Dietary Patterns and Risk for Type 2 Diabetes Mellitus in U.S. Men

Rob M. van Dam, MSc; Eric B. Rimm, ScD; Walter C. Willett, MD; Meir J. Stampfer, MD; and Frank B. Hu, MD

Background: The role of diet in the development of type 2 diabetes mellitus remains unsettled.

Objective: To examine the association between major dietary patterns and risk for type 2 diabetes mellitus.

Design: Prospective cohort study.

Setting: United States.

Participants: 42 504 male health professionals, 40 to 75 years of age, without diagnosed diabetes, cardiovascular disease, or cancer at baseline.

Measurements: Using factor analysis based on data from food-frequency questionnaires, we identified and validated two major dietary patterns that we labeled "prudent" (characterized by higher consumption of vegetables, fruit, fish, poultry and whole grains) and "western" (characterized by higher consumption of red meat, processed meat, French fries, high-fat dairy products, refined grains, and sweets and desserts). Relative risks and 95% CIs were adjusted for potential confounders, including body mass index (BMI), physical activity, and cigarette smoking.

Results: During 12 years of follow-up (466 508 person-years), we documented 1321 cases of type 2 diabetes. The prudent dietary pattern score was associated with a modestly lower risk for type 2 diabetes (relative risk for extreme quintiles, 0.84 [CI, 0.70 to 1.00]). In contrast, the western dietary pattern score was associated with an increased risk for type 2 diabetes (relative risk, 1.59 [CI, 1.32 to 1.93]; $P < 0.001$ for trend). A high score for the western dietary pattern combined with low physical activity (relative risk comparing extreme quintiles of dietary pattern score and physical activity, 1.96 [CI, 1.35 to 2.84]) or obesity (relative risk for BMI $\geq$ 30 kg/m^2 vs. <25 kg/m^2, 11.2 [CI, 8.07 to 15.6]) was associated with a particularly high risk for type 2 diabetes.

Conclusion: Our findings suggest that a western dietary pattern is associated with a substantially increased risk for type 2 diabetes in men.

Ann Intern Med. 2002;136:201-209. www.annals.org
For author affiliations, current addresses, and contributions, see end of text.

The prevalence of type 2 diabetes mellitus is rapidly increasing in the United States (1, 2) and worldwide (3). Ecologic studies (4), migration studies (4, 5), and analyses of secular trends (4, 6) suggest that adoption of a western diet may be associated with increased incidence of type 2 diabetes. However, these studies could not separate the effects of diet from those of other risk factors, such as obesity and physical activity.

Studies of the relation between diet and incidence of type 2 diabetes on an individual level have predominantly focused on intake of macronutrients and fiber (7). However, these dietary factors alone probably explain only part of the effect of diet on glucose metabolism. Other nutrients, additives, contaminants, and unknown compounds and the physical properties of foods and interactions between nutrients (for example, with respect to bioavailability) may play a role in the deterioration of glucose metabolism. Hence, the examination of effects of food consumption may be an important complementary approach. Recently, the analysis of patterns of food consumption by using factor analysis has emerged as a useful tool for elucidating relationships between diet and health (8–11). The overall dietary pattern may affect health more than individual foods and nutrients do. Furthermore, dietary patterns reflect the way foods are consumed in reality, which may provide insight into possibilities for dietary changes and may facilitate the translation of findings to public health recommendations. Recent dietary intervention studies have indicated that interventions focused on dietary patterns can decrease blood pressure (12) and reduce cardiovascular complications (13).

Data on dietary patterns and risk for type 2 diabetes are sparse. Although two cross-sectional studies of dietary patterns and diabetes have been conducted (11, 14), these studies incompletely controlled for confounding by other lifestyle factors. Therefore, we prospectively examined the association between major dietary patterns and risk for type 2 diabetes in a large prospective study of U.S. professional men with extensive, repeatedly obtained information on diet and other lifestyle factors.

METHODS

Participants

The Health Professionals Follow-up Study started in 1986, when 51 529 male health professionals (dentists, veterinarians, pharmacists, optometrists, osteopathic physicians, and podiatrists) completed a detailed mailed questionnaire on medical history, diet, and other potential risk factors for major diseases. The participants lived in all 50 U.S. states, were predominantly white, and were 40 to 75 years of age in 1986. We excluded from the current analysis 1595 men who did not satisfy the a priori criteria of reporting daily energy intake of 3.3 to 17.6 MJ (800 to 4200 kcal/24 h) and leaving fewer than 70 responses blank of the 131 food items on the diet questionnaire (<5% had >11 blanks). We also excluded men who reported having diabetes, cardiovascular disease (myocardial infarction, angina pectoris, coronary artery surgery, or stroke), or cancer (except nonmelanoma skin cancer) at baseline because having these diagnoses may affect diet or reporting of diet. After exclusions, the study sample comprised 42 504 men, who were followed for incidence of type 2 diabetes and other diseases for 12 years (1986 to 1998). Every 2 years, the participants received questionnaires by mail that asked for updated information on exposures and newly diagnosed diseases. The follow-up rate for potential person-years was about 97% for nonfatal events. We identified deaths by reports from family members, coworkers, or postal authorities or through systematic searches of the National Death Index.

