Dietary patterns and breast cancer risk in women participating in the Black Women's Health Study[1–4]

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ABSTRACT

Background: No studies have examined dietary patterns and breast cancer risk in a large cohort of African American women.

Objective: We investigated the association between dietary patterns and breast cancer risk in the Black Women's Health Study.

Design: This is a prospective cohort study of 50,778 participants followed biennially from 1995 through 2007. During 443,742 person-years of follow-up, 1094 incident cases of breast cancer were identified. Factor analysis was used to derive food patterns based on 69 food variables. We used Cox regression models to obtain incident rate ratios (IRRs) for breast cancer in relation to quintiles of each of the 2 dietary patterns, with adjustment for other breast cancer risk factors.

Results: Through factor analysis, we identified 2 dietary patterns: Western (refined grains, processed meat, and sweets) and prudent (whole grains, vegetables, fruit, and fish). The prudent diet was weakly associated with lower breast cancer risk overall; *P* for trend = 0.06. In analyses stratified by body mass index (BMI; in kg/m^2), the prudent dietary pattern was associated with a significantly lower risk of breast cancer in women with a BMI <25 (IRR: 0.64; 95% CI: 0.43, 0.93; *P* for trend = 0.01). The prudent dietary pattern was also associated with a significantly lower risk of breast cancer in premenopausal women (IRR: 0.70; 95% CI: 0.52, 0.96; *P* for trend = 0.01), and we found a significant inverse association for the prudent dietary pattern and estrogen receptor–negative breast cancer (IRR: 0.52; 95% CI: 0.28, 0.94; *P* for trend <0.01).

Conclusion: Our findings suggest that the prudent dietary pattern may protect against breast cancer in some black women. *Am J Clin Nutr* 2009;90:621–8.

INTRODUCTION

Breast cancer is the leading cancer and the second leading cause of cancer death among women in the United States (1). Numerous epidemiologic studies have linked individual foods with risk of breast cancer (2). An increased risk of breast cancer has been shown to be associated with high intakes of red meat (3, 4), animal fat (5), and refined carbohydrates (6). Foods found to be inversely associated with breast cancer risk include fruit and vegetables (ie, dark yellow-orange and dark green) (6, 7), cereal and olive oil (4), and dairy products and dietary calcium (8). However, results from other studies contradict these findings (9–14). Dietary patterns may be more representative of the various foods consumed daily than individual food items, and studies of single nutrients and foods cannot disaggregate the individual effects of highly correlated foods (15). We identified 17 studies that examined dietary patterns and breast cancer risk (15–31). A reduced breast cancer risk has been shown to be associated with a "prudent" dietary pattern (16, 22), a traditional southern dietary pattern (15), "stew" and "traditional" dietary patterns (26), a salad vegetable pattern (27, 29), a vegetable/soy pattern (31), and Native Mexican and Mediterranean patterns (25). All of these dietary patterns were higher in fruit and vegetables and lower in animal fats than the typical "Western" diet. Increased breast cancer risk has been associated with a Western or Western-like dietary pattern (17, 18, 20–22), starch-rich pattern (19), high glycemic load pattern (32), fatty food pattern (22, 28), and an "alcoholic" drinker dietary pattern, characterized by intake of wine, liquor, and beer (30). Finally, one study found that a dietary pattern high in pork, processed meats, and potato was inversely associated with breast cancer risk (23). To our knowledge, the association between dietary patterns and breast cancer has not previously been studied in African American women. Studies suggest that African Americans consume more energy-dense foods and have lower intakes of vegetables, potassium, and calcium, which places them at greater risk of obesity, chronic disease, and cancer (33, 34). Also, premenopausal African Americans have a higher incidence of breast cancer, poorer prognostic tumor features, and higher breast cancer mortality than do white women (35). The objective of the present study was to identify major dietary patterns in a cohort of African American women and examine their associations with risk of breast cancer.

