

# Dietary Patterns Are Associated with Predicted Cardiovascular Disease Risk in an Urban Mexican Adult Population<sup>1–3</sup>

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## Abstract

**Background:** Dietary patterns may predict cardiovascular disease (CVD) risk more accurately than does consumption of specific nutrients or foods.

**Objective:** We evaluated the association between Mexican adults' dietary patterns and development of a >10% risk of 10-y CVD (using the Framingham risk score) over 7 y of follow-up.

**Methods:** This prospective cohort study included 1196 men and women aged 20–80 y with a 10-y predicted risk <10% and without a CVD diagnosis at baseline in 2004–2007. Data on sociodemographic, lifestyle, and medical history factors were collected with a self-administered questionnaire. Dietary intake was evaluated by using a semiquantitative food-frequency questionnaire. The relations between dietary patterns and predicted CVD were analyzed by using pooled logistic regression models.

**Results:** With the use of factor analysis, we identified 3 major dietary patterns in participants' dietary data. The "prudent" pattern was characterized by high positive loadings for the consumption of fresh fruit, vegetables, and whole grains. The "meat/fish" pattern showed positive loadings for the consumption of red meat, processed meat, eggs, fats, fish, and poultry. Finally, the "refined foods" pattern featured positive loadings for corn tortillas, refined grains, soft drinks, and alcohol. After adjustment for potential confounders, compared with participants in the lowest quintile of the prudent pattern, those in the highest quintile had a lower RR of 10-y CVD (RR: 0.40; 95% CI: 0.20, 0.79; *P*-trend = 0.006). In contrast, participants in the highest quintile of the refined-foods pattern had a greater risk of elevated 10-y CVD (RR: 2.98; 95% CI: 1.46, 6.10; *P*-trend = 0.020) than did those in the lowest quintile. Finally, the meat/fish dietary pattern was not significantly associated with 10-y CVD.

**Conclusion:** Our data suggest that the prudent pattern is associated with a reduced risk of 10-y CVD, whereas the refined-foods pattern may increase 10-y CVD in Mexican adults. *J Nutr* 2016;146:90–7.

**Keywords:** predicted cardiovascular disease risk, dietary patterns, factor analysis, adult population, Mexico, Health Workers Cohort Study

## Introduction

Cardiovascular disease (CVD)<sup>9</sup> is the leading cause of death, a major cause of disability worldwide (1), and the main cause of

mortality in Mexican adults (2). Multiple epidemiologic studies suggest that lifestyle, especially diet, significantly influences CVD occurrence (3). In response, traditional nutritional epidemiology research has focused primarily on investigating the CVD risk posed by specific dietary components (single foods, beverages, or nutrients). This research revealed that fruit, vegetables, whole grains, and nuts play an important role in preventing CVD, mainly by providing dietary fiber and antioxidants (3, 4). Similarly, MUFA- and PUFA-rich diets (featuring olive oil and fish) have been shown to have beneficial effects on lipid profiles and to reduce CVD risk (5–7). *trans* FAs, saturated fats, dietary cholesterol, red meat, and other dietary components have been associated with increased CVD risk (3).

However, these individual dietary factors explain only part of the relation between diet and CVD risk. Dietary intake is a

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<sup>3</sup> Supplemental Tables 1 and 2 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>.

<sup>9</sup> Abbreviations used: BP, blood pressure; CVD, cardiovascular disease; DBP, diastolic blood pressure; PCA, principal components analysis; SBP, systolic blood pressure.

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complex exposure variable, because meals consist of multiple foods and have the combined effect of all of those foods' constituent nutrients. This has made it difficult to investigate the separate effects of individual nutrients consumed simultaneously. As a result, dietary pattern analysis has emerged in nutritional epidemiology research. Assessing dietary patterns may shed more light on diet-disease relations (8–14) and generate dietary advice more suited to real-world eating behavior (10).

Studies from around the world have examined the role of specific dietary patterns on the risk of CVD (11, 15, 16) and its risk factors (17–21). Previous research in the Mexican population documented significant associations between dietary patterns, defined by using principal components analysis (PCA), and metabolic syndrome, obesity, insulin resistance, and gastric cancer (22–25). However, to our knowledge, an analysis of the relation between dietary patterns and CVD risk in the Mexican population has not yet been conducted.

Therefore, the objective of the present study was to evaluate the association between major dietary patterns, derived by PCA, on the risk of 10-y CVD in a Mexican population. We hypothesized that of the 3 dietary patterns we previously identified in this population (22–25), the dietary pattern characterized by greater intakes of whole grains, fruit, vegetables, and legumes would be negatively associated with CVD risk, whereas the dietary pattern characterized by greater intakes of red or processed meats, refined grains, pastries, and sweetened beverages would be positively associated with higher risk of CVD, independently of demographic and lifestyle factors.

