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Dietary patterns and breast cancer risk in Asian American women[1–3]

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ABSTRACT

Background: The role of diet as a cause of breast cancer in Asian Americans has not been adequately studied.

Objective: We investigated the association between dietary patterns and breast cancer risk in Asian Americans.

Design: This population-based case-control study in Los Angeles County compared dietary patterns between 1248 Asian American women with incident breast cancer and 1148 age-, ethnicity-, and neighborhood-matched controls. The relation between dietary patterns and serum concentrations of estrogens, androgens, and sex hormone–binding globulin (SHBG) was investigated in 2172 postmenopausal control women.

Results: We used a scoring method proposed by Trichopoulou et al (1) and found that adherence to a Mediterranean diet was inversely associated with risk; the odds ratio (OR) was 0.65 (95% CI: 0.44, 0.95) in women with the highest scores ($\geq$8; most adherent) compared with those with the lowest scores (0–3; *P* for trend = 0.009), after adjustment for key covariates. We also used factor analysis and identified 3 dietary patterns (Western-meat/starch, ethnic-meat/starch, and vegetables/soy). In a combined index of the 3 patterns, women who were high consumers of Western and ethnic meat/starch and low consumers of the vegetables/soy diets showed the highest risk (OR: 2.19; 95% CI: 1.40, 3.42; *P* for trend = 0.0005). SHBG concentrations were 23% lower in women with a high intake of the meat/starch pattern and a low intake of the vegetables/soy pattern than in those with a low intake of the meat/starch pattern and a high intake of the vegetables/soy pattern (*P* for trend = 0.069).

Conclusion: Our results suggest that a diet characterized by a low intake of meat/starches and a high intake of legumes is associated with a reduced risk of breast cancer in Asian Americans. *Am J Clin Nutr* 2009;89:1145–54.

INTRODUCTION

The role of diet in the etiology of breast cancer remains controversial despite extensive studies, most of which were conducted in Western white populations. The conclusions of most studies that have investigated risk associations in relation to specific macronutrients and micronutrients suggest that any effect of diet on breast cancer may be weak (2). However, several findings, including a positive association between red meat intake and risk in premenopausal women in the Nurses' Health Study (NHS) II (3), a role of dietary fat and risk in postmenopausal women in recent large cohort studies conducted in the United States (4) and Europe (5), and lower breast cancer risk in the dietary intervention arm of the Women's Health Initiative trial (6), emphasize the importance of identifying new avenues to study the role of diet and breast cancer. In the past decade, interest has shifted to dietary patterns, because they can accommodate the complex interplay of nutrients within a diet. Risk of breast cancer in relation to dietary patterns, identified using factor analysis, has been investigated in studies conducted in Western populations in the United States and Europe (7–13), in Uruguay (14), in Japan (15), and in China (16). These studies typically identified patterns that are labeled as "meat/starch" or "healthy or prudent." Results from these studies are mixed; the strongest evidence of any significant effects of dietary patterns is from the 3 case-control studies conducted in non-Western populations (14–16). Trichopoulou et al (1) also assessed risk patterns in relation to adherence to the Mediterranean diet using a linear 10-point dietary score. The main features of the Mediterranean diet are a high consumption of vegetables, fruit, legumes, and grains; a moderate consumption of dairy products and ethanol (mainly wine); and a low consumption of meat and meat products and potatoes. Adherence to a Mediterranean diet and risk of heart disease and cancer (17–21) has been investigated in different populations. The Mediterranean diet has much in common with the traditional Asian diet in that both diets are marked by a high intake of vegetables, fruit, and legumes and a low intake of fat and meat (22). If the dietary components of a Mediterranean diet have an etiologic role and are not markers of some other aspect of the Mediterranean lifestyle that is associated with risk, then demonstration of risk relations with a Mediterranean diet in a non-Mediterranean country or study population should strengthen the overall evidence of its role. Hence, we investigated the relation between dietary patterns and risk of breast cancer among Asian American women in Los Angeles County using factor analysis and the Mediterranean diet composite food score.

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SUBJECTS AND METHODS

Subjects

The methods used in this case-control study were described previously (23, 24). Briefly, this population-based case-control study included women who were identified as Chinese, Japanese, or Filipino between the ages of 25 and 74 y at the time of diagnosis of an incident breast cancer in the period January 1995 through December 2001. Cases were identified through the Los Angeles County Cancer Surveillance Program, the population-based cancer registry that is a member of the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program and the statewide California Cancer Registry. Of the 2221 women (784 Chinese, 585 Japanese, 852 Filipino women), 1384 (492 Chinese, 384 Japanese, 508 Filipinos) were interviewed, 507 (193 Chinese, 143 Japanese, 171 Filipinos) declined to be interviewed, 42 (8 Chinese, 15 Japanese, 19 Filipinos) had died, and 288 (91 Chinese, 43 Japanese, 154 Filipinos) could not be located.

The 1225 controls (514 Chinese, 331 Japanese, 380 Filipinos) were selected from the neighborhoods where the cancer cases resided at the time of diagnosis. A well-established, standard algorithm was used to identify neighborhood controls that the University of Southern California Epidemiology Program has used in numerous case-control studies (25). This algorithm defines a specified sequence of houses to be visited in the neighborhoods where index cases lived at the time of diagnosis. We sought to interview as the control the first eligible resident in the sequence. If the first eligible control subject refused to participate, the second eligible one in the sequence was asked, and so on. Letters were left when no one was home, and follow-up was by mail and telephone (if a number could be determined). Controls were sought to frequency-match to the cases on specific Asian ethnicities and 5-y age groups. On average, a suitable control was identified after contacting 65 households. Of the controls interviewed, 62% were the first identified eligible control, 21% were the second identified eligible control, and 17% were the third or later eligible control.