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SUBJECTS AND METHODS

The Black Women's Health Study (BWHS) is a prospective cohort study of African American women from across the United States. In 1995, 59,000 women aged 21–69 y enrolled by responding to health questionnaires mailed to subscribers of a popular lifestyle magazine (*Essence*) targeted to African American women, members of several African American professional associations, and friends of early respondents. Most respondents (88%) were subscribers to *Essence* magazine. Respondents completed 14-page questionnaires that probed for information on demographics, health status, and medical and lifestyle variables. The baseline questionnaire obtained information on adult height, current weight, demographic characteristics, reproductive history, medical history, use of medications, use of cigarettes and alcohol, and usual diet. Since 1995, follow-up questionnaires have been sent every 2 y to update information on reproductive history and other exposures and identify new occurrences of serious illnesses, such as breast cancer. Over 6 cycles of follow-up, response rates have averaged >80%.

Assessment of dietary intake

The food-frequency questionnaire (FFQ) included in the 1995 baseline questionnaire was based on the short National Cancer Institute's FFQ developed by Block et al (36). The FFQ was used to assess average food intake over the previous year. Based on pilot studies among BWHS participants, several additional food items were added to the short form, and the final FFQ contained 68 items. Details concerning the development of the questionnaire can be found elsewhere (36). The women were asked to choose from 9 frequency categories, ranging from "never or <1 per month" to "2 or more per day" for each food. Total caloric intake was calculated by summing intake from all foods. In the validation study, we found that the FFQ provided estimates of total fat, saturated fat, protein, carbohydrates, dietary fiber, calcium, iron, vitamin C, folate, and β-carotene that were well correlated with individual intakes assessed by food diaries and records (37). A separate question on the baseline questionnaire that was not part of the FFQ asked about alcoholic beverage consumption (drinks/wk).

Other variables

Data were collected on age, cigarette smoking status, years of education, alcohol intake, family history of breast cancer (in a mother or sibling), age at first birth, parity, weight, height, menopausal status, and strenuous exercise (h/wk). Body mass index (BMI; in kg/m^2) was calculated as weight/height^2. Data on education, height, and family history were collected in 1995. All of the other variables were updated with each biennial questionnaire.

Case ascertainment

Incident breast cancers were ascertained by self-report in the 1997, 1999, 2001, 2003, 2005, and 2007 questionnaires or through the National Death Index if the woman died of breast cancer before she was able to report the disease on a questionnaire. An attempt was made to obtain medical records for all participants who reported incident breast cancer. To date, we have obtained medical record or cancer registry data for 1151 incident cases and confirmed the diagnosis for 1144 (99.4%), which indicated that self-report of breast cancer is accurate in the BWHS (38). Estrogen and progesterone receptor status for cases were obtained from pathology reports. The procedures followed were in accordance with the ethical standards of Boston University School of Public Health and approval was obtained from Boston University Institutional Review Board.

Statistical analysis

For the present analysis, women were included if they completed the 1995 baseline FFQ, had missing data on ≤10 questions on the FFQ, had a total caloric intake in the range of 500 to 3800 kcal/d, had completed at least one follow-up questionnaire, and did not report breast cancer at baseline.

The 69 food items in our data (including alcohol) were suitably grouped into 29 foods groups (**Table 1**). A food that did not fit into any of the groups was left as an individual item or was not included. Principal factor analysis was used to derive food patterns (food clusters) based on the 29 food groups. PROC FACTOR in SAS version 9.1 with option METHOD = PRINCIPAL was used. The function ROTATE = VARIMAX was used for the

TABLE 1
Rotated factor loadings for food groups

	Dietary pattern	
Food group	Western	Prudent
Cruciferous vegetables	−0.01	0.65[1]
Other vegetables	−0.06	0.75[1]
Tomatoes	−0.06	0.48[1]
Fruit	−0.14	0.61[1]
Whole grains	−0.03	0.54[1]
Cereals	0.11	0.28
Low-fat dairy products	−0.07	0.39
Fish	0.09	0.48[1]
Poultry	0.19	0.36
Other fruit	0.22	0.11
Juice	0.01	0.34
Beverages	0.25	0.21
Refined grains	0.47[1]	0.23
High-fat dairy products	0.46[1]	0.08
Total meat	0.65[1]	−0.02
Processed meat	0.62[1]	−0.05
Eggs	0.41[1]	0.06
Margarine, butter, and mayonnaise	0.40[1]	−0.07
Chili	0.15	0.27
Potato	0.36	0.27
French fries	0.55[1]	−0.10
Sweets	0.47[1]	0.05
Soda	0.42[1]	−0.19
Soup	0.06	0.41[1]
Beans	−0.01	0.49[1]
Pasta	0.09	0.35
Snacks	0.45[1]	−0.04
Nuts	0.18	0.21
Alcohol	0.10	−0.01
Variance explained[2]	3.65	2.71