Assessment of Dietary Patterns

To assess dietary intake, we used a 131-item semiquantitative food-frequency questionnaire in 1986, 1990, and 1994. The questionnaire specified for each food a commonly used unit or portion size, and the participants were asked to indicate how often, on average, they had consumed a given amount of the specified food during the past year. Each such item provided nine possible responses, ranging from "never or less than once a month" to "6 or more times per day." To reduce within-respondent variation and to best represent long-term dietary habits, we averaged the dietary intakes from all available dietary questionnaires up to the start of each follow-up interval (the average of the 1986 and 1990 dietary intakes was related to incidence of type 2 diabetes between 1990 and 1994) (15).

To identify dietary patterns, we applied factor analysis to data from the food-frequency questionnaire. First, we grouped the food items on the questionnaire into 37 predefined food groups. We combined food items that were similar in nutrient profile and culinary use (for example, spinach, iceberg or head lettuce, and romaine or leaf lettuce were combined into "green leafy vegetables"). We classified a food item individually if its composition differed substantially from that of other foods (for example, eggs or pizza) or if we suspected that it represents a particular dietary habit (for example, wine or French fries).

Second, we applied the principal components program for factor analysis of SAS software (16) with varimax rotation to the 37 food groups. Factor analysis aggregates correlated variables. The obtained factors are linear combinations of the included variables, explaining as much of the variation in the original variables as possible. Our analysis retained two factors based on the eigenvalue of the factors, the Scree test, and the interpretability of the derived factors, and we labeled these diets as the "prudent" pattern and the "western" pattern (9).

Third, for each participant, we calculated a prudent pattern score and a western pattern score by summing the standardized intake of foods, weighted by the factor loadings of the foods (17). We used these scores to rank participants according to the degree to which they conformed to each dietary pattern. We divided dietary pattern scores into quintiles; thus, on this basis of his scores, each participant was grouped in a prudent-pattern quintile and a western-pattern quintile.

To test the validity and reproducibility of the assessment of the dietary pattern scores by the food-frequency questionnaire, we examined a subgroup of 127 participants (9). The Pearson correlation coefficient (corrected for week-to-week variation in the diet records) for a comparison between the responses to the food-frequency questionnaire and the diet records was 0.52 for the prudent dietary pattern and 0.74 for the western dietary pattern.

Assessment of Nondietary Exposures

The 1986 questionnaire and each biennial follow-up questionnaire assessed weight, smoking status, and physical activity. Participants provided information on age, diagnosis of hypertension and hypercholesterolemia, and height in 1986 and on family history of diabetes in 1987. The criterion for family history of type 2 diabetes was having at least one first-degree relative with a diagnosis of diabetes after 30 years of age. For each participant, we determined a physical activity level, measured in weekly metabolic equivalent hours, on the basis of reported time spent on various activities, which we weighted according to intensity level (18). The validity of self-reported weight (19) and physical activity (18) in this cohort has been reported previously.

Ascertainment of Type 2 Diabetes Mellitus

We mailed a supplementary questionnaire on symptoms, diagnostic tests, and medication use to participants who indicated on any biennial follow-up questionnaire that he had received a diagnosis of diabetes mellitus. Confirmation of diabetes required at least one of the following: 1) an elevated plasma glucose level (fasting plasma glucose level $\geq$ 7.8 mmol/L [$\geq$140 mg/dL]; random plasma glucose level $\geq$ 11.1 mmol/L [$\geq$200 mg/dL]; or plasma glucose level after 2 hours or more during an oral glucose tolerance test $\geq$ 11.1 mmol/L [$\geq$200 mg/dL]) plus at least one classic symptom (excessive thirst, polyuria, weight loss, or hunger); 2) at least two elevated plasma glucose levels measured on different occasions; or 3) treatment with insulin or oral hypoglycemic medication. We excluded men who reported having type 1 diabetes on the supplementary questionnaire. These criteria for diabetes are consistent with those from the World Health Organization in 1985 (20). We did not use the current diabetes classification of the American Diabetes Association (21) because among the men in our study, most cases of diabetes were diagnosed before these criteria were published. The validity of our assessment of type 2 diabetes was verified with medical records in a subsample of 71 participants. A physician blinded to the information on the supplementary questionnaire reviewed the records according to the diagnostic criteria. Of the 71 participants classified as having type 2 diabetes, 12 had incomplete medical records—for example, absent laboratory data (n = 2) or only one set of laboratory data (n = 9). We confirmed the classification of type 2 diabetes in 57 (97%) of the other 59 other men in the subsample. One participant denied having diabetes, and another participant lacked evidence of diabetes in his submitted records.

