Diet quality indexes and mortality in postmenopausal women: the Iowa Women's Health Study[1–4]

Jaakko Mursu, Lyn M Steffen, Katie A Meyer, Daniel Duprez, and David R Jacobs Jr

ABSTRACT

Background: A priori diet scores such as the Alternative Healthy Eating Index (AHEI) and the food-based a priori diet quality score predict chronic disease risk.

Objective: We compared the AHEI and a priori diet quality score relative to mortality.

Design: Postmenopausal women who were free of diabetes, cardiovascular disease (CVD), and cancer in the Iowa Women's Health Study (in 1986, n = 29,634 with a mean ± SD age of 61.4 ± 4.2 y; in 2004, n = 15,076 with a mean ± SD age of 79.7 ± 4.0 y). A food-frequency questionnaire was used. Through 31 December 2008, 10,343 total, 3646 CVD, 3207 cancer, and 2888 inflammatory-related deaths were identified through record linkage. HRs were computed for quartiles of each diet score at baseline and 2004. To compare scores, the residual of each score given the other score was computed by using linear regression.

Results: At baseline, indexes had a correlation of 0.65. For the AHEI, the multivariable-adjusted HRs (95% CIs) for total, CVD, cancer, and inflammatory-related mortality were 0.82 (0.77, 0.87), 0.79 (0.72, 0.88), 0.88 (0.79, 0.98), and 0.76 (0.68, 0.84), respectively. The a priori score had corresponding HRs of 0.80 (0.76, 0.85), 0.79 (0.72, 0.88), 0.86 (0.77, 0.95), and 0.75 (0.67, 0.84), respectively. Each score added information to the other score for total, CVD mortality, and inflammatory-related mortality. In 2004, both scores predicted total, CVD, and inflammatory-related mortality, and the a priori score also predicted cancer mortality. The a priori score added independent information for all outcomes except cancer, whereas the AHEI added information only for total mortality.

Conclusion: Two correlated diet quality scores predicted total and disease-specific mortality, but their residuals also predicted complementarily. *Am J Clin Nutr* 2013;98:444–53.

INTRODUCTION

Chronic diseases such as cardiovascular disease (CVD)[5] and cancer remain the main causes of premature death in Western countries (1). A substantial proportion of these deaths may be preventable by a healthy diet (2–4). Studies that assessed the role of nutrition in chronic diseases have focused on specific components of foods, often single nutrients. Although these studies have provided evidence about the role of deficient vitamins and minerals in human health, this approach has limitations when applied in generally sufficient diets. People do not eat isolated nutrients but, rather, foods with complex combinations of nutrients that may have interactive or synergistic properties embedded in dietary patterns (2, 5). Also, because nutritional compounds are often intercorrelated, the effect of a single nutrient is challenging to isolate. Recent studies have shifted focus to food groups and dietary patterns, which are fundamental units of diet that directly provide practical information to prevent chronic diseases.

Food patterns defined a priori such as the Alternative Healthy Eating Index (AHEI) (6, 7) and the Mediterranean diet score (8) have been shown to predict total and CVD morbidity and mortality (8–10) in observational studies, although for cancer, the risk reduction has been more modest (7, 10). We have created a pattern called the a priori diet quality score, which has been shown to predict myocardial infarction (11), type 2 diabetes (12), and several markers of CVD (13, 14). One novelty of our diet quality score is that it is based solely on foods rather than nutrients and, thus, is practical for the translation to dietary recommendations. It is also exhaustive, including all foods listed on the food-frequency questionnaire (FFQ), which enables complete analyses of dietary factors as risk factors for chronic diseases.

The aim of our study was to assess the relations of the AHEI (7) and food-based a priori diet quality score (11–14) to total and disease-specific mortality in older women of the Iowa Women's Health Study (IWHS). Because the literature on which the creators of the scores implicitly relied was largely based on

[1] From the Divisions of Epidemiology and Community Health (JM, LMS, KAM, and DRJ) and Cardiology, Department of Medicine (DD), University of Minnesota, Minneapolis, MN; the Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland (JM); and the Department of Nutrition, School of Medicine, University of Oslo, Oslo, Norway (DRJ).

[2] Sponsors did not play a role in the conception, design, or conduct of the study; collection, management, analysis, and interpretation of data; or preparation, review, and approval of the manuscript.

[3] Supported in part by grant R01 CA39742 from the National Cancer Institute, grant 131209 from the Academy of Finland, a grant from the Finnish Cultural Foundation, and the Fulbright program's Research Grant for a Junior Scholar (the last 3 of which were granted to JM).

[4] Address reprint requests and correspondence to DR Jacobs Jr, Division of Epidemiology and Community Health, University of Minnesota, 1300 South Second Street, Suite 300, Minneapolis, MN 55454. E-mail: jacob004@umn.edu.

[5] Abbreviations used: AHEI, Alternative Healthy Eating Index; ARR, absolute risk reduction; CVD, cardiovascular disease; FFQ, food-frequency questionnaire; ICD, International Classification of Diseases; IWHS, Iowa Women's Health Study; WHR, waist-to-hip ratio.

Received November 27, 2012. Accepted for publication April 30, 2013.

First published online June 19, 2013; doi: 10.3945/ajcn.112.055681.

CVD, we hypothesized that higher quality scores would be inversely related to total and CVD mortality. On the basis of earlier work with coffee and whole-grain foods in the IWHS (15, 16), our hypothesis was that the score would relate less strongly to cancer and would not relate to acute and injury-related mortality but might be inversely related to inflammatory-related mortality. Therefore, we formed a composite of causes of death selected a priori for which inflammation, oxidative stress, and chronic infection have been documented as a significant pathologic factor.

SUBJECTS AND METHODS

Participants and study design

The IWHS was designed to examine associations between several host, dietary, and lifestyle factors and the incidence of cancer in postmenopausal women (17). At the study baseline in 1986, 41,836 women aged 55–69 y completed a 16-page self-administered questionnaire. Of these women, 99% of subjects were white, and 99% of subjects were postmenopausal. Compared with nonrespondents, respondents were slightly younger, had lower BMI (in kg/m^2), and more likely lived in rural areas (18). The IWHS was approved by the University of Minnesota Institutional Review Board, and the return of the questionnaire was considered informed consent in concordance with prevailing practice in 1986.

For the analyses, we excluded subjects who self-reported to be premenopausal (n = 569); had diabetes (n = 2747), CVD (n = 4115), or cancer (n = 3830); or did not adequately complete an FFQ at baseline in 1986 (n = 3096). At baseline, we had 29,634 women available for the calculation of the AHEI and a priori diet score and, thus, for analyses. For 2004 follow-up analyses, 15,076 women with complete baseline and FFQ follow-up data were included.

Dietary information

Food intake was assessed at baseline in 1986 and 2004 follow-ups by using a validated 127-food-item Harvard FFQ (19, 20). Food-composition values were obtained from the Harvard University Food Composition Database derived from US Department of Agriculture sources, supplemented with manufacturer information, and later updated to reflect marketplace changes (19).