[1] Factor loading ≥0.40.
[2] Twenty-two percent of the variance is explained by the 2 dietary patterns.

rotation of the factors by an orthogonal transformation. A combination of eigenvalues (>1), the Scree test, and factor interpretability was used in determining the number of retained factors (21). A factor loading of ≥0.35 was used to identify the primary factor on which the items are loaded. For each woman, we calculated factor scores for each dietary pattern (29). The factor scores were derived by weighting each food group proportionally to its involvement in a dietary pattern. The more involved a variable is, the higher the weight. Variables unrelated to a dietary pattern would be weighted close to zero. To determine the score for a woman on a pattern, the woman's data on each food group were multiplied by the pattern weight for that food group. The sum of these products for all the food groups yields the factor score for the dietary pattern. Selected nutrients and food groups were regressed against quintiles of dietary patterns adjusted for age as a continuous variable (**Table 2**).

Menopausal status was classified as premenopausal, postmenopausal, or unknown. Women were classified as premenopausal if they had a hysterectomy but retained one or both ovaries and their current age was less than the 10th percentile of age at natural menopause (age: 43 y). Women were classified as postmenopausal if they were greater than the 90th percentile of age at natural menopause in the cohort (age: 56 y) and as unknown menopausal status if aged 43–56 y. Person-years of follow-up were computed for each participant as the amount of time since baseline in 1995 until the first of one of the following events: breast cancer diagnosis, loss to follow-up, death, and end of the 10-y follow-up period. PROC PHREG was used to fit questionnaire cycle–stratified Cox regression models to determine the multivariate-adjusted incidence rate ratios (with the corresponding 95% CIs) for the association between the dietary factors and breast cancer incidence. Quintiles of the 2 dietary factors were assessed, and the lowest quintile was used as the reference category.

TABLE 2
Age-adjusted baseline nutrients and food groups by quintile (Q)

	Western			Prudent		
Variable	Q1	Q3	Q5	Q1	Q3	Q5
Nutrient consumption						
Energy (kcal)	1056	1362	2453	1497	1493	2003
Cholesterol (mg)	137	190	318	206	233	256
Protein (g)	42.4	51.9	77.8	46.5	59.7	73.7
Total carbohydrates (g)	149	177	252	163	162	253
Total fat (g)	32.2	48.0	96.2	53.7	56.8	58.6
Total saturated fat (g)	9.4	15.3	28.6	17.4	16.9	18.1
β-Carotene (μg)	3657	3316	3676	1980	3811	5485
Fiber (g/d)	10.3	9.8	11.8	6.0	10.0	16.4
Lycopene (μg)	768	752	726	564	750	940
Folate (μg)	213	210	239	140	205	327
Food (servings/wk)						
Total vegetables	9	7	7	3	6	15
Fruit	10	7	7	3	8	14
Whole grains	5	4	4	2	3	7
Processed meat	1	3	5	4	2	3
Poultry	2	3	3	2	3	5
Meat	1	2	4	2	2	2
Fish	2	1	2	1	2	3
High-fat dairy products	4	7	16	7	8	7