Statistical Analysis

We used pooled logistic regression analyses with 2-year intervals to estimate the adjusted relative risk for each quintile compared with the lowest quintile of intake. With short time intervals and low rates of events, this approach gives results very similar to those of Cox proportional hazards analyses (22). Participants who died or received a diagnosis of diabetes during a 2-year cycle were censored at the end of that 2-year period and were not entered in any subsequent 2-year cycle. We used the cumulative average of available dietary assessments up to the start of each 2-year follow-up interval (15). We stopped updating a participant's dietary data at the beginning of the time interval during which he received a diagnosis of hypertension, hypercholesterolemia, cancer (except nonmelanoma skin cancer), or cardiovascular diseases, because subsequent changes in personal dietary habits could confound the relationship between diet and diabetes (15). Analyses using only dietary information from 1986 and analyses relating diet in 1986 to incidence of type 2 diabetes between 1992 and 1998 yielded essentially the same results.

To reduce residual confounding, the same cumulative updating approach was used for physical activity and alcohol intake by using the information of all available assessments. During follow-up, we also updated participant data on body mass index (BMI) and smoking status using the most recent data for each 2-year interval. We included categorical variables in the models as binary indicator variables. To further reduce extraneous variation or confounding due to variation in body size, physical activity, or metabolic efficiency or caused by over- or underreporting, we adjusted dietary pattern scores for total energy intake using the residual method (23). We tested for linear trends across categories of dietary intake by assigning each participant the median value for the category and modeling this value as a continuous variable. All P values reported are two sided.

Role of the Funding Sources

The funding sources had no role in the collection, analysis, or interpretation of the data or in the decision to submit the paper for publication.

RESULTS

Table 1 shows correlations between food intake and dietary pattern factors in 1986, 1990, and 1994. The correlation between a food and a factor increased as the contribution of that food to the corresponding dietary pattern score increased. The factor that we labeled the prudent dietary pattern was characterized by high consumption of vegetables, legumes, fruit, whole grains,

Table 1. **Pearson Correlation Coefficients for the Relationship between Food Intake and Factors Representing Dietary Patterns: Health Professionals Follow-up Study—1986, 1990, and 1994***

Foods	Correlation Coefficient					
	1986		1990		1994	
	Prudent Pattern	Western Pattern	Prudent Pattern	Western Pattern	Prudent Pattern	Western Pattern
Vegetables†	0.75	–	0.70	–	0.71	–
Dark-yellow vegetables	0.64	–	0.66	–	0.65	–
Cruciferous vegetables	0.63	–	0.66	–	0.63	–
Green leafy vegetables	0.63	–	0.63	–	0.63	–
Legumes	0.63	–	0.63	–	0.62	–
Fruit	0.60	–	0.62	–	0.58	–
Tomatoes	0.54	–	0.52	–	0.51	–
Fish	0.51	–	0.48	–	0.46	–
Whole grains	0.38	–	0.37	–	0.38	–
Poultry	0.36	–	0.35	–	0.32	–
Salad dressing‡	0.32	–	0.33	–	0.23	0.18
Fruit juice	0.32	–	0.32	–	0.30	–
Low-fat dairy products	0.16	–	0.17	–	0.19	–
Wine	–	–	–	–	–	–
Low-sugar drinks	–	–	–	–	–	–
Red meat	–	0.64	–	0.66	–	0.66
Processed meat	–	0.61	–	0.63	–	0.60
Refined grains	–	0.48	–	0.42	0.18	0.38
French fries	–	0.48	–	0.49	–	0.48
High-fat dairy products	–	0.47	–	0.52	–	0.50
Sweets and desserts	–	0.42	–	0.44	–	0.50
Eggs	–	0.42	–	0.48	–	0.46
Condiments	0.16	0.41	0.16	0.28	0.19	0.41
High-sugar drinks	–	0.36	–	0.35	–	0.32
Snacks§	–	0.36	–	0.33	–	0.33
Mayonnaise	–	0.34	–	0.36	–	0.35
Butter	–	0.34	–	0.36	–	0.37
Pizza	–	0.29	–	0.29	–	0.30
Margarine	–	0.29	–	0.30	–	0.34
Potatoes	0.26	0.29	0.31	0.26	0.33	0.29
Chowder or cream soup	–	0.22	–	0.24	–	0.31
Nuts	0.18	0.22	0.17	0.24	0.17	0.28
Coffee	–	0.27	–	0.23	–	0.23
Beer	–	0.16	–	–	–	0.15
Tea	–	–	–	–	–	–
Liquor	–	–	–	–	–	–
Organ meats	–	–	–	–	–	–

* With the orthogonal rotation used, correlations are identical to factor loading matrix; to simplify data presentation, loadings with an absolute value less than 0.15 are not shown. We used the same food groups as in previous analyses (10) except for "garlic" and "other soups," which were not included in the present study because intake of these foods was not assessed in 1990 or 1994, and "cold breakfast cereal," which was included in the whole-grains group (if ≥25% of weight consisted of whole grains or bran [24]) or the refined-grains group.
† Vegetables other than dark-yellow, cruciferous, or leafy-green vegetables.
‡ Salad dressing other than mayonnaise.
§ Potato or corn chips, crackers, popcorn.

fish, and poultry; the western dietary pattern was characterized by high consumption of red meat, processed meat, refined grains, French fries, high-fat dairy products, sweets and desserts, high-sugar drinks, and eggs.