AHEI

The year 2010 version of the AHEI was calculated on the basis of values of the following 11 components: vegetables (servings/d), whole fruit (servings/d), whole grains (g/d), sugar-sweetened beverages and fruit juices (servings/d), nuts and legumes (servings/d), red and processed meat (servings/d), *trans* fatty acids (percentage of energy), long-chain (n−3) fatty acids EPA + DHA (mg/d), PUFAs (percentage of energy), sodium (mg/d), and alcohol intake (servings/d) as described previously (7). Each component had a potential to contribute 0–10 points to the total AHEI score (lower *trans* fatty acids, sugar-sweetened beverages, and sodium received higher scores). The total AHEI score was the sum of all component scores with a potential range from 0 to 110, with higher scores indicating a healthier diet. The AHEI was calculated separately at baseline and the 2004 follow-up.

A priori diet quality score

We created the a priori score from 34 food groups on the basis of 10 (12), 35 (11, 14), 46 (21), and 47 (13) food-item versions of the score used previously. Each food group was rated as favorable or positive (n = 17), neutral (n = 7), or adverse or negative (n = 10) on the basis of the literature and expert judgment regarding its role on risk of chronic disease. The positively rated food groups were beans and legumes, beer, coffee, fish, fruit, green vegetables, low-fat dairy, liquor, oil, other vegetables, poultry, seeds, and nuts, soy products, tea, tomato, whole grains, and wine. The negatively rated food groups were butter, fried foods, fried potatoes, red meat, liver, processed meat, salty snacks, soft drinks, sweets, and whole-fat dairy. The remaining neutrally rated food groups were chocolate, diet soft drinks, eggs, fruit juice, margarine, potatoes, and refined grains. Food groups considered to be either beneficial or harmful on health were categorized in quartiles. In food groups with a large proportion of nonconsumers, nonconsumers were coded as 0, and consumers were categorized into tertiles. The a priori diet quality score was calculated by summing category scores (0–3) of positively rated food groups with the reverse scores (3–0) of the negatively rated food groups. The theoretical maximum score was 81, with a higher score indicating a healthier diet. Compared with previous studies that used the a priori score (11–14), the IWHS implementation did not include food groups for avocados, grain desserts, lean fish, lean red meats, meal replacements, pickled foods, sauces, shellfish, soups, sugar substitutes, sweet breads, and yellow vegetables. The a priori diet quality score was calculated separately at baseline and the 2004 follow-up.

Ascertainment and classification of mortality

Deaths through 31 December 2008 were identified annually through the State Health Registry of Iowa or National Death Index for subjects who did not respond to follow-up questionnaires or had emigrated from Iowa. The underlying cause of death was assigned by state vital registries via the International Classification of Diseases (ICD). We defined *1*) CVD (ICD-9 codes 390–459 or ICD-10 codes I00–I99; *2*) cancer (codes 140–239 or C00–D48), *3*) inflammatory-related cause of death, including infection, benign tumor, endocrine, nutritional, and metabolic disorders (except diabetes, hyperlipidemia, or obesity), nervous system, respiratory, digestive, genitourinary, skin, and subcutaneous tissue, and selected connective tissue disorders and arthropathies (ICD-9 codes 1–139, 240–249, 251–271, 273–277, 279–359, 460–629, 680–714, or 720; ICD-10 codes A, B, E00–E09, E15–E64, E67–E77, E79–E90, F, G, H, J, K, L, M00–M14, M30–M36, M45–M46, or N); and *4*) other and external cause of death, including sense organ, osteoarthritis, congenital anomalies, signs and symptoms, injuries, accidents, and suicides deaths. Diabetes, hyperlipidemia, or obesity-related deaths (n = 157 at 1986 and n = 36 at 2004) were included only in all-cause deaths. The follow-up duration was calculated as the time from the baseline date to the date of death or the earlier of the last follow-up contact or 31 December 2008.

Other measurements

The baseline questionnaire included questions concerning potential confounders, including age, height, education, place of residence (living on a farm or rural area other than a farm or city), high blood pressure, weight, hormone-replacement therapy, physical activity, and smoking. As previously described (22), physical activity was characterized as participating in moderate or vigorous activities less than a few times a month, a few times a month or 1 time/wk, or ≥2 times/wk. Waist and hip circumferences were measured by each participant by using a fixed protocol (23). The 2004 questionnaires included similar questions except that the educational level, place of residence, and waist and hip circumferences were not reassessed. Blood lipids and blood pressure were not measured at any survey.

Statistical analyses

Continuous variables were compared by using ANOVA, and categorical variables were measured by using chi-square tests. Cumulative mortality rates across quartiles of diet score assessed at baseline were examined. Absolute risk reduction (ARR) was calculated by multiplying absolute risk in the reference group by the multivariable-adjusted HR change in the comparison group. Cox proportional hazards regression analyses were used to explore the relation between diet indexes and outcomes. In the minimally adjusted model 1, we adjusted the association for age (continuous) and energy intake (continuous), whereas in the multivariable model 2, we further adjusted for marital status (currently married or never married or divorced or widowed), education (less than high school, high school, or more than high school), place of residence (living on a farm or other), high blood pressure (yes or no), BMI (continuous), waist-to-hip ratio (WHR, continuous), hormone-replacement therapy (current, past, or never), physical activity (low, moderate, or high), and smoking (current, past, or never). For baseline analyses, covariate data obtained from baseline were used. For analyses that started in 2004, current data were used for all covariates except the level of education, place of residence, and WHR. When current data were unavailable, information from 1986 was used. In the analyses with continuous exposure variables, HRs were shown for each SD change. A trend was computed by treating exposure as a continuous variable. To test the complementary prediction of the 2 scores, we computed residuals in simple linear regression (the AHEI given a priori score and the a priori score given the AHEI), then carried out Cox models for mortality regressed on the main effect of the AHEI simultaneously with the residual effect of the a priori score given the AHEI, and then reversed the procedure (ie, the a priori score main effect in the same model as the AHEI given the a priori score). Both regressions input the same predictive information as the single simultaneous regression by using the 2 main effects as predictors but allowed the analyst to assess additional information of each variable conditional on noting the full statistical effect of the other. Analyses were performed with PC-SAS software (version 9.2; SAS Institute Inc).

RESULTS

During the mean follow-up time of 20.3 ± 4.9 y, 10,343 (35% of 29,634 women at risk) total, 3646 (12%) CVD, 3207 (11%) cancer, 2888 (10%) inflammatory, and 445 (2%) external cause–related deaths were identified. At baseline in 1986, the mean (±SD) AHEI was 40.0 ± 10.4, the a priori score was 38.4 ± 8.2, and the correlation between these 2 indexes was 0.65. In a cross-tabulation of each score in quartiles, we showed an exact agreement in 43% of women, and near agreement in 84% of women. We showed that κ was equal to 0.24, which attributed the expected cases along the diagonal to chance. At the 2004 follow-up, in 15,076 women who had complete baseline information and dietary information from the follow-up, the mean AHEI was 40.0 ± 10.4, and the a priori score was 38.9 ± 7.7. The tracking correlation for baseline and follow-up scores was 0.42 ($P < 0.001$) for the AHEI and 0.55 ($P < 0.001$) for the a priori diet quality score. *See* Supplemental Table S1 under "Supplemental data" in the online issue for the 2 diet scores with a list of AHEI and the a priori score items that correspond to each diet category.

Women with a higher AHEI were more likely to be older, nonsmoking, physically more active, more educated, use estrogen replacement therapy, have lower BMI and WHR, and higher self-rated health and less likely to live on a farm or be married than were women with a lower AHEI (**Table 1**). In addition, women with a higher AHEI were more likely to have a healthier diet, as expected (**Table 2**). For the a priori score, physical (Table 1) and dietary characteristics (Table 2) were similar to those of the AHEI. Similar patterns were seen for both scores at the 2004 follow-up.