The variables included in the Cox regression were age (continuous), BMI (continuous), alcohol intake (0, 1–3, 4–13, or ≥14 drinks/wk), education (≤12, 13–15, or ≥16 y), age at menarche (<11, 11–12, or ≥13 y), parity and age at first live birth (nulliparous; parity 1–2 and age at first birth <25 y; parity 1–2 and age at first birth 25–29 y; parity 1–2 and age at first birth ≥30 y; parity ≥3 and age at first birth <25 y; parity ≥3 and age at first birth 25–29 y; and parity ≥3 and age at first birth ≥30 y), family history of breast cancer (yes or no), strenuous physical activity (none, ≤2 h/wk, or >2 h/wk), energy intake (quintiles), menopausal status (pre- or postmenopausal), smoking status (never, former, or current), and female hormone use (yes or no). These covariates were selected based on known or potential associations with breast cancer risk. In this analysis, age, BMI, age at first live birth, parity, alcohol intake, strenuous physical activity, smoking history, menopausal status, and female hormone use were handled as time-varying covariates and henceforth updated biennially. Additional analyses were performed within strata of BMI at baseline (BMI ≥30, 25–29, or <25), smoking status at baseline (smoking ever or never), and women and estrogen receptor (ER) and progesterone receptor (PR) tumor status. The Andersen-Gill data structure was used to update the time-dependent covariates with the EXACT option in SAS used to handle tied event times. All analyses were conducted by using SAS statistical software (version 9.1; SAS Institute, Inc, Cary, NC).

RESULTS

Two major dietary patterns, which explained 22% of the variance in the food groups, emerged from factor analysis. The factor loading matrix for the 2 patterns, for which a positive loading indicates a positive association between the dietary pattern and the food group and a negative loading indicates an inverse association, is shown in Table 1. The first factor is labeled the "Western" dietary pattern and is characterized by a high loading of refined grains, high-fat dairy products, meat and processed meat, eggs, margarine, butter and mayonnaise, potato, French fries, sweets, soda, and snacks. The second factor is labeled the "prudent" dietary pattern and is characterized by higher intakes of cruciferous and other vegetables, fruit, whole grains, cereals, beans, low-fat dairy products, fish, and poultry. The 2 dietary patterns also emerged for pre- and postmenopausal women and explained 22.1% and 21.6%, respectively, of the variance in the food groups. Similarly, these 2 patterns also manifested among normal-weight (BMI <25), overweight (BMI 25–29), and obese (BMI ≥30) women, which explained 22.4%, 21.8%, and 21.6%, respectively, of the variance in the food groups.

Nutrients and food servings per week were assessed across quintiles for the Western and prudent dietary patterns (Table 2). Women in the highest quintile for the Western dietary pattern had higher intakes of total energy, protein, total fat, and saturated fat, whereas women in the highest quintile for the prudent dietary pattern had higher intakes of β-carotene, fiber, lycopene, and folate.

We assessed which potential risk factors for breast cancer were associated with obtaining a high score on the Western and prudent patterns (data not shown) by computing the mean standardized factor scores for categories of selected variables. Women who scored high on the Western dietary pattern were younger, weighed more, were less educated, were more often smokers and drinkers, and exercised less than those who scored low on this pattern. Compared with those who scored low on this dietary pattern, women with high prudent scores were older; more educated, less likely to smoke, and had higher levels of strenuous physical activity. Other potential risk factors for breast cancer (family history of breast cancer, parity, and age at first birth) did not differ in their associations with Western and prudent dietary patterns. Furthermore, from **Table 3**, ≈75% were nondrinkers, 65% were never smokers, and 77% were premenopausal. The participants' mean age and BMI were 38.5 y and 27.9, respectively.

During 443,742 person-years of follow-up from 1995 through 2007, 1094 incident breast cancer cases were identified. Of the 1094 case subjects, 558 (51%) were diagnosed before age 50 y. As shown in **Table 4**, the Western dietary pattern was not significantly associated with breast cancer risk. The prudent dietary pattern was nonsignificantly inversely associated with breast cancer risk: the IRR for quintile 5 relative to quintile 1 was 0.86 (95% CI, 0.68, 1.08), and the *P* for trend across quintiles was 0.06.

The Western dietary pattern was unrelated to breast cancer risk within strata of BMI (<25, 25–29, and ≥30) (**Table 5**). However, the prudent dietary pattern was inversely associated with breast cancer risk in women with a BMI <25: relative to quintile 1, the IRR was 0.64 (95% CI: 0.43, 0.93) for quintile 5 and the *P* for trend was 0.01. There were no significant associations among women with a BMI 25–29 or a BMI ≥30.