The correlations between foods and factors representing dietary patterns were similar over time. Furthermore, the Pearson correlation between the prudent pattern score was 0.59 between 1986 and 1990, 0.60 between 1990 and 1994, and 0.55 between 1986 and 1994; for the western pattern scores, the Pearson correlation was 0.69 between 1986 and 1990, 0.72 between 1990 and 1994, and 0.64 between 1986 and 1994.

Table 2 shows the baseline characteristics of the study sample according to quintile of dietary pattern scores. On average, men with higher prudent-pattern scores were older, more physically active, less likely to be smokers, and more likely to have hypercholesterolemia. The cross-sectional association with hypercholesterolemia probably reflects changes in diet after diagnosis. Higher prudent-pattern scores were associated with higher intakes of protein, cereal fiber, and magnesium and lower intakes of alcohol, saturated fat, and total fat. Men with higher western-pattern scores generally were younger, had a higher BMI and a lower level of physical activity, were less likely to have hypercholesterolemia, and were more likely to be smokers and of Northern European ancestry. Furthermore, higher western-pattern scores were associated with higher intakes of fat and alcohol and lower intakes of cereal fiber and magnesium. **Table 2** also shows the mean intake of foods by quintiles of the dietary pattern scores. Men in the lowest quintile of the western pattern score, for example, had low mean intakes of unprocessed red meat (<3 servings/wk), processed meat (<1 serving/wk), and high-fat dairy products (<5 servings/wk) and high intakes of fish (>4 servings/wk), whole grains (>2 servings/d), vegetables (>4 servings/d), and fruits (>3 servings/d).

During 466 508 person-years of follow-up, we ascertained 1321 cases of type 2 diabetes. As shown in **Table 3**, the prudent pattern was associated with modestly reduced risk for type 2 diabetes (multivariate relative risk for highest vs. lowest quintiles, 0.84 [95% CI, 0.70 to 1.00]). In contrast, higher western-pattern scores were associated with substantially increased risk for type 2 diabetes (relative risk, 1.59 [CI, 1.32 to 1.93]; $P <$ 0.001 for trend). Adjustment for physical activity, cigarette smoking, alcohol consumption, hypercholesterolemia, hypertension, ancestry, and family history of diabetes did not substantially change the associations (**Table 3**). Results were essentially the same when the analysis was limited to symptomatic type 2 diabetes ($n = 810$).

Table 2. **Baseline Characteristics of the Study Sample, according to Quintile of Energy-Adjusted Dietary-Pattern Score: Health Professionals Follow-up Study, 1986***

Variable	Prudent Dietary Pattern			Western Dietary Pattern		
	Quintile 1	Quintile 3	Quintile 5	Quintile 1	Quintile 3	Quintile 5
Age, *y*	51.5	53.7	55.0	55.1	53.4	52.2
Ancestry, %						
Northern European	72.2	69.4	66.9	67.3	68.7	73.4
Southern European	22.3	24.3	25.7	24.5	24.1	22.1
Other	4.4	5.0	5.5	6.4	5.5	3.6
Body mass index, *kg/m²*	25.6	25.5	25.2	24.7	25.6	26.0
Physical activity, *METs/wk*†	15.1	20.5	27.9	29.2	19.4	15.1
Current smoking, %	17.3	8.3	5.2	3.2	8.0	18.4
Family history of diabetes, %	18.5	19.7	21.0	19.1	20.4	19.2
Hypertension, %	18.3	19.3	20.2	18.6	19.7	19.0
Hypercholesterolemia, %	7.7	10.2	12.8	13.6	9.9	7.4
Dietary intake						
Alcohol, *g/d*	15.0	10.8	9.7	9.6	11.7	13.2
Protein, *% of energy intake*	16.8	18.7	19.9	19.4	18.5	17.5
Total fat, *% of energy intake*	35.2	32.4	28.2	26.4	32.1	37.2
Saturated fat, *% of energy intake*	12.8	11.1	9.0	8.5	11.0	13.3
Linoleic acid, *% of energy intake*	4.9	5.2	5.1	4.7	5.1	5.5
Cereal fiber, *g/d*	4.7	6.0	6.8	7.7	5.7	4.4
Magnesium, *mg/d*	300	349	420	418	345	307
Food consumption, *servings/d*						
Vegetables	1.8	2.9	5.8	4.4	3.0	2.8
Fruit	1.1	1.7	3.0	3.2	1.6	1.2
Whole grains	0.9	1.4	2.0	2.4	1.2	0.9
Refined grains	1.6	1.1	1.1	1.0	1.1	1.8
Fish	0.2	0.4	0.6	0.6	0.4	0.3
Poultry	0.2	0.3	0.5	0.5	0.3	0.3
Red meat	0.9	0.6	0.4	0.4	0.6	0.9
Processed meat	0.6	0.3	0.2	0.1	0.3	0.7
High-fat dairy products	1.4	0.9	0.8	0.7	0.9	1.5
Sweets and desserts	1.7	1.0	0.8	0.9	1.1	1.4

* Scores are standardized to the age distribution of the total study sample (except for age); quintiles are based on dietary pattern scores adjusted for total energy intake by using the residual method; quintile 1 is the lowest quintile, quintile 3 is the medium quintile, and quintile 5 is the highest quintile. MET = metabolic equivalent hour.
† One metabolic equivalent represents the energy expended at rest.

