

Adherence to the *Dietary Guidelines for Americans* and risk of major chronic disease in men^{1–5}

Marjorie L McCullough, Diane Feskanich, Eric B Rimm, Edward L Giovannucci, Alberto Ascherio, Jayachandran N Variyam, Donna Spiegelman, Meir J Stampfer, and Walter C Willett

ABSTRACT

Background: The *Dietary Guidelines for Americans* and the food guide pyramid aim to reduce the risk of major chronic disease in the United States, but data supporting their overall effectiveness are sparse. The healthy eating index (HEI) measures the concordance of dietary patterns with these guidelines.

Objective: We tested whether a high HEI score (range: 0–100; 100 is best) calculated from a validated food-frequency questionnaire (HEI-f) could predict lower risk of major chronic disease in men.

Design: A cohort of US male health professionals without major disease completed detailed questionnaires on food intake and other risk factors for heart disease and cancer in 1986 and repeatedly during the 8-y follow-up. Major chronic disease outcome was defined as incident major cardiovascular disease (stroke or myocardial infarction, $n = 1092$), cancer ($n = 1661$), or other non-trauma-related deaths ($n = 366$).

Results: The HEI-f was weakly inversely associated with risk of major chronic disease [comparing highest with lowest quintile of the HEI-f, relative risk (RR) = 0.89; 95% CI: 0.79, 1.00; $P < 0.001$ for trend]. The HEI-f was associated with moderately lower risk of cardiovascular disease (RR = 0.72; 95% CI: 0.60, 0.88; $P < 0.001$) but was not associated with lower cancer risk.

Conclusions: The HEI-f was only weakly associated with risk of major chronic disease, suggesting that improvements to the HEI may be warranted. Further research on the HEI could have implications for refinements to the *Dietary Guidelines for Americans* and the food guide pyramid. *Am J Clin Nutr* 2000;72:1223–31.

KEY WORDS Diet, nutrition, diet quality, healthy eating index, food guide pyramid, dietary guidelines, *Dietary Guidelines for Americans*, cardiovascular disease, stroke, heart disease, myocardial infarction, cancer, men, chronic disease prevention, cancer prevention, cardiovascular disease prevention

INTRODUCTION

Diet plays a major role in the development of cardiovascular disease (CVD) and cancer (1, 2)—the leading causes of death in the United States (3)—but the contributions made by specific aspects of the diet are unclear. Analyses of diet and disease have traditionally focused on understanding etiology and thus have appropriately investigated individual nutrients in relation to spe-

See corresponding article on page 1214.

cific disease endpoints (4). Much has been learned from this approach. However, public health nutrition policy should also consider complete dietary patterns (5) and their overall impact on health. The former was the rationale for developing the *Dietary Guidelines for Americans* (6) and its companion educational tool, the food guide pyramid (7, 8).

The healthy eating index (HEI) measures adherence to the major recommendations of the *Dietary Guidelines for Americans* and the food guide pyramid and was created to monitor the dietary status of Americans and to serve as the basis of nutrition promotion activities for the population (9). Although the HEI has been used to characterize changes in diet quality over time (10) and to describe US eating patterns and their determinants (11), the value of this index for predicting health outcomes has not been examined.

The purpose of this study was to evaluate whether adherence to the *Dietary Guidelines for Americans*, measured by calculating the HEI from food-frequency questionnaires (FFQs), predicts improved health outcomes. We addressed this question in the Health Professionals Follow-up Study (HPFS), a large prospective cohort study of men, by using a composite of major CVD, cancer, and non-trauma-related death as the outcome measure.

¹From the Departments of Nutrition, Epidemiology, and Biostatistics, Harvard School of Public Health, Boston; the Channing Laboratory, Brigham and Women's Hospital and Harvard Medical School, Boston; and the Economic Research Service, US Department of Agriculture, Washington, DC.

²The views expressed in this article are those of the authors and do not necessarily reflect the views of the Economic Research Service or the US Department of Agriculture.

³Supported by research grants HL 35464 and CA55075 from the National Institutes of Health and cooperative agreement 43-3AEM-8-80062 with the Economic Research Service of the US Department of Agriculture.

⁴Address reprint requests to WC Willett, Department of Nutrition, Harvard School of Public Health, 665 Huntington Avenue, Boston, MA 02115.

⁵Address correspondence to ML McCullough, Epidemiology and Surveillance Research, The American Cancer Society, 1599 Clifton Avenue, NE, Atlanta, GA 30309. E-mail: mmccullo@cancer.org.

Received July 26, 1999.

Accepted for publication April 21, 2000.

TABLE 1

Healthy eating index (HEI) scoring criteria and HEI scores in the Continuing Survey of Food Intakes by Individuals (CSFII) and the Health Professionals Follow-up Study (HPFS)¹

Component	Criteria for maximum score of 10 ²		Criteria for minimum score of 0	CSFII (1996) ³	HPFS
	Men 19–50 y	Men ≥51 y			
				<i>HEI score</i>	<i>HEI-f score</i>
1) Grains (servings/d)	11	9.1	0	6.7 ⁴	4.7 ± 2.1 ⁵
2) Vegetables (servings/d)	5	4.2	0	6.3	8.2 ± 2.1
3) Fruit (servings/d)	4	3.2	0	3.8	6.7 ± 2.9
4) Milk (servings/d)	2	2	0	5.4	6.1 ± 2.9
5) Meat (servings/d)	2.8	2.5	0	6.4	8.5 ± 1.9
6) Total fat (% of total energy)		≤30	≥45	6.9	7.6 ± 2.6
7) Saturated fat (% of total energy)		<10	≥15	6.4	7.0 ± 3.3
8) Cholesterol (mg/d)		<300	≥450	7.9	7.7 ± 3.5
9) Sodium (mg/d)		<2400	≥4800	6.3	6.8 ± 3.5
10) Variety (no. of different food items over 3 d)		≥16	≤6	7.6	5.1 ± 3.1 ⁶
Total score ⁷				64	68.3 ± 11.5

¹Scoring criteria were adapted from Kennedy et al (9).

²Recommended servings for age and sex groups were extrapolated from recommended energy requirements (20).

³*n* = 4800 male and female Americans aged >2 y (19).

⁴ \bar{x} .

⁵ $\bar{x} \pm$ SD.

⁶Variety scored by quantile of number of unique foods consumed per month.