When we stratified by menopausal status (**Table 6**), among premenopausal women, the IRR for quintile 5 of the prudent dietary pattern relative to quintile 1 was 0.70 (95% CI: 0.52, 0.96), and the *P* for trend was 0.01; there was little evidence of a trend among postmenopausal women (*P* = 0.61). We also examined the association of dietary patterns with breast cancer risk within categories of smoking (data not shown). There were no trends across quintile of Western diet in ever smokers or never smokers, nor was there a trend across quintiles of prudent diet among ever smokers (data not shown). However, among never smokers for the prudent dietary pattern, the IRR was 0.54 (95% CI: 0.31, 0.93) for quintile 4 and 0.73 (95% CI: 0.42, 1.25) for quintile 5; the *P* for trend was 0.08.

We stratified by ER and PR status (data not shown). Hormone receptor status was available for 629 tumors, of which 400 (37%) were ER-positive, 229 (21%) were ER- negative, 317 (29%) were PR-positive and 304 (28%) were PR-negative. The Western dietary pattern was not associated with hormone receptor status. The prudent dietary pattern was inversely associated with ER-negative breast tumors; the IRR was 0.52 (95% CI: 0.28, 0.94) for quintile 5 relative to quintile 1, and the *P* for trend was <0.01. The same inverse pattern was found for PR-negative tumors and for the combination of ER-/PR-negative breast tumors: for quintile 5 relative to quintile 1, the IRRs were 0.66 (95% CI: 0.39, 1.09; *P* for trend = 0.03) and 0.66 (95% CI: 0.34, 1.26; *P* for trend = 0.04), respectively. The risk of ER-positive hormone receptor cancer was not associated with either dietary pattern.

TABLE 3
Baseline characteristics of Black Women's Health Study participants[1]

Variable	Value
Age (y)	38.5 ± 10.6[2]
BMI (kg/m^2)	27.9 ± 6.6
Education (%)	
≤12 y	17.6
13–15 y	36.1
≥16 y	46.1
Smoking (%)	
Never	65.1
Current	15.8
Former	19.0
Alcohol consumption (%)	
0 drinks/wk	75.1
1–3 drinks/wk	12.8
4–13 drinks/wk	10.2
≥14 drinks/wk	2.0
Family history of breast cancer (%)	6.5
Age at menarche (y)	12.3 ± 1.6
Age at first live birth (y)	22.3 ± 5.0
Menopausal status (%)	
Premenopausal	77.2
Postmenopausal	16.6
Strenuous physical activity (%)	
None	31.4
≤2 h/wk	38.3
>2 h/wk	27.1

[1] Percentages do not add to 100% because of missing values.
[2] Mean ± SD (all such values).

TABLE 4
Multivariate-adjusted incidence rate ratios (IRRs) and 95% CIs for breast cancer according to the 2 major dietary patterns by quintile (Q)

Dietary pattern	Cases	Person-years	Adjusted IRR[1] (95% CI)
Western			
Q1	239	89,113	1.00
Q2	241	89,075	1.10 (0.90, 1.33)
Q3	229	89,223	1.09 (0.89, 1.34)
Q4	200	88,536	1.01 (0.80, 1.27)
Q5	185	87,795	1.06 (0.81, 1.37)
P for trend[2]			0.86
Prudent dietary pattern			
Q1	182	87,582	1.00
Q2	202	88,780	0.97 (0.78, 1.20)
Q3	239	89,353	0.99 (0.79, 1.22)
Q4	214	89,554	0.80 (0.64, 0.99)
Q5	257	88,473	0.86 (0.68, 1.08)
P for trend[2]			0.06

[1] Model adjusted for age (continuous), BMI (continuous), alcohol intake (0, 1–3, 4–13, or ≥14 drinks/wk), education (≤12, 13–15, or ≥16 y), age at menarche (<11, 11–12, or ≥13 y), parity and age at first live birth (nulliparous; parity 1–2 and age at first birth <25 y; parity 1–2 and age at first birth 25–29 y; parity 1–2 and age at first birth ≥30 y; parity ≥3 and age at first birth <25 y; parity ≥3 and age at first birth 25–29 y; and parity ≥3 and age at first birth ≥30 y), family history of breast cancer (yes or no), strenuous physical activity (none, ≤2 h/wk, or >2 h/wk), energy intake (quintiles), menopausal status (pre- or postmenopausal), smoking status (never, former, or current), and female hormone use (yes or no).
[2] Obtained for each dietary pattern by including in the model a variable representing the median value for each quintile.

