



Original Contribution

Multiple Healthful Dietary Patterns and Type 2 Diabetes in the Women's Health Initiative

Elizabeth M. Cespedes*, Frank B. Hu, Lesley Tinker, Bernard Rosner, Susan Redline, Lorena Garcia, Melanie Hingle, Linda Van Horn, Barbara V. Howard, Emily B. Levitan, Wenjun Li, JoAnn E. Manson, Lawrence S. Phillips, Jinnie J. Rhee, Molly E. Waring, and Marian L. Neuhouser

* Correspondence to Dr. Elizabeth M. Cespedes, Departments of Nutrition and Epidemiology, Harvard T.H. Chan School of Public Health, 665 Huntington Avenue, 3rd Floor, Boston, MA 02115 (e-mail: emc611@mail.harvard.edu).

Initially submitted April 1, 2015; accepted for publication September 4, 2015.

The relationship between various diet quality indices and risk of type 2 diabetes (T2D) remains unsettled. We compared associations of 4 diet quality indices—the Alternate Mediterranean Diet Index, Healthy Eating Index 2010, Alternate Healthy Eating Index 2010, and the Dietary Approaches to Stop Hypertension (DASH) Index—with reported T2D in the Women's Health Initiative, overall, by race/ethnicity, and with/without adjustment for overweight/obesity at enrollment (a potential mediator). This cohort ($n = 101,504$) included postmenopausal women without T2D who completed a baseline food frequency questionnaire from which the 4 diet quality index scores were derived. Higher scores on the indices indicated a better diet. Cox regression was used to estimate multivariate hazard ratios for T2D. Pearson coefficients for correlation among the indices ranged from 0.55 to 0.74. Follow-up took place from 1993 to 2013. During a median 15 years of follow-up, 10,815 incident cases of T2D occurred. For each diet quality index, a 1-standard-deviation higher score was associated with 10%–14% lower T2D risk ($P < 0.001$). Adjusting for overweight/obesity at enrollment attenuated but did not eliminate associations to 5%–10% lower risk per 1-standard-deviation higher score ($P < 0.001$). For all 4 dietary indices examined, higher scores were inversely associated with T2D overall and across racial/ethnic groups. Multiple forms of a healthful diet were inversely associated with T2D in these postmenopausal women.

Alternate Healthy Eating Index; Alternate Mediterranean Diet Index; Dietary Approaches to Stop Hypertension Index; dietary patterns; health disparities; Healthy Eating Index; type 2 diabetes; women's health

Abbreviations: AHEI-2010, Alternate Healthy Eating Index 2010; aMED, Alternate Mediterranean Diet; BMI, body mass index; DASH, Dietary Approaches to Stop Hypertension; FFQ, food frequency questionnaire; HEI-2010, Healthy Eating Index 2010; MET, metabolic equivalent of task; SD, standard deviation; T2D, type 2 diabetes; WHI, Women's Health Initiative.

Diet quality contributes to type 2 diabetes (T2D) risk and potentially to racial/ethnic disparities in the burden of diabetes, making identification of the optimal diet (or diets) for T2D prevention a public health priority (1). While much nutrition research focuses on single nutrients or foods, the combinations and quantities in which these are consumed have synergistic and cumulative effects. Moreover, isolating individual exposures may not provide a realistic picture of dietary patterns or health impact, since typically dietary changes involve substituting one component for another (2). Numerical indices measuring adherence to a priori dietary patterns offer

one approach for examining the totality of diet. As was noted in the 2010 Dietary Guidelines for Americans, such dietary pattern analysis may lead more easily to public health recommendations (3).

As a recent systematic review highlighted (4), the ability to compare associations across different dietary indices is limited by differences in modeling approaches and scoring methods (e.g., median population intakes vs. fixed cutoffs). The current study addresses this limitation: The Women's Health Initiative (WHI) offers the opportunity to calculate multiple, standardized dietary indices in the same cohort. Further, the

cohort's ethnic diversity complements existing research, which has been conducted primarily among European-descent populations, with few exceptions (5–7). Since African Americans and Hispanic/Latinos develop diabetes more often and at younger ages than non-Hispanic whites (8), examining the benefit of healthful dietary patterns in these populations is of particular importance.

Our study extends research by Qiao et al. (6) examining associations of the Alternate Healthy Eating Index (AHEI) with T2D in WHI. Here we strengthen the approach by 1) excluding women participating in the WHI Dietary Modification Trial, who probably changed their diets because of intervention and had systematically higher fat intakes due to eligibility criteria; 2) calculating scores on the updated AHEI-2010; and 3) presenting results for 3 additional indices: the Alternate Mediterranean Diet (aMED) Index, Healthy Eating Index 2010 (HEI-2010), and the Dietary Approaches to Stop Hypertension (DASH) Index.

In sum, this study examined associations of 4 commonly used dietary indices with incident T2D among diverse postmenopausal women. We standardized indices for comparability and stratified by race/ethnicity. We hypothesized that all indices would be inversely associated with T2D in all racial/ethnic groups.

METHODS

Study population

The design and methods of the WHI have been published elsewhere (9–11). From 1993 to 1998, postmenopausal women 50–79 years of age were recruited into clinical trials or an observational study ($n = 161,808$). When the first phase of WHI ended (2004–2005), participants were invited to join WHI Extension 1 (2005–2010) and later Extension 2 (2010–2015). Participants continue to be followed for various health outcomes. This analysis includes follow-up through September 20, 2013.

Written informed consent was obtained. Procedures were approved by institutional review boards at all participating institutions. A standardized written protocol, centralized staff training, and quality assurance visits by the clinical coordinating center ensured uniform data collection. Our analytical sample was drawn from women participating in the WHI Observational Study and the Calcium and Vitamin D and Hormone Therapy trials. We excluded women in both arms of the WHI Dietary Modification Trial ($n = 48,835$) due to the likelihood of dietary changes and the systematically higher (>32%) intake of energy from fat that was part of the trial's eligibility criteria. We additionally excluded women with missing dietary intake data or missing information on prevalent diabetes at baseline ($n = 844$). Of the remaining 112,129 women, we excluded those with prevalent diabetes outside of pregnancy ($n = 6,585$) and implausible energy intakes (<600 kcal/day or >5,000 kcal/day; $n = 4,040$). Women excluded for having outlying energy intakes (versus those included) had the same mean age and body mass index (BMI) but were less likely to be non-Hispanic white (61% vs. 85%) or college-educated (25% vs. 41%). Our analytical sample included 101,504 women.