Data collection

In-person interviews were conducted by using a standardized structured questionnaire that covered demographic characteristics and migration history, menstrual and reproductive history, body size, physical activity, and diet history. Subjects were asked about their height and weight at age 18 y, at age 30 y, and at each decade thereafter. The food-frequency questionnaire (FFQ) we used was modeled after the validated diet instrument used in the Multiethnic Cohort Study being conducted in Hawaii and Los Angeles (26). The FFQ asked about usual intake of 174 foods and beverages. Subjects could select from 8 choices of frequency of intake (never, 1 time/mo, 2–3 times/mo, 1 time/wk, 2–3 times/wk, 4–6 times/wk, once per day, $\geq$2 times/d) and 3 choices of portion sizes. Subjects could select multiple servings of particular portion sizes and visual aids; different-sized bowls, plates, and glasses were displayed to facilitate the assessment.

Mediterranean Diet Score

Briefly, a value of zero or one is assigned to each of 6 presumed beneficial dietary components, based on whether the subject's intake level is below or above the median value (grams per 1000 kcal) for all controls. Conversely, a value of one or zero is assigned to each of 4 presumed detrimental dietary components, based on whether the subject's intake level is below or above the median value for all controls. The scores across the 10 dietary components are summed to form the Mediterranean Diet Score (range: 0–10), with a low score indicating nonadherence to a typical Mediterranean diet. The 6 beneficial dietary components are vegetables, legumes, fruit/nuts, cereals, fish and seafood, and a high ratio of monounsaturated lipids to saturated lipids. The 4 detrimental dietary components are meat products, milk/dairy products, carbohydrates, and alcohol (**Appendix A**).

Breast cancer risk is significantly and inversely associated with high soy intake during adolescence and adult life in this study population (23, 27). Thus, we also examined the joint effects of a lifetime soy exposure index and a modified Soy-Free Mediterranean Diet Score (all soy foods were excluded from the computational steps, which otherwise were identical to the original index) on breast cancer risk (**Table 1**, bottom).

Principal components analysis

We used factor analysis to identify distinct dietary patterns in control women. Extraction of principal components was followed by orthogonal rotation (28). Dietary intakes (in g/d) of each of the 174 food and beverage items were input variables to the principal components analysis (PCA). Based on an eigenvalue criteria $>$1.0, scree plot analysis, and factor interpretability, 3 factors (ie, dietary patterns) were identified. Factors were labeled descriptively according to the dietary pattern they generally represented: Western meat/starch (pattern 1), ethnic meat/starch (pattern 2), and vegetables/soy (pattern 3; **Appendix B**). The Western meat/starch pattern was characterized by pasta with meat, beef taco, beef burrito, pizza, meatballs, hamburger, fried potatoes, baked potatoes, mashed potatoes, pancake, bagels, and other items. The ethnic meat/starch pattern was characterized by pork/fish soups, liver, pork spareribs, salted/dried fish, fried shellfish, chicken wings, rice, fried/Spanish rice, fried noodles, fried dim sum, and other foods. The vegetables/soy pattern was characterized by green beans/peas, carrots, cabbage, bean sprouts, green peppers, bok choy, fresh tofu, fresh soybeans, soybean milk, and other items. For each dietary pattern, a component score was calculated as a linear composite of the foods with loading scores of $\geq$0.30 multiplied by the corresponding intake of the food (in g/d). To estimate risk of breast cancer associated with each of the 3 dietary patterns separately, we first categorized density scores (scores per 1000 kcal) for each pattern into quartiles according to their distributions among control women. The 3 dietary patterns in combination were then examined. We hypothesized that a high intake of meat/starch is associated with elevated breast cancer risk, and we assigned scores of 1, 2, 3, or 4 respectively to quartiles 1 to 4 for both the Western and ethnic meat/starch patterns. In addition, we hypothesized that a high intake of vegetables/soy is inversely associated with risk; therefore, for the vegetables/soy pattern, scores of -1, -2, -3, and -4, respectively, were assigned to quartiles 1 to 4. We examined risk associations in relation to the meat/starch patterns (ie, patterns 1 and 2) combined: a score of 8 represents women with a high (quartile 4) intake of both the Western meat/starch and ethnic meat/starch patterns, whereas a score of 2 represents

TABLE 1

Multivariate-adjusted relative risks (RRs) of breast cancer in relation to the Mediterranean Diet Score and the Soy-free Mediterranean Diet Score