## Methods

**Study population.** The Health Workers Cohort Study is a longitudinal study investigating relations between lifestyle and health. It was established in 2004 when 10,769 Mexican employees and their relatives from 3 health and academic institutions in Morelos and Mexico states responded to a questionnaire on health-related factors. Study design, methodology, and participants' characteristics were previously described (22, 26). Seven years after the initiation of the study, ~2500 of those

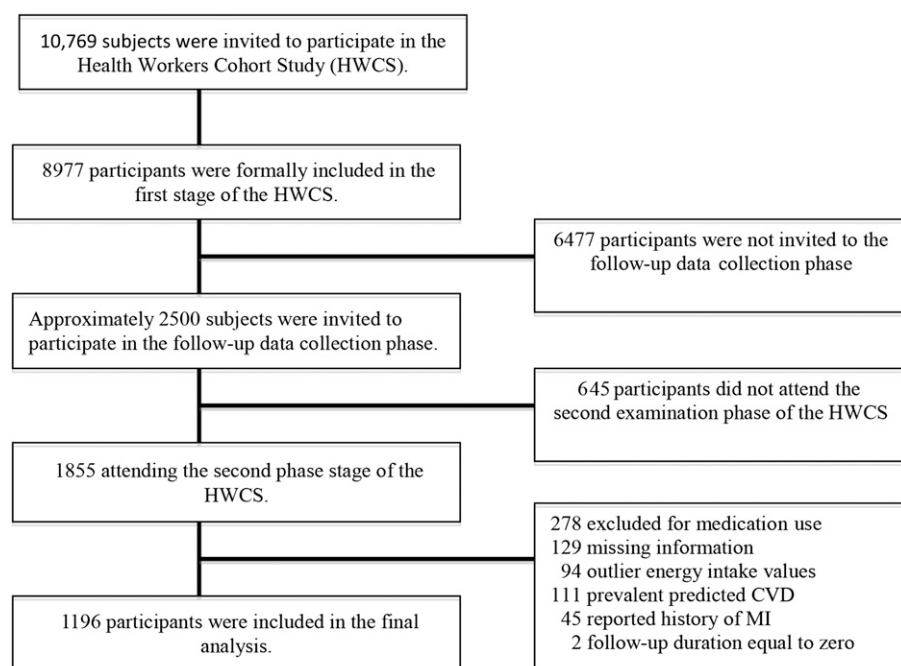
original Health Workers Cohort Study participants were invited to participate in a follow-up data collection phase, which took place from 2012 to 2013. Of these, participants attending the second examination ( $n = 1855$ ) were eligible for the current investigation. Both participants who attended the second, follow-up examination and those who did not were similar in age, percentage of women and men, and other important characteristics.

Data on sociodemographic, lifestyle, and medical history factors were collected with a self-administered questionnaire; and anthropometric measurements and clinical evaluations were taken at baseline and at the end of follow-up. For the present analysis, we excluded participants with missing information on blood pressure (BP), with >10% blank items on their dietary intake information, or with missing serum lipid, lipoprotein, and glucose concentrations. Participants with outlier energy intake values (outside the range of 600–7000 kcal/d) were eliminated by using the SD method (27). We also excluded participants who reported a history of myocardial infarction or stroke, because these diseases could have led them to alter their diet and lifestyle. We excluded those who were taking medications that would affect serum lipid, lipoprotein, or glucose concentrations or blood pressure. In addition, we eliminated those whose follow-up duration was equal to zero or missing. Finally, we also excluded individuals who had elevated predicted CVD risk ( $\geq 10\%$  risk in 10 y) at baseline (Figure 1). Consequently, our final analysis included 1196 participants (299 men and 897 women).

This study was planned and performed according to the guidelines of the Declaration of Helsinki. All participating institutions' research ethics committees approved the study protocol and informed consent forms, and written informed consent was obtained from all participants.

**Dietary intake and dietary pattern assessment.** We used a previously validated (28) and self-administered semiquantitative FFQ to assess dietary intake in the previous year. The FFQ included data regarding the consumption of 116 food items. For each food, a commonly used portion size (e.g., 1 slice of bread or 1 cup of coffee) was specified, and participants reported the frequency with which they had consumed each specific food during the previous year. Response options were 1 of 10 mutually exclusive possibilities, ranging from "never" to "6 or more times per day." For our analysis, the reported frequency for each food item was converted into daily intake. Total energy intake was computed by summing the energy intakes from all foods (29).

The energy intake from each food group was converted into a percentage of total energy intake per day and standardized by  $z$  score



**FIGURE 1** Participant flow diagram. CVD, cardiovascular disease; MI, myocardial infarction.

(30); all food items received the same weighting within a food group. The percentage of contribution to total energy intake was used. This was calculated for each food item consumed and then adding the contribution from all of the foods within the same food group. Foods and beverages from the FFQ were categorized into 28 food groups that were used to derive dietary patterns with the use of PCA. Details of the food groupings used to derive the dietary patterns are described elsewhere (22, 23). Briefly, the basis for placing a food item in a certain food group (Supplemental Table 1) was the similarity of nutrients. Some groups were defined according to the amount of sugar added (e.g., sweetened beverages). Other groups were defined according to their lipid profile (e.g., seeds or margarine). Finally, some food items were considered individually as a food group, because their nutrient profiles were unique, were consumed frequently, or had a unique culinary use (e.g., tortillas, eggs, and orange juice) (22, 23).

To derive the dietary patterns and to determine the factor loadings of each of the 28 groups, a factor analysis of the main components was used (31). The factors were orthogonally rotated (varimax rotation) to keep them uncorrelated and to improve the interpretation. Factors with eigenvalues  $>1.5$  were retained after assessment of graphic analysis and interpretability. Each factor was defined by a subset of at least 5 food groups with an absolute loading  $\geq 0.2$  (considering that  $\geq 0.2$  loadings contributed significantly to the dietary pattern) (22, 23, 30). The factor scores for each dietary pattern were estimated by adding the consumption of the food groups weighted by their loading factor, and each participant received a score for each of the 3 identified patterns.

**Assessment of anthropometric and clinical variables.** Body weight was measured with a calibrated electronic scale (model BC-533; Tanita) with participants wearing minimal clothing and no shoes. Height was measured by using a conventional stadiometer (Seca), with barefoot participants standing with their shoulders in a normal position; measurements were taken with the tape in a horizontal plane perpendicular to the vertical scale, touching the top of the head at the moment of inspiration. BMI was computed as a ratio of weight in kilograms divided by height in meters squared. BP was measured with an automatic digital BP monitor (Citizens Systems). Participants were seated with their right arm resting at heart level. BP categories were defined according to the Fifth Joint National Committee on Hypertension as follows (32): optimal BP [systolic BP (SBP)  $<120$  and diastolic BP (DBP)  $<80$  mm Hg], normal BP (SBP of  $120$ – $129$  or DBP of  $80$ – $84$  mm Hg), high-normal BP (SBP of  $130$ – $139$  or DBP of  $85$ – $89$  mm Hg), hypertension stage I (SBP of  $140$ – $159$  or DBP of  $90$ – $99$  mm Hg), and hypertension stages II–IV (SBP of  $\geq 160$  or DBP of  $\geq 100$  mm Hg). All measurement procedures were performed by nurses trained to use standardized procedures (reproducibility was evaluated, resulting in concordance coefficients between 0.83 and 0.90).

**Biomarker assessment.** After a 12-h fast, a venous blood sample was collected from each participant at baseline and follow-up evaluations. Plasma TGs were measured with a colorimetric method after enzymatic hydrolysis performed with the lipase technique. HDL cholesterol was measured with the clearance method. This method showed excellent correlation with the reference method ( $r = 0.99$ ). Non-HDL-cholesterol lipoprotein was removed in the first step of the reaction (clearance step). LDL cholesterol was also measured with the clearance method; correlation studies on the LDL-cholesterol clearance method produced a coefficient of  $r = 0.985$  with ultracentrifugation; total cholesterol was measured with the colorimetric method after enzymatic assay. The results of these clinical tests were obtained by using Instrumentation Laboratory ILab 600 Chemistry Analyzer and reagents. LDL and HDL cholesterol were classified into the following categories:  $<100$ ,  $100$ – $129$ ,  $130$ – $159$ ,  $160$ – $189$ , and  $\geq 190$  mg/dL for LDL cholesterol and  $<35$ ,  $35$ – $44$ ,  $45$ – $59$ , and  $\geq 60$  mg/dL for HDL cholesterol. Type 2 diabetes was diagnosed as fasting glucose  $\geq 126$  mg/dL or by self-report (33).

**Assessment of other variables.** Information on participants' socio-demographic characteristics (e.g., age, sex, and education), medical history, and lifestyle, including consumption of alcohol and tobacco, and physical activity level was collected with self-administered questionnaires.

Physical activity was assessed by using a validated physical activity questionnaire, adapted for use in the Mexican population (34). Participants reported the time spent in a typical week on activities such as running and walking during the previous year. Each activity was given a value in metabolic equivalent tasks (METs) and total metabolic equivalent tasks per week were computed.

**CVD risk assessment.** CVD risk was calculated using a recalibration of the Framingham ischemic heart disease prediction score (33, 35). We first estimated the predicted risk of total CVD, applying the  $\beta$ -coefficients from the Cox proportional hazards model obtained from the Framingham population by Wilson et al. (33), which included age, current smoking, type 2 diabetes, BP regardless of hypertension treatment (predefined BP categories), serum LDL cholesterol (predefined categories), and HDL cholesterol (predefined categories). We used this equation to calculate each participant's 10-y predicted probability of CVD.

The main outcome of the current analysis was the development of a  $>10\%$  risk of CVD in the 7 y between baseline and follow-up data collection. We defined participants as at low CVD risk when they had  $<10\%$  risk in 10 y (36).

**Statistical analysis.** Descriptive analyses of the main variables of interest (including age, BMI, physical activity, and total energy intake) across quintiles of each dietary pattern score were performed. ANOVA was used to evaluate mean differences across quintiles of each dietary pattern. The chi-square test was used to determine differences in the distribution of qualitative variables across quintiles of each dietary pattern.

Multivariate linear models were fitted to evaluate the relation of dietary patterns on cardiovascular risk factors, in which these variables were analyzed as continuous. In this case, the increments of the cardiovascular risk factors were considered for each unit increase in dietary pattern score. To compute RR, we used pooled logistic regression with 2-y intervals (37), which is approximately equivalent to Cox regression for time-dependent covariates when the event is rare. We fitted 4 different models. The first model was adjusted for age ( $<30$ ,  $30$ – $39$ ,  $40$ – $49$ ,  $50$ – $59$ ,  $60$ – $69$ , and  $\geq 70$  y), sex, time (four 2-y periods), and energy intake (quintiles). The second multivariable model added smoking (never, past, and current), physical activity (min/d), postmenopausal hormone use (yes or no), use of multivitamin supplements (yes or no), parental history of myocardial infarction (yes or no), and history of hypertension (yes or no). The final multivariable-adjusted model further controlled for BMI (in  $\text{kg}/\text{m}^2$ ;  $<25$  and  $\geq 25$ ). To assess the overall trend of ORs across increasing quintiles of dietary pattern scores, we computed the Mantel-Haenszel extension chi-square test (38). All  $P$  values presented are 2-sided;  $P < 0.05$  was considered significant. All statistical analyses were performed by using the STATA statistical software package, version 11.0, for Windows (Stata Corp).

## Results

Food intake data for 28 predefined food groups were entered into the factor analysis procedure. According to the criteria indicated above, a 3-factor solution was obtained, which explained 21.9% of the total variance (Supplemental Table 2). The "prudent" dietary pattern accounted for 9.6% of the variance and was characterized by high positive loadings for the consumption of fresh fruit (0.74), vegetables (0.60), and whole grains (0.37) and negative loadings for refined grains ( $-0.38$ ) and soft drinks ( $-0.45$ ). The second pattern, which we labeled the "meat/fish" dietary pattern, showed positive loadings for the consumption of red meat, processed meat, eggs, fats, fish, and poultry and negative loadings for pastries ( $-0.36$ ) and corn tortillas ( $-0.42$ ), accounting for 6.6% of the variance. Last, the "refined foods" dietary pattern accounted for 5.7% of the total variance and was represented mainly by negative loadings for high-fat dairy products ( $-0.65$ ) and red meat ( $-0.24$ ) and

**TABLE 1** Baseline characteristics by quintile within dietary pattern categories in the Health Workers Cohort Study, 2004–2007<sup>1</sup>

Variables	Prudent pattern					Meat/fish pattern					Refined-foods pattern				
	Total (n = 1196)	Q1 (n = 240)	Q3 (n = 239)	Q5 (n = 239)	P	Q1 (n = 240)	Q3 (n = 239)	Q5 (n = 239)	P	Q1 (n = 240)	Q3 (n = 239)	Q5 (n = 239)	P		
Participant characteristics															
Women, %	75.0	71.7	73.2	79.1	0.33	90.0	78.7	57.7	<0.001	82.9	73.6	64.9	<0.001		
Age, y	41.1 ± 13.0 <sup>2</sup>	41.1 ± 12.6	39.9 ± 12.9	41.9 ± 13.1	0.19	45.0 ± 12.0	42.0 ± 12.6	34.6 ± 13.4	<0.001	36.2 ± 14.0	40.9 ± 12.1	44.7 ± 12.6	<0.001		
Physical activity, min/d	26.0 ± 5.0	26.2 ± 4.9	26.5 ± 5.0	25.5 ± 5.1	0.20	23.8 ± 4.1	25.5 ± 4.6	29.1 ± 5.1	<0.001	26.9 ± 5.2	26.2 ± 4.9	25.6 ± 5.2	0.019		
Current smoker, %	17.0	15.0	16.7	19.3	<0.001	14.2	13.0	27.6	<0.001	15.4	18.8	20.5	0.08		
Multivitamin supplement use, %	37.2	29.1	41.2	43.7	0.013	44.3	33.7	32.7	0.13	41.5	36.4	35.4	0.53		
Parental history of MI, %	19.3	17.5	20.5	20.5	0.14	23.3	23.9	16.7	0.019	19.2	21.8	15.1	0.25		
BMI, kg/m <sup>2</sup>	25.6 ± 4.2	25.7 ± 3.9	25.6 ± 4.3	25.7 ± 4.5	0.22	25.7 ± 3.9	25.7 ± 3.9	25.5 ± 4.5	0.58	24.4 ± 4.2	26.0 ± 4.3	26.1 ± 3.7	<0.001		
Hypertension, <sup>3</sup> %	6.9	8.3	6.7	5.0	0.68	6.7	6.7	7.5	0.68	3.8	8.4	11.3	0.001		
Hypercholesterolemia, <sup>4</sup> %	38.8	37.5	40.1	39.7	0.96	49.2	36.8	28.0	<0.001	31.7	39.8	44.4	0.07		
Hypertriglyceridemia, <sup>5</sup> %	37.2	40.8	35.9	33.5	0.33	38.8	37.2	32.2	0.32	25.0	38.1	50.2	<0.001		
Incident predicted CVD risk, <sup>6</sup> %	16.5	20.8	16.7	9.6	<0.001	16.2	13.8	15.5	0.79	9.2	17.2	24.3	<0.001		
Dietary intake															
Energy, kcal/d	2188 ± 916	2329 ± 948	2192 ± 870	1936 ± 909	<0.001	2281 ± 986	2205 ± 848	2222 ± 1066	0.017	2480 ± 1086	2162 ± 867	1866 ± 762	<0.001		
Carbohydrate, % of energy	60.5 ± 8.7	66.1 ± 7.8	61.4 ± 6.8	54.0 ± 8.4	<0.001	68.0 ± 8.2	60.2 ± 6.9	54.2 ± 7.9	<0.001	56.9 ± 8.8	62.5 ± 8.5	63.2 ± 7.4	<0.01		
Protein, % of energy	14.3 ± 2.7	12.8 ± 2.5	14.6 ± 2.2	16.3 ± 2.8	0.67	13.6 ± 3.3	14.2 ± 2.1	14.4 ± 2.8	0.07	14.9 ± 3.0	13.9 ± 2.6	13.5 ± 2.2	<0.001		
Total fat, % of energy	25.2 ± 7.3	21.1 ± 6.5	24.0 ± 6.0	29.7 ± 7.3	0.61	18.4 ± 6.5	25.6 ± 6.1	31.4 ± 6.3	<0.001	28.2 ± 7.4	23.6 ± 7.1	23.3 ± 6.9	<0.001		
Saturated fat, % of energy	8.9 ± 2.5	8.1 ± 2.8	8.8 ± 2.3	9.6 ± 2.5	0.76	6.9 ± 2.1	9.1 ± 2.1	10.3 ± 2.6	<0.001	11.1 ± 2.8	8.5 ± 2.1	7.1 ± 1.5	<0.001		
MUFAs, % of energy	9.9 ± 2.5	8.2 ± 2.2	10.0 ± 2.0	11.7 ± 2.6	0.016	8.4 ± 2.6	10.0 ± 2.0	11.8 ± 2.7	<0.001	11.6 ± 2.9	9.5 ± 2.2	8.9 ± 2.0	<0.001		
PUFAs, % of energy	4.2 ± 1.1	3.5 ± 0.9	4.3 ± 0.9	5.5 ± 1.0	<0.01	3.8 ± 1.2	4.1 ± 0.9	5.0 ± 1.2	<0.001	4.1 ± 1.1	4.2 ± 1.1	4.4 ± 1.2	<0.001		
Alcohol use, g/d	4.1 ± 9.9	2.4 ± 4.8	4.9 ± 6.0	5.5 ± 10.7	<0.001	2.4 ± 5.0	4.9 ± 15.1	5.5 ± 10.8	<0.01	2.0 ± 3.4	3.7 ± 8.6	5.8 ± 15.5	<0.001		
Dietary folate, µg/d	396 ± 228	390 ± 255	394 ± 221	408 ± 256	0.63	589 ± 298	380 ± 178	290 ± 160	<0.001	446 ± 256	415 ± 238	323 ± 200	<0.001		
Fiber, g/d	16.9 ± 10.2	16.7 ± 11.0	17.3 ± 9.9	15.9 ± 10.7	0.35	22.7 ± 13.4	17.1 ± 9.7	13.2 ± 7.2	<0.001	17.8 ± 11.9	16.7 ± 8.9	16.6 ± 11.1	0.59		

<sup>1</sup> ANOVA was used for statistical comparisons between quantitative variables, and chi-square test was used for statistical comparisons between qualitative variables. CVD, cardiovascular disease; MI, myocardial infarction; Q, quintile.

<sup>2</sup> Mean ± SD (all such values).

<sup>3</sup> Systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg.

<sup>4</sup> Total cholesterol ≥200 mg/dL.

<sup>5</sup> TGs ≥150 mg/dL.

<sup>6</sup> More than 10% risk of 10-y predicted CVD (based on the Framingham risk score).

**TABLE 2** Mean differences in concentrations of blood lipids and other cardiometabolic biomarkers according to dietary pattern in the Health Workers Cohort Study<sup>1</sup>

	Dietary pattern		
	Prudent (−3.6 to 3.1 z scores)	Meat/fish (−2.8 to 5.0 z scores)	Refined (−5.4 to 4.4 z scores)
TGs, <sup>2</sup> mg/dL	−8.1 (−13.9, −2.2)	−2.0 (−8.3, 4.3)	10.8 (4.5, 17.1)
Total cholesterol, <sup>3</sup> mg/dL	−4.2 (−7.0, −1.5)	−0.1 (−2.5, 2.4)	2.2 (−0.6, 4.8)
LDL cholesterol, <sup>4</sup> mg/dL	−3.0 (−5.4, −0.6)	1.2 (−0.9, 3.3)	0.3 (−2.0, 2.5)
HDL cholesterol, <sup>5</sup> mg/dL	0.4 (−0.2, 1.1)	−0.2 (−1.0, 0.5)	−0.3 (−0.9, 0.5)
Glucose, <sup>6</sup> mg/dL	−1.3 (−2.8, 0.2)	0.7 (−0.8, 1.9)	1.9 (0.4, 3.2)
Diastolic blood pressure, <sup>7</sup> mm Hg	−0.2 (−0.9, 0.4)	−0.05 (−0.7, 0.6)	0.7 (0.1, 1.4)
Systolic blood pressure, <sup>8</sup> mm Hg	−0.3 (−1.1, 0.7)	0.2 (−0.7, 1.1)	1.1 (0.1, 2.0)

<sup>1</sup> Values are  $\beta$  (95% CI) adjusted for age (<30, 30–39, 40–49, 50–59, 60–69, and  $\geq 70$  y), sex, time, smoking status (never, past, and current smoking), alcohol consumption (nondrinker, moderate, and heavy), multivitamin use (yes or no), physical activity (min/d), postmenopausal hormone use (yes or no), BMI ( $\text{kg}/\text{m}^2$ ; <25 or  $\geq 25$ ), and energy intake (quintiles).

<sup>2–6</sup> Additionally adjusted by baseline concentrations of <sup>2</sup>TGs, <sup>3</sup>total cholesterol, <sup>4</sup>LDL cholesterol, <sup>5</sup>HDL cholesterol, and <sup>6</sup>glucose.

<sup>7,8</sup> Additionally adjusted by baseline <sup>7</sup>diastolic blood pressure and <sup>8</sup>systolic blood pressure.

positive loadings for corn tortillas (0.60), refined grains (0.35), soft drinks (0.22), and alcohol (0.22).

The present analysis included data from a sample of 1196 participants, 75% of whom were female. During the  $\sim 7$  y between the baseline and follow-up assessments, 16.5% of the participants showed a >10% risk of predicted CVD. At baseline in 2004–2007, those in the highest quintile of the prudent pattern tended to be current smokers, take multivitamins, and have a lower likelihood of a >10% risk of 10-y CVD than those in the lowest quintile of the prudent dietary pattern (Table 1). In addition, the adults in the highest compared with lowest quintile had lower intakes of carbohydrates and higher intakes of MUFAs and PUFAs. Those in the highest quintile of the meat/fish dietary pattern were younger and more likely to be men, to smoke, to exercise, and to have a lower prevalence of parental history of myocardial infarction than those in the lowest quintile. They had higher intakes of MUFAs and PUFAs and lower intakes of fiber and folate than did those in the lowest quintile. Finally, participants in the highest quintile of the refined-foods dietary pattern were older, more likely to be men, and to have a greater likelihood of hypertension, hypertriglyceridemia, and a >10% risk of 10-y predicted CVD than those in the lowest quintile. These participants also had relatively higher intakes of carbohydrates and lower intakes of monounsaturated fat, protein, and folate than did those in the lowest quintile.

Table 2 shows the results of multiple linear regression analysis, which evaluated the relation between dietary patterns on cardiovascular risk factors. After adjusting for age, sex, time, smoking status, alcohol consumption, multivitamin use, phys-

ical activity, postmenopausal status, hormone use, BMI, and total energy intake, we found that subjects' TG and glucose concentrations increased with each unit (z score) increase in their refined-foods dietary pattern score [ $\beta = 10.8$  (95% CI: 4.5, 17.1) and  $\beta = 1.9$  (95% CI: 0.4, 3.2), respectively]. Conversely, subjects' HDL cholesterol concentrations decreased with each unit increase in their refined-foods dietary pattern score ( $\beta = -0.3$ ; 95% CI: -0.9, 0.5). In addition, we observed a decrease in subjects' TG and total cholesterol concentrations for each unit increase in their prudent dietary pattern score [ $\beta = -8.1$  (95% CI: -13.9, -2.2) and  $\beta = -4.2$  (95% CI: -7.0, -1.5), respectively].

After adjustment for age, sex, duration of follow-up, smoking status, multivitamin use, physical activity, postmenopausal status, hormone use, BMI, and total energy intake, the multivariate RRs across quintiles of prudent dietary pattern scores were 1.0, 0.93, 0.84, 0.64, and 0.40 ( $P$ -trend = 0.006). In the multivariate analysis, participants in the highest quintile of scores in the refined-foods dietary pattern were more likely to develop elevated 10-y predicted CVD than were those in the lowest quintile (RR: 2.98; 95% CI: 1.46, 6.10;  $P$ -trend = 0.020). The meat/fish dietary pattern was not significantly associated with 10-y CVD (Table 3).

## Discussion

Three major dietary patterns emerged through factor analysis. The prudent dietary pattern, characterized by high intakes of whole grains, fresh fruit, and fresh vegetables and lower

**TABLE 3** RRs (95% CIs) of developing a Framingham CVD risk score of >10%, by dietary pattern quintile in the Health Workers Cohort Study<sup>1</sup>

Dietary pattern	Dietary pattern quintile scores					$P$ -trend <sup>2</sup>
	Q1 ( $n = 240$ )	Q2 ( $n = 239$ )	Q3 ( $n = 239$ )	Q4 ( $n = 239$ )	Q5 ( $n = 239$ )	
Prudent	1.0	0.93 (0.46, 2.00)	0.84 (0.48, 1.45)	0.64 (0.29, 1.26)	0.40 (0.20, 0.79)	0.006
Meat/fish	1.0	1.02 (0.58, 1.81)	0.99 (0.53, 1.86)	1.12 (0.62, 2.08)	1.22 (0.64, 2.33)	0.981
Refined-foods	1.0	1.61 (0.63, 4.1)	2.39 (1.17, 4.87)	2.71 (1.11, 6.05)	2.98 (1.46, 6.10)	0.020

<sup>1</sup> Values were adjusted for age (<30, 30–39, 40–49, 50–59, 60–69, and  $\geq 70$  y), sex, time (four 2-y periods), energy intake (quintiles), smoking (never, past, and current), multivitamin use (yes or no), parental history of myocardial infarction (yes or no), history of hypertension (yes or no), physical activity (min/d), BMI ( $\text{kg}/\text{m}^2$ ; <25 or  $\geq 25$ ), and postmenopausal hormone use (yes or no). CVD, cardiovascular disease; Q, quintile.

<sup>2</sup> To assess the overall trend of ORs across increasing quintiles of dietary pattern scores, we computed the Mantel-Haenszel extension chi-square test.

consumption of refined grains, soft drinks, and sugars was significantly associated with a lower 10-y CVD in individuals with low CVD risk at baseline. In contrast, the refined-foods dietary pattern, reflecting high consumption of refined grains, corn tortillas, alcohol, and soft drinks, was linked to a higher 10-y predicted CVD. In contrast to our hypothesis, the meat/fish dietary pattern, characterized by intakes of red and processed meats and fats, was not significantly associated with predicted CVD.

The patterns we found overlapped with those identified in previous studies conducted in different populations. Such studies found a vegetable-rich pattern similar to our prudent dietary pattern, generally labeled as “healthy” or “prudent” (11, 16, 21, 39), and “Western” or “traditional” patterns with contents resembling those in our meat/fish and refined-foods dietary patterns (11, 40).

The relation of dietary patterns with CVD has been analyzed in different countries around the world (11, 15, 16, 40). In the present study, the prudent pattern was associated with lower 10-y CVD (RR: 0.40; 95% CI: 0.20, 0.77; *P*-trend = 0.006). This is consistent with results from the Health Professionals Follow-Up Study (11), in which the prudent dietary pattern (mainly represented by frequent consumption of fruit, vegetables, and whole grains) was significantly associated with a decreased risk of ischemic heart disease (RR: 0.75; 95% CI: 0.59, 0.95; *P*-trend = 0.02). An inverse relation between prudent or healthy dietary patterns and incident CVD was also found in Swedish (41) and US (40, 42) women. The dietary pattern approach cannot identify the particular nutrients accountable for the observed differences in disease risk. However, on the basis of findings from previous single-nutrient/food studies (4), we suggest that the protection afforded by the prudent dietary pattern may derive from the dietary fiber and the antioxidant and anti-inflammatory vitamins, minerals, and phytonutrients present in vegetables, fruit, and whole grains.

Some foods that loaded positively in our refined-foods dietary pattern (refined grains, soft drinks, alcohol) were comparable to those reported in the Western dietary pattern, which were correlated with greater CVD risk and incident ischemic heart disease in the US Nurses' Health Study (40) and the Health Professionals Follow-Up Study (11). Our finding that the refined-foods dietary pattern was linked to augmented CVD risk is of considerable concern, because this pattern is becoming increasingly common due to the growing accessibility and consumption of refined cereals and soft drinks (43, 44). The adverse relation of the refined-foods dietary pattern may be due to added sugars and the high glycemic index of refined grains, because these nutrients are highly present in the food groups that comprise that dietary pattern. This information is congruent with recent findings linking the consumption of added sugar to higher risk of CVD (45, 46).

The lack of association between the meat/fish dietary pattern, characterized mainly by red meat, processed meat, and fats, might seem surprising. These food groups have contributed to diets labeled “Western” in other populations and have been shown to have negative associations with heart disease risk (11, 40). However, we found only a weak and nonsignificant association (*P* = 0.981), which could be attributed to the fish present in the meat/fish dietary pattern and the lack of a link with the processed foods usually seen together with meat and processed meat in other studies with Western dietary patterns. Rather, it is noteworthy that those in the lowest quintile of this pattern had a high carbohydrate intake. This variation in food combinations from wealthier countries reveals the dietary

inadequacies likely experienced by low-income participants with inadequate access to animal-based foods. Furthermore, those in the highest quintile of this pattern still have mean saturated fat intakes that are <10% of energy, which is lower than those reported in the United States (47).

It is important to mention some methodologic limitations that affect the interpretation of our results. First, the use of PCA to estimate the dietary patterns has some weaknesses. It requires decisions with regard to the number and types of foods that make up the groups and the cutoffs in the number of patterns. In addition, the use of the FFQ provides an indirect measure of eating patterns (48). However, the participants in our study were categorized into concrete groups of foods on the basis of usual long-term intake, and this method has been shown to be useful for evaluating the relation of diet to a disease outcome (49). Second, measurement errors in dietary intakes are unavoidable; these errors are likely random in prospective studies such as this one and not systematically related to the outcome of interest, but this could have led to an underestimation of the association between dietary patterns and predicted CVD. However, the FFQ used here has been validated (28) and can reasonably reflect long-term dietary intake. Third, participants may change their diets after they develop some intermediate diseases, leading to a conservative estimate. Nevertheless, excluding participants with histories of diabetes or CVD reduced the possibility of bias from this source and generated similar null associations. Furthermore, although we adjusted for potential confounding factors, the presence of residual or unmeasured confounding is possible. In addition, the participants in this cohort study are adults from a specific segment of the Mexican population: working class, seemingly healthy individuals. Although these adults cannot be considered representative of the Mexican adult population as a whole, they may be considered representative of middle- to low-income men and women residing in the urban areas of central Mexico. Finally, the prospective design of our study reduces the possibility of recall or selection bias.

In conclusion, these findings on the association between dietary patterns and CVD risk in a Mexican population extend the literature that has focused on Western countries. Studying the link between dietary patterns and CVD risk is especially relevant for the Mexican adult population, because of both their high incidence of CVD and the unique dietary characteristics that may lead to variance with findings from other parts of the world. We found that a dietary pattern characterized by a high consumption of vegetables, fresh fruit, and whole grains and lower consumption of refined grains and soft drinks was robustly associated with a lower 10-y predicted CVD in this population. In contrast, a dietary pattern mainly represented by a high consumption of refined grains (processed foods), corn tortillas, and soft drinks was significantly associated with higher CVD. Although it is necessary to implement large-scale primary prevention trials focused on dietary patterns and CVD risk, observational findings such as these constitute an important basis for dietary recommendations, government programs, and negotiations with industry that can help Mexican people make healthy food choices.

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ED-G and JS designed the study and secured funding; JS conducted the research; ED-G and MF performed the statistical analyses; ED-G wrote the manuscript; ED-G, KLT, and SB critically reviewed the manuscript; and ED-G, KLT, MF, and SB reviewed and commented on the manuscript. All authors read and approved the final version of the manuscript.

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