Table 3. **Relative Risk for Type 2 Diabetes Mellitus, according to Quintile of Dietary Pattern Scores: Health Professionals Follow-up Study, 1986–1998***

Variable	Quintile					*P* Value for Trend
	1 (Lowest)	2	3	4	5 (Highest)	
Prudent pattern score						
Cases of type 2 diabetes, *n/person-years*	291/93 180	229/93 359	286/93 326	263/93 342	252/93 302	
Age- and BMI-adjusted relative risk†	1	0.76	0.94	0.88	0.84	>0.2
Multivariate relative risk (95% CI)‡	1	0.77 (0.65–0.92)	0.96 (0.81–1.14)	0.89 (0.75–1.06)	0.84 (0.70–1.00)	0.2
Western pattern score						
Cases of type 2 diabetes, *n/person-years*	177/93 341	231/93 320	274/93 330	278/93 323	361/93 195	
Age- and BMI-adjusted relative risk†	1	1.18	1.29	1.26	1.54	<0.001
Multivariate relative risk (95% CI)‡	1	1.22 (1.00–1.48)	1.34 (1.10–1.63)	1.34 (1.10–1.63)	1.59 (1.32–1.93)	<0.001

* BMI = body mass index.
† Models were adjusted for age (5-year categories), body mass index (<23.0, 23.0–23.9, 24.0–24.9, 25.0–26.9, 27.0–28.9, 29.0–30.9, 31.0–32.9, 33.0–34.9, or ≥35 kg/m^2), total energy intake (quintiles), and time period (6 periods).
‡ Also adjusted for physical activity (quintiles of metabolic equivalents), cigarette smoking (never, past, or current smoking of 1–14, 15–24, or ≥25 cigarettes/d), alcohol consumption (0, 0.1–4, 5–14, 15–29, or ≥30 g/d), ancestry (Northern European, Southern European, or other), hypercholesterolemia (yes or no), hypertension (yes or no), and family history of type 2 diabetes mellitus (yes or no).

We also examined whether consumption of specific foods with large contributions to the dietary-pattern scores was associated with the risk for type 2 diabetes. Of the foods that characterized the prudent pattern, whole grains had the strongest inverse association with the risk for type 2 diabetes (multivariate relative risk for extreme quintiles, 0.77 [CI, 0.64 to 0.93]; $P = 0.001$ for trend). Consumption of fruit, vegetables, and fish was not appreciably associated with the risk for type 2 diabetes. The foods with major contributions to the western pattern were all positively associated with the risk for type 2 diabetes. (The relative risks for extreme quintiles were: for unprocessed red meat, 1.27 [CI, 1.04 to 1.55]; for processed meat, 1.48 [CI, 1.23 to 1.77]; for refined grains, 1.37 [CI, 1.13 to 1.66]; and for high-fat dairy products, 1.17 [CI, 0.97 to 1.41].) When these western-pattern–associated foods were modeled simultaneously, consumption of processed meat and refined grains remained significantly associated with the risk for type 2 diabetes (relative risk for consumption of processed meat, 1.38 [CI, 1.14 to 1.68]; $P < 0.001$ for trend; relative risk for consumption of refined grains, 1.32 [CI, 1.09 to 1.60]; $P = 0.04$ for trend).