In Cox proportional hazards regression models, both the AHEI and a priori diet quality score assessed at baseline predicted total, CVD, cancer, and inflammatory-related mortality. In age- and energy-adjusted models, women in the highest quartile of the AHEI had RR for total mortality of 0.76 (ARR: 9.1%) compared with that of women in the lowest quartile (**Table 3**). The association was attenuated to 0.82 (ARR: 6.8%) after multivariable adjustment (model 2). For CVD, cancer, and inflammatory-related mortality, the associations were similarly attenuated, and multivariable adjusted HRs were 0.79 (ARR: 2.8%) and 0.88 (ARR: 1.4%), and 0.76 (ARR: 2.7%), respectively.

In the age- and energy-adjusted models, women in the highest compared with lowest quartiles of the a priori score had an HR of 0.73 (ARR: 10.3%) for total mortality, and additional adjustment attenuated RR to 0.80 (ARR: 7.6%) (Table 3). For CVD, cancer, and inflammatory-related mortality, multivariable-adjusted HRs were 0.79 (ARR: 2.8%), 0.86 (ARR: 1.7%), and 0.75 (ARR: 2.7%), respectively. The AHEI and a priori diet quality score were not significantly related to mortality from other and external causes.

To test whether each score predicted mortality independently of the other, we compared the prediction of the complementary alternate-pattern residuals per SD change at baseline. In the multivariable-adjusted analyses, an increase of one SD of the a priori diet quality score and AHEI residuals were associated with an identical 4% (95% CI: 2%, 6%) decrease in total mortality (**Table 4**). Both residuals significantly predicted also CVD and inflammatory-related mortality, whereas for cancer, neither residual added statistically significant information to the other score.

In 15,076 surviving women with complete baseline and FFQ follow-up data, mortality was reported in 1671 cases from all causes, and there were 625 CVD, 466 cancer, 456 inflammatory-related, and 88 other or external cause deaths. In multivariable-adjusted models, the a priori diet quality score predicted mortality outcomes significantly and more strongly than was the case by using 1986 as the baseline, whereas the AHEI significantly predicted total, CVD, and inflammatory-related mortality (**Table 5**). With 2004 as the baseline, the a priori score residual added

TABLE 1
Characteristics of 29,634 Iowa Women's Health Study participants at study baseline in 1986 across quartiles of the AHEI and a priori diet quality score[1]

	AHEI quartiles				A priori diet quality score quartiles			
	1	2	3	4	1	2	3	4
Diet score cutoffs	<38.8	38.8–45.7	>45.7–52.8	>52.8	≤32	33–38	39–43	≥44
Women (*n*)	7408	7409	7409	7408	7295	7950	6442	7946
AHEI	33.1 ± 4.5[2]	42.3 ± 2.0	49.1 ± 2.0	59.5 ± 5.6	28.8 ± 7.2	35.6 ± 1.7	40.9 ± 1.4	48.7 ± 4.1
A priori diet quality score	32.3 ± 6.4	36.5 ± 6.5	39.9 ± 6.7	45.0 ± 7.2	28.1 ± 3.5	33.6 ± 7.7	37.1 ± 7.9	43.3 ± 8.6
Age (y)	61.2 ± 4.1	61.5 ± 4.2	61.5 ± 4.2	61.4 ± 4.2	61.2 ± 4.1	61.5 ± 4.2	61.6 ± 4.2	61.4 ± 4.2
Current smoker (%)	17.3	15.4	14.5	13.4	17.8	15.4	15.1	12.5
Currently married (%)	79.8	77.7	77.2	76.8	79.1	77.3	77.1	77.9
Lives on a farm (%)	25.5	21.6	18.4	14.3	25.7	22.2	19.2	13.0
Current HRT (%)	9.7	10.5	11.7	14.1	9.2	10.9	11.7	14.1
Education (%)								
1–12 y	21.1	19.0	16.2	12.6	23.6	19.2	16.1	10.2
High school graduate	45.2	43.3	42.1	38.1	48.6	44.2	41.5	34.9
Beyond high school	33.7	37.7	41.7	49.3	27.7	36.6	42.5	54.9
High BP (%)	34.4	33.8	33.1	30.5	32.0	34.1	33.1	32.7
BMI (kg/m^2)	27.0 ± 5.1	26.9 ± 4.9	26.8 ± 4.9	26.2 ± 4.6	27.1 ± 5.2	27.0 ± 5.0	26.7 ± 4.8	26.2 ± 4.5
Waist-to-hip ratio	0.84 ± 0.08	0.84 ± 0.08	0.83 ± 0.08	0.82 ± 0.08	0.84 ± 0.08	0.84 ± 0.08	0.83 ± 0.08	0.82 ± 0.08
Physical activity index (%)								
Less than a few times a month	27.0	21.6	18.1	12.5	31.2	22.5	16.0	9.8
A few times a month or 1 time/wk	31.3	29.2	27.5	23.3	31.4	30.3	27.6	22.2
≥2 times/wk	41.8	49.2	54.5	64.1	37.4	47.3	56.5	68.0
Self-rated health (%)								
Excellent	23.4	25.9	30.2	34.2	22.2	25.3	28.3	37.3
Good	64.0	64.0	61.0	59.1	64.9	63.7	63.4	56.5
Fair	11.6	9.4	8.3	6.3	11.7	10.3	7.9	5.9
Poor	1.0	0.7	0.5	0.4	1.1	0.8	0.4	0.3

[1] *P* values from *t* tests for continuous variables or the chi-square test for categorical variables were <0.001 in all cases across the AHEI and a priori score except for currently married (0.02) and high BP (0.01). AHEI, Alternative Healthy Eating Index; BP, blood pressure; HRT, hormone-replacement therapy.
[2] Mean ± SD (all such values).

information to the AHEI main effect for total, CVD, and inflammatory-related mortality, whereas the AHEI residual added information to the a priori score main effect for total mortality only (*see* Supplemental Table S2 under "Supplemental data" in the online issue).

DISCUSSION

In this longitudinal investigation from the IWHS, both the AHEI and a priori diet quality score predicted total and disease-specific mortality in older women. At baseline, these 2 different a priori indexes produced very similar findings, whereas at the 2004 follow-up, when the surviving women were, on average, ∼80 y old, the a priori diet quality score tended to be more strongly related to outcomes. The a priori score predicted total and disease-specific mortality outcomes, whereas the AHEI significantly predicted total, CVD, and inflammatory-related mortality. Also, at 2004, the a priori score tended to add to predictiveness for total, CVD, and inflammatory-related mortality, independent of the AHEI, whereas the AHEI significantly added predictiveness to the a priori score only for total mortality.