At enrollment, participants reported information on demographic factors, health behaviors, and medical histories using self-administered questionnaires. We categorized the following covariates: age (50–54, 55–59, 60–69, or 70–79 years); race/ethnicity (non-Hispanic white, black, Hispanic/Latina, other, or missing ($n = 275$)); education (less than high school, less than college, college, postgraduate education, or missing ($n = 798$)); and smoking (never smoker, past smoker, current smoker, or missing ($n = 1,355$)). Physical activity was self-reported using the WHI physical activity inventory, which has been shown to be reliable (weighted- κ range, 0.67–0.71) and valid in comparison with accelerometer data ($r = 0.73$) (11). We calculated metabolic equivalent of task (MET)-hours/week and categorized them into quintiles (0–<2, 2–<7, 7–13, >13–23, or >23 MET-hours/week, or missing ($n = 2,132$)). Postmenopausal hormone therapy (unopposed estrogen and/or estrogen + progesterone) in the form of pills or patches was self-reported and classified as never use, past use, current use, or missing ($n = 2,119$). At the clinic visit, trained staff measured weight, height, and waist circumference during expiration at the torso's narrowest section, using a standardized protocol. BMI was calculated as weight in kilograms divided by squared height in meters and categorized as <18.5, 18.5–24.9, 25.0–29.9, 30.0–34.9, 35.0–39.9, ≥ 40 , or missing ($n = 1,070$).

Diet assessment

The exposure was diet quality as measured by each of 4 numeric indices (aMED, HEI-2010, AHEI-2010, and DASH). The foods and nutrients composing these indices were self-reported at enrollment using an FFQ developed and validated for WHI (12, 13). The FFQ included 122 composite and single-food line items asking about consumption frequency and portion size, 19 adjustment questions related to fat intake/type, and 4 summary questions about usual intakes of fruits and vegetables and added fats, used for comparison with the line item information. The FFQ was designed for application in multiethnic and geographically diverse populations, and has been shown to produce reliable estimates comparable to 8 days of intake data from four 24-hour dietary recalls and 4-day food records (13). The nutrient database used to analyze the FFQ was derived from the Nutrition Data System for Research, 2005 version (University of Minnesota, Minneapolis, Minnesota) (14). The Minnesota system provides nutrient information for more than 140 nutrients and compounds, including energy, saturated fat, and sodium. We calculated each diet quality index using dietary data in units of MyPyramid equivalents by establishing a customized link (15) between the Minnesota system and the MyPyramid Equivalents Database, version 2.0 (US Department of Agriculture) (16). MyPyramid Equivalents translate foods, as eaten, into standardized quantities; for example, a MyPyramid equivalent is an amount nutritionally equal to 1 cup (240 g, 237 mL) in the vegetable, fruit, and dairy components or 1 ounce (28.4 g) in grains or protein foods.

Four indices were examined as predictors of T2D. The aMED Index reflects a Mediterranean-style dietary pattern characterized by high consumption of minimally processed plant-based foods; olive oil as the principal source of fat;

Table 1. Characteristics of Participants by Category of Standardized Alternate Mediterranean Diet Score, Women's Health Initiative ($n = 101,504$), 1993–2013

	Category of Standardized aMED Score					
	SD < -1 ($n = 18,912$)		SD -1 to 1 ($n = 71,680$)		SD > 1 ($n = 10,912$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
aMED score ^a	2 (0–2)		4 (3–6)		7 (7–9)	
Age, years	63 (7)		64 (7)		64 (7)	
Body mass index ^b	28 (6)		27 (6)		26 (5)	
Physical activity, MET-hours/week	9 (12)		14 (14)		19 (16)	
Total energy intake, kcal/day	1,350 (534)		1,596 (603)		1,826 (561)	
Alcoholic beverages, drinks/week	0.36 (0.9)		0.43 (0.84)		0.49 (0.68)	
Neighborhood SES ^c	75 (9)		76 (8)		78 (7)	
Hormone therapy						
Current use		36		42		47
Past use		25		23		22
Never use		37		35		31
Smoking status						
Current smoker		12		6		3
Past smoker		38		43		46
Never smoker		49		50		49
Race/ethnicity						
Non-Hispanic white		83		85		89
Black		8		7		5
Hispanic/Latina		6		3		1
Asian		2		3		3
Other		1		1		1
College graduate		28		42		58
Family history of diabetes		31		30		27

Abbreviations: aMED, Alternate Mediterranean Diet; MET, metabolic equivalent of task; SD, standard deviation; SES, socioeconomic status.

^a Values are presented as median (range).

^b Weight (kg)/height (m)².

^c Neighborhood SES was a composite measure based on census tract data regarding adult high school education rates, male unemployment, neighborhood poverty, number of female-headed households with children, and median household income (31). Scores could range from 20 to 100.

low-to-moderate consumption of dairy products, fish, and poultry; low consumption of red meat; and low-to-moderate consumption of wine (17, 18). HEI-2010 was created by the Department of Agriculture and the National Cancer Institute to align with the 2010 Dietary Guidelines for Americans (3, 19, 20). AHEI-2010 was adapted from the intakes recommended in the Dietary Guidelines to incorporate foods and nutrients predictive of chronic disease risk, including greater intake of vegetables and fruits, whole grains, nuts and legumes, long-chain ω -3 fatty acids, and polyunsaturated fatty acids; lower intake of sugar-sweetened beverages and fruit juices, red/processed meat, *trans*-fat, and sodium; and moderate alcohol consumption (21). The DASH Index, which was based on controlled-feeding studies (22, 23) that administered a diet rich in vegetables, fruits, and low-fat dairy products, includes whole grains, poultry, fish, and nuts and tends to be lower in saturated fat, red meat, sweets, sugar-containing

beverages, and sodium (24, 25). Details on the components of each diet quality index, their contributions to the total index scores, and study-specific cutpoints are shown in Web Table 1 (available at <http://aje.oxfordjournals.org/>).