DISCUSSION

We found a weak inverse association between the prudent dietary pattern and overall breast cancer risk and found no

TABLE 5
Multivariate-adjusted incidence rate ratios (IRRs) and 95% CIs for breast cancer according to BMI and 2 major dietary patterns, by quintile (Q)[1]

	Cases	Person-years	Adjusted IRR[2] (95% CI)
BMI <25 kg/m^2			
Western dietary pattern			
Q1	83	30,811	1.00
Q2	65	29,013	1.14 (0.82, 1.56)
Q3	62	27,802	1.14 (0.81, 1.60)
Q4	43	26,591	0.86 (0.58, 1.28)
Q5	46	27,282	0.92 (0.59, 1.45)
P for trend[3]			0.49
Prudent dietary pattern			
Q1	55	28,165	1.00
Q2	56	28,140	0.76 (0.53, 1.08)
Q3	65	28,581	0.85 (0.60, 1.19)
Q4	51	28,751	0.61 (0.42, 0.88)
Q5	72	27,862	0.64 (0.43, 0.93)
P for trend[3]			0.01
BMI 25 to <30 kg/m^2			
Western dietary pattern			
Q1	79	31,453	1.00
Q2	86	31,352	0.99 (0.72, 1.38)
Q3	83	29,501	1.06 (0.74, 1.49)
Q4	78	28,906	1.03 (0.70, 1.50)
Q5	60	26,352	1.04 (0.66, 1.59)
P for trend[3]			0.89
Prudent dietary pattern			
Q1	61	27,143	1.00
Q2	70	29,518	1.06 (0.72, 1.57)
Q3	86	29,770	1.16 (0.79, 1.70)
Q4	75	30,605	0.95 (0.63, 1.41)
Q5	94	30,528	1.11 (0.73, 1.66)
P for trend[3]			0.88
BMI ≥30 kg/m^2			
Western dietary pattern			
Q1	77	26,849	1.00
Q2	90	28,710	1.19 (0.82, 1.70)
Q3	84	31,920	1.06 (0.72, 1.57)
Q4	79	33,039	1.14 (0.75, 1.73)
Q5	79	34,161	1.25 (0.78, 1.98)
P for trend[3]			0.47
Prudent dietary pattern			
Q1	66	32,274	1.00
Q2	76	31,122	1.17 (0.80, 1.70)
Q3	88	31,002	1.02 (0.69, 1.50)
Q4	88	30,198	0.96 (0.64, 1.42)
Q5	90	30,083	0.94 (0.62, 1.42)
P for trend[3]			0.44

[1] *P* value for BMI × prudent dietary pattern interaction = 0.61; *P* value for BMI × Western dietary pattern interaction = 0.63.

[2] Model adjusted for age (continuous), alcohol intake (0, 1–3, 4–13, or ≥14 drinks/wk), education (≤12, 13–15, or ≥16 y), age at menarche (<11, 11–12, or ≥13 y), parity and age at first live birth (nulliparous; parity 1–2 and age at first birth <25 y; parity 1–2 and age at first birth 25–29 y; parity 1–2 and age at first birth ≥30 y; parity ≥3 and age at first birth <25 y; parity ≥3 and age at first birth 25–29 y; and parity ≥3 and age at first birth ≥30 y), family history of breast cancer (yes or no), strenuous physical activity (none, ≤2 h/wk, or >2 h/wk), energy intake (quintiles), menopausal status (pre- or postmenopausal), smoking status (never, former, or current), and female hormone use (yes or no).