⁷Total score can range from 0 (worst) to 100 (best).

SUBJECTS AND METHODS

Study population

The HPFS is a prospective investigation of dietary etiologies of heart disease and cancer among 51 529 men who were aged 40–75 y in 1986. The cohort consists of US dentists (57.6%), veterinarians (19.6%), pharmacists (8.1%), optometrists (7.3%), osteopathic physicians (4.3%), and podiatrists (3.1%); health professionals were chosen for their ability to respond to health-related questionnaires with a high degree of accuracy. At the beginning of the study in 1986, participants completed a detailed dietary and medical history questionnaire sent to them by mail. Every 2 y, we send the subjects follow-up questionnaires to obtain up-to-date information on smoking habits, physical activity, weight, and other risk factors and to ask about newly diagnosed medical conditions. The study is being conducted according to the ethical guidelines of Brigham and Women's Hospital, Boston.

Dietary assessment

Dietary intake data were collected in 1986 and 1990 with a 131-item semiquantitative FFQ. For each item on the FFQ, a common serving size was specified (eg, one-half cup carrots or 1 cup milk) and participants were asked how often, on average, they consumed this serving size of the item during the previous year. The FFQ provided 9 frequency responses ranging from "never or less than once per month" to "6 or more times per day." Separate questions were asked about types of fats and oils used in cooking and the addition of salt to food during cooking and at the table. We computed nutrient intakes by multiplying the frequency of consumption of each food by the nutrient content of the specified portion and then summing the nutrient contributions from all the foods. Nutrient values were obtained from the Harvard University Food Composition Database, derived from US Department of Agriculture sources (17, 18), and supplemented with information from food manufacturers and published research.

The development, reproducibility, and validity of the FFQ were documented in detail (4, 12–14). Dietary factors assessed by the FFQ correlated with biochemical measures in blood and adipose tissue (15, 16) and predicted disease risk (4). For the validation study, the FFQ was administered twice in 1986 (at a 1-y interval) to 127 randomly selected cohort members living in the Boston area. We compared intakes from the FFQs with 2 sets of weighed, 1-wk food records that were recorded 6 mo apart. Pearson correlation coefficients between the FFQs and food records for energy-adjusted nutrients were 0.67 for total fat, 0.75 for saturated fat, 0.60 for sodium, and 0.76 for cholesterol (14); these data were corrected for week-to-week variation in food records. For individual food items, the mean Pearson correlation coefficient between the food records and FFQ, corrected for within-person weekly variation, was 0.63.

The healthy eating index

The HEI was originally developed by using 24-h-recall and food-record data from the Continuing Survey of Food Intakes by Individuals (CSFII), a nationwide survey conducted by the Agricultural Research Service of the US Department of Agriculture (19). The HEI consists of 10 equally weighted components, each representing different dietary recommendations from the food guide pyramid (7) and the 1995 *Dietary Guidelines for Americans* (6) (Table 1). Each component has a minimum score of 0 and a maximum score of 10 (for perfect adherence); intermediate degrees of adherence are calculated proportionately. The scores from the 10 components are then added to obtain a total HEI score ranging from 0 (worst) to 100 (best).

The first 5 components in Table 1 quantify adherence to serving recommendations for 5 food groups: grains (bread, cereal, rice, and pasta), vegetables, fruit, milk (milk, yogurt, and cheese), and meat (meat, poultry, fish, dry beans, eggs, and nuts) (10). The recommended numbers of servings vary by age and sex and are extrapolated from energy recommendations of the food guide pyramid. In Table 1, the serving recommendations for determining HEI scores that are applicable to the men in this study are listed. For example, the recommended number of daily

servings of grains for men ≤ 50 y is 11. Those men meeting or exceeding this goal earned a score of 10 (best) and those not consuming any grains received a score of 0. Five and one-half servings (half of the goal) earned a score of 5.

We calculated an HEI score for each cohort member in 1986 and 1990 by using their responses to the FFQs and guidelines from the *Healthy Eating Index Final Report* (20). Food items on the FFQ were assigned to their appropriate food groups after applying serving size conversion factors when appropriate to conform to the serving size definitions in the food guide pyramid. These serving size definitions were obtained from the database for the CSFII (19). Consistent with US Department of Agriculture protocol (20), recipes and foods from our nutrient database were disaggregated into their component parts and individual foods were assigned to the appropriate food groups. The average daily number of servings of foods from each food group was then totaled. Energy-adjusted food intake was not used, because such adjustment is not used in the original US Department of Agriculture HEI scoring system. This system accounts for general differences in energy requirements (eg, serving recommendations are different for different combinations of sex and age.)

Only food and recipe constituents that contributed toward the 5 food groups were counted when calculating average daily number of food servings. For example, consistent with the HEI, the potato portion of French fries (by weight) was counted toward the vegetable group and the apple and flour in apple pie were counted toward the fruit and grain groups, respectively. The fat content of these foods was considered later in the components for total fat, saturated fat, and cholesterol. Sugar content was not considered.

Values for the components total fat, saturated fat, cholesterol, and sodium were obtained from our nutrient database. Total and saturated fat intakes were expressed as percentages of total energy. Cholesterol and sodium were calculated by using raw nutrient totals; energy was controlled for in the multivariate model. For the component variety, it was necessary to use a different calculation method than was used in the original HEI, which scored variety by the number of unique food items consumed during a 3-d period. Because the FFQ is designed to measure long-term intake and contains a fixed number of food items, we scored variety as the number of unique foods consumed at least once per month. To do this, we grouped similar foods together by using HEI guidelines (20). For example, skim milk and whole milk were grouped together, whereas chicken and beef were counted separately. The total number of unique foods consumed per month (range: 23–63) among all participants was then divided into 11 quantiles to calculate cutoffs for assigning variety component scores of 0–10. For example, those subjects with the smallest number of unique foods consumed per month received a score of 0, and those with the highest number received a score of 10.

Hereafter, we refer to the HEI calculated from FFQs as the HEI-f. In the group of 127 men who also completed food records (13, 14), the Pearson correlation coefficient relating the HEI-f to the HEI computed from the food records, corrected for week-to-week variation in food records, was 0.72 (95% CI: 0.59, 0.81).

Outcome ascertainment

We identified incident diseases and deaths that occurred after subjects returned the baseline 1986 questionnaire until 1 February 1994. The primary endpoint, major chronic disease, was defined as CVD, cancer, or death not resulting from trauma, whichever came first.