Food score	Cases/controls	Adjusted RR (95% CI)	
		Model 1[1]	Model 2[2]
Mediterranean diet score			
0–3	294/257	1.00	1.00
4	258/215	0.97 (0.74, 1.26)	0.98 (0.75, 1.28)
5	249/224	0.93 (0.71, 1.21)	0.94 (0.72, 1.23)
6	224/212	0.83 (0.63, 1.10)	0.87 (0.66, 1.16)
7	138/143	0.75 (0.54, 1.03)	0.78 (0.57, 1.07)
≥8	78/94	0.65 (0.44, 0.95)	0.70 (0.48, 1.04)
P for trend		0.009	0.033
Soy-free Mediterranean diet score			
0–3	263/238	1.00	1.00
4	258/230	1.00 (0.74, 1.26)	0.97 (0.74, 1.26)
5	276/238	0.96 (0.76, 1.30)	0.99 (0.75, 1.29)
6	236/211	0.79 (0.73, 1.27)	0.97 (0.73, 1.29)
7	141/147	0.69 (0.58, 1.09)	0.81 (0.58, 1.12)
≥8	67/81	0.66 (0.46, 1.04)	0.72 (0.48, 1.09)
P for trend		0.078	0.14
Lifetime soy[3]			
Low/low	465/339	1.00	1.00
Low/high	178/150	0.87 (0.65, 1.16)	0.91 (0.68, 1.23)
High/low	175/170	0.71 (0.53, 0.95)	0.72 (0.54, 0.96)
High/high	423/486	0.57 (0.44, 0.75)	0.62 (0.48, 0.82)
P (3 df)		0.0003	0.003

[1] Adjusted for the following covariates: age, Asian ethnicity, education, birthplace, years of residence in the United States, years of physical activity, marital status, parity, age at menarche, type of menopause, age at menopause, and recent BMI (in quartiles).

[2] Adjusted for the same covariates as in model 1 plus waist-hip ratio, intake of tea (never, green tea only, black tea only, and green and black tea), and history of diabetes.

[3] "Low/low" indicates a less than weekly soy intake during adolescence and <6.24 mg isoflavones/1000 kcal in adult life, "low/high" indicates a less than weekly soy intake during adolescence and ≥6.24 mg isoflavones/1000 kcal in adult life, "high/low" indicates a greater than weekly or weekly soy intake during adolescence and <6.24 mg isoflavones/1000 kcal in adult life, and "high/high" indicates a greater than or weekly soy intake during adolescence and ≥6.24 mg isoflavones/1000 kcal in adult life.

subjects with a low (quartile 1) intake of both meat/starch patterns. Also, we investigated risk associations with all 3 patterns combined. In this combined analysis, a combined score of 7 is assigned to a woman with a high intake (quartile 4) of the Western meat/starch pattern (score: 4), a high intake (quartile 4) of the ethnic meat/starch pattern (score: 4), and a low intake (quartile 1) of the vegetables/soy pattern (score: −1). In contrast, a combined score of −2 is given to someone with a low intake (quartile 1) of the Western meat/starch pattern (score: 1), a low intake (quartile 1) of the ethnic meat/starch pattern (score: 1), and a high intake (quartile 4) of the vegetables/soy pattern (score: −4).

Blood hormone analysis

Study participants were asked to donate a blood specimen at the completion of the interview. Blood specimens were provided by 72% of cases and 64% of controls. For the blood hormone analysis in control women, we selected only those subjects who were naturally postmenopausal and were not using any menopausal hormones at the time of blood collection ($n = 221$).

Serum concentrations of estradiol (E2), estrone (E1), testosterone (T), and androstenedione (A) were measured by using previously described radioimmunoassay methods (29, 30). The interassay CVs were between 9% and 12%. Free [ie, nonprotein sex hormone–binding globulin (SHBG) and albumin-bound] testosterone and free E2 were calculated on the basis of measured total testosterone and total E2 concentrations, respectively, and SHBG concentrations and an assumed average concentration for albumin (31). This method has been found to have high validity compared with direct measurements (32). SHBG was measured by a chemiluminescent immunometric assay on the Immulite analyzer (Siemens Medical Solutions, Los Angeles, CA). The SHBG interassay CV was 8%.

Statistical analysis

Of the 1384 cases and 1225 controls we interviewed, we excluded 136 cases and 77 controls from the final analyses because of an incomplete interview (22 cases, 3 controls), previous cancer (44 cases, 35 controls), or missing information on body size, menstrual or pregnancy history, or one of the other adjustment covariates (70 cases, 39 controls). The results presented below are based on 1248 cases and 1148 controls who have complete information on FFQ as well as the covariates included for adjustment. We calculated odds ratios as estimates of relative risk (RR), their corresponding 95% CIs and *P* values by conditional logistic regression methods, with matched sets defined jointly by reference age (<39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, or ≥70 y) and specific Asian ethnicity (Chinese, Japanese, or Filipino). All regression models included the following covariates: years of residence in the United States (US born; >20 y, 11–20 y, or ≤10 y), education (less than high school, high school, some college, or college graduate), age at menarche (<12, 12–13, or ≥14 y), parity (0, 1, 2, 3, or ≥4 births), marital status (ever or never married), current body mass index (BMI; quartiles of controls), years of regular (ie, ≥1 h/wk) recreational physical activity (<5, 5–9, 10–19, or ≥20 h), total calories (continuous), menopausal status [premenopausal, natural menopause, bilateral oophorectomy, simple hysterectomy, hormone therapy (HT) menopause, ie, naturally menopausal but age unknown due to starting HT before periods had stopped], and age at menopause (≤44, 45–49, 50–54, or ≥55 y). Selected regression models included the following additional covariates: waist-hip ratio (WHR; quartiles of controls), intake of tea (none, black tea only, green tea only, or green and black tea), and history of diabetes (no or yes). We conducted case-control comparisons for all subjects combined and separately for premenopausal and postmenopausal women. To examine the potential effect modification of the diet score–breast cancer association by menopausal status, migration history, body size, and tea intake interaction terms were tested. *P* values <5% are considered statistically significant, and all *P* values quoted are 2-sided. All analyses were performed by using EPILOG Windows (version 1.01s) statistical software system (Pasadena, CA) and the SAS statistical software system (version 8.0; SAS Institute, Cary, NC).