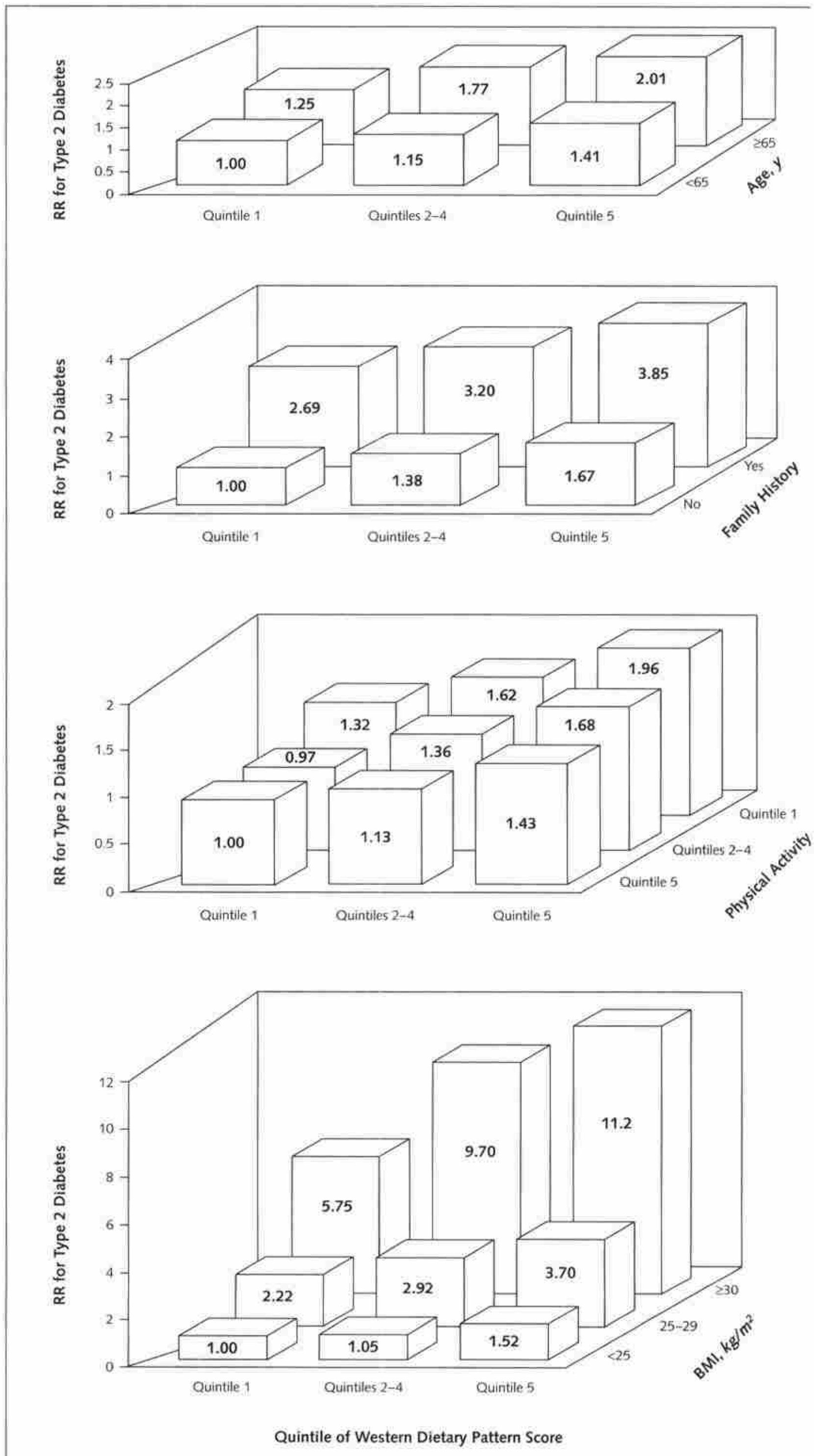
The western dietary pattern was consistently associated with type 2 diabetes in subgroups divided according to age, family history of diabetes, BMI, and physical activity (**Figure**). The combination of a high Western-pattern score with low physical activity was associated with a relative risk (for comparison of combinations of extreme quintiles) of 1.96 (CI, 1.35 to 2.84). We found that the risk for type 2 diabetes was highest for obese men (BMI ≥ 30 kg/m^2) in the highest quintile of the western dietary-pattern scores (relative risk compared with BMI < 25 kg/m^2 and lowest quintile of western pattern score, 11.2 [CI, 8.07 to 15.6]).

DISCUSSION

In this large prospective study of U.S. professional men, we identified two major dietary patterns—a *prudent pattern*, characterized by a high consumption of vegetables, fruits, fish, poultry, and whole grains and a *western pattern*, characterized by a high consumption of red meat, processed meat, high-fat dairy products, French fries, refined grains, and sweets and desserts. The prudent diet was associated with a modestly lower risk for type 2 diabetes. The western pattern, however, was associated with a substantially higher risk for type 2 diabetes. This association was stronger than that for individual foods and was independent of BMI, physical activity, family history of diabetes, and age. The combination of a western dietary pattern with a low level of physical activity or obesity was associated with a particularly high risk for type 2 diabetes.

Two cross-sectional studies examined the association between dietary patterns and type 2 diabetes. In the first, which studied Native Canadians, a "junk food" pattern was associated with a higher prevalence of impaired glucose tolerance and type 2 diabetes (14). In the second, which evaluated a British population, a dietary pattern characterized by high consumption of fruit and vegetables and low consumption of processed meat and fried foods was inversely associated with previously un-

Figure. **Relative risk (*RR*) for type 2 diabetes mellitus, according to combinations of the western dietary pattern score and other risk factors.**



Data are from the Health Professionals Follow-up Study, 1986–1998. Relative risks were multivariate adjusted for the variables listed in the text below Table 3, except the stratifying variable. BMI = body mass index.

diagnosed type 2 diabetes (11). These studies were limited by lack of control for physical activity, which tends to be associated with dietary patterns (8, 11). In one study that had results consistent with our findings, a traditional Pima Indian diet improved glucose tolerance compared with a modern western diet in a short-term dietary intervention (25). Moreover, in a previous subsample analysis of the Health Professionals Follow-up Study, the western dietary pattern was positively associated with fasting plasma insulin level and C-peptide level (26).

In some studies, saturated and total fat intake have been associated with hyperinsulinemia, hyperglycemia, or type 2 diabetes, but the results have not been consistent (7). In our study, saturated and total fat were not appreciably associated with the risk for type 2 diabetes (27). Therefore, the association between the western dietary pattern and type 2 diabetes cannot be explained by saturated or total fat intake. Compared with the contribution of these nutrients, low intake of cereal fiber and magnesium probably contributed more to the increased risk for type 2 diabetes associated with the western pattern (27–29). However, given the magnitude of the association between the western pattern and the risk for type 2 diabetes, it seems unlikely that a single dietary factor was responsible for the observed association.