Diabetes ascertainment

Participants were asked about ever having physician-diagnosed “sugar diabetes” or “high blood sugar” outside of pregnancy. Women who responded “yes” at baseline were excluded. At each semiannual (WHI Clinical Trial) or annual (WHI Observational Study) contact, all participants were asked, “Since the date given on the front of this form, has a doctor prescribed for the first time any of the following pills or treatments?” Choices included “pills for diabetes” and “insulin shots for diabetes.” Thus, only incident treated diabetes was ascertained, defined as a self-report of physician-diagnosed diabetes

Table 2. Characteristics of Participants by Category of Standardized Healthy Eating Index 2010 Score, Women's Health Initiative ($n = 101,504$), 1993–2013

	Category of Standardized HEI-2010 Score					
	SD < -1 ($n = 16,617$)		SD -1 to 1 ($n = 68,716$)		SD > 1 ($n = 16,171$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
HEI-2010 score ^a	50 (18–55)		68 (55–77)		80 (77–95)	
Age, years	62 (7)		64 (7)		65 (7)	
Body mass index ^b	29 (7)		27 (6)		26 (5)	
Physical activity, MET-hours/week	8 (12)		14 (14)		18 (15)	
Total energy intake, kcal/day	1,818 (766)		1,550 (567)		1,427 (455)	
Alcoholic beverages, drinks/week	0.38 (1.08)		0.44 (0.81)		0.41 (0.65)	
Neighborhood SES ^c	74 (9)		76 (8)		77 (8)	
Hormone therapy						
Current use		34		43		46
Past use		26		23		23
Never use		38		34		31
Smoking status						
Current smoker		15		6		3
Past smoker		38		43		44
Never smoker		46		50		52
Race/ethnicity						
Non-Hispanic white		78		86		89
Black		12		6		6
Hispanic/Latina		7		3		2
Asian		2		3		2
Other		2		1		1
College graduate		27		42		52
Family history of diabetes		33		30		27

Abbreviations: HEI-2010, Healthy Eating Index 2010; MET, metabolic equivalent of task; SD, standard deviation; SES, socioeconomic status.

^a Values are presented as median (range).

^b Weight (kg)/height (m)².

^c Neighborhood SES was a composite measure based on census tract data regarding adult high school education rates, male unemployment, neighborhood poverty, number of female-headed households with children, and median household income (31). Scores could range from 20 to 100.

treated with oral medication or insulin (26, 27). Time to diabetes was defined as number of days from enrollment to the return of the questionnaire in which diabetes was first reported. The accuracy of self-reported diabetes in WHI has been assessed using medication and laboratory data and found to be valid (28).

Statistical analysis

Scores on each diet quality index were categorized into quintiles to examine potential nonlinear associations. To facilitate comparison across indices with markedly different ranges of scores (e.g., aMED (range, 0–9) vs. HEI-2010 (range, 0–100)), we report results 1) per 10% increment in the theoretical score (e.g., per 1-point increase in aMED vs. per 10-point increase in HEI-2010) and 2) per 1-standard-deviation (1-SD) increase (after standardizing each of the indices to the

normal distribution). Mean values, standard deviations, and frequencies for demographic and lifestyle characteristics of the study sample were calculated by category of the standardized indices (<–1, –1 to 1, and >1 SD unit). To assess the extent to which quantification of a healthy diet was similar across scores, we calculated Pearson coefficients for correlations between the indices and the proportions of participants who fell into the top or bottom quintiles on all 4 indices.

Participants were followed from enrollment until death, loss to follow-up, or the most recent follow-up for WHI Extension 2, which took place on September 20, 2013. Participants who did not consent to participation in either WHI extension but were alive at study closeout were censored on those dates (September 12, 2005, and September 30, 2010, respectively). Overall, of those eligible, 77% consented to Extension 1 and 87% consented to Extension 2 (29).

Table 3. Characteristics of Participants by Category of Standardized Alternate Healthy Eating Index 2010 Score, Women's Health Initiative ($n = 101,504$), 1993–2013

	Category of Standardized AHEI-2010 Score					
	SD < -1 ($n = 16,824$)		SD -1 to 1 ($n = 67,872$)		SD > 1 ($n = 16,808$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
AHEI-2010 score ^a	35 (13–39)		50 (39–61)		66 (61–94)	
Age, years	63 (7)		64 (7)		64 (7)	
Body mass index ^b	29 (6)		27 (6)		26 (5)	
Physical activity, MET-hours/week	8 (11)		13 (14)		20 (16)	
Total energy intake, kcal /day	1,718 (627)		1,552 (603)		1,520 (537)	
Alcoholic beverages, drinks/week	0.32 (0.99)		0.43 (0.84)		0.52 (0.63)	
Neighborhood SES ^c	74 (9)		76 (8)		78 (8)	
Hormone therapy						
Current use		34		42		48
Past use		26		24		22
Never use		38		34		30
Smoking status						
Current smoker		12		6		3
Past smoker		35		42		50
Never smoker		52		50		45
Race/ethnicity						
Non-Hispanic white		80		85		89
Black		12		6		4
Hispanic/Latina		5		4		2
Asian		1		3		3
Other		1		1		1
College graduate		26		41		56
Family history of diabetes		33		30		27

Abbreviations: AHEI-2010, Alternate Healthy Eating Index 2010; MET, metabolic equivalent of task; SD, standard deviation; SES, socioeconomic status.

^a Values are presented as median (range).

^b Weight (kg)/height (m)².

^c Neighborhood SES was a composite measure based on census tract data regarding adult high school education rates, male unemployment, neighborhood poverty, number of female-headed households with children, and median household income (31). Scores could range from 20 to 100.

We estimated multivariable-adjusted hazard ratios and 95% confidence intervals for T2D with Cox proportional hazards models, using person-days since enrollment as the underlying time metric and modeling dietary indices categorically and continuously as described above. The proportional hazards assumption was assessed by examining plots of weighted Schoenfeld residuals with log person-months; no evidence of violation was found for any of the dietary indices. Models adjusted for age, educational attainment, race/ethnicity, smoking status, family history of diabetes, hormone therapy, total daily energy intake, physical activity quintile, and study arm.

Given obesity's potential role as a mediator of the diet-T2D relationship, we examined the change in the multivariable-adjusted hazard ratios with and without BMI category as a covariate. We also stratified analyses by race/ethnicity, physical

activity tertile, and baseline overweight (BMI 25.0–29.9) or obesity (BMI ≥ 30.0) status (reference group, BMI <25.0) and tested for interactions of each index with these variables using likelihood ratio tests and Wald χ^2 tests.

In sensitivity analyses, we excluded participants with prevalent cardiovascular disease or cancer (excluding skin cancer) at baseline ($n = 12,913$), since these conditions could lead to an altered diet. We also considered modeling age as continuous in years and stratifying by age group. We considered adjusting for hypertension, waist circumference, waist:hip ratio, coffee intake (which has an inverse association with T2D (30)), geographic region, and neighborhood socioeconomic status (a composite measure based on census tract data regarding adult high school education rates, male unemployment, neighborhood poverty, number of female-headed households with children, and median household income (31)).