Hormone measurements were transformed logarithmically to achieve approximate normal distributions for statistical analysis. Of the 221 postmenopausal control women, 9 had E1 values >125 pg/mL or E2 values >75 pg/mL (indicating postmenopausal hormone use) and were excluded from the analysis. Geometric mean values were computed for concentrations of serum estrogens (total and free E2 and E1), androgens (total and free testosterone and androstenedione) and SHBG. Analysis of covariance models were used to investigate the relation between geometric mean hormone concentrations and the various dietary patterns by including specific Asian ethnicity, years of residence in the United States (>20 y, 11–20 y, or ≤10 y), age at natural menopause, age at menarche, parity, recent BMI, WHR, and diabetes. We previously reported significantly lower SHBG concentrations and higher free testosterone concentrations in the control women who had diabetes than in those without diabetes after BMI, WHR, and other covariates were adjusted for (24).

RESULTS

The mean intake of the 10 food indexes used to construct the Mediterranean Diet Score among breast cancer cases ($n = 1248$) and control subjects ($n = 1148$) is shown in **Table 2**. To assess their influence on breast cancer risk, RRs of breast cancer per 1 SD (in controls) unit of change in the food indexes are shown. Risk of breast cancer decreased in association with a high intake of vegetables and legumes. Results were statistically significant for vegetables (RR per 89 g/1000 kcal was 0.90, $P = 0.027$) and legumes (RR per 30 g/1000 kcal was 0.87, $P = 0.0036$). In contrast, risk of breast cancer increased in association with a high intake of carbohydrates (RR per 81 g/1000 kcal was 1.10, $P = 0.038$) and meat (RR per 23 g/1000 kcal was 1.12, $P = 0.014$).

Breast cancer risk in association with the composite Mediterranean Diet Score is shown in Table 1 (top). In all subjects combined, there was a significant trend of decreasing risk with increasing score (*P* for trend = 0.009); women with the highest score (≥8) showed a significant 35% risk reduction compared with women with scores ≤3. Breast cancer risk in association, jointly, with an index of lifetime soy intake and a modified Soy-Free Mediterranean Diet Score (*see* above) is also shown in Table 1 (bottom). Breast cancer risk was inversely associated with a high soy intake ($P = 0.0003$) and with a high Soy-Free Mediterranean Diet Score (*P* for trend = 0.078); the latter finding was of borderline statistical significance. This inverse risk pattern remained after further adjustment for WHR, tea intake, and history of diabetes (second column of RRs).

TABLE 2
Levels of food indexes used in the composition of the Mediterranean Diet Score among breast cancer cases and controls and relative risks (RRs)

Food indexes[2]	Intake[1]: All cases ($n = 1248$)	Intake[1]: All controls ($n = 1148$)	Adjusted RR per 1 SD (95% CI)[3]
	$g \cdot 1000\ kcal^{-1} \cdot d^{-1}$		
Vegetables	128.5	138.2 ± 89	0.90 (0.82, 0.99)
Legumes	24.3	28.0 ± 30	0.87 (0.79, 0.95)
Fruits and nuts	148.7	148.5 ± 109	0.95 (0.86, 1.04)
Cereals	24.3	25.3 ± 42	0.94 (0.86, 1.03)
Fish/shellfish	18.3	18.2 ± 16	0.99 (0.90, 1.07)
Monounsaturated/saturated	1.25	1.27 ± 0.25	1.02 (0.87, 1.19)
Total carbohydrates	182.8	175.6 ± 81	1.10 (1.01, 1.21)
Dairy products	81.8	84.2 ± 86	0.99 (0.90, 1.08)
Meat	37.2	35.4 ± 23	1.12 (1.02, 1.22)
Alcohol	7.5	8.2 ± 3.7	0.99 (0.91, 1.08)

[1] All values are means or means ± SDs.
[2] *See* Appendix A.
[3] Adjusted for the following covariates: age, Asian ethnicity, education, birthplace, years of residence in the United States, years of physical activity, marital status, parity, age at menarche, type of menopause, age at menopause, and recent BMI.

PCA identified 3 distinct dietary patterns: Western meat/starch, ethnic meat/starch, and vegetables/soy (Appendix B). The 3 factors accounted for 21% of the variance in the 174 food and beverage items (ie, 7.7%, 7.2%, and 6.5% respectively for the 3 patterns). When we investigated risk associations with the 3 dietary patterns separately, breast cancer risk was not associated with increasing intake of the Western meat/starch pattern (*P* for trend = 0.27). However, breast cancer risk increased significantly with increasing intake of the ethnic meat/starch pattern (*P* for trend = 0.008) and decreased significantly with increasing intake of the vegetables/soy pattern (*P* for trend = 0.013; **Table 3**). The elevated risk was enhanced when we examined the intake of both the Western meat/starch and ethnic meat/starch patterns combined (patterns 1 + 2); the RR for the highest intake of both diets (score: ≥7) compared with the lowest intake of both diets (score: ≤3) was 1.61 (95% CI: 1.08, 2.39). When we examined all 3 dietary patterns combined, there was a significant trend of increasing risk with increasing scores (*P* for trend = 0.0005). Women with a high intake of the meat/starch pattern and a low intake of the vegetables/soy pattern (combined score: ≥6) showed a 2-fold increased risk (RR: 2.19; 95% CI: 1.40, 3.42) compared with women with a low intake of the meat/starch pattern and a high intake of the vegetables/soy pattern (combined score: ≤0). Risk associations were attenuated slightly when we further adjusted for WHR, tea intake, and history of diabetes (second column of RR). We evaluated the possibility of effect modification by menopausal status, body size, and intake of green tea. None of the interaction terms was statistically significant (data not shown).