The prospective design and high rate of follow-up of our study minimize the possibility of recall bias or bias due to loss of follow-up. Diet was inevitably measured with error, and this undoubtedly resulted in some misclassifications. As diet was assessed before type 2 diabetes developed, such misclassification should be nondifferential between cases and noncases and would tend toward underestimation of the true effect. We confirmed self-reported diabetes by a supplementary questionnaire. Validation of assessment of type 2 diabetes with medical records indicated that diabetes reporting was accurate in this medically knowledgeable population. Because screening for blood glucose level was not feasible in this large cohort, underdiagnosis of diabetes is likely. However, compared with the general population, the degree of underdiagnosis was probably smaller in these health professionals with ready access to medical care. Moreover, underascertainment of type 2 diabetes, if not associated with exposure, would not be expected to affect relative risk estimates (30). We also considered the possibility that diet was associated with the likelihood to be screened for diabetes. However, when we restricted the analyses to symptomatic cases, the findings were similar, arguing against presence of surveillance bias.

Principal component analysis requires several arbitrary decisions on the selection of included variables, the number of retained factors, the method of rotation, and the labels of the factors (31). We did multiple sensitivity analyses to examine whether these choices affected the reproducibility of our findings (10). We identified a similar prudent and western pattern by randomly dividing the sample in two groups, including in our analysis all individual food items on the questionnaire (instead of the 37 food groups), retaining three instead of two factors, using the maximum likelihood method instead of the principal component method, and using an oblique (resulting in correlated factors) instead of orthogonal rotation (10). Furthermore, our analysis showed that the reproducibility of the dietary pattern scores over 4-year and 8-year periods is high compared with the long-term reproducibility reported for nutrients (15). Finally, other large U.S. studies (8, 32), which have included women and socioeconomically diverse participants (8), also have identified a prudent and a western pattern that were largely characterized by the same foods. Yet, the association in our study between dietary patterns and the risk for type 2 diabetes needs to be confirmed for women and various ethnic groups.

In conclusion, our findings indicate that major dietary patterns identified by factor analysis can predict risk for type 2 diabetes. Obesity is known to be a strong risk factor for type 2 diabetes (33). However, in addition to balancing total energy intake with expenditure to prevent weight gain, avoiding a western dietary pattern may substantially reduce risk for type 2 diabetes in men.

From Harvard School of Public Health, Brigham and Women's Hospital, and Harvard Medical School, Boston, Massachusetts, and National Institute for Public Health and the Environment, the Netherlands.

Grant Support: By CA 55075 and HL 35464 from the U.S. National Institutes of Health. Dr. Hu was supported in part by a Research Award from the American Diabetes Association.

Requests for Single Reprints: Frank B. Hu, MD, Department of Nutrition, Harvard School of Public Health, 665 Huntington Avenue, Boston, MA 02115; e-mail, frank.hu@channing.harvard.edu.

Current Author Addresses: Mr. Van Dam: Department of Chronic Diseases Epidemiology, National Institute for Public Health and the Environment, Box 1, 3720 BA Bilthoven, the Netherlands.

Drs. Rimm, Willett, Hu, and Stampfer: Harvard School of Public Health, 665 Huntington Avenue, Boston, MA 02115.

Author Contributions: Conception and design: R.M. van Dam, E.B. Rimm, W.C. Willett, F.B. Hu.
Analysis and interpretation of the data: R.M. van Dam, E.B. Rimm, W.C. Willett, M.J. Stampfer, F.B. Hu.
Drafting of the article: R.M. van Dam, F.B. Hu.
Critical revision of the article for important intellectual content: R.M. van Dam, E.B. Rimm, W.C. Willett, M.J. Stampfer, F.B. Hu.
Final approval of the article: R.M. van Dam, E.B. Rimm, W.C. Willett, M.J. Stampfer, F.B. Hu.
Provision of study materials or patients: W.C. Willett.
Statistical expertise: R.M. van Dam, W.C. Willett, F.B. Hu.
Obtaining of funding: E.B. Rimm, W.C. Willett, F.B. Hu.
Administrative, technical, or logistic support: E.B. Rimm, W.C. Willett, F.B. Hu.
Collection and assembly of data: E.B. Rimm, W.C. Willett.

References

1. **Burke JP, Williams K, Gaskill SP, Hazuda HP, Haffner SM, Stern MP.** Rapid rise in the incidence of type 2 diabetes from 1987 to 1996: results from the San Antonio Heart Study. Arch Intern Med. 1999;159:1450-6. [PMID: 10399896]

2. **Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, et al.** Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988-1994. Diabetes Care. 1998;21:518-24. [PMID: 9571335]

3. **King H, Aubert RE, Herman WH.** Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care. 1998;21:1414-31. [PMID: 9727886]

4. **Himsworth HP.** Diet and the incidence of diabetes mellitus. Clin Sci. 1935; 2:117-48.