Table 4. Characteristics of Participants by Category of Standardized DASH Diet Score, Women's Health Initiative ($n = 101,504$), 1993–2013

	Category of Standardized DASH Score					
	SD < -1 ($n = 18,376$)		SD -1 to 1 ($n = 65,216$)		SD > 1 ($n = 17,912$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
DASH diet score ^a	17 (8–19)		24 (20–28)		30 (29–38)	
Age, years	62 (7)		64 (7)		64 (7)	
Body mass index ^b	29 (6)		27 (6)		26 (5)	
Physical activity, MET-hours/week	8 (11)		14 (14)		20 (16)	
Total energy intake, kcal/day	1,596 (638)		1,551 (607)		1,637 (529)	
Alcoholic beverages, drinks/week	0.38 (0.88)		0.45 (0.85)		0.38 (0.71)	
Neighborhood SES ^c	73 (10)		76 (8)		78 (7)	
Hormone therapy						
Current use		34		43		46
Past use		26		23		22
Never use		38		34		32
Smoking status						
Current smoker		14		6		2
Past smoker		37		43		45
Never smoker		47		50		52
Race/ethnicity						
Non-Hispanic white		75		87		91
Black		14		6		4
Hispanic/Latina		7		3		2
Asian		3		3		2
Other		2		1		1
College graduate		25		42		56
Family history of diabetes		33		30		27

Abbreviations: DASH, Dietary Approaches to Stop Hypertension; MET, metabolic equivalent of task; SD, standard deviation; SES, socioeconomic status.

^a Values are presented as median (range).

^b Weight (kg)/height (m)².

^c Neighborhood SES was a composite measure based on census tract data regarding adult high school education rates, male unemployment, neighborhood poverty, number of female-headed households with children, and median household income (31). Scores could range from 20 to 100.

All statistical analyses were conducted using SAS, version 9.3 (SAS Institute, Inc., Cary, North Carolina). All tests were 2-sided, with statistical significance set at $P < 0.05$.

RESULTS

Univariate correlations between the 4 dietary indices were moderate to strong (all P 's < 0.001), ranging from 0.55 (between HEI-2010 and aMED) to 0.74 (between HEI-2010 and DASH). Different dietary indices quantified high-quality diets differently. Few women scored consistently high (in the top quintile of every score; $n = 5,417$ (5%)) or low (in the bottom quintile of every score; $n = 5,501$ (5%)). Among adjacent quintiles, there was greater agreement: 18,291 (18%) women scored in one of the top 2 quintiles and 17,013 (17%) scored in one of the bottom 2 quintiles on each of the 4 indices.

Across dietary indices, compared with women with poor-quality diets (SD of a given dietary index < -1), women with better-quality diets (SD > 1) were older, had lower BMI, and were more likely to be physically active, college-educated, non-Hispanic white, and current users of hormone therapy; they were also less likely to have a family history of diabetes (Tables 1–4; Web Table 2).

During a median 14.9 years of follow-up, we observed 10,815 incident T2D cases. In multivariable-adjusted models, better-quality diets were associated with lower risk of T2D regardless of the dietary index examined (Table 5): A 1-SD increase in a woman's score on any index, wherein higher scores indicate better-quality diets, was associated with a 10%–14% lower risk of T2D ($P < 0.001$). Additional adjustment for BMI category attenuated but did not eliminate these associations (5%–10% lower risk; $P < 0.001$). Comparing the

Table 5. Hazard Ratios for Diabetes According to Category of Diet Quality as Measured by 4 Diet Quality Indices, Women's Health Initiative (*n* = 101,504), 1993–2013^{a,b}

Diet Quality Index and Increment	Score		No. of Cases	Total No. of Women	Hazard Ratio for Diabetes					
	Minimum	Maximum			Age-Adjusted ^c		Multivariable-Adjusted ^d		Multivariable- and BMI-Adjusted ^e	
					HR	95% CI	HR	95% CI	HR	95% CI
aMED Index										
Per 1-SD increase					0.84	0.83, 0.86	0.90	0.88, 0.92	0.95	0.95, 0.97
Per 10% increase					0.91	0.90, 0.92	0.94	0.93, 0.96	0.97	0.96, 0.98
Quintile										
1	0	2	2,305	18,936	1.00	Referent	1.00	Referent	1.00	Referent
2	3	3	1,953	18,129	0.82	0.77, 0.87	0.87	0.82, 0.92	0.90	0.85, 0.96
3	4	4	2,245	20,301	0.81	0.77, 0.86	0.90	0.85, 0.95	0.95	0.90, 1.01
4	5	5	1,961	18,869	0.74	0.69, 0.78	0.85	0.80, 0.91	0.92	0.87, 0.98
5	6	9	2,366	25,392	0.61	0.58, 0.65	0.74	0.70, 0.79	0.85	0.80, 0.90
HEI-2010										
Per 1-SD increase					0.77	0.76, 0.79	0.89	0.87, 0.91	0.93	0.92, 0.95
Per 10% increase					0.79	0.78, 0.80	0.90	0.88, 0.92	0.94	0.92, 0.96
Quintile										
1	18	57	2,777	20,325	1.00	Referent	1.00	Referent	1.00	Referent
2	57	65	2,267	20,326	0.74	0.70, 0.78	0.88	0.83, 0.93	0.92	0.87, 0.97
3	65	70	2,115	20,325	0.65	0.61, 0.68	0.84	0.79, 0.89	0.90	0.85, 0.95
4	70	76	1,925	20,326	0.56	0.53, 0.59	0.77	0.73, 0.82	0.85	0.80, 0.91
5	76	95	1,746	20,325	0.49	0.46, 0.52	0.72	0.67, 0.76	0.83	0.78, 0.89
AHEI-2010										
Per 1-SD increase					0.77	0.75, 0.78	0.87	0.85, 0.89	0.92	0.90, 0.94
Per 10% increase					0.77	0.75, 0.78	0.87	0.85, 0.89	0.92	0.90, 0.94
Quintile										
1	13	41	2,786	20,325	1.00	Referent	1.00	Referent	1.00	Referent
2	41	47	2,348	20,326	0.79	0.75, 0.83	0.90	0.85, 0.95	0.93	0.88, 0.99
3	47	53	2,107	20,325	0.67	0.63, 0.71	0.82	0.77, 0.87	0.87	0.82, 0.92
4	53	60	1,923	20,326	0.58	0.55, 0.62	0.77	0.72, 0.81	0.84	0.79, 0.90
5	60	94	1,666	20,325	0.48	0.45, 0.51	0.68	0.64, 0.72	0.78	0.73, 0.83