We investigated the relations between circulating concentrations of SHBG, E1, E2, free E2, testosterone, and free testosterone and androstenedione and dietary patterns in a subset

TABLE 3
Relative risks (RRs) of breast cancer in relation to factor analysis–based patterns of food intake, by quartile (Q)

		Adjusted RR (95% CI)	
	Cases/controls	Model 1[1]	Model 2[2]
Pattern 1: Western meat/starch			
Q1 (score 1)	290/277	1.0	1.0
Q2 (score 2)	344/307	1.11 (0.86, 1.42)	1.09 (0.84, 1.41)
Q3 (score 3)	337/275	1.29 (0.98, 1.71)	1.26 (0.95, 1.66)
Q4 (score 4)	270/286	1.14 (0.83, 1.56)	1.10 (0.80, 1.51)
P for trend		0.27	0.40
Pattern 2: ethnic meat/starch			
Q1 (score 1)	245/294	1.0	1.0
Q2 (score 2)	282/277	1.24 (0.96, 1.60)	1.28 (0.99, 1.66)
Q3 (score 3)	336/287	1.44 (1.10, 1.87)	1.43 (1.10, 1.87)
Q4 (score 4)	378/287	1.46 (1.09, 1.95)	1.41 (1.05, 1.89)
P for trend		0.008	0.019
Pattern 3: vegetables/soy			
Q1 (score −1)	350/283	1.0	1.0
Q2 (score −2)	330/290	0.91 (0.71, 1.16)	0.94 (0.74, 1.20)
Q3 (score −3)	315/288	0.88 (0.68, 1.14)	0.92 (0.71, 1.20)
Q4 (score −4)	246/284	0.69 (0.52, 0.91)	0.72 (0.54, 0.96)
P for trend		0.013	0.039
Patterns 1 + 2			
Score			
≤3	123/141	1.00	1.00
4	208/216	1.10 (0.79, 1.53)	1.05 (0.75, 1.46)
5	418/394	1.28 (0.93, 1.75)	1.21 (0.88, 1.66)
6	322/274	1.40 (0.99, 1.96)	1.30 (0.92, 1.84)
≥7	170/120	1.61 (1.08, 2.39)	1.49 (1.00, 2.23)
P for trend		0.008	0.023
Patterns 1 + 2 + 3			
Score			
≤0	170/203	1.00	1.00
1	158/156	1.31 (0.95, 1.81)	1.26 (0.91, 1.74)
2	207/189	1.49 (1.09, 2.04)	1.44 (1.05, 1.98)
3	223/217	1.37 (1.00, 1.88)	1.30 (0.94, 1.79)
4	221/191	1.62 (1.15, 2.27)	1.51 (1.07, 2.12)
5	164/128	1.78 (1.22, 2.60)	1.63 (1.12, 2.39)
≥6	98/61	2.19 (1.40, 3.42)	2.03 (1.29, 3.19)
P for trend		0.0005	0.003

[1] Adjusted for the following covariates: age, Asian ethnicity, education, birthplace, years of residence in the United States, years of physical activity, marital status, parity, age at menarche, type of menopause, age at menopause, and recent BMI (in quartiles).

[2] Adjusted for the same covariates as in model 1 plus waist-hip ratio, intake of tea (never, green tea only, black tea only, or green and black tea), and history of diabetes.

of postmenopausal control women who were naturally menopausal and were nonusers of menopausal hormones (24). After adjustment for age, Asian ethnicity, education, birthplace, years living in the United States, age at menopause, age at menarche, number of births, recent BMI, and diabetes, SHBG concentrations increased significantly with increasing intake of a diet high in vegetables/soy (pattern 3; *P* for trend = 0.004). Although SHBG concentrations decreased with increasing intake of a diet rich in meat/starch (patterns 1 + 2); this finding was not statistically significant (*P* for trend = 0.39; **Table 4**).When we considered the intake of all 3 dietary patterns together, women with the highest scores (ie, high meat/starch and low vegetables/soy) had SHBG concentrations that were 23% lower (33.5 nmol/L) than those of women with the lowest score (43.7 nmol/L) (ie, low meat/starch and high vegetables/soy patterns); this finding was of borderline statistical significance (*P* for trend = 0.069). There were no clear associations between concentrations of estrogen (E1, E2, free E2), total testosterone, and free testosterone and androstenedione and the 3 dietary patterns combined. A high Mediterranean Diet Score had a borderline association with SHBG concentrations; women with Mediterranean Diet Scores ≥7 had a 28% higher SHBG concentration (39.8 nmol/L) than did women with low (0–3) scores (31.7 nmol/L; *P* for trend = 0.11; data not shown).