5. **Kawate R, Yamakido M, Nishimoto Y, Bennett PH, Hamman RF, Knowler WC.** Diabetes mellitus and its vascular complications in Japanese migrants on the Island of Hawaii. Diabetes Care. 1979;2:161-70. [PMID: 520120]

6. **Pratley RE.** Gene-environment interactions in the pathogenesis of type 2 diabetes mellitus: lessons learned from the Pima Indians. Proc Nutr Soc. 1998; 57:175-81. [PMID: 9656318]

7. **Hu FB, van Dam RM, Liu S.** Diet and risk of Type II diabetes: the role of types of fat and carbohydrate. Diabetologia. 2001;44:805-17. [PMID: 11508264]

8. **Slattery ML, Boucher KM, Caan BJ, Potter JD, Ma KN.** Eating patterns and risk of colon cancer. Am J Epidemiol. 1998;148:4-16. [PMID: 9663397]

9. **Hu FB, Rimm E, Smith-Warner SA, Feskanich D, Stampfer MJ, Ascherio A, et al.** Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. Am J Clin Nutr. 1999;69:243-9. [PMID: 9989687]

10. **Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D, Willett WC.** Prospective study of major dietary patterns and risk of coronary heart disease in men. Am J Clin Nutr. 2000;72:912-21. [PMID: 11010931]

11. **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. [PMID: 10884714]

12. **Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al.** A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336:1117-24. [PMID: 9099655]

13. **de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N.** Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. Circulation. 1999;99:779-85. [PMID: 9989963]

14. **Gittelsohn J, Wolever TM, Harris SB, Harris-Giraldo R, Hanley AJ, Zinman B.** Specific patterns of food consumption and preparation are associated with diabetes and obesity in a Native Canadian community. J Nutr. 1998;128: 541-7. [PMID: 9482761]

15. **Hu FB, Stampfer MJ, Rimm E, Ascherio A, Rosner BA, Spiegelman D, et al.** Dietary fat and coronary heart disease: a comparison of approaches for adjusting for total energy intake and modeling repeated dietary measurements. Am J Epidemiol. 1999;149:531-40. [PMID: 10084242]

16. SAS/STAT User's Guide, version 6. 4th ed. Cary, NC: SAS Institute; 1989.

17. **Kim J-O, Mueller CW.** Factor Analysis: Statistical Methods and Practical Issues. Thousand Oaks, CA: Sage Publications; 1978.

18. **Chasan-Taber S, Rimm EB, Stampfer MJ, Spiegelman D, Colditz GA, Giovannucci E, et al.** Reproducibility and validity of a self-administered physical activity questionnaire for male health professionals. Epidemiology. 1996;7:81-6. [PMID: 8664406]

19. **Rimm EB, Stampfer MJ, Colditz GA, Chute CG, Litin LB, Willett WC.** Validity of self-reported waist and hip circumferences in men and women. Epidemiology. 1990;1:466-73. [PMID: 2090285]

20. WHO Study Group on Diabetes Mellitus. Diabetes Mellitus: Report of a WHO Study Group. Geneva: World Health Organization; 1985.

21. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 1997;20:1183-97. [PMID: 9203460]

22. **D'Agostino RB, Lee ML, Belanger AJ, Cupples LA, Anderson K, Kannel WB.** Relation of pooled logistic regression to time dependent Cox regression analysis: the Framingham Heart Study. Stat Med. 1990;9:1501-15. [PMID: 2281238]

23. **Willett W.** Nutritional Epidemiology. 2nd ed. New York: Oxford Univ Pr; 1998:273-301.

24. **Jacobs DR Jr, Meyer KA, Kushi LH, Folsom AR.** Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. Am J Clin Nutr. 1998;68:248-57. [PMID: 9701180]

25. **Swinburn BA, Boyce VL, Bergman RN, Howard BV, Bogardus C.** Deterioration in carbohydrate metabolism and lipoprotein changes induced by modern, high fat diet in Pima Indians and Caucasians. J Clin Endocrinol Metab. 1991;73:156-65. [PMID: 2045466]

26. **Fung TT, Rimm EB, Spiegelman D, Rifai N, Tofler GH, Willett WC, et al.** Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. Am J Clin Nutr. 2001;73:61-7. [PMID: 11124751]

27. **Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, et al.** Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care. 1997;20:545-50. [PMID: 9096978]

28. **Salmerón J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC.** Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA. 1997;277:472-7. [PMID: 9020271]

29. **Meyer KA, Kushi LH, Jacobs DR Jr, Slavin J, Sellers TA, Folsom AR.** Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. Am J Clin Nutr. 2000;71:921-30. [PMID: 10731498]

30. **Rothman KJ, Greenland S.** Modern Epidemiology. 2nd ed. Philadelphia: Lippincott-Raven; 1998;115-134.

31. **Martinez ME, Marshall JR, Sechrest L.** Invited commentary: Factor analysis and the search for objectivity. Am J Epidemiol. 1998;148:17-9. [PMID: 9663398]

32. **Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB.** Dietary patterns and the risk of coronary heart disease in women. Arch Intern Med. 2001;161: 1857-62. [PMID: 11493127]

33. **Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al.** Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med. 2001;345:790-7. [PMID: 11556298]