Only a few studies have assessed the relation of the AHEI on total mortality or CVD risk (6, 7, 10, 24), and in most of the studies (6, 10, 24), the original version (year 2002) of the AHEI was used. In the Health Professional's Follow-up Study and Nurses' Health Study, men in the highest compared with lowest quintile of the AHEI (2002) had a multiadjusted HR of 0.80 (95% CI: 0.71, 91) for a major chronic disease, whereas for women, the HR was 0.89 (0.82, 0.96), respectively (6). Risk reductions were particularly strong for CVD incidence and men [in men, HR: 0.61 (0.49, 0.75); in women, HR: 0.72 (0.60, 0.86)]. Similar findings were shown in the Whitehall II Study, in which participants (*n* = 7319, of whom 30% were women) in the highest third of the AHEI (2002) had ∼ 25% lower risk of total and ∼ 40% lower CVD mortality than did women in the lowest third of the AHEI (2002) (10). In the Women's Health Initiative Observational Study (*n* = 93,676), women in the highest compared with lowest quintiles of the AHEI (2002) had HRs of 0.77 (95% CI: 0.70, 0.84) and 0.70 (95% CI: 0.59, 0.82) for incident CVD and heart failure (24). Recently, the updated version of the AHEI (7) was shown to predict risk of chronic diseases in the Health Professionals Follow-Up Study and Nurses' Health Study (7). Highest compared with lowest quintiles of the AHEI (2010) had pooled multiadjusted HRs of 0.81 (95% CI: 0.77, 0.85) and 0.76 (95% CI: 0.71, 0.81) for a major chronic disease and CVD, respectively (7). These inverse associations were slightly stronger for women than men.

Also, the a priori diet quality score has been shown to predict several outcomes (11–14). In a Norwegian case-control study, higher scores of the 35 food-group version of the a priori diet score were inversely related with risk of myocardial infarction (11). In the Oslo Diet and Exercise Study, subjects with increased

TABLE 2

Dietary characteristics of 29,634 Iowa Women's Health Study participants at study baseline in 1986 across quartiles of the AHEI and a priori diet quality score[1]

	AHEI quartiles				A priori diet quality score quartiles			
	1	2	3	4	1	2	3	4
Diet score cutoffs	<29.0	29.0–35.1	35.2–41.9	>41.9	≤32	33–38	39–43	≥44
Women (*n*)	7408	7409	7409	7408	7295	7950	6442	7946
AHEI	24.2 ± 3.7[2]	32.0 ± 1.8	38.4 ± 1.9	48.4 ± 5.4	28.8 ± 7.2	35.6 ± 1.7	40.9 ± 1.4	48.7 ± 4.1
A priori diet quality score	32.3 ± 6.4	36.5 ± 6.5	39.9 ± 6.7	45.0 ± 7.2	28.1 ± 3.5	33.6 ± 7.7	37.1 ± 7.9	43.3 ± 8.6
Energy intake (kcal/d)	1857 ± 581	1780 ± 597	1772 ± 626	1799 ± 553	1802 ± 619	1791 ± 638	1791 ± 611	1822 ± 553
AHEI component (points)								
Vegetables	4.5 ± 2.6	5.4 ± 2.5	6.1 ± 2.6	7.1 ± 2.5	4.0 ± 2.1	5.2 ± 2.4	6.2 ± 2.4	7.6 ± 2.2
Fresh fruit	3.4 ± 2.1	4.3 ± 2.4	4.8 ± 2.5	5.8 ± 2.6	3.3 ± 2.2	4.2 ± 2.4	4.9 ± 2.5	5.9 ± 2.4
Whole grains	2.8 ± 2.3	3.7 ± 2.6	4.5 ± 2.9	5.7 ± 3.0	2.8 ± 2.5	3.9 ± 2.8	4.5 ± 2.9	5.5 ± 2.9
SSBs and fruit juice	1.8 ± 3.0	3.0 ± 3.6	4.0 ± 3.8	5.4 ± 3.8	3.5 ± 3.8	3.5 ± 3.8	3.6 ± 3.8	3.6 ± 3.8
Nuts and legumes	1.6 ± 1.9	2.4 ± 2.5	3.2 ± 3.1	4.8 ± 3.6	2.2 ± 2.7	2.8 ± 3.0	3.2 ± 3.1	3.8 ± 3.2
Red or processed meat	2.3 ± 2.6	3.3 ± 2.9	4.0 ± 3.0	5.1 ± 3.0	2.6 ± 2.8	3.3 ± 2.9	3.8 ± 3.0	4.9 ± 2.9
trans Fat	4.3 ± 1.7	3.7 ± 1.6	3.3 ± 1.5	2.6 ± 1.6	4.3 ± 1.7	3.7 ± 1.6	3.3 ± 1.5	2.6 ± 1.6
EPA + DHA	3.7 ± 2.9	5.3 ± 3.2	6.2 ± 3.2	7.4 ± 3.0	4.1 ± 3.2	5.1 ± 3.3	5.9 ± 3.2	7.3 ± 2.9
PUFA	4.0 ± 1.6	4.6 ± 3.0	5.1 ± 1.7	5.8 ± 2.0	4.8 ± 1.8	4.9 ± 1.8	4.9 ± 1.9	5.0 ± 1.9