[3] Obtained for each dietary pattern by including in the model a variable representing the median value for each quintile.

association between the Western pattern and overall breast cancer risk. Results from other studies have been mixed. Some studies have found inverse associations of breast cancer risk with a prudent dietary pattern, or with patterns similar to the prudent diet (14, 19, 20, 22, 31), and positive associations with the Western dietary pattern (16, 19, 24, 26). In a Japanese study, women in the highest quartile of the prudent dietary pattern had a 27% decreased risk of breast cancer when compared with the lowest quartile (22). A study of US white women enrolled in the Breast Cancer Detection Demonstration Project found that the traditional southern dietary pattern (eg, cooked greens, cooked beans and legumes, cabbage, sweet potato, cornbread, and fried

TABLE 6
Multivariate-adjusted incidence rate ratios (IRRs) and 95% CIs for breast cancer according to menopausal status and 2 major dietary patterns, by quintile (Q)[1]

	Cases	Person-years	Adjusted IRR[2] (95% CI)
Premenopausal			
Western dietary pattern			
Q1	90	53,136	1.00
Q2	116	58,714	1.13 (0.87, 1.48)
Q3	103	60,931	1.08 (0.81, 1.43)
Q4	103	61,771	1.03 (0.76, 1.40)
Q5	97	63,197	1.14 (0.80, 1.61)
P for trend[3]			0.70
Prudent dietary pattern			
Q1	98	65,530	1.00
Q2	111	62,897	0.96 (0.73, 1.24)
Q3	116	60,222	0.97 (0.74, 1.26)
Q4	99	56,613	0.79 (0.59, 1.04)
Q5	85	52,487	0.70 (0.52, 0.96)
P for trend[3]			0.01
Postmenopausal			
Western dietary pattern			
Q1	119	25,853	1.00
Q2	99	20,667	1.03 (0.75, 1.42)
Q3	84	18,694	0.92 (0.64, 1.32)
Q4	77	17,417	0.90 (0.61, 1.34)
Q5	63	15,649	0.95 (0.60, 1.49)
P for trend[3]			0.62
Prudent dietary pattern			
Q1	53	12,945	1.00
Q2	66	16,062	1.00 (0.64, 1.57)
Q3	93	19,609	1.00 (0.64, 1.54)
Q4	83	23,495	0.77 (0.49, 1.21)
Q5	147	26,169	1.19 (0.76, 1.84)
P for trend[3]			0.66

[1] *P* value for menopause × Western dietary pattern interaction <0.0001; *P* value for menopause × prudent dietary pattern interaction <0.0001.

[2] Model adjusted for age (continuous), BMI (continuous), alcohol intake (0, 1–3, 4–13, or ≥14 drinks/wk), education (≤12, 13–15, or ≥16 y), age at menarche (<11, 11–12, or ≥13 y), parity and age at first live birth (nulliparous; parity 1–2 and age at first birth < 25 y; parity 1–2 and age at first birth 25–29 y; parity 1–2 and age at first birth ≥30 y; parity ≥3 and age at first birth <25 y; parity ≥3 and age at first birth 25–29 y; and parity ≥3 and age at first birth ≥30 y), family history of breast cancer (yes or no), strenuous physical activity (none, ≤2 h/wk, or >2 h/wk), energy intake (quintiles), smoking status (never, former, or current), and female hormone use (yes or no).

[3] Obtained for each dietary pattern by including in the model a variable representing the median value for each quintile.

chicken) was associated with a decreased risk, but only in postmenopausal women (15). Another study conducted in Uruguay identified 3 patterns—" traditional" (boiled meat, grains, cooked vegetables, and tubers), "healthy" (white meat, raw vegetables, cooked vegetables, and fruit), and "stew" (boiled meat and legumes)—that were significantly associated with reduced risk, whereas the Western dietary pattern was associated with an elevated risk (26). Finally, in a study of Hispanic and non-Hispanic women, both the Western and prudent dietary patterns were associated with an increased breast cancer risk, whereas the native Mexican dietary pattern (Mexican cheeses, soups, meat dishes, legumes, and tomato) and the Mediterranean dietary pattern were inversely associated with risk (25).

We found a significant inverse association of the prudent dietary pattern with breast cancer risk among women with a BMI <25, but no association in heavier women. These findings are generally consistent with those of other dietary pattern studies that stratified by BMI. One study found an inverse association of the "salad vegetables" dietary pattern with breast cancer risk among women with a BMI <25, whereas there was no evidence of a protective effect among women with a BMI >25 (29). Another study observed an interaction between BMI and the traditional southern dietary pattern, such that women with a high traditional southern dietary pattern and a BMI <25 had a greater reduction in breast cancer risk (15). Last, premenopausal women with a BMI <25 and who consumed a high native Mexican dietary pattern had greater reductions in breast cancer risk than did women with a BMI >25 (25). Although the mechanism is not clear, these results suggest that the protective effect of the prudent or traditional diet is present largely among thinner women.