CVD was defined as myocardial infarction (MI) or stroke (fatal or nonfatal for both). We asked all men who reported incident MI or stroke on their 1988, 1990, 1992, and 1994 questionnaires to confirm the report and provide permission to review their medical records. Study physicians who were unaware of the subjects' risk factor status reviewed the records. MI was confirmed by using the following World Health Organization criteria (21): compatible symptoms plus either typical electrocardiographic changes or elevation of cardiac enzymes. Stroke was confirmed if characterized by a typical neurologic defect of sudden or rapid onset, lasting ≥ 24 h, that was attributable to a cerebrovascular event (22). When medical records were unavailable, confirmation was based on other sources (such as a verbal report from the subject); this occurred for 3.9% of MI cases and 8.5% of stroke cases.

As with CVD, we sought to confirm all cancer diagnoses by using medical records, which were reviewed by physicians who were unaware of the subjects' risk factor status. A total of 10–15% of confirmations were not based on medical records but instead were based on other confirmatory evidence. We included all confirmed cancers except nonmelanoma skin cancer and low-grade, organ-confined prostate cancer (below stage C and below Gleason grade 7) (23) because of the relatively low mortality from these highly prevalent lesions and because diet appears to be more of a risk factor for more aggressive forms of prostate cancer.

All deaths except those from external causes (eg, accidents and suicides) were included in the composite major chronic disease endpoint. Deaths were reported by next of kin, coworkers, or postal authorities or through the National Death Index (24). Nonresponding participants were assumed to be alive if not listed in the National Death Index. We attempted to confirm each cause of death by using medical records or autopsy reports.

Statistical analyses

We excluded men with implausibly high or low reported energy intake (outside the range of 3360 to 17640 kJ), those who left ≥ 70 questions blank on the FFQ, and men who reported previous cancer (other than nonmelanoma skin cancer), MI, stroke, angina, other heart disease (eg, aortic stenosis, heart rhythm disturbances), diabetes, or renal disease. After all exclusions, 38622 men remained.

Each participant contributed follow-up time lasting from the return of his baseline questionnaire in 1986 until either the diagnosis of CVD or cancer, death, or February 1, 1994. Confirmed cases were excluded from subsequent follow-up; thus, each person could contribute only one diagnosed CVD or cancer endpoint to the analysis and the cohort at risk included only those free of disease at the beginning of each 2-y follow-up interval. Overall follow-up, on the basis of eligible person-years through 1994, was 97% complete.

HEI-f quintiles were defined by using a cumulative average scoring method (25). This method used repeated measures of diet to capture long-term intake and to reduce measurement error due to intraindividual variation in diet over time (25). With this method, quintiles of the 1986 HEI-f score were used to predict outcome during the period 1986–1990 and quintiles derived from an average of the 1986 and 1990 HEI-f scores were related to outcome from 1990 to 1994. If no questionnaire was completed in 1990, the 1986 HEI-f score was carried forward. Because intentional changes in diet resulting from the diagnosis of angina, hypercholesterolemia, diabetes, and hypertension may confound the associations between diet and disease, in the

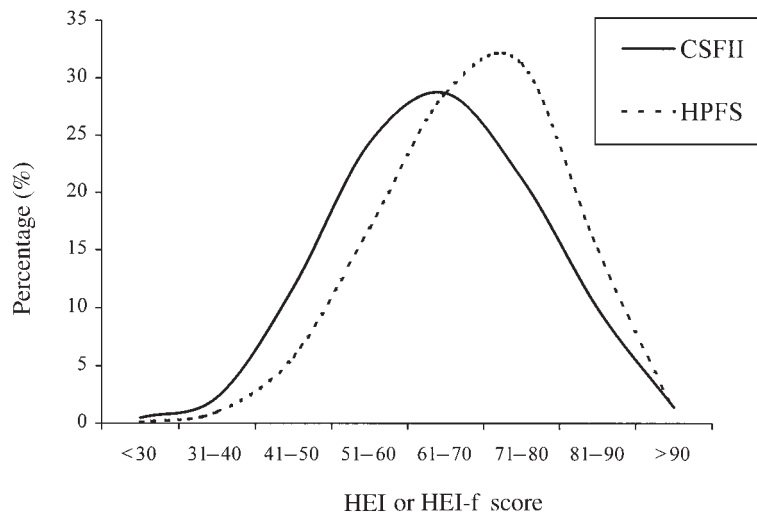


FIGURE 1. Distribution of total healthy eating index scores calculated from food-frequency questionnaires (HEI-f) in the Health Professionals Follow-up Study (HPFS) cohort compared with HEI scores from the Continuing Survey of Food Intakes by Individuals (CSFII) population (19).

primary analysis we did not update diet for men who reported these conditions between 1986 and 1990. Quintile cutoffs were computed separately for the 2 time periods. The trend test was computed by using the median quintile value from the average of all interval scores and was modeled as a continuous regression variable. Because long-term diet is relevant in analyses of diet and chronic disease, we also examined baseline HEI-f scores in relation to all outcomes.

We calculated relative risks (RRs) as the incidence of major chronic disease, CVD, or cancer among men in each quintile of HEI-f score divided by the incidence for those in the lowest quintile of HEI-f score, adjusting for age. To adjust for ≥ 2 covariates, we used pooled logistic regression (26) with 2-y time increments to calculate odds ratios as estimates of RRs. This approach is used regularly in prospective cohort analyses with repeated measures of exposure and was shown to closely approximate Cox regression analysis with time-dependent covariates (27).

In addition to analysis by quintiles of HEI-f, we examined each outcome according to US Department of Agriculture–defined HEI categories of poor (< 51), needs improvement (51–80), and good (> 80). To examine the influence of individual HEI-f components on the incidence of major chronic disease, CVD, and cancer, each of the 10 components was entered individually into the final multivariate model as a continuous (0–10) variable. We then conducted stepwise regression to determine which HEI-f components were most strongly and independently associated with the outcomes of interest. We tested for interactions by stratifying according to age ($<$ or ≥ 60 y), smoking (never, past, or current), and multivitamin use (yes or no). To formally test for interactions, models with and without interaction terms were compared by using likelihood ratio tests (28).