Table continues

Table 5. Continued

Diet Quality Index and Increment	Score		No. of Cases	Total No. of Women	Hazard Ratio for Diabetes					
	Minimum	Maximum			Age-Adjusted ^c		Multivariable-Adjusted ^d		Multivariable- and BMI-Adjusted ^e	
					HR	95% CI	HR	95% CI	HR	95% CI
DASH Index										
Per 1-SD increase					0.75	0.73, 0.76	0.86	0.84, 0.88	0.90	0.89, 0.92
Per 10% increase					0.82	0.81, 0.83	0.90	0.89, 0.92	0.93	0.92, 0.95
Quintile										
1	8	19	2,623	18,402	1.00	Referent	1.00	Referent	1.00	Referent
2	20	22	2,219	19,781	0.71	0.67, 0.75	0.84	0.79, 0.89	0.87	0.82, 0.92
3	23	25	2,499	24,094	0.60	0.57, 0.63	0.77	0.73, 0.82	0.83	0.78, 0.88
4	26	28	2,013	21,418	0.51	0.48, 0.54	0.70	0.66, 0.75	0.77	0.72, 0.82
5	29	38	1,476	17,932	0.43	0.40, 0.46	0.64	0.60, 0.68	0.74	0.69, 0.80

Abbreviations: AHEI-2010, Alternate Healthy Eating Index 2010; aMED, Alternate Mediterranean Diet; BMI, body mass index; DASH, Dietary Approaches to Stop Hypertension; GED, General Educational Development; HEI-2010, Healthy Eating Index 2010; HR, hazard ratio; MET, metabolic equivalent of task; SD, standard deviation; WHI, Women's Health Initiative.

^a The total number of incident cases of diabetes was 10,815; the unadjusted incidence rate was 8.26 cases/1,000 person-years.

^b All *P* values for trend, computed using the median value of each quintile of diet quality index to construct a continuous variable, were less than 0.001.

^c Results were adjusted for age (50–54, 55–59, 60–69, or 70–79 years).

^d Results were additionally adjusted for covariates measured at the screening visit, including race/ethnicity (non-Hispanic black, Hispanic, other, non-Hispanic white (referent), or missing), educational attainment (less than high school/GED, some college or vocational training, college graduation, or postgraduate education (referent)), quintile of recreational physical activity (MET-hours/week), postmenopausal hormone use (current, former, or never use (referent)), family history of diabetes (yes/no), smoking status (current, former, or never smoker (referent)), study arm (randomization status for the WHI Calcium and Vitamin D Trial, the WHI Hormone Therapy Trials, or no assignment (referent)), and dietary energy intake (kcal/day).

^e Results were additionally adjusted for BMI (weight (kg)/height (m)²) category (<18.5, 18.5–24.9, 25.0–29.9, 30.0–34.9, 35.0–39.9, or ≥40).

Table 6. Multivariable- and BMI-Adjusted Hazard Ratios^a for Incident Diabetes According to Diet Quality Index Score and Race/Ethnicity, Women's Health Initiative (*n* = 101,504), 1993–2013^b

Diet Quality Index and Racial/Ethnic Group	Mean (SD) Index Score	Mean (SD) z Score ^c	Increment of Diet Quality Index				<i>P</i> for Trend ^e	<i>P</i> for Interaction ^f
			Per 1-SD Increase		Quintile 5 vs. Quintile 1 ^d			
			HR	95% CI	HR	95% CI		
aMED Index								
White	4 (2)	0.02 (1)	0.97	0.94, 0.99	0.90	0.84, 0.97	0.04	Referent
Black	4 (2)	−0.16 (0.97)	0.93	0.87, 0.99	0.82	0.68, 0.99	0.06	0.34
Hispanic/Latina	4 (2)	−0.36 (0.91)	0.76	0.68, 0.84	0.37	0.25, 0.54	0.0004	<0.001
Asian	5 (2)	0.23 (0.94)	0.87	0.75, 0.996	0.68	0.45, 1.05	0.14	0.52
AHEI-2010								
White	51 (11)	0.03 (1)	0.92	0.90, 0.94	0.80	0.74, 0.86	<0.001	Referent
Black	46 (11)	−0.36 (0.99)	0.93	0.87, 0.99	0.78	0.63, 0.96	0.03	0.38
Hispanic/Latina	48 (10)	−0.26 (0.93)	0.78	0.71, 0.87	0.46	0.32, 0.66	<0.001	<0.001
Asian	53 (10)	0.27 (0.91)	0.90	0.78, 1.03	0.81	0.53, 1.24	0.04	0.9
HEI-2010								
White	67 (11)	0.05 (0.98)	0.95	0.92, 0.97	0.86	0.80, 0.93	<0.001	Referent
Black	63 (12)	−0.33 (1.13)	0.93	0.88, 0.99	0.74	0.60, 0.90	0.01	0.81
Hispanic/Latina	62 (11)	−0.44 (1.05)	0.85	0.78, 0.93	0.75	0.53, 1.05	<0.001	0.001
Asian	67 (10)	0.06 (0.91)	0.91	0.79, 1.05	0.80	0.52, 1.23	0.04	0.85
DASH Index								
White	24 (5)	0.07 (0.98)	0.91	0.89, 0.93	0.77	0.71, 0.83	<0.001	Referent
Black	21 (5)	−0.53 (1.06)	0.92	0.86, 0.97	0.70	0.54, 0.90	0.004	0.47
Hispanic/Latina	22 (5)	−0.44 (1)	0.83	0.75, 0.91	0.54	0.37, 0.79	<0.001	0.0007
Asian	24 (4)	−0.08 (0.96)	0.87	0.76, 0.996	0.70	0.44, 1.10	0.06	0.97

Abbreviations: AHEI-2010, Alternate Healthy Eating Index 2010; aMED, Alternate Mediterranean Diet; BMI, body mass index; CI, confidence interval; DASH, Dietary Approaches to Stop Hypertension; GED, General Educational Development; HEI-2010, Healthy Eating Index 2010; HR, hazard ratio; IR, incidence rate; MET, metabolic equivalent of task; PY, person-years; SD, standard deviation; WHI, Women's Health Initiative.

^a Results were adjusted for covariates measured at the screening visit: age (50–54, 55–59, 60–69, or 70–79 years), education (less than high school/GED, some college or vocational training, college graduation, or postgraduate education (referent)), quintile of recreational physical activity (MET-hours/week), postmenopausal hormone use (current, former, or never use (referent)), family history of diabetes (yes/no), smoking status (current, former, or never smoker (referent)), study arm (randomization status for the WHI Calcium and Vitamin D Trial, the WHI Hormone Therapy Trials, or no assignment (referent)), dietary energy intake (kcal/day), and BMI (weight (kg)/height (m)²) category (<18.5, 18.5–24.9, 25.0–29.9, 30.0–34.9, 35.0–39.9, or ≥40).

^b For white women (*n* = 86,442), there were 8,549 cases, and the crude IR was 7.49 cases/1,000 PY. For black women (*n* = 7,021), there were 1,208 cases, and the crude IR was 15.56 cases/1,000 PY. For Hispanic/Latina women (*n* = 3,675), there were 549 cases, and the crude IR was 13.95 cases/1,000 PY. For Asian women (*n* = 2,621), there were 282 cases, and the crude IR was 9.58 cases/1,000 PY.

^c Indices were standardized as z scores (overall mean = 0; SD, 1) for comparability.

^d Quintile of diet index score in its native units.

^e Computed using the median value of each quintile of diet quality index to construct a continuous variable.

^f Wald test *P* value for the product of indicator variables for each nonreference racial/ethnic group (black, Hispanic/Latina, and Asian) with the continuous, standardized values of each dietary index (aMED, HEI-2010, AHEI-2010, and DASH). Each dietary index represents a different model, but all 3 product terms for the interaction of racial/ethnic group (black, Hispanic/Latina, or Asian) with the dietary index of interest were included in the same model (e.g., aMED × black + aMED × Latina + aMED × Asian). For all dietary indices, likelihood ratio tests rejected the null hypothesis (*P* > 0.05) that the simpler model (without interaction terms) fitted the data better than a model including product terms for the interaction of the multicategory variable race/ethnicity with each of the continuous, standardized dietary index scores.

top and bottom quintiles, higher indices were associated with a lower relative risk of T2D—between 26% (aMED; 95% confidence interval: 21, 30) and 36% (DASH; 95% confidence interval: 32, 40).

Associations were comparable across categories of BMI and tertile of physical activity, with no evidence that the association of any index with T2D varied by level of adiposity

or activity. However, the magnitude of the inverse association between diet quality and T2D varied by race/ethnicity (likelihood ratio test: *P* < 0.01 for interaction between race/ethnicity and each dietary index; Table 6). Higher dietary indices were inversely associated with T2D among all women before BMI adjustment. Notably, non-Hispanic white and Asian women had the highest median scores on each of the dietary indices

and the lowest crude rates of T2D, while Hispanic/Latina women and black women had the lowest median scores and highest crude rates of T2D. Before BMI adjustment, each 1-SD higher dietary index score was associated with 8%–13% lower T2D risk among non-Hispanic white women. Associations were stronger among Hispanic/Latina women (each 1-SD higher score was associated with an 18%–25% lower T2D risk; P for interaction < 0.05). The association of diet quality with T2D did not differ significantly between non-Hispanic white women and black women (9%–11% lower T2D risk per 1-SD higher dietary index score) or Asian women (16%–18% lower T2D risk). After BMI adjustment, the inverse trends in aMED score were statistically insignificant among black women and Asian women, and DASH score was statistically insignificant among Asian women, the smallest subgroup (Table 6).

Sensitivity analyses adding alternative and additional measures of adiposity, hypertension, geographic region, neighborhood socioeconomic status, or coffee intake to the models did not materially alter the results, either overall or when stratified by race/ethnicity. Analyses excluding women with baseline cardiovascular disease and cancer yielded virtually identical hazard ratios and conclusions (Web Table 3).

DISCUSSION

In this study of the diverse postmenopausal women participating in WHI, a 1-SD higher score on any of the 4 dietary indices examined was associated with a 10%–14% lower risk of T2D, suggesting that each index captures important characteristics of a healthful dietary pattern. The method used to achieve a healthy diet may vary, as there are multiple ways to achieve high scores on each index. For example, a 10-point increase in the AHEI-2010 could be achieved by eliminating sugar-sweetened beverages or reducing intake of red/processed meat to < 2.5 ounces/day (< 31 g/day). Despite this flexibility, defining characteristics are shared across multiple indices and are likely to be pillars of healthy diets, including high intakes of fruits, vegetables, whole grains, nuts, legumes, and unsaturated fats and low intakes of red and processed meat, sodium, sugar-sweetened beverages, and *trans*-fat.

After adjustment for overweight/obesity status at enrollment, each dietary index remained associated with a 5%–10% lower risk of T2D. These attenuated but still significant associations suggest that overweight/obesity explains much (but not all) of the association of diet with T2D risk. The magnitude of the estimated inverse association with T2D was lowest for aMED and highest for DASH. Higher scores for the aMED Index may have weaker inverse associations with T2D than higher scores on the DASH Index, because in the United States most monounsaturated fat comes from intake of meat; thus, the potential benefits of plant-source monounsaturated fat (a signature component of a Mediterranean diet) may be confounded by meat intake. Consistent with this, omission of the monounsaturated fatty acid:saturated fatty acid ratio strengthened rather than attenuated associations with T2D.

While higher-quality diets were associated inversely with T2D in all groups examined, Hispanic/Latina women appeared to benefit most from incremental increases in diet

quality compared with non-Hispanic white women. Other studies have also detected statistical differences by race/ethnicity. As noted above, Qiao et al. (6) found that AHEI was associated inversely with T2D only among white and Hispanic/Latina women in WHI. However, Qiao et al. used the original AHEI (we used the updated AHEI-2010) and included participants in the WHI Dietary Modification Trial (6), which could have biased results because of the trial's eligibility requirements ($> 32\%$ of energy derived from fat, a key component of the AHEI) and changes in diet due to the intervention. Similar to our findings, in the Multiethnic Cohort Study, Jacobs et al. (5) observed significant inverse associations between higher DASH scores and T2D among non-Hispanic whites, Japanese-American women, and Native Hawaiian men, even after BMI adjustment. However, higher scores on the AHEI-2010 and aMED indices were inversely associated only among non-Hispanic whites, and the HEI-2010 did not show an inverse association in any group (5). By contrast, in our study, all indices showed inverse associations with T2D in all racial/ethnic groups before adjustment for BMI at baseline; after adjustment for baseline BMI, all indices other than aMED and DASH maintained a statistically significant and inverse trend. After BMI adjustment, aMED score was nonsignificant only in black or Asian women and DASH score only in Asian women.

Clinical studies have suggested differences between African-American and Hispanic/Latina women as compared with non-Hispanic white women with respect to insulin sensitivity and β -cell responsiveness according to level of body fat, and also location of body fat depots (32–35). However, racial/ethnic differences in this study may reflect statistical differences in the incidence of T2D and distribution of the dietary indices by racial/ethnic group rather than biological differences. Racial/ethnic differences may also be driven by differing patterns of consumption in the foods making up the dietary indices, not just their distribution; for example, in the Multi-Ethnic Study of Atherosclerosis, Gao et al. (36) reported significant ethnic differences for all nutrients except saturated fat when intakes based on the DASH guidelines were compared between white, Chinese-American, African-American, and Hispanic adults. Independent of the underlying reason for racial/ethnic differences, the finding that these commonly used dietary indices had inverse associations with T2D regardless of the racial/ethnic group examined supports the content validity and potential utility of these indices in diverse populations.

Our study had several strengths. The prospective design of WHI and the large, ethnically diverse sample strengthened the internal and external validity of the findings and allowed for subgroup analyses by race/ethnicity and measured BMI. Our study also had limitations. T2D was self-reported rather than adjudicated. Self-reported diabetes in this cohort has been shown to be valid (28); however, our definition of T2D did not include those treated with diet and exercise alone. Additionally, since approximately 28% of cases of diabetes in the United States are undiagnosed (8), some diabetic individuals were probably misclassified as noncases; this would most likely have biased our results towards the null. Further, the indices, as well as the FFQs used to measure their components, were initially developed in majority European-descent populations; consumption of culturally specific foods

may not have been completely captured. The most likely impact of this misclassification would have been to underestimate associations among racial/ethnic minorities. Additionally, though a strength of this study was a sample size adequate to stratify results by race/ethnicity, in relative terms there were few Asian women ($n = 2,621$; 281 events). As in any observational study, the possibility of residual confounding by health consciousness remains even after careful control for many possible confounders. However, our results were robust to adjustment for measured confounders, including neighborhood socioeconomic status, and analyses excluding participants with prevalent chronic disease at baseline yielded near-identical results.

In conclusion, higher-quality diets as characterized by each of the 4 dietary indices were inversely associated with T2D in all racial/ethnic groups. While multiple factors (e.g., fruits, vegetables, and whole grains) overlap in these dietary indices, highlighted features also differ (e.g., inclusion of sugar-sweetened beverages or emphasis on fat quality). This suggests that while overall diet quality is beneficial for prevention of T2D, preventive interventions may be tailored to tastes, preferences, or cultures.

ACKNOWLEDGMENTS

Author affiliations: Departments of Nutrition and Epidemiology, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (Elizabeth M. Cespedes, Frank B. Hu); Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, Washington (Lesley Tinker, Marian L. Neuhouser); Channing Division of Network Medicine, Harvard Medical School and Department of Biostatistics, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (Bernard Rosner); Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, Massachusetts (Susan Redline); Department of Public Health Sciences, University of California Davis, Davis, California (Lorena Garcia); Department of Nutritional Sciences, University of Arizona, Tucson, Arizona (Melanie Hingle); Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, Illinois (Linda Van Horn); MedStar Health Research Institute, Hyattsville, Maryland (Barbara V. Howard); Department of Epidemiology, School of Public Health, University of Alabama, Birmingham, Alabama (Emily B. Levitan); Department of Medicine, University of Massachusetts Medical School, Amherst, Massachusetts (Wenjun Li); Division of Preventive Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts (JoAnn E. Manson); Atlanta Veterans Affairs Medical Center, Decatur, Georgia (Lawrence Phillips); Division of Endocrinology and Metabolism, Department of Medicine, School of Medicine, Emory University, Atlanta, Georgia (Lawrence Phillips); Division of Nephrology, Department of Medicine, School of Medicine, Stanford University, Palo Alto, California (Jinnie J. Rhee); and Departments of Quantitative Health Sciences and Obstetrics and Gynecology, University of Massachusetts Medical School, Worcester, Massachusetts (Molly E. Waring).

This work was supported by National Institute of Diabetes and Digestive and Kidney Diseases grants T32 DK007703 (E.M.C.) and KL2TR000160 (M.E.W.). The Women's Health Initiative is funded by the National Heart, Lung, and Blood Institute (contracts HHSN268201100046C, HHSN268201100001C, HHSN268201100002C, HHSN268201100003C, HHSN268201100004C, and HHSN271201100004C). Additional support was received from Food and Drug Administration grant RO1FD003527 (L.S.P.); Department of Veterans Affairs grant HSR&D IIR 07-138 (L.S.P.); National Institutes of Health grants R21DK099716 (L.S.P.), DK066204 (L.S.P.), U01DK091958 (L.S.P.), and U01DK098246 (L.S.P.); and Cystic Fibrosis Foundation grant PHILLI12A0 (L.S.P.).

Within the past several years, E.B.L. has received research support from Amgen, Inc. (Thousand Oaks, California), though the activity was unrelated to this project. L.S.P. has served on scientific advisory boards for Boehringer Ingelheim (Ingelheim am Rhein, Germany) and Janssen Pharmaceutica (Beerse, Belgium) and is receiving or has received research support from Merck & Company (Kenilworth, New Jersey), Amylin Pharmaceuticals (San Diego, California), Eli Lilly & Company (Indianapolis, Indiana), Novo Nordisk (Bagsvaerd, Denmark), Sanofi-Pasteur (Lyon, France), PhaseBio Pharmaceuticals, Inc. (Malvern, Pennsylvania), Roche, Inc. (Basel, Switzerland), and the Cystic Fibrosis Foundation (Bethesda, Maryland). In the past, L.S.P. was a speaker for Novartis International AG (Basel, Switzerland) and Merck but has not been for the last several years. He is also a cofounder of a company, DIASYST LLC (Atlanta, Georgia), which aims to develop and commercialize diabetes management software programs. These activities involve diabetes but had nothing to do with the current research.

REFERENCES

- Esposito K, Chiodini P, Maiorino MI, et al. Which diet for prevention of type 2 diabetes? A meta-analysis of prospective studies. *Endocrine*. 2014;47(1):107–116.
- Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol*. 2002;13(1):3–9.
- Center for Nutrition Policy and Promotion, US Department of Agriculture. Dietary Guidelines—2010. <http://www.cnpp.usda.gov/dietary-guidelines-2010>. Updated January 31, 2011. Accessed November 5, 2014.
- Center for Nutrition Policy and Promotion, US Department of Agriculture. *A Series of Systematic Reviews on the Relationship Between Dietary Patterns and Health Outcomes*. Alexandria, VA: US Department of Agriculture; 2014. <http://www.nel.gov/vault/2440/web/files/DietaryPatterns/DPRptFullFinal.pdf>. Accessed November 5, 2014.
- Jacobs S, Harmon BE, Boushey CJ, et al. A priori-defined diet quality indexes and risk of type 2 diabetes: the Multiethnic Cohort. *Diabetologia*. 2015;58(1):98–112.
- Qiao Y, Tinker L, Olenzki BC, et al. Racial/ethnic disparities in association between dietary quality and incident diabetes in postmenopausal women in the United States: the Women's Health Initiative 1993–2005. *Ethn Health*. 2014;19(3):328–347.
- Zamora D, Gordon-Larsen P, He K, et al. Are the 2005 Dietary Guidelines for Americans associated with reduced risk of type 2 diabetes and cardiometabolic risk factors? Twenty-year