We found a nonsignificant inverse association for prudent dietary pattern with breast cancer risk among never smokers and no association among women who had ever smoked. There was no association with Western diet regardless of smoking status. Results from other studies on effect modification by smoking are inconsistent (39–41). A study of US white women observed an inverse association between the prudent dietary pattern and postmenopausal breast cancer risk among smokers, but the test for interaction was not significant; the Western dietary pattern had a positive association with breast cancer risk among smokers at baseline (21). A study of Japanese women found that a prudent dietary pattern was associated with a significant inverse risk of nonsmokers and a nonsignificant inverse risk among current smokers (22).

We observed a significant inverse association of a prudent diet with breast cancer risk among premenopausal women. Evidence on potential effect modification by menopausal status is sparse and conflicting. Additionally, very few studies examined dietary patterns in premenopausal women (16). A recent study found a protective effect of a vegetable/soy dietary pattern in postmenopausal Asian American women (31). In a Chinese study, the "meat-sweet" pattern was associated with an increased risk among postmenopausal women (16), and a US study found a decreased risk of a pattern similar to the prudent diet with breast cancer risk among postmenopausal women (14).

We found a significant inverse association between the prudent dietary pattern and ER-negative, PR-negative, and ER−/PR− negative tumors. Very few studies assessed dietary patterns and nutrient according to ER/PR subtypes. One study observed a significantly lower risk of ER-negative tumors in women eating the prudent dietary pattern (21). Other studies reported inverse associations for high intakes of fruit and vegetables (42), lignans (24), and folate (43) and for healthy diet-quality indexes (44) with ER-negative tumors. These studies suggest that dietary patterns high in fruit, vegetables, and whole grains, which are rich sources of compounds such as isothiocyanates, antioxidants, flavonoids, folate, and phytoestrogens, all of which have been associated with a decreased cancer risk (45–47), may protect against ER-negative breast cancer. Our results support these findings. Hormone receptor–negative tumors are clinically aggressive, and women with these tumors have poorer survival rates (48, 49) Understanding the association between dietary factors and hormone receptor status is very important because premenopausal African American women have a higher prevalence of hormone receptor–negative tumors than do their white counterparts (48, 49).

Assessment of dietary patterns may prove to be more informative than assessment of individual food items, but the method has limitations. Factor analysis, which we used to identify dietary patterns, involves subjective decisions in selecting the food groups, number of factors, the method of rotation, and labeling of dietary patterns (23, 50). However, the dietary patterns identified in this study are similar to those that have been reported in several previous studies (17, 21, 29, 30). The strength of our study was its large sample of black women and its diversity of diets from across the United States. Another strength was the prospective design, ie, the collection of dietary data before the occurrence of the breast cancers precludes biased reporting. Although we used a validated FFQ, measurement error is a recognized limitation of FFQs. Imprecision in the reporting of individual items may have been overcome to some extent by our use of dietary patterns. Measurement errors, if random, would have tended to attenuate associations. Although we controlled for known confounders in the analyses, it is possible that our dietary patterns may be correlated with other lifestyle behaviors not measured. Furthermore, the prudent dietary pattern may be a proxy for a healthy lifestyle, because the women who scored high for this dietary plan were of normal weight, smoked less, and exercised more. Almost all women in the BWHS graduated from high school or completed a higher level of education; thus, whereas the study represents the educational levels of most US black women, it does not represent the ≈15% of black women nationally who have not graduated from high school (51).

In summary, in this first study of dietary patterns and risk of breast cancer in African American women, we found no overall association between the Western dietary pattern and breast cancer risk. There was an inverse association between the prudent dietary pattern and the overall breast cancer risk, with a stronger association for thinner women. The prudent dietary pattern was also inversely associated with premenopausal breast cancer and ER-negative and PR-negative tumors. Additional studies among African American women are needed to confirm or reject these results and to examine associations with the hormone receptor status of the tumor and the modification of effects by menopausal status, smoking, and other factors.

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