Sodium	4.6 ± 3.0	5.0 ± 3.1	5.1 ± 3.1	4.9 ± 3.1	5.1 ± 3.1	5.0 ± 3.2	4.9 ± 3.1	4.6 ± 3.0
Alcohol	1.2 ± 2.6	1.9 ± 3.3	2.6 ± 3.7	3.7 ± 4.1	1.2 ± 2.6	1.9 ± 3.3	2.6 ± 3.7	3.7 ± 4.1
A priori diet quality score components (servings/wk)								
Beans and legumes	0.5 ± 0.6	0.5 ± 0.7	0.5 ± 0.9	0.6 ± 0.8	0.4 ± 0.6	0.5 ± 0.7	0.5 ± 0.9	0.6 ± 0.9
Tofu	0.0 ± 0.1	0.0 ± 0.2	0.0 ± 0.3	0.1 ± 0.7	0.0 ± 0.1	0.0 ± 0.2	0.0 ± 0.4	0.1 ± 0.6
Nuts	1.2 ± 1.9	1.8 ± 2.6	2.5 ± 3.3	4.1 ± 4.8	1.7 ± 2.9	2.2 ± 3.4	2.5 ± 3.6	3.0 ± 3.8
Green leafy vegetables	3.4 ± 2.9	4.4 ± 3.6	5.1 ± 4.5	6.4 ± 5.1	2.8 ± 2.7	4.0 ± 3.5	5.1 ± 3.8	7.2 ± 5.2
Other vegetables	13.0 ± 8.3	15.9 ± 10.4	18.2 ± 11.9	21.6 ± 12.9	11.3 ± 7.3	15.2 ± 9.7	18.4 ± 11.3	23.5 ± 12.8
Tomato	1.7 ± 2.5	2.0 ± 2.9	2.2 ± 3.2	2.5 ± 3.1	1.4 ± 2.4	1.9 ± 2.9	2.3 ± 3.2	2.8 ± 3.2
Potatoes	3.8 ± 3.2	3.2 ± 2.5	2.9 ± 2.4	2.7 ± 2.2	3.4 ± 2.9	3.2 ± 2.8	3.0 ± 2.4	2.9 ± 2.3
Fresh fruit	9.8 ± 7.4	12.3 ± 8.1	14.1 ± 9.1	17.1 ± 9.9	9.3 ± 7.2	12.2 ± 8.4	14.2 ± 9.1	17.3 ± 9.3
Fried foods	2.8 ± 2.4	2.3 ± 2.2	2.1 ± 2.2	1.7 ± 2.0	3.4 ± 2.4	2.5 ± 2.2	1.9 ± 2.0	1.1 ± 1.6
French fries	0.4 ± 0.7	0.4 ± 0.7	0.4 ± 0.6	0.3 ± 0.6	0.6 ± 0.8	0.4 ± 0.6	0.3 ± 0.5	0.2 ± 0.4
Butter	2.8 ± 6.2	1.9 ± 4.9	1.7 ± 4.6	1.4 ± 4.0	3.8 ± 7.0	2.0 ± 5.0	1.2 ± 3.6	0.8 ± 2.9
Margarine	8.6 ± 8.3	9.1 ± 8.3	9.4 ± 8.4	9.7 ± 8.4	8.8 ± 8.8	9.5 ± 8.6	9.6 ± 8.3	8.9 ± 7.7
Oil and vinegar dressing	0.6 ± 1.2	0.8 ± 1.6	1.0 ± 1.9	1.5 ± 2.6	0.3 ± 0.8	0.6 ± 1.5	1.0 ± 1.8	1.8 ± 2.7
Low-fat dairy	7.4 ± 8.0	7.6 ± 7.9	7.4 ± 7.4	7.9 ± 7.5	4.6 ± 6.6	6.9 ± 7.3	8.4 ± 7.8	10.2 ± 7.9
High-fat dairy	11.9 ± 12.0	10.2 ± 10.0	9.6 ± 9.5	8.8 ± 8.6	13.9 ± 12.6	10.3 ± 10.3	8.8 ± 8.6	7.6 ± 7.0
Fish	0.9 ± 1.4	1.4 ± 1.8	1.8 ± 2.2	2.5 ± 2.2	0.9 ± 1.3	1.3 ± 1.5	1.7 ± 2.0	2.7 ± 2.6
Poultry	1.4 ± 1.4	1.7 ± 1.9	1.9 ± 1.9	2.4 ± 2.3	1.2 ± 1.5	1.6 ± 1.9	1.9 ± 1.7	2.6 ± 2.2
Red meat	7.3 ± 4.4	6.2 ± 4.0	5.5 ± 3.7	4.5 ± 3.2	6.8 ± 4.2	6.2 ± 4.2	5.8 ± 3.8	4.8 ± 3.3
Processed meat	2.7 ± 3.1	2.1 ± 2.3	1.7 ± 2.0	1.3 ± 1.8	2.7 ± 2.9	2.1 ± 2.6	1.8 ± 2.2	1.2 ± 1.6
Liver	0.2 ± 0.8	0.3 ± 0.8	0.3 ± 0.6	0.3 ± 0.7	0.3 ± 0.7	0.3 ± 0.9	0.3 ± 0.6	0.3 ± 0.7
Eggs	2.4 ± 2.4	2.1 ± 2.0	2.1 ± 2.0	2.1 ± 2.1	2.3 ± 2.4	2.2 ± 2.1	2.2 ± 2.2	2.1 ± 1.9
Whole grains	7.4 ± 6.7	9.9 ± 7.7	12.2 ± 8.7	15.7 ± 10.1	7.4 ± 7.2	10.3 ± 8.2	12.2 ± 8.8	15.1 ± 9.5
Refined grains	11.3 ± 8.7	9.0 ± 7.6	7.9 ± 7.2	6.4 ± 6.2	11.1 ± 8.8	9.1 ± 7.9	7.7 ± 6.9	6.5 ± 6.0
Salty snacks	3.6 ± 6.2	3.6 ± 5.9	4.0 ± 6.6	4.4 ± 7.3	4.3 ± 6.8	3.9 ± 6.4	3.8 ± 6.4	3.5 ± 6.4
Chocolate	0.9 ± 2.4	0.7 ± 1.9	0.7 ± 1.8	0.7 ± 1.8	1.0 ± 2.3	0.8 ± 2.1	0.7 ± 1.8	0.5 ± 1.7
Sweets without chocolate	8.6 ± 8.8	8.4 ± 8.4	8.0 ± 7.9	7.6 ± 8.5	10.6 ± 9.9	8.8 ± 8.5	7.5 ± 7.7	5.8 ± 6.5
Fruit juices	6.2 ± 5.5	5.4 ± 5.1	4.7 ± 5.1	3.5 ± 4.4	4.4 ± 4.9	4.9 ± 5.2	5.1 ± 5.1	5.4 ± 5.4
Low-calorie beverages	1.3 ± 3.5	1.4 ± 3.3	1.6 ± 3.7	2.0 ± 3.9	1.2 ± 3.5	1.4 ± 3.5	1.7 ± 3.9	1.9 ± 3.7
High-sugar beverages	2.4 ± 4.1	1.5 ± 2.8	1.1 ± 2.2	0.7 ± 1.5	2.3 ± 3.9	1.5 ± 3.0	1.1 ± 2.3	0.7 ± 1.8
Coffee	12.1 ± 13.4	11.7 ± 13.1	12.1 ± 13.3	12.4 ± 13.4	11.4 ± 13.5	12.2 ± 13.5	12.3 ± 13.4	12.4 ± 12.8
Tea	3.0 ± 6.4	2.9 ± 5.9	3.0 ± 6.1	3.1 ± 6.3	2.1 ± 5.4	2.9 ± 6.1	3.3 ± 6.5	3.7 ± 6.4
Beer	0.8 ± 4.1	0.5 ± 2.8	0.6 ± 2.5	0.6 ± 1.8	0.5 ± 2.9	0.6 ± 2.9	0.7 ± 3.1	0.7 ± 2.7
Liquor	0.9 ± 3.8	0.8 ± 3.0	0.8 ± 2.6	1.1 ± 2.5	0.4 ± 2.2	0.7 ± 2.8	1.0 ± 3.2	1.4 ± 3.6
Wine	0.3 ± 1.8	0.4 ± 1.6	0.6 ± 2.0	1.0 ± 2.0	0.2 ± 1.1	0.4 ± 1.4	0.6 ± 1.8	1.2 ± 2.6