DISCUSSION

This study was one of the first to examine dietary patterns in relation to breast cancer risk in Asian American women—a group experiencing rapid increases in breast cancer incidence. Our results suggest that adherence to a Mediterranean diet may favorably influence the risk of breast cancer (Table 1; top); this finding cannot be explained by the effect of soy because a Mediterranean diet score that excluded soy intake had similar beneficial effects on risk, with only very slight attenuation in the risk estimates (Table 1; bottom). Vegetables and legumes were the individual components that were significantly associated with risk reduction, whereas total carbohydrates and all meats combined (red and white meats and processed meats combined) were the individual components that were significantly associated with risk elevation (Table 2). Comparable results were observed when we used the factor analysis technique to examine dietary pattern and breast cancer risk. A dietary pattern rich in vegetables/soy (pattern 3) was significantly inversely associated with risk, whereas a dietary pattern rich in meat/starches (patterns 1 + 2) was significantly positively associated with risk. The risk pattern in relation to a combined index representing the 3 dietary patterns was also significantly associated with risk. Women who were high consumers of meat/starches and low consumers of vegetables/soy had a significantly elevated breast cancer risk (Table 3). The generally similar conclusions reached by using the 2 different approaches to comprehensively describe the effects of diet on breast cancer risk are strengths of this study. The correlation between blood SHBG concentrations and dietary patterns in a subset of control women (Table 4) adds credibility to our overall results.

Before discussing the significance of these findings, several limitations of the study should be mentioned. One limitation was that we interviewed 63% of the cases we contacted. We compared cases we interviewed with those we did not interview in terms of

TABLE 4
Dietary patterns and circulating blood hormone concentrations in naturally postmenopausal control women, by quartile (Q)[1]

	SHBG	E1	E2	Free E2	Testosterone	Free testosterone	Androstenedione
	nmol/L	*pg/mL*	*pg/mL*	*pg/mL*	*ng/dL*	*pg/mL*	*pg/mL*
Pattern 3: vegetables/soy							
Q1 (*n* = 44)	33.3 ± 1.11	30.7 ± 1.08	9.5 ± 1.09	0.26 ± 1.16	18.2 ± 1.11	4.0 ± 1.14	457 ± 1.10
Q2 (*n* = 45)	31.5 ± 1.11	30.9 ± 1.08	10.2 ± 1.09	0.44 ± 1.16	17.4 ± 1.11	5.3 ± 1.14	517 ± 1.09
Q3 (*n* = 56)	38.2 ± 1.10	31.1 ± 1.07	10.3 ± 1.08	0.34 ± 1.14	19.0 ± 1.10	4.8 ± 1.12	512 ± 1.08
Q4, high (*n* = 51)	44.8 ± 1.10	33.5 ± 1.07	9.7 ± 1.08	0.32 ± 1.15	19.6 ± 1.10	4.0 ± 1.13	519 ± 1.09
P for trend	0.004	0.38	0.82	0.59	0.38	0.69	0.30
Patterns 1 + 2: Western/ethnic meat/starch							
Score							
≤3 (*n* = 29)	40.7 ± 1.13	32.0 ± 1.09	10.3 ± 1.10	0.28 ± 1.19	19.8 ± 1.13	4.2 ± 1.17	536 ± 1.11
4 (*n* = 30)	38.2 ± 1.13	30.4 ± 1.09	9.8 ± 1.10	0.32 ± 1.18	20.2 ± 1.13	4.5 ± 1.16	438 ± 1.10
5 (*n* = 62)	36.6 ± 1.11	33.8 ± 1.07	10.4 ± 1.08	0.31 ± 1.15	18.5 ± 1.11	4.7 ± 1.13	511 ± 1.09
6 (*n* = 56)	38.8 ± 1.11	31.3 ± 1.08	9.9 ± 1.09	0.40 ± 1.16	18.5 ± 1.11	4.8 ± 1.14	513 ± 1.09
7–8, high (*n* = 19)	33.7 ± 1.15	29.8 ± 1.11	9.1 ± 1.12	0.36 ± 1.22	16.2 ± 1.15	4.1 ± 1.19	499 ± 1.12
P for trend	0.39	0.69	0.59	0.17	0.21	0.93	0.95
Patterns 1+ 2 + 3							
Score							
≤0 (*n* = 33)	43.7 ± 1.13	29.9 ± 1.09	9.8 ± 1.10	0.26 ± 1.18	18.9 ± 1.13	3.7 ± 1.16	492 ± 1.11
1 (*n* = 33)	36.9 ± 1.12	34.1 ± 1.08	9.4 ± 1.09	0.30 ± 1.17	21.5 ± 1.12	5.1 ± 1.15	513 ± 1.10
2 (*n* = 29)	44.8 ± 1.13	32.9 ± 1.09	11.0 ± 1.10	0.41 ± 1.19	18.1 ± 1.13	4.3 ± 1.16	502 ± 1.11
3 (*n* = 33)	32.2 ± 1.13	33.8 ± 1.09	11.2 ± 1.10	0.42 ± 1.18	19.0 ± 1.13	5.3 ± 1.16	539 ± 1.10
4 (*n* = 38)	35.6 ± 1.12	27.6 ± 1.08	9.6 ± 1.10	0.40 ± 1.17	15.1 ± 1.12	4.3 ± 1.15	449 ± 1.10
≥5 (*n* = 30)	33.5 ± 1.13	32.5 ± 1.09	9.3 ± 1.10	0.28 ± 1.19	18.8 ± 1.13	4.4 ± 1.16	526 ± 1.11
P for trend	0.069	0.78	0.86	0.39	0.28	0.64	1.00

[1] All values are adjusted least-squares means ± SEMs. SHBG, sex hormone–binding globulin; E1, estrone; E2, estradiol. ANOVA models were used and adjusted for age, Asian ethnicity, education, birthplace, years of residence in the United States, age at menopause, age at menarche, number of births, recent BMI, and diabetes.

age, social class (based on census tract of residence), birthplace, and tumor stage at diagnosis and found few differences (23). The problem of measurement error in dietary assessment, particularly in relation to using FFQs in case-control studies, is well recognized. The possibility of bias is always a concern in case-control studies, but we believe this issue was lessened in the present analysis because it was less likely that the breast cancer patients selectively recalled differentially specific foods/food groups that are included in the 3 dietary patterns or are in the Mediterranean diet score because these are composite measures. Nondifferential misclassification would tend to bias our findings toward the null, which suggests that the results we observed may be even stronger. Whereas prospective cohort studies are preferred, few cohort studies that included multiple Asian ethnic groups have been conducted in the United States; only Japanese Americans were included in the Multiethnic Cohort Study in Hawaii and Los Angeles County (26). In addition, most cohort studies conducted in Western and Asian populations included few premenopausal women. Close to 50% of the breast cancer cases in this Asian American population were premenopausal at the time of cancer diagnosis (23).

Some differences exist between the Asian diet and the Mediterranean diet, most notably the lower intakes of cereals and olive oil/monounsaturated oil in the Asian diet and higher intakes of legumes. Thus, there is subjectivity involved in deciding which individual foods/food groups to include in each dietary component and the appropriate cutoffs. Although potatoes were commonly consumed in Western populations, this food group was not commonly consumed in our Asian American population; instead, we used a total carbohydrate component that included starches commonly consumed in a Western and an Asian diet. Using factor analysis to identify dietary patterns also requires arbitrary decisions including determining the number of dietary patterns, labeling the patterns, and interpreting these patterns. The 3 patterns only explained 21% of the variance; whereas this finding is comparable with the findings of other dietary studies, it underscores the challenges of reducing highly interrelated and complex dietary variables.

A significant beneficial effect of a Mediterranean diet on risk of cancer in women was recently reported in the Greek European Prospective Investigation into Cancer cohort; breast cancer accounted for 37% of the cancers in females in this study (17). Key dietary components of the Mediterranean Diet Score such as vegetables (33–35), legumes (36, 37), olive oil (35), meat (3, 38, 39), and carbohydrates (40, 41) implicated in our study were associated with breast cancer risk in some previous studies. Studies conducted in white populations in the United States and Europe reported few consistent or significant findings between breast cancer risk and dietary patterns (7–11, 13), whereas results from case-control studies conducted in Asian and other populations tend to be more supportive. In Japan, breast cancer risk reduced significantly in association with a high intake of a prudent diet (ie, represented by intake of vegetables, fruit, soybean curd, and fish) and increased in association with a "fatty diet" (represented by intake of meat, ham, sausage, and other fatty foods) (15). In Shanghai, China, high consumers of a "meat-sweet" dietary pattern (represented by meats, particularly pork, poultry, organ meats, beef, lamb, candy, dessert, and others) experienced a statistically significantly 30% elevated risk of breast cancer, but risk was unrelated to intake of the vegetable-soy pattern (represented by different vegetables, soy-based products, and freshwater fish) (16). In a study in the southwestern US, breast cancer risk in Hispanic and non-Hispanic white women was inversely associated with a native Mexican diet and the Mediterranean dietary pattern, whereas a Western diet pattern was associated with elevated risk (12). In Uruguay, risk of breast cancer doubled among those in the highest quartile of intake of a Western pattern (represented by fried meat, barbecue, and processed meat), whereas risk was reduced by about half among women in the highest quartile of intake of a healthy pattern (represented by white meat, raw vegetables, cooked vegetables, and total fruit) (14).

We found significantly lower SHBG concentrations in postmenopausal control women with a high intake of the meats/starch pattern and a low intake of the vegetables/soy pattern (patterns 1 + 2 + 3; Table 4); these results are compatible with findings from the NHS, which also reported lower concentrations of SHBG in association with the Western pattern, represented by higher intakes of red and processed meats, refined grains, sweets, and desserts. In Asian American women, the reduction in SHBG concentrations remained statistically significant after adjustment for BMI, whereas the findings weakened after adjustment for BMI in the NHS (42). Among Asian American women, we did not observe any significant associations between dietary patterns and circulating estrogen concentrations with or without adjustment for BMI. In the NHS, total estrogen and free estradiol concentrations were significantly higher in women with a high intake of the Western pattern diet, but these effects were largely eliminated after adjustment for BMI (42). Our findings on SHBG and dietary patterns contribute to the literature on possible effects of specific nutrients and food groups on circulating hormones and binding proteins (43–47). Additional investigation of the effects of dietary patterns on estrogen metabolism is warranted (48, 49).

Our study represents one of the first large population-based epidemiologic studies in Asian American women designed specifically to investigate the role of dietary factors and breast cancer risk. Our results suggest that the risk of breast cancer is lower among Asian women who are adherent to a Mediterranean diet. Results from our factor analysis also showed that soy/vegetable-rich diets are beneficial foods and meat/starch diets are risk-enhancing foods. Thus, changes in dietary patterns may have contributed, in part, to the increasing trend of breast cancer incidence in Asian Americans and in Asia. Additional cohort studies of dietary pattern and breast cancer risk in Asian populations are needed.

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REFERENCES

1. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med 2003;348:2599–608.
2. Michels KB, Mohllajee AP, Roset-Bahmanyar E, Beehler GP, Moysich KB. Diet and breast cancer: a review of the prospective observational studies. Cancer 2007;109:2712–49.
3. Cho E, Chen WY, Hunter DJ, et al. Red meat intake and risk of breast cancer among premenopausal women. Arch Intern Med 2006;166: 2253–9.
4. Thiebaut AC, Kipnis V, Chang SC, et al. Dietary fat and postmenopausal invasive breast cancer in the National Institutes of Health-AARP Diet and Health Study cohort. J Natl Cancer Inst 2007;99:451–62.
5. Sieri S, Krogh V, Ferrari P, et al. Dietary fat and breast cancer risk in the European Prospective Investigation into Cancer and Nutrition. Am J Clin Nutr 2008;88:1304–12.
6. Prentice RL, Caan B, Chlebowski RT, et al. Low-fat dietary pattern and risk of invasive breast cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA 2006;295:629–42.
7. Terry P, Suzuki R, Hu FB, Wolk A. A prospective study of major dietary patterns and the risk of breast cancer. Cancer Epidemiol Biomarkers Prev 2001;10:1281–5.
8. Velie EM, Schairer C, Flood A, He JP, Khattree R, Schatzkin A. Empirically derived dietary patterns and risk of postmenopausal breast cancer in a large prospective cohort study. Am J Clin Nutr 2005;82: 1308–19.
9. Adebamowo CA, Hu FB, Cho E, Spiegelman D, Holmes MD, Willett WC. Dietary patterns and the risk of breast cancer. Ann Epidemiol 2005; 15:789–95.
10. Fung TT, Hu FB, Holmes MD, et al. Dietary patterns and the risk of postmenopausal breast cancer. Int J Cancer 2005;116:116–21.
11. Männistö S, Dixon LB, Balder HF, et al. Dietary patterns and breast cancer risk: results from three cohort studies in the DIETSCAN project. Cancer Causes Control 2005;16:725–33.
12. Murtaugh MA, Sweeney C, Giuliano AR, et al. Diet patterns and breast cancer risk in Hispanic and non-Hispanic white women: the Four-Corners Breast Cancer Study. Am J Clin Nutr 2008;87:978–84.
13. Sieri S, Krogh V, Pala V, et al. Dietary patterns and risk of breast cancer in the ORDET cohort. Cancer Epidemiol Biomarkers Prev 2004;13: 567–72.
14. Ronco AL, De Stefani E, Boffetta P, Deneo-Pellegrini H, Acosta G, Mendilaharsu M. Food patterns and risk of breast cancer: a factor analysis study in Uruguay. Int J Cancer 2006;119:1672–8.
15. Hirose K, Matsuo K, Iwata H, Tajima K. Dietary patterns and the risk of breast cancer in Japanese women. Cancer Sci 2007;98:1431–8.
16. Cui X, Dai Q, Tseng M, Shu XO, Gao YT, Zheng W. Dietary patterns and breast cancer risk in the Shanghai Breast Cancer study. Cancer Epidemiol Biomarkers Prev 2007;16:1443–8.
17. Benetou V, Trichopoulou A, Orfanos P, et al. Conformity to traditional Mediterranean diet and cancer incidence: the Greek EPIC cohort. Br J Cancer 2008;99:191–5.
18. Bosetti C, Gallus S, Trichopoulou A, et al. Influence of the Mediterranean diet on the risk of cancers of the upper aerodigestive tract. Cancer Epidemiol Biomarkers Prev 2003;12:1091–4.
19. Dalvi TB, Canchola AJ, Horn-Ross PL. Dietary patterns, Mediterranean diet, and endometrial cancer risk. Cancer Causes Control 2007;18: 957–66.
20. Goulet J, Lamarche B, Nadeau G, Lemieux S. Effect of a nutritional intervention promoting the Mediterranean food pattern on plasma lipids, lipoproteins and body weight in healthy French-Canadian women. Atherosclerosis 2003;170:115–24.
21. La Vecchia C. Mediterranean diet and cancer. Public Health Nutr 2004; 7:965–8.
22. Woo J, Woo KS, Leung SS, et al. The Mediterranean score of dietary habits in Chinese populations in four different geographical areas. Eur J Clin Nutr 2001;55:215–20.
23. Wu AH, Yu MC, Tseng CC, Pike MC. Body size, hormone therapy and risk of breast cancer in Asian-American women. Int J Cancer 2007;120: 844–52.
24. Wu AH, Yu MC, Tseng CC, Stanczyk FZ, Pike MC. Diabetes and risk of breast cancer in Asian-American women. Carcinogenesis 2007;28: 1561–6.
25. Pike MC, Peters RK, Cozen W, et al. Estrogen-progestin replacement therapy and endometrial cancer. J Natl Cancer Inst 1997;89:1110–6.
26. Stram DO, Hankin JH, Wilkens LR, et al. Calibration of the dietary questionnaire for a multiethnic cohort in Hawaii and Los Angeles. Am J Epidemiol 2000;151:358–70.
27. Wu AH, Wan P, Hankin J, Tseng CC, Yu MC, Pike MC. Adolescent and adult soy intake and risk of breast cancer in Asian-Americans. Carcinogenesis 2002;23:1491–6.
28. Kline P. An easy guide to factor analysis. New York, NY: Routledge, 1994.
29. Goebelsmann UBG, Gale JA, Kletzky OA, Nakamura RM, Coulsom AH, Korelitz JJ. Serum gonadotropin, testosterone, estradiol and estrone levels prior to and following bilateral vasectomy. New York, NY: Academic Press, 1979:165.
30. Probst-Hensch NM, Pike MC, McKean-Cowdin R, Stanczyk FZ, Kolonel LN, Henderson BE. Ethnic differences in post-menopausal plasma oestