“Beans, tomatoes, and refined grains”	78/4,311	68/3,394	75/4,433	83/4,446	109/4,344		
Model 1†	1.00	0.92 (0.66–1.28)	1.02 (0.74–1.41)	1.07 (0.77–1.48)	1.25 (0.87–1.81)	0.004	1.18 (1.06–1.32)
Model 2‡	1.00	0.92 (0.66–1.28)	1.02 (0.74–1.41)	1.06 (0.76–1.47)	1.23 (0.85–1.78)	0.004	1.18 (1.06–1.32)
Model 3§	1.00	0.99 (0.71–1.38)	1.09 (0.78–1.51)	1.09 (0.78–1.52)	1.28 (0.88–1.84)	0.003	1.19 (1.06–1.33)
“Whole grains and fruit”	108/4,328	82/4,341	76/4,435	76/4,410	71/4,414		
Model 1†	1.00	0.76 (0.56–1.02)	0.71 (0.52–0.97)	0.71 (0.52–0.98)	0.63 (0.45–0.89)	0.002	0.84 (0.75–0.94)
Model 2‡	1.00	0.76 (0.56–1.02)	0.73 (0.53–1.09)	0.72 (0.52–0.99)	0.66 (0.47–0.93)	0.005	0.85 (0.76–0.95)
Model 3§	1.00	0.74 (0.54–1.09)	0.76 (0.55–1.03)	0.77 (0.56–1.07)	0.73 (0.52–1.04)	0.05	0.89 (0.79–1.00)
A priori, low-risk food pattern	106/4,263	84/4,401	101/4,336	68/4,451	54/4,477		
Model 1†	1.00	0.88 (0.65–1.20)	1.16 (0.86–1.57)	0.79 (0.57–1.10)	0.59 (0.42–0.84)	0.02	0.88 (0.80–0.98)
Model 2‡	1.00	0.93 (0.69–1.26)	1.22 (0.90–1.66)	0.83 (0.60–1.16)	0.62 (0.44–0.88)	0.04	0.87 (0.81–0.99)
Model 3§	1.00	1.00 (0.73–1.33)	1.31 (0.94–1.74)	0.92 (0.66–1.29)	0.72 (0.51–1.03)	0.18	0.93 (0.84–1.03)

Data are *n* or HR (95% CI).  $P_{\text{trend}}$  calculated with dietary pattern modeled as a continuous variable (score units). \*For incident type 2 diabetes per 1-SD change in dietary pattern score. For the “beans, tomatoes, and refined grains” and “whole grains and fruit” dietary patterns, SD was 1.00. For the a priori, low-risk food pattern, SD was 3.8. †For incident type 2 diabetes with quintile 1 as the reference category adjusted for energy intake (kcal/day), study center (California, Minnesota, Maryland, New York, Illinois, or North Carolina), age (years), sex, and race/ethnicity (white, black, Chinese, or Hispanic). ‡For incident type 2 diabetes with quintile 1 as the reference category adjusted for the above plus education (less than a high school degree, a high school degree, and more than a high school degree), active leisure-time physical activity (MET minutes per week), inactive leisure-time physical activity (MET minutes per week), current smoking status (yes or no), smoking pack-years, and current weekly supplement use (yes or no). §For incident type 2 diabetes with quintile 1 as the reference category adjusted for the above plus waist circumference (cm). ||The a priori, low type 2 diabetes–risk food pattern is the sum of servings per day from 10 food groups (standardized to mean 0.00, SD 1.00). Positive (+1) weights were assigned to whole grains, fruit, vegetables, nuts/seeds, low-fat dairy, and coffee. Negative (–1) weights were assigned to red meat, processed meat, high-fat dairy, and regular soda.

0.004; model 2), whereas scores on the “whole grains and fruit” dietary pattern were associated with low risk of type 2 diabetes ( $P_{\text{trend}} = 0.005$ ; model 2) (Table 2). For a 1-SD increase in score on the “beans, tomatoes, and refined grains” dietary pattern, the risk of type 2 diabetes was 18% greater (model 2). For a 1-SD increase in score on the “whole grains and fruit” dietary pattern, the risk of type 2 diabetes was 15% lower (model 2). Estimates were slightly attenuated after additional adjustment for waist circumference (model 3, Table 2). However, neither adjustment for change in body weight nor change in waist circumference over follow-up materially impacted risk estimates (data not shown). Other PCA-derived dietary patterns were not significantly associated with type 2 diabetes risk (data not shown).

**An a priori–defined low-diabetes-risk food pattern score and risk of type 2 diabetes**

Higher scores on the a priori low-risk food pattern were associated with lower risk of type 2 diabetes (Table 2). Participants in the 5th quintile had a 38% lower risk of type 2 diabetes compared with those in the lowest quintile low-risk food pattern (HR 0.62 [95% CI 0.44–0.88];

model 2). Each 1-SD increase in score corresponded to a 13% lower risk of type 2 diabetes (0.87 [0.81–0.99];  $P_{\text{trend}} = 0.04$ ; model 2). Baseline differences in waist circumference partly explained the association between type 2 diabetes and the low-risk food pattern ( $P_{\text{trend}}$  after adjustment = 0.18; model 3), although adjustment for change in body weight did not impact results ( $P_{\text{trend}}$  after adjustment = 0.03; data not shown).

No individual food group component of the low-risk food pattern was significantly associated with risk of type 2 diabetes. Although individual food groups were not significantly associated with type 2 diabetes risk, estimates were generally in the hypothesized direction, i.e., the five food groups anticipated to lower risk showed HRs <1.00, and the four food groups anticipated to increase risk showed HRs >1.00 (data not shown).

**Interactions**

There were no significant interactions between any of the dietary patterns studied and sex, BMI, or waist circumference ( $P$  for interaction >0.2 for all tested interactions; data not shown). When stratified by race/ethnicity, HR (95% CI) per 1-SD on the “beans, tomatoes, and refined grains”

pattern were 1.25 (0.95–1.63), 0.95 (0.47–1.93), 1.04 (0.77–1.40), and 1.17 (1.00–1.37) for white, Chinese, black, and Hispanic individuals, respectively. For a 1-SD difference in “whole grains and fruit” dietary pattern score, these values were 0.83 (0.67–1.02), 0.57 (0.36–0.91), 0.86 (0.69–1.06), and 0.97 (0.80–1.19), respectively, and for a 1-SD difference in low-risk food pattern score, these values were 0.86 (0.72–1.04), 0.56 (0.35–0.90), 0.88 (0.72–1.07), and 1.03 (0.86–1.22), respectively. CIs of these stratum-specific estimates were large, and the formal test for interaction between race/ethnicity and each of these three dietary patterns was not statistically significant ( $P > 0.16$ ).

**Influence of race/ethnicity**

To further explore the role of race/ethnicity, we applied a PCA separately in each race/ethnic group. A two-pattern solution consisting of a conventionally healthy dietary pattern and a conventionally unhealthy dietary pattern emerged in each ethnic group. Nine food groups were shared across the four race/ethnic groups’ healthy dietary patterns, and ten food groups were shared across the four race/ethnic groups’ unhealthy dietary patterns



(see online appendix). However, there were not significant associations between these empirical, race/ethnic-specific dietary patterns and risk of type 2 diabetes (data not shown), likely due to limited power within each race/ethnic strata.

**CONCLUSIONS**—There were three main findings of our study. First, high scores on the “whole grains and fruit” PCA dietary pattern were associated with lower risk of type 2 diabetes, whereas high scores on the “beans, tomatoes, and refined grains” PCA dietary pattern were associated with greater risk of type 2 diabetes. Second, a dietary score based on the intake of 10 food groups previously shown to be independently related to type 2 diabetes in other populations was associated with lower type 2 diabetes risk. Third, these findings were not modified by race/ethnicity, and dietary pattern analyses conducted within each race/ethnic group suggested many similarities in overall dietary intake among race/ethnic groups.

Consistent with our findings are those of previous observational studies showing an inverse association between risk of type 2 diabetes and intake of whole grains (1), nuts/seeds (2), and green leafy vegetables (6), all of which were important components of our “whole grains and fruit” dietary pattern. Also similar to our findings are those from previous cohort studies, in which similarly composed dietary patterns were inversely associated with type 2 diabetes (19,20) or other measures of metabolic dysfunction with risk factors that closely mirror those for type 2 diabetes (21–23). In contrast, the “beans, tomatoes, and refined grains” dietary pattern was positively associated with type 2 diabetes risk. Although both beans and tomatoes have positive nutrient attributes, these foods are correlated with less-favorable food groups in MESA, such as refined grains, high-fat dairy foods, and red-meat (other key contributors to high scores on the “beans, tomatoes, and refined grains” pattern). A similar observation was made in another large cohort study (19). It is possible that the beneficial components of beans and tomatoes (e.g., fiber, phytoestrogens, vitamin C, and lycopene) are countered by the less than beneficial nutritional components of high-fat dairy and red meat (e.g., saturated fat and iron) or are negatively impacted by food groups such as refined grains due to nutrient displacement (e.g., fiber) (17).

Although empirical dietary pattern analysis has successfully identified dietary patterns associated with disease risk, patterns that maximally explain variation in intake (as is the goal of PCA) may not maximally explain variation in the outcome of interest (24). To circumvent this, we chose an a priori approach and calculated a food pattern score based on the intake of 10 individual food groups previously associated with type 2 diabetes risk (1–7,9–12) that we found to be significantly inversely associated with risk of type 2 diabetes in MESA. Interestingly, although the type 2 diabetes risk estimates for each of the component food groups were in the hypothesized direction, no individual food group was independently associated with type 2 diabetes risk. This observation supports the hypothesis that the effects of single foods or nutrients may be too small to detect individually, but their cumulative effects may be sufficiently large to detect and be deemed statistically significant.

Few other studies have been able to investigate the contribution of racial/ethnic diversity to dietary pattern analysis in cohorts such as MESA, where multiple race/ethnic groups were surveyed with uniform assessment tools (21,25,26). In additional studies, dietary patterns were derived with data from all represented race/ethnic groups, likely due to limited power within race/ethnic strata—a limitation we share here. We did examine the race/ethnic-specific PCA dietary patterns and their associations with incident diabetes; our data showed more similarities than differences across race/ethnic groups.

Limitations of our analysis should also be noted. First, imperfections in dietary assessment are generally thought to be randomly distributed among categories of the outcome (nondifferential); nevertheless, systematic imperfections that could bias risk estimates toward or away from the null value are possible. Second, although we tried various methods to characterize dietary patterns, we cannot conclude that other dietary patterns not represented by the posteriori or a priori patterns we presented here are importantly related to type 2 diabetes. Third, as mentioned, our race/ethnic-stratified analyses were limited by small sample sizes, especially in the Chinese participants. Thus, it is possible that estimates that were not statistically significant could have been significant with a larger sample size. Last, risk estimates may be con-

founded by demographic and nondietary lifestyle factors, which may have been inadequately captured within the covariates we included in our multivariable models.

In conclusion, we found that an empirically derived dietary pattern characterized by high intake of whole grains, fruit, nuts/seeds, and green leafy vegetables was inversely associated with risk of incident type 2 diabetes in a large, prospective study including white, black, Hispanic, and Chinese men and women. Similarly, an a priori composite food pattern score capturing high intake of whole grains, nuts/seeds, vegetables, low-fat dairy, and coffee and low intake of red meat, processed meat, white potatoes, high-fat dairy, and soda was associated with lower risk of incident type 2 diabetes. These findings underscore the importance of the collective influence of multiple food groups in the development of type 2 diabetes and suggest that individuals wishing to decrease the risk of type 2 diabetes, regardless of their race/ethnicity, focus on increasing intake of several key food groups, including whole grains, low-fat dairy, fruits and vegetables, and nuts/seeds and on decreasing their intake of red/processed meats, high-fat foods, and sugared soda beverages.

**Acknowledgments**—This research was supported by contracts N01-HC-95159 through N01-HC-95165 and N01-HC-95169 from the National Heart, Lung, and Blood Institute.

The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions (a full list of participating Multi-Ethnic Study of Atherosclerosis investigators and institutions can be found at <http://www.mesa-nhlbi.org>).

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