- findings from the CARDIA study. *Diabetes Care*. 2011;34(5):1183–1185.
8. National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. *Diabetes Report Card 2014*. Atlanta, GA: Centers for Disease Control and Prevention; 2015. <http://www.cdc.gov/diabetes/pdfs/library/diabetesreportcard2014.pdf>. Updated 2015. Accessed August 15, 2015.
 9. The Women's Health Initiative Study Group. Design of the Women's Health Initiative clinical trial and observational study. *Control Clin Trials*. 1998;19(1):61–109.
 10. Hays J, Hunt JR, Hubbell FA, et al. The Women's Health Initiative recruitment methods and results. *Ann Epidemiol*. 2003;13(9 suppl):S18–S77.
 11. Langer RD, White E, Lewis CE, et al. The Women's Health Initiative Observational Study: baseline characteristics of participants and reliability of baseline measures. *Ann Epidemiol*. 2003;13(9 suppl):S107–S121.
 12. Block G, Hartman AM, Dresser CM, et al. A data-based approach to diet questionnaire design and testing. *Am J Epidemiol*. 1986;124(3):453–469.
 13. Patterson RE, Kristal AR, Tinker LF, et al. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. *Ann Epidemiol*. 1999;9(3):178–187.
 14. Schakel SF, Sievert YA, Buzzard IM. Sources of data for developing and maintaining a nutrient database. *J Am Diet Assoc*. 1988;88(10):1268–1271.
 15. George SM, Irwin ML, Smith AW, et al. Postdiagnosis diet quality, the combination of diet quality and recreational physical activity, and prognosis after early-stage breast cancer. *Cancer Causes Control*. 2011;22(4):589–598.
 16. Bowman SA, Friday JE, Moshfegh AJ. *MyPyramid Equivalents Database, 2.0 for USDA Survey Foods, 2003–2004: Documentation and User Guide*. Beltsville, MD: Agricultural Research Service, US Department of Agriculture; 2008. http://www.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/mped/mped2_doc.pdf. Updated September 2008. Accessed November 5, 2014.
 17. Trichopoulou A, Costacou T, Bamia C, et al. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med*. 2003;348(26):2599–2608.
 18. Fung TT, McCullough ML, Newby PK, et al. Diet-quality scores and plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr*. 2005;82(1):163–173.
 19. Guenther PM, Casavale KO, Reedy J, et al. Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet*. 2013;113(4):569–580.
 20. Guenther PM, Reedy J, Krebs-Smith SM. Development of the Healthy Eating Index-2005. *J Am Diet Assoc*. 2008;108(11):1896–1901.
 21. Chiuve SE, Fung TT, Rimm EB, et al. Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr*. 2012;142(6):1009–1018.
 22. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med*. 1997;336(16):1117–1124.
 23. Sacks FM, Obarzanek E, Windhauser MM, et al. Rationale and design of the Dietary Approaches to Stop Hypertension trial (DASH). A multicenter controlled-feeding study of dietary patterns to lower blood pressure. *Ann Epidemiol*. 1995;5(2):108–118.
 24. Fung TT, Chiuve SE, McCullough ML, et al. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern Med*. 2008;168(7):713–720.
 25. National Heart, Lung, and Blood Institute. *Lowering Your Blood Pressure With DASH*. Bethesda, MD: National Heart, Lung, and Blood Institute; 2009. http://www.nhlbi.nih.gov/files/docs/public/heart/dash_atglance.pdf. Updated August 2009. Accessed November 3, 2014.
 26. de Boer IH, Tinker LF, Connelly S, et al. Calcium plus vitamin D supplementation and the risk of incident diabetes in the Women's Health Initiative. *Diabetes Care*. 2008;31(4):701–707.
 27. Tinker LF, Bonds DE, Margolis KL, et al. Low-fat dietary pattern and risk of treated diabetes mellitus in postmenopausal women: the Women's Health Initiative randomized controlled dietary modification trial. *Arch Intern Med*. 2008;168(14):1500–1511.
 28. Margolis KL, Lihong Q, Brzyski R, et al. Validity of diabetes self-reports in the Women's Health Initiative: comparison with medication inventories and fasting glucose measurements. *Clin Trials*. 2008;5(3):240–247.
 29. Women's Health Initiative Clinical Coordinating Center. *Women's Health Initiative 2014 Annual Progress Report*. Seattle, WA: Fred Hutchinson Cancer Research Center; 2014. https://www.whi.org/researchers/data/_layouts/15/WopiFrame.aspx?sourcedoc=/researchers/data/WHI%20Progress%20Reports/2014%20Annual.pdf&action=default. Updated August 29, 2014. Accessed August 16, 2015.
 30. Jiang X, Zhang D, Jiang W. Coffee and caffeine intake and incidence of type 2 diabetes mellitus: a meta-analysis of prospective studies. *Eur J Nutr*. 2014;53(1):25–38.
 31. Shih RA, Ghosh-Dastidar B, Margolis KL, et al. Neighborhood socioeconomic status and cognitive function in women. *Am J Public Health*. 2011;101(9):1721–1728.
 32. Chandler-Laney PC, Phadke RP, Granger WM, et al. Adiposity and β -cell function: relationships differ with ethnicity and age. *Obesity (Silver Spring)*. 2010;18(11):2086–2092.
 33. Ingram KH, Lara-Castro C, Gower BA, et al. Intramyocellular lipid and insulin resistance: differential relationships in European and African Americans. *Obesity (Silver Spring)*. 2011;19(7):1469–1475.
 34. Lawrence JC, Newcomer BR, Buchthal SD, et al. Relationship of intramyocellular lipid to insulin sensitivity may differ with ethnicity in healthy girls and women. *Obesity (Silver Spring)*. 2011;19(1):43–48.
 35. Weiss R, Dziura JD, Burgert TS, et al. Ethnic differences in beta cell adaptation to insulin resistance in obese children and adolescents. *Diabetologia*. 2006;49(3):571–579.
 36. Gao SK, Fitzpatrick AL, Psaty B, et al. Suboptimal nutritional intake for hypertension control in 4 ethnic groups. *Arch Intern Med*. 2009;169(7):702–707.