Criteria for inclusion of covariates in the model included a priori knowledge of risk factors in this cohort or a change of $> 10\%$ in the β -coefficient for the HEI-f outcome association. Self-reported nondietary covariates, including age (in 5-y categories), leisure time physical activity (in metabolic equivalents), cigarette smoking (never, past, or current amount smoked), and body mass index in kg/m^2 (quintiles) were updated every 2 y. Total energy intake and alcohol consumption (both in quintiles)

were assessed at baseline and in 1990. In general, the same baseline exclusions were used for each outcome and the same covariates were included in the final models. Exceptions were that the diagnoses of hypercholesterolemia and hypertension (both binary) at baseline were included in multivariate models for major chronic disease and CVD outcomes only, and use of multivitamins (yes or no) and vitamin E [yes (defined as ≥ 100 IU/d) or no] were included in the CVD models only. All reported *P* values are two-tailed. We present RRs and 95% CIs.

RESULTS

During the period 1986–1994, we documented 3119 major chronic disease endpoints. These included 1092 CVD events, 1661 cancers, and 366 deaths not resulting from CVD, cancer, or trauma.

The mean total HEI-f score from the cumulatively updated diet measure was 68.3 ± 11.5 (range: 21.6–97.4) and the 10th and 90th percentiles were 52.7 and 82.6, respectively. On average, participants came closest to meeting the recommendation for the meat group, with a mean (\pm SD) score of 8.5 ± 1.9 . The food group with the lowest average score was grains (4.7 ± 2.1) (Table 1). The SDs indicated substantial variation for all component scores within the cohort. Because the HEI-f score for variety was determined on the basis of the distribution in the HPFS cohort, the mean score was predetermined to be 5.0. As shown in Table 1, the mean total HEI-f score in this cohort was comparable to the total HEI scores in the CSFII (10), although scores in the HPFS were higher for some components (eg, fruit, vegetables, and meat) and lower for others (eg, grains and variety). The distribution of total HEI-f scores in the HPFS cohort was shifted slightly toward higher values but overlapped considerably with scores in the CSFII population, which included men, women, and children (Figure 1).

Baseline characteristics of the HPFS cohort are shown according to HEI-f quintiles in Table 2. Higher HEI-f scores were generally associated with other healthful lifestyle behaviors: subjects with high scores exercised more, were less likely to smoke, and were more likely to take multivitamin

TABLE 2

Baseline (1986) diet and lifestyle characteristics and risk factors by quintile of healthy eating index score calculated from food-frequency questionnaires (HEI-f) for 38 622 male health professionals¹

	HEI-f quintile				
	1	2	3	4	5
HEI-f score	52.2 (21.6–58.2) ²	62.4 (58.2–66.0)	69.2 (66.0–72.2)	75.4 (72.3–78.7)	82.8 (78.7–97.4)
Age (y)	51.7 ± 9.1 ³	52.4 ± 9.2	53.1 ± 9.4	54.0 ± 9.5	54.7 ± 9.4
BMI (in kg/m ²)	26.0 ± 3.5	25.7 ± 3.5	25.4 ± 3.2	25.3 ± 3.2	24.9 ± 3.1
Physical activity (METs/d) ⁴	14.7 ± 21.0	17.5 ± 23.1	19.8 ± 24.3	22.5 ± 28.1	26.8 ± 29.3
Current smoker (%)	18	12	9	6	4
Current multivitamin user (%)	37	38	41	45	47
Current vitamin E user (≥100 IU) (%)	8	9	11	13	15
Lives alone (%)	8	6	6	5	5
Diagnosis of hypertension (%)	17	18	19	19	19
Diagnosis of high cholesterol (%)	7	8	10	11	12
Has a routine physical (%) ⁵	45	50	51	53	56
Has a routine cholesterol test (%) ⁵	48	53	54	57	61
Contributors to HEI score					
Milk (servings/d)	1.4 ± 1.3	1.4 ± 1.1	1.4 ± 1.1	1.6 ± 1.1	1.8 ± 1.1
Fruit (servings/d)	1.4 ± 1.1	2.1 ± 1.4	2.8 ± 1.8	3.5 ± 1.9	4.5 ± 2.2
Vegetables (servings/d)	3.7 ± 2.0	4.1 ± 2.0	4.4 ± 2.1	4.9 ± 2.2	5.4 ± 2.1
Grains (servings/d)	3.7 ± 1.9	4.3 ± 2.1	4.7 ± 2.2	5.0 ± 2.2	5.6 ± 2.3
Meats (servings/d)	2.9 ± 1.4	2.7 ± 1.3	2.7 ± 2.2	2.7 ± 1.7	2.7 ± 1.1
Variety (no. of unique foods/mo)	37 ± 8	42 ± 8	44 ± 8	47 ± 7	51 ± 7
Total fat (% of energy)	38.4 ± 5.2	33.8 ± 5.0	31.2 ± 4.9	29.5 ± 4.7	27.1 ± 4.4
Saturated fat (% of energy)	13.9 ± 2.5	11.8 ± 2.2	10.6 ± 2.1	9.8 ± 1.9	8.9 ± 1.7
Sodium (mg/d)	3465 ± 1764	3313 ± 1643	3237 ± 1549	3180 ± 1417	3012 ± 1115
Cholesterol (mg/d)	357 ± 183	313 ± 150	291 ± 128	279 ± 108	265 ± 77
Other dietary components					
Alcohol intake (g/d)	10.3 ± 14.5	12.5 ± 16.9	12.0 ± 16.9	11.6 ± 15.4	10.4 ± 13.6
Total energy intake (kJ)	7920 ± 2840	8104 ± 2745	8238 ± 2623	8502 ± 2431	9012 ± 2113
Monounsaturated fat (% of energy)	14.8 ± 2.3	13.0 ± 2.3	11.9 ± 2.2	11.2 ± 2.1	10.2 ± 2.0
Polyunsaturated fat (% of energy)	6.3 ± 1.8	6.0 ± 1.6	5.9 ± 1.5	5.8 ± 1.4	5.5 ± 1.3
Dietary fiber (g/d)	14.9 ± 6.2	17.8 ± 6.9	20.3 ± 7.8	23.2 ± 8.4	27.5 ± 9.5
Glycemic load ⁶	99 ± 42	114 ± 44	124 ± 46	134 ± 45	151 ± 46

¹Test for trend was significant at $P < 0.001$ for all variables except alcohol intake and diagnosis of hypertension for which $P < 0.05$.

²Median with range in parentheses.

³ $\bar{x} \pm$ SD.

⁴Metabolic equivalents (METs) are defined for each type of physical activity as a multiple of the metabolic equivalent of sitting quietly for 1 h [eg, a subject who jogs (7 METs/h) 1 h/d 3 times/wk would have an MET score of 21 for that activity.]

⁵From 1988 questionnaire.

⁶An indicator of blood glucose response induced by total carbohydrate intake.

supplements and supplemental vitamin E. In addition, energy consumption was higher and body mass index was slightly lower in the higher HEI-f quintiles. Subjects in the higher quintiles were slightly older, were more likely to seek routine physical exams and blood cholesterol tests, and were more likely to have been diagnosed with hypercholesterolemia. Of the dietary components that contributed to the total HEI-f score, meat and milk intake remained relatively constant across the quintiles whereas all other components increased or decreased in the expected direction. Other dietary factors that varied with HEI-f score included dietary fiber (positively associated) and monounsaturated fat (negatively associated). Glycemic load, which represents intake of foods that elicit an increased plasma glucose response relative to defined quantities of carbohydrate (29), was positively associated with HEI-f score. Polyunsaturated fat intake did not vary markedly with HEI-f score.

Comparing men in the highest HEI-f quintile with those in the lowest quintile, the age-adjusted RR of major chronic dis-

ease was 0.74 (95% CI: 0.67, 0.83) (**Table 3**). After controlling for risk factors other than diet (full multivariate-adjusted model), the association was substantially weakened (RR = 0.89; 95% CI: 0.79, 1.00), primarily as a result of confounding by smoking (as indicated by the model adjusted for smoking only). After excluding the first 2 y of follow-up to remove those subjects who may have had preexisting disease, the association between HEI-f score and risk of major chronic disease was slightly more inverse (RR = 0.83; 95% CI: 0.72, 0.95). In analyses stratified by smoking status (never, past, or current), age (< or ≥60 y), or current multivitamin use (yes or no), the association between the HEI-f score and risk of major chronic disease was similar for the different categories (eg, age < or ≥60 y). The results were similar when body mass index was not included in the model (RR = 0.88; 95% CI: 0.78, 0.99 for men in the highest quintile compared with those in the lowest) and when we continued updating the dietary data if a participant was diagnosed with hypertension or hypercholesterolemia (RR = 0.87; 95% CI: 0.77, 0.99).

TABLE 3

Relative risk (RR) of major chronic disease by quintile of healthy eating index score calculated from food-frequency questionnaires (HEI-f) for 38622 men enrolled in the Health Professionals Follow-up Study (1986–1994)¹

	HEI-f quintile					<i>P</i> for trend ²
	1	2	3	4	5	
Person-years	63080	57978	54759	53523	51548	—
Major chronic disease (no. cases)	764	630	592	539	594	—
Age-adjusted RR	1.0	0.84 (0.76, 0.94) ³	0.78 (0.70, 0.87)	0.68 (0.61, 0.76)	0.74 (0.67, 0.83)	<0.001
Multivariate analyses						
Adjusted for smoking ⁴	1.0	0.93 (0.83, 1.04)	0.85 (0.76, 0.95)	0.75 (0.67, 0.84)	0.84 (0.75, 0.94)	<0.001
Full multivariate adjusted ⁵	1.0	0.95 (0.85, 1.06)	0.87 (0.78, 0.97)	0.77 (0.69, 0.87)	0.89 (0.79, 1.00)	<0.001
Full multivariate adjusted, 1988–1994 ^{5,6}	1.0	0.98 (0.86, 1.11)	0.88 (0.78, 1.01)	0.76 (0.66, 0.87)	0.83 (0.72, 0.95)	<0.001

¹Major chronic disease was defined as cardiovascular disease ($n = 1092$), cancer ($n = 1661$), or death from non-trauma-related causes ($n = 366$), whichever came first.

²Test for trend over the quintiles of HEI-f when the median value per quintile was used.

³95% CI in parentheses.

⁴Adjusted for age (5-y categories), smoking (never, past, 1–14 cigarettes/d, 15–24 cigarettes/d, ≥ 25 cigarettes/d), and time period.

⁵Adjusted for variables listed in footnote 4 plus body mass index (quintiles), alcohol intake (7 categories), physical activity (6 categories of metabolic equivalents), history of hypertension or hypercholesterolemia at baseline, and total energy intake (quintiles).

⁶Chronic disease endpoints from 1986–1988 were excluded.

The relations between HEI-f results and incidences of CVD and cancer as specific endpoints are summarized in **Table 4**. Men in the highest HEI-f quintile compared with those in the lowest quintile had a 28% lower risk of CVD (RR = 0.72; 95% CI: 0.60, 0.88). For cancer, a slightly positive, although not significant, association was observed for those in the top HEI-f quintile (RR = 1.12; 95% CI: 0.95, 1.31).

We also examined chronic disease risk in relation to the pre-defined US Department of Agriculture classifications for HEI scores (**Table 5**). When the category of good scores was used as the reference, those with poor scores had a slight increase in risk of major chronic disease (RR = 1.17; 95% CI: 1.00, 1.37) and an increased risk of CVD (RR = 1.36; 95% CI: 1.06, 1.74). Men in the needs-improvement category were not at increased risk.

In **Table 6** we present the risk of major chronic disease that is associated with a 5-point increase in the HEI-f components, with each component considered individually in the final multivariate model. Higher saturated fat and total fat scores (representing lower intakes) and higher fruit scores (representing more servings) were all individually related to reduced risk of major chronic disease ($P < 0.05$). Other HEI-f components were not associated with risk. In stepwise regression analyses controlling for energy intake, alcohol intake, and nondietary risk factors, saturated fat was the only HEI-f component that remained in the final model for major chronic disease risk. For a 5-point increase in saturated fat score, indicating lower saturated fat intake, RR = 0.91 (95% CI: 0.86, 0.96). In stepwise analyses of CVD risk, both the fruit and saturated fat components remained in the model. For a 5-point

TABLE 4

Relative risk (RR) of cardiovascular disease (CVD) and cancer by quintiles of healthy eating index score calculated from food-frequency questionnaires (HEI-f) for 38622 men enrolled in the Health Professionals Follow-up Study (1986–1994)

	HEI-f quintile					<i>P</i> for trend ¹
	1	2	3	4	5	
CVD ²						
Person-years	64555	59277	56040	54653	52867	—
Total no. cases	325	235	205	178	196	—
Age-adjusted RR	1.0	0.75 (0.63, 0.89) ³	0.66 (0.55, 0.78)	0.55 (0.46, 0.66)	0.59 (0.49, 0.71)	<0.001
Multivariate adjusted RR ⁴	1.0	0.86 (0.73, 1.03)	0.74 (0.62, 0.89)	0.63 (0.52, 0.76)	0.72 (0.60, 0.88)	<0.001
Cancer ⁵						
Person-years	64570	59095	55587	54321	52364	—
Total no. cases	350	321	346	319	360	—
Age-adjusted RR	1.0	0.94 (0.81, 1.10)	1.01 (0.87, 1.18)	0.88 (0.76, 1.03)	0.99 (0.86, 1.15)	0.79
Multivariate-adjusted RR	1.0	1.01 (0.86, 1.18)	1.06 (0.91, 1.24)	0.99 (0.85, 1.17)	1.12 (0.95, 1.31)	0.27

¹Test for trend over the quintiles of HEI-f when the median value per quintile was used.

²CVD ($n = 1139$) was defined as fatal and nonfatal myocardial infarction and stroke.

³95% CI in parentheses.

⁴Controlled for age (5-y categories), body mass index (quintiles), smoking (never, past, 1–14 cigarettes/d, 15–24 cigarettes/d, ≥ 25 cigarettes/d), alcohol intake (7 categories), physical activity (6 categories), total energy intake (quintiles), time period, multivitamin use, vitamin E use, and diagnosis of hypercholesterolemia and hypertension at baseline.

⁵Cancer ($n = 1702$) was defined as all cancers except nonaggressive prostate cancer (<stage C and <grade 7) and nonmelanoma skin cancer; the multivariate model includes the same covariates as the CVD model except it does not include multivitamin use, vitamin E use, and diagnosis of hypertension or hypercholesterolemia at baseline.

TABLE 5

Risk of major chronic disease, cardiovascular disease (CVD), and cancer according to US Department of Agriculture classification of healthy eating index (HEI) scores

	HEI score >80: good	HEI score of 51–80: needs improvement	HEI score <51: poor
Major chronic disease ¹	1.0	0.96 (0.87, 1.06) ²	1.17 (1.00, 1.37)
CVD ³	1.0	1.05 (0.88, 1.24)	1.36 (1.06, 1.74)
Cancer ⁴	1.0	0.88 (0.78, 1.01)	1.04 (0.84, 1.29)

¹Adjusted for age, body mass index, smoking, alcohol intake, energy intake, physical activity, time period, and diagnosis of hypertension or hypercholesterolemia at baseline.

²Relative risk with 95% CI in parentheses.

³Adjusted for the same variables as the major chronic disease model and also controlled for multivitamin and vitamin E use.

⁴Adjusted for the same variables as the major chronic disease model, except for multivitamin and vitamin E use and diagnosis of hypertension or hypercholesterolemia at baseline.

increase, the fruit component was associated with reduced risk of CVD (RR = 0.87; 95% CI: 0.77, 0.98), as was the saturated fat component (RR = 0.91; 95% CI: 0.83, 1.00). For cancer, no individual HEI components were associated with risk.

DISCUSSION

Although the food guide pyramid has been evaluated as a teaching tool for the reduction of chronic disease risk factors through diet (30), little is known about the effect of adopting the dietary guidelines on actual chronic disease incidence. The HEI, a measure of diet quality based on the *Dietary Guidelines for Americans* and the food guide pyramid, offers an approach for evaluating these relations. In this large, prospective study in men, we found only a weak inverse association between an estimate of the HEI score (HEI-f) and the risk of major chronic disease, after accounting for substantial confounding by smoking. Men in the highest HEI-f quintile were 11% less likely to develop CVD or cancer or to die from other non-trauma-related causes than were those in the lowest HEI-f quintile. This slight protective relation with overall chronic disease risk appears to have been driven mainly by a lower risk of CVD, rather than a lower risk of cancer. Men in the highest HEI-f quintile compared with those in the lowest quintile had a 28% lower risk of CVD, but there was no association between HEI-f score and cancer incidence.

Several factors may explain the stronger association observed for CVD than for cancer. In general, more HEI components have been consistently associated with reduced CVD or CVD risk factors than with cancer, because more is known about dietary risk factors for CVD. Also, the duration of the study may not have captured the appropriate latency period to detect an association between diet and overall cancer risk. However, the results were unchanged whether baseline or cumulative diet was used in the analyses, and associations between specific dietary factors and cancer were previously detected during this 8-y follow-up period (31–33).

One possible explanation for the weak association between the HEI-f score and overall chronic disease risk might be that dietary intake was measured poorly by the methods that we used; measurement errors would tend to underestimate associations between diet and disease. However, for the foods and nutrients included in the HEI, the FFQ we used was shown to rank individuals similarly using different assessment methods (12–16) so that important associations would not be missed in a study of this size. In this same population, controlling for similar confounders, various aspects of diet clearly predicted the incidence of heart disease and cancer (31–35). Further, in the

age-adjusted analysis, the HEI-f did predict major chronic disease outcome, indicating that the dietary questionnaire can capture individual intake and can predict disease outcome. The weak association in the multivariate analysis is therefore not a result of an inability of the FFQ to measure individual differences in diet, but rather a result of associations between these differences in diet and smoking and other lifestyle variables that are strong risk factors for chronic disease.

The HEI was originally developed by using 3 d of dietary intake results (2 food records and one 24-h recall) from the CSFII (19). Because FFQs are designed to measure long-term intake and necessarily contain a fixed list of foods, one may anticipate that HEI scores would vary greatly between methods. We did not find this to be the case, however, and our scores were generally comparable with those reported for CSFII participants. In addition, the HEI-f correlated well with the HEI calculated from two 1-wk food records in a subset of the HPPS population. Furthermore, our method of calculating the variety component was apparently not a limitation because our approach yielded variety scores that were inversely associated with disease outcome.