[1] *P* values from *t* tests for continuous variables were <0.001 in all cases across the AHEI and across a priori score except for energy intake (0.003). AHEI, Alternative Healthy Eating Index; SSB, sugar-sweetened beverage.

[2] Mean ± SD (all such values).

TABLE 3
AHEI and a priori diet quality score assessed in the 29,634 Iowa Women's Health Study participants at study baseline in 1986 and risk of total and disease-specific mortality[1]

	Quartile of score				
	1	2	3	4	*P*-trend
Total mortality					
AHEI					
Cases (*n*)	2797	2631	2534	2281	—
Participants (*n*)	7408	7409	7409	7408	—
Person-years	148,451	149,029	150,989	152,946	—
Model 1	1	0.94 (0.89, 0.99)[2]	0.85 (0.81, 0.90)	0.76 (0.71, 0.80)	<0.001
Model 2	1	0.98 (0.92, 1.03)	0.90 (0.85, 0.95)	0.82 (0.77, 0.87)	<0.001
A priori score					
Cases (*n*)	2785	2889	2225	2444	—
Participants (*n*)	7295	7950	6442	7946	—
Person-years	145,413	160,541	131,256	164,184	—
Model 1	1	0.89 (0.85, 0.94)	0.82 (0.78, 0.87)	0.73 (0.69, 0.77)	<0.001
Model 2	1	0.93 (0.88, 0.98)	0.87 (0.82, 0.92)	0.80 (0.76, 0.85)	<0.001
CVD mortality					
AHEI					
Cases (*n*)	980	989	915	762	—
Participants (*n*)	7408	7409	7409	7408	—
Person-years	148,451	149,029	150,989	152,946	—
Model 1	1	0.95 (0.87, 1.04)	0.87 (0.79, 0.95)	0.71 (0.65, 0.78)	<0.001
Model 2	1	0.99 (0.91, 1.09)	0.91 (0.83, 1.00)	0.79 (0.72, 0.88)	<0.001
A priori score					
Cases (*n*)	984	1024	789	849	—
Participants (*n*)	7295	7950	6442	7946	—
Person-years	145,413	160,541	131,256	164,184	—
Model 1	1	0.88 (0.81, 0.96)	0.80 (0.73, 0.88)	0.71 (0.64, 0.77)	<0.001
Model 2	1	0.92 (0.84, 1.01)	0.86 (0.78, 0.95)	0.79 (0.72, 0.88)	<0.001
Cancer mortality					
AHEI					
Cases (*n*)	858	835	757	757	—
Participants (*n*)	7408	7409	7409	7408	—
Person-years	148,451	149,029	150,989	152,946	—
Model 1	1	0.95 (0.87, 1.05)	0.85 (0.77, 0.94)	0.84 (0.76, 0.92)	0.006
Model 2	1	1.00 (0.90, 1.10)	0.90 (0.81, 0.99)	0.88 (0.79, 0.98)	<0.001
A priori score					
Cases (*n*)	865	845	688	809	—
Participants (*n*)	7295	7950	6442	7946	—
Person-years	145,413	160,541	131,256	164,184	—
Model 1	1	0.87 (0.79, 0.95)	0.85 (0.77, 0.94)	0.80 (0.73, 0.88)	<0.001
Model 2	1	0.88 (0.80, 0.98)	0.89 (0.80, 0.98)	0.86 (0.77, 0.95)	0.025
Inflammatory-related mortality					
AHEI					
Cases (*n*)	819	735	705	629	—
Participants (*n*)	7408	7409	7409	7408	—
Person-years	148,451	149,029	150,989	152,946	—
Model 1	1	0.86 (0.78, 0.95)	0.81 (0.73, 0.89)	0.70 (0.63, 0.78)	<0.001
Model 2	1	0.89 (0.80, 0.99)	0.85 (0.76, 0.94)	0.76 (0.68, 0.84)	<0.001
A priori score					
Cases (*n*)	784	843	611	650	—
Participants (*n*)	7295	7950	6442	7946	—
Person-years	145,413	160,541	131,256	164,184	—
Model 1	1	0.92 (0.84, 1.01)	0.79 (0.71, 0.88)	0.68 (0.61, 0.75)	<0.001
Model 2	1	0.96 (0.87, 1.06)	0.83 (0.74, 0.93)	0.75 (0.67, 0.84)	<0.001
Other and external mortality[3]					
AHEI					
Cases (*n*)	92	124	123	106	—
Participants (*n*)	7408	7409	7409	7408	—
Person-years	148,451	149,029	150,989	152,946	—
Model 1	1	1.29 (0.98, 1.68)	1.25 (0.95, 1.64)	1.06 (0.80, 1.39)	0.98
Model 2	1	1.36 (1.03, 1.81)	1.34 (1.01, 1.77)	1.16 (0.86, 1.56)	0.56

(Continued)

TABLE 3 (*Continued*)

	Quartile of score				
	1	2	3	4	*P*-trend
A priori score					
Cases (*n*)	105	133	105	102	—
Participants (*n*)	7295	7950	6442	7946	—
Person-years	145,413	160,541	131,256	164,184	—
Model 1	1	1.09 (0.84, 1.40)	1.02 (0.78, 1.34)	0.80 (0.61, 1.05)	0.18
Model 2	1	1.14 (0.87, 1.48)	1.02 (0.76, 1.36)	0.85 (0.63, 1.15)	0.36

[1] *P*-trend was based on Cox proportional hazards regression analyses with exposure as a continuous variable. Model 1 was adjusted for age and energy intake. Model 2 was adjusted for age, energy intake, marital status, education, place of residence, high blood pressure, BMI, waist-to-hip ratio, hormone-replacement therapy, physical activity, and smoking. AHEI, Alternative Healthy Eating Index; CVD, cardiovascular disease.

[2] HR; 95% CI in parentheses (all such values).

[3] Accident, injury, and suicide mortality plus other causes not included in cardiovascular disease, cancer, or inflammatory-related mortality.

scores of the 35 food-group version of the diet score had more favorable changes in body size variables, systolic blood pressure, and blood lipid, glucose, insulin, and adiponectin concentrations (14). In the Multi-Ethnic Study of Atherosclerosis, participants with the higher of the 47 food-group version of the score had a lower urinary albumin:creatinine ratio, common carotid intimamedia thickness, measures of adiposity, and inflammatory marker, triacylglycerol, and insulin concentrations (13). Also, in the Multi-Ethnic Study of Atherosclerosis, compared with participants in the lowest quintile of the 10 food-item version of the score, participants in the highest quintile had an HR of 0.62 (95% CI: 0.44–0.88) for type 2 diabetes (12).

In most studies, the AHEI has not been shown to be significantly related to risk of cancer (6, 10), although in a recent study that used the updated version of the AHEI, subjects in the highest compared with lowest quintile had a pooled multiadjusted HR of 0.94 (95% CI: 0.89, 0.98) for cancer (7). This association was slightly stronger for women than men. We showed that the AHEI was significantly related to cancer mortality, although risk reductions were more modest than those shown for CVD mortality. Similar findings were also shown for the a priori diet quality score, for which the relation for cancer has not been previously explored to our knowledge.

Adherence to a Mediterranean diet has been shown to be inversely related to cancer mortality in a meta-analysis of >1.5 million subjects (8).The Mediterranean diet, AHEI, and a priori diet quality score all share similar characteristics. The consumption of vegetables, fruit, legumes, cereals, and fish and

TABLE 4
Prediction of total and disease-specific mortality per SD increase of a dietary pattern and its complementary alternate pattern residual in 29,634 Iowa Women's Health Study participants at study baseline in 1986[1]

	Regression examining a priori residual		Regression examining AHEI residual	
	Main effect	Residual	Main effect	Residual
	AHEI (SD: 10.4)	A priori given AHEI (SD: 7.9)	A priori (SD: 8.2)	AHEI given a priori (SD: 6.2)
Total mortality				
Model 1	0.89 (0.88, 0.91)[2]	0.95 (0.93, 0.97)	0.89 (0.87, 0.91)	0.95 (0.94, 0.97)
Model 2	0.92 (0.91, 0.94)	0.96 (0.94, 0.98)	0.92 (0.90, 0.94)	0.96 (0.94, 0.98)
CVD mortality				
Model 1	0.88 (0.85, 0.91)	0.96 (0.92, 0.99)	0.87 (0.84, 0.90)	0.95 (0.92, 0.98)
Model 2	0.92 (0.89, 0.95)	0.97 (0.93, 1.00)	0.91 (0.88, 0.95)	0.96 (0.93, 1.00)
Cancer mortality				
Model 1	0.93 (0.90, 0.96)	0.99 (0.95, 1.03)	0.93 (0.90, 0.97)	0.96 (0.93, 1.00)
Model 2	0.95 (0.92, 0.99)	0.99 (0.95, 1.03)	0.96 (0.92, 1.00)	0.97 (0.94, 1.01)
Inflammatory-related mortality				
Model 1	0.87 (0.84, 0.90)	0.91 (0.88, 0.95)	0.86 (0.83, 0.90)	0.95 (0.91, 0.98)
Model 2	0.90 (0.87, 0.94)	0.93 (0.89, 0.97)	0.90 (0.86, 0.94)	0.95 (0.91, 0.98)
Other and external mortality[3]				
Model 1	0.97 (0.89, 1.07)	0.93 (0.84, 1.03)	0.94 (0.85, 1.03)	1.02 (0.93, 1.12)
Model 2	1.00 (0.91, 1.11)	0.93 (0.83, 1.03)	0.95 (0.86, 1.06)	1.04 (0.94, 1.14)

[1] Cox proportional hazards regression analyses with main effect and residual diet score exposures as continuous variables. Model 1 was adjusted for age and energy intake. Model 2 was adjusted for age, energy intake, marital status, education, place of residence, high blood pressure, BMI, waist-to-hip ratio, hormone-replacement therapy, physical activity, and smoking. AHEI, Alternative Healthy Eating Index; CVD, cardiovascular disease.

[2] HR; 95% CI in parentheses (all such values).

[3] Accident, injury, and suicide mortality plus other causes not included in CVD, cancer, or inflammatory-related mortality.

TABLE 5
AHEI and a priori diet quality score assessed in 15,076 Iowa Women's Health Study participants in 2004 and risk of total and disease-specific mortality[1]

	Quartile of score				
	1	2	3	4	*P*-trend
Total mortality					
AHEI					
Cases (*n*)	534	477	381	279	—
Participants (*n*)	3769	3769	3769	3769	—
Model 1	1	0.89 (0.79, 1.01)[2]	0.72 (0.63, 0.82)	0.53 (0.46, 0.62)	<0.001
Model 2	1	1.00 (0.88, 1.14)	0.87 (0.76, 1.00)	0.70 (0.60, 0.81)	<0.001
A priori score					
Cases (*n*)	581	426	422	242	—
Participants (*n*)	3726	3684	4130	3536	—
Model 1	1	0.73 (0.64, 0.83)	0.65 (0.57, 0.73)	0.43 (0.37, 0.50)	<0.001
Model 2	1	0.87 (0.77, 0.99)	0.79 (0.70, 0.91)	0.58 (0.49, 0.68)	<0.001
CVD mortality					
AHEI					
Cases (*n*)	195	185	141	104	—
Participants (*n*)	3769	3769	3769	3769	—
Model 1	1	0.96 (0.78, 1.17)	0.75 (0.60, 0.93)	0.57 (0.45, 0.72)	<0.001
Model 2	1	1.05 (0.85, 1.29)	0.89 (0.71, 1.12)	0.72 (0.56, 0.93)	0.002
A priori score					
Cases (*n*)	213	172	150	90	—
Participants (*n*)	3726	3684	4130	3536	—
Model 1	1	0.81 (0.66, 0.99)	0.64 (0.52, 0.78)	0.45 (0.35, 0.58)	<0.001
Model 2	1	0.95 (0.77, 1.17)	0.77 (0.62, 0.96)	0.61 (0.47, 0.80)	<0.001
Cancer mortality					
AHEI					
Cases (*n*)	136	113	119	98	—
Participants (*n*)	3769	3769	3769	3769	—
Model 1	1	0.83 (0.64, 1.06)	0.87 (0.68, 1.11)	0.72 (0.55, 0.93)	0.004
Model 2	1	0.88 (0.68, 1.13)	0.95 (0.73, 1.22)	0.83 (0.63, 1.09)	0.037
A priori score					
Cases (*n*)	143	106	135	82	—
Participants (*n*)	3726	3684	4130	3536	—
Model 1	1	0.74 (0.57, 0.95)	0.84 (0.66, 1.06)	0.59 (0.45, 0.77)	<0.001
Model 2	1	0.86 (0.66, 1.11)	0.97 (0.75, 1.24)	0.70 (0.52, 0.94)	0.028
Inflammatory-related mortality					
AHEI					
Cases (*n*)	160	136	96	64	—
Participants (*n*)	3769	3769	3769	3769	—
Model 1	1	0.85 (0.68, 1.07)	0.59 (0.46, 0.77)	0.40 (0.30, 0.53)	<0.001
Model 2	1	1.03 (0.81, 1.31)	0.83 (0.64, 1.09)	0.61 (0.44, 0.82)	<0.001
A priori score					
Cases (*n*)	189	100	110	57	—
Participants (*n*)	3726	3684	4130	3536	—
Model 1	1	0.52 (0.41, 0.67)	0.51 (0.40, 0.64)	0.30 (0.22, 0.40)	<0.001
Model 2	1	0.68 (0.53, 0.88)	0.69 (0.54, 0.89)	0.46 (0.33, 0.63)	<0.001
Other and external mortality[3]					
AHEI					
Cases (*n*)	29	28	20	11	—
Participants (*n*)	3769	3769	3769	3769	—
Model 1	1	0.97 (0.58, 1.63)	0.72 (0.41, 1.27)	0.41 (0.21, 0.83)	0.02
Model 2	1	1.06 (0.62, 1.81)	0.84 (0.47, 1.51)	0.49 (0.23, 1.00)	0.26
A priori score					
Cases (*n*)	29	29	19	11	—
Participants (*n*)	3726	3684	4130	3536	—
Model 1	1	1.01 (0.60, 1.69)	0.61 (0.34, 1.08)	0.42 (0.21, 0.85)	0.020
Model 2	1	1.08 (0.64, 1.83)	0.64 (0.35, 1.16)	0.49 (0.23, 1.01)	0.064

[1] *P*-trend was based on Cox proportional hazards regression analyses with exposure as a continuous variable. Model 1 was adjusted for age and energy intake. Model 2 was adjusted for age, energy intake, marital status, education, place of residence, high blood pressure, BMI, waist-to-hip ratio, hormone-replacement therapy, physical activity, and smoking. AHEI, Alternative Healthy Eating Index; CVD, cardiovascular disease.

[2] HR; 95% CI in parentheses (all such values).

[3] Accident, injury, and suicide mortality plus other causes not included in cardiovascular disease, cancer, or inflammatory-related mortality.

a moderate intake of red wine during meals above the median consumption of the population has generally been considered to be part of a Mediterranean diet (8). Less-strong risk reductions that have been shown for cancer in general may be because the cause and dietary influence vary by cancer type (25). Furthermore, the AHEI (7) and a priori diet quality score (11–14) were created mainly on the basis of the current knowledge regarding diet and CVD.

We showed, in agreement with previous IWHS analyses of whole-grain foods (15) and coffee (16), that the a priori diet quality score was strongly and inversely related to mortality from inflammatory-related causes. Similar findings were shown also for the AHEI. Previously, adherence to a Mediterranean diet has been shown to be associated with reduced risk of Parkinson disease and Alzheimer disease (8). These results merit additional study in the IWHS by using more-nuanced a priori scores.

Main strengths of the study were the relatively large sample size and longitudinal setting and extensive adjustment for potential confounders. Also, we used 2 independent dietary assessments as exposures, which were shown to be relatively stable (eg, the tracking correlation between the 1986 and 2004 reports of the a priori score was 0.55). These tracking correlations are generally higher than those for individual foods or nutrients. The current study also had limitations. First, the AHEI and a priori diet quality score are both based on current knowledge about the role of food in the prevention of chronic diseases, particularly CVD. Each score may best be regarded as a representation of a good diet rather than an optimal diet, and studies such as the current one may help to add information about the complex relation of diet to disease. Also, although there is general agreement that certain Mediterranean-type diet patterns are associated with reduced chronic disease risk (8), we do not think that there is a single optimal diet score that applies to all situations. A history of usages of the a priori diet quality score was published as supplementary material (21). There has been substantial agreement about the rating of individual food groups in raters, although emerging information would suggest that ratings for a few food groups might be altered, such as for chocolate, refined grain, and eggs. As currently formulated, the score predicts well, but any of numerous possible alterations in ratings could potentially yield an even better prediction. Second, both indexes were associated with other factors related with indicators of good health, and although we adjusted for potential confounders, we could not fully exclude the possibility of residual confounding. Randomized clinical trial settings would be ideal to avoid confounding and prove causality, but for foods or diets, such an approach is, for the most part, unrealistic, although the recently published Prevención con Dieta Mediterránea study (26) proved that such studies are possible. Third, because the study population consisted largely of older white women, a generalization to other populations should be done cautiously. The analysis of the broad cause of death categories was both a strength and limitation. The strength was that it capitalized on broad pathologic commonalities. The limitation was that it missed important pathologic distinctions in which diet patterns may have been related more specifically to some more-specific causes.

In conclusion, we showed that both the AHEI and a priori diet quality score predicted total and disease-specific mortality in older women. These scores also had complementary predictive importance, and thus, study of these 2 patterns, both representing good diets, may yield new information about an even better diet.

The authors' responsibilities were as follows—DRJ: designed the research; JM and DRJ: performed statistical analyses, interpreted data, wrote the manuscript, and had primary responsibility for the final content of the manuscript; and all authors: provided essential materials, critically revised the manuscript for important intellectual content, and read and approved the final manuscript. None of the authors had a conflict of interest.

REFERENCES

1. Miniño AM, Xu J, Kochanek KD, Tejada-Vera B. Death in the United States, 2007. NCHS data brief, no 26. Hyattsville, MD: National Center for Health Statistics, 2009.
2. Hu FB. Diet and lifestyle influences on risk of coronary heart disease. Curr Atheroscler Rep 2009;11:257–63.
3. World Cancer Research Fund/American Institute for Cancer Research. Food, nutrition, and physical activity, and the prevention of cancer: a global perspective. Washington, DC: AICR, 2007.
4. Mente A, de Koning L, Shannon HS, Anand SS. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. Arch Intern Med 2009;169:659–69.
5. Jacobs DR Jr, Tapsell LC. Food, not nutrients, is the fundamental unit in nutrition. Nutr Rev 2007;65:439–50.
6. McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB, Spiegelman D, Hunter DJ, Colditz GA, Willett WC. Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr 2002;76:1261–71.
7. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough ML, Wang M, Stampfer MJ, Willett WC. Alternative dietary indices both strongly predict risk of chronic disease. J Nutr 2012;142:1009–18.
8. Sofi F, Cesari F, Abbate R, Gensini GF, Casini A. Adherence to Mediterranean diet and health status: meta-analysis. BMJ 2008;337: a1344.
9. Arvaniti F, Panagiotakos DB. Healthy indexes in public health practice and research: a review. Crit Rev Food Sci Nutr 2008;48:317–27.
10. Akbaraly TN, Ferrie JE, Berr C, Brunner EJ, Head J, Marmot MG, Singh-Manoux A, Ritchie K, Shipley MJ, Kivimaki M. Alternative Healthy Eating Index and mortality over 18 y of follow-up: results from the Whitehall II cohort. Am J Clin Nutr 2011;94:247–53.
11. Lockheart MS, Steffen LM, Rebnord HM, Fimreite RL, Ringstad J, Thelle DS, Pedersen JI, Jacobs DR Jr. Dietary patterns, food groups and myocardial infarction: a case-control study. Br J Nutr 2007;98: 380–7.
12. Nettleton JA, Steffen LM, Ni H, Liu K, Jacobs DR Jr. Dietary patterns and risk of incident type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). Diabetes Care 2008;31:1777–82.
13. Nettleton JA, Schulze MB, Jiang R, Jenny NS, Burke GL, Jacobs DR Jr. A priori-defined dietary patterns and markers of cardiovascular disease risk in the Multi-Ethnic Study of Atherosclerosis (MESA). Am J Clin Nutr 2008;88:185–94.
14. Jacobs DR Jr, Sluik D, Rokling-Andersen MH, Anderssen SA, Drevon CA. Association of 1-y changes in diet pattern with cardiovascular disease risk factors and adipokines: results from the 1-y randomized Oslo Diet and Exercise Study. Am J Clin Nutr 2009;89:509–17.
15. Jacobs DR Jr, Andersen LF, Blomhoff R. Whole-grain consumption is associated with a reduced risk of noncardiovascular, noncancer death attributed to inflammatory diseases in the Iowa Women's Health Study. Am J Clin Nutr 2007;85:1606–14.
16. Andersen LF, Jacobs DR Jr, Carlsen MH, Blomhoff R. Consumption of coffee is associated with reduced risk of death attributed to inflammatory and cardiovascular diseases in the Iowa Women's Health Study. Am J Clin Nutr 2006;83:1039–46.
17. Folsom AR, Kushi LH, Anderson KE, Mink PJ, Olson JE, Hong CP, Sellers TA, Lazovich D, Prineas RJ. Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's Health Study. Arch Intern Med 2000;160:2117–28.
18. Bisgard KM, Folsom AR, Hong CP, Sellers TA. Mortality and cancer rates in nonrespondents to a prospective study of older women: 5-year follow-up. Am J Epidemiol 1994;139:990–1000.
19. Willett WC, Sampson L, Brown ML, Stampfer MJ, Rosner B, Hennekens CH, Speizer FE. The use of a self-administered questionnaire to assess diet four years in the past. Am J Epidemiol 1988;127:188–99.
20. Munger RG, Folsom AR, Kushi LH, Kaye SA, Sellers TA. Dietary assessment of older Iowa women with a food frequency questionnaire:

nutrient intake, reproducibility, and comparison with 24-hour dietary recall interviews. Am J Epidemiol 1992;136:192–200.
21. Sijtsma FP, Meyer KA, Steffen LM, Shikany JM, Van Horn L, Harnack L, Kromhout D, Jacobs DR Jr. Longitudinal trends in diet and effects of sex, race, and education on dietary quality score change: the Coronary Artery Risk Development in Young Adults study. Am J Clin Nutr 2012; 95:580–6.
22. Park K, Harnack L, Jacobs DR Jr. Trends in dietary supplement use in a cohort of postmenopausal women from Iowa. Am J Epidemiol 2009; 169:887–92.
23. Kushi LH, Kaye SA, Folsom AR, Soler JT, Prineas RJ. Accuracy and reliability of self-measurement of body girths. Am J Epidemiol 1988; 128:740–8.
24. Belin RJ, Greenland P, Allison M, Martin L, Shikany JM, Larson J, Tinker L, Howard BV, Lloyd-Jones D, Van Horn L. Diet quality and the risk of cardiovascular disease: the Women's Health Initiative (WHI). Am J Clin Nutr 2011;94:49–57.
25. World Cancer Research Fund / American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington DC: AICR, 2007. Available from: http://www.aicr.org/site/PageServer?pagename=research_science_expert_report (cited 30 March 2013).
26. Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, Gómez-Gracia E, Ruiz-Gutiérrez V, Fiol M, Lapetra J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med 2013;368:1279–90.