Another possible explanation for our modest findings is that important food components are not represented by the HEI and HEI-f, or that some components of the HEI are not important in relation to major chronic disease risk. Although our findings for CVD were in the expected direction, it seems certain that some HEI-f components could be refined and improved in several ways to strengthen the association of the HEI with major chronic

TABLE 6


Relation of a 5-point increase in individual healthy eating index components to risk of major chronic disease

Diet component	Amount represented by higher score	Risk of major chronic disease ¹
Milk	More servings	1.03 (0.96, 1.10)
Fruit	More servings	0.93 (0.87, 1.00)
Vegetable	More servings	0.91 (0.83, 1.01)
Grains	More servings	0.95 (0.85, 1.06)
Meats	More servings	1.00 (0.89, 1.12)
Total fat	Less total fat	0.92 (0.86, 0.99)
Saturated fat	Less saturated fat	0.91 (0.86, 0.96)
Cholesterol	Less cholesterol	0.95 (0.89, 1.01)
Sodium	Less sodium	0.99 (0.93, 1.06)
Variety	More variety	0.94 (0.88, 1.00)

¹Relative risk (RR) with 95% CI in parentheses. Each component was added individually into the multivariate model, adjusting for age, smoking, body mass index, alcohol intake, physical activity, diagnosis of hypertension or hypercholesterolemia at baseline, total energy intake, and time period.

disease outcomes. For example, the dietary guidelines consider total fat intake >30% of energy to be detrimental, regardless of the fat source. This limitation on all types of fat ignores the benefits of unsaturated fats (36–39). In general, total fat intake has not been associated with increased cancer risk independent of total energy intake (32, 33, 40, 41). Also, low-fat, high-carbohydrate diets can have adverse metabolic effects (42–44) and neither the original HEI nor the HEI-f distinguish between differences in refinement of grains, which can have important effects on the risk of heart disease and type 2 diabetes (45, 46). Potato consumption makes an important contribution to the vegetable score in the HEI-f ($\approx 28\%$ of the vegetable score in this cohort), and the evidence does not support an inverse relation between potato consumption and risk of any type of cancer (2, 47). Further, the meat group in the food guide pyramid includes red meat, which was associated with CVD and several types of cancer (32, 33, 48–50), together with foods that were associated with reduced disease risk (eg, fish and nuts) (33, 51). The saturated fat component of the HEI-f score had the strongest independent inverse association with major chronic disease outcome (a higher score represents lower saturated fat intake) and both the saturated fat and fruit components of the HEI-f were independently associated with a reduced risk of CVD. This suggests that the HEI might be more predictive if certain components were eliminated or revised. Future analyses that would use modifications of the HEI could address these issues.

Our cohort was well educated, had a relatively homogeneous socioeconomic status, and was mostly white. However, this did not appear to reduce the variation in the HEI-f scores. This lack of diversity has the advantage of reducing confounding by variables that are related to socioeconomic status and that are difficult to control. However, because the response of some chronic disease risk factors to dietary interventions may vary by race (52), these analyses should be replicated in other populations.

In summary, a diet pattern reflecting concordance with the 1995 *Dietary Guidelines for Americans* and the food guide pyramid, as assessed by the HEI-f, was only weakly inversely associated with overall risk of major chronic disease outcomes. Because various aspects of diet that were previously shown to influence risk of cancer or CVD are not represented by the HEI, these findings suggest that the *Dietary Guidelines for Americans* and the food guide pyramid, on which the HEI is based, may need to be redesigned. Future dietary guidelines should be evaluated for their efficacy in reducing chronic disease risk. 

We are indebted to the participants of the Health Professionals Follow-up Study for their continued dedication to this project. We thank Frank Hu for his thoughtful feedback and suggestions and Al Wing and Karen Corsano for computer assistance. Mitzi Wolff, Mira Kaufman, Betsy Frost-Hawes, and Kerry Demers made invaluable contributions to the maintenance of the HPFS database and Laura Sampson and Paula Tocco contributed food composition data for HEI-f tabulation. We are also grateful for help with HEI coding provided by Peter Basiotis and Katie Fleming.

REFERENCES

- Committee on Diet and Health, National Research Council. Diet and health: implications for reducing chronic disease risk. Washington, DC: National Academy Press, 1989.
- World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research, 1997.
- National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. Chronic diseases and their risk factors, the nation's leading causes of death. December 1999, section 1. World Wide Web: <http://www.cdc.gov/nccd-php/statbook/statbook.htm> (accessed 31 July 2000).
- Willett WC. Nutritional epidemiology. 2nd ed. New York: Oxford University Press, 1998.
- Kant AK. Indexes of overall diet quality: a review. *J Am Diet Assoc* 1996;96:785–91.
- US Department of Health and Human Services, US Department of Agriculture. Dietary guidelines for Americans. 4th ed. Washington, DC: US Government Printing Office, 1995.
- US Department of Agriculture. The food guide pyramid. Hyattsville, MD: Human Nutrition Information Service, 1992. (Publication HG252.)
- Kennedy E, Meyers L, Layden W. The 1995 dietary guidelines for Americans: an overview. *J Am Diet Assoc* 1996;96:234–7.
- Kennedy ET, Ohls J, Carlson S, Fleming K. The healthy eating index: design and applications. *J Am Diet Assoc* 1995;95:1103–8.
- Bowman SA, Lino M, Gerrior SA, Basiotis PP. The healthy eating index: 1994–1996. Washington, DC: US Department of Agriculture, Center for Nutrition Policy and Promotion, 1998.
- Variyam J, Blaylock J, Smallwood D. USDA's healthy eating index and nutrition information. Washington, DC: Economic Research Service, US Department of Agriculture, 1998. (Technical Bulletin no. 1866.)
- Willett WC, Sampson L, Stampfer MJ, et al. Reproducibility and validity of a semiquantitative food frequency questionnaire. *Am J Epidemiol* 1985;122:51–65.
- Feskanih D, Rimm EB, Giovannucci EL, et al. Reproducibility and validity of food intake measurements from a semiquantitative food frequency questionnaire. *J Am Diet Assoc* 1993;93:790–6.
- Rimm EB, Giovannucci EL, Stampfer MJ, Colditz GA, Litin LB, Willett WC. Reproducibility and validity of an expanded self-administered semiquantitative food frequency questionnaire among male health professionals. *Am J Epidemiol* 1992;135:1114–26.
- Michaud DS, Giovannucci EL, Ascherio A, et al. Associations of plasma carotenoid concentrations and dietary intake of specific carotenoids in samples of two prospective cohort studies using a new carotenoid database. *Cancer Epidemiol Biomarkers Prev* 1998;7:283–90.
- Hunter DJ, Rimm EB, Sacks FM, et al. Comparison of measures of fatty acid intake by subcutaneous fat aspirate, food frequency questionnaire, and diet records in a free-living population of US men. *Am J Epidemiol* 1992;135:418–27.
- Adams CF. Nutritive value of American foods in common units. Agricultural handbook no. 456. Washington, DC: Agricultural Research Service, US Department of Agriculture, 1975.
- Consumer and Food Economic Institute. Composition of foods. Agriculture handbook no. 8 series, 1976–1989. Washington, DC: US Government Printing Office, 1989.
- US Department of Agriculture, Agricultural Research Service. 1995–1996 Continuing Survey of Food Intakes by Individuals and 1994–1996 Diet and Health Knowledge Survey and related materials. Washington, DC: US Department of Agriculture, Agricultural Research Service, 1998. (CD-ROM.)
- Kennedy E, Ohls J, Carlson S, Fleming K. The healthy eating index final report. Alexandria, VA: Food and Nutrition Service, US Department of Agriculture, 1994. (contract no. 53-3198-3-0381994 submitted by Mathematica Policy Research Inc, Princeton, NJ, PR 94-65.)
- Rose GA, Blackburn H. Cardiovascular survey methods. *World Health Organ Monogr Ser* 1982;58.
- Walker AE, Robins M, Weinfeld FD. The National Survey of Stroke. Clinical findings. *Stroke* 1981;12(suppl):I13–44.
- Catalona WJ, Aviola LV. Diagnosis, staging, and surgical management of prostatic carcinoma. *Arch Intern Med* 1987;147:361–3.
- Stampfer MJ, Willett WC, Speizer FE, et al. Test of the National Death Index. *Am J Epidemiol* 1984;119:837–9.



25. Hu FB, Stampfer MJ, Rimm E, 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.
26. Cupples LA, D'Agostino RB, Anderson K, Kannel WB. Comparison of baseline and repeated measure covariate techniques in the Framingham Heart Study. *Stat Med* 1988;7:205–22.
27. D'Agostino RB, Lee MLT, 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.
28. Hosmer D, Lemeshow S. *Applied logistic regression*. New York: John Wiley and Sons, 1989.
29. Jenkins DJ, Wolever TM, Taylor RH, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* 1981;34:362–6.
30. Gambera PJ, Schneeman BO, Davis PA. Use of the food guide pyramid and US dietary guidelines to improve dietary intake and reduce cardiovascular risk in active-duty Air Force members. *J Am Diet Assoc* 1995;95:1268–73.
31. Giovannucci E, Ascherio A, Rimm EB, Stampfer MJ, Colditz GA, Willett WC. Intake of carotenoids and retinol in relation to risk of prostate cancer. *J Natl Cancer Inst* 1995;87:1767–76.
32. Giovannucci E, Rimm EB, Colditz GA, et al. A prospective study of dietary fat and risk of prostate cancer. *J Natl Cancer Inst* 1993;85:1571–9.
33. Giovannucci E, Rimm EB, Stampfer MJ, Colditz GA, Ascherio A, Willett WC. Intake of fat, meat, and fiber in relation to risk of colon cancer in men. *Cancer Res* 1994;54:2390–7.
34. Rimm EB, Ascherio A, Giovannucci E, Spiegelman D, Stampfer MJ, Willett WC. Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. *JAMA* 1996;275:447–51.
35. Rimm EB, Willett WC, Hu FB, et al. Folate and vitamin B₆ from diet and supplements in relation to risk of coronary heart disease among women. *JAMA* 1998;279:359–64.
36. Hu FB, Stampfer MJ, Manson JE, et al. Dietary intake of alpha-linolenic acid and risk of fatal ischemic heart disease among women. *Am J Clin Nutr* 1999;69:890–7.
37. Sacks FM, Willett WC. More on chewing the fat—the good fat and the good cholesterol. *N Engl J Med* 1991;325:1740–2.
38. Siguel EN, Lerman RH. Role of essential fatty acids: dangers in the US Department of Agriculture dietary recommendations (“pyramid”) and in low-fat diets. *Am J Clin Nutr* 1994;60:973–4.
39. Kennedy E, Shaw A, Davis C. Essential fatty acids and USDA's food guide pyramid. *Am J Clin Nutr* 1995;62:645–7.
40. Willett WC. Diet and health: what should we eat? *Science* 1994;264:532–7.
41. Howe GR, Aronson KJ, Benito E, et al. The relationship between dietary fat intake and risk of colorectal cancer—evidence from the combined analysis of 13 case-control studies. *Cancer Causes Control* 1997;8:215–28.
42. Reaven GM. Looking at the world through LDL-cholesterol-colored glasses. *J Nutr* 1986;116:1143–7.
43. Mensink RP, Katan MB. Effect of monounsaturated fatty acids versus complex carbohydrates on high-density lipoprotein in healthy men and women. *Lancet* 1987;1:122–5.
44. Katan MB, Grundy SM, Willett WC. Should a low-fat, high-carbohydrate diet be recommended for everyone? Beyond low-fat diets. *N Engl J Med* 1997;337:563–7.
45. 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.
46. Salmeron 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.
47. Steinmetz KA, Potter JD. Vegetables, fruit and cancer. I. Epidemiology. *Cancer Causes Control* 1991;2:325–57.
48. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Speizer FE. Relation of meat, fat, and fiber intake to the risk of colon cancer in a prospective study among women. *N Engl J Med* 1990;323:1664–72.
49. Kune S, Kune GA, Watson LF. Case-control study of dietary etiologic factors: the Melbourne Colorectal Cancer Study. *Nutr Cancer* 1987;9:21–42.
50. Le Marchand L, Kolonel LN, Wilkens LR, Myers BC, Hirohata T. Animal fat consumption and prostate cancer: a prospective study in Hawaii. *Epidemiology* 1994;5:276–82.
51. Hu F, Stampfer MJ, Manson JE, et al. Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study. *BMJ* 1998;317:1341–5.
52. Svetkey LP, Simons-Morton D, Vollmer WM, et al. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. *Arch Intern Med* 1999;159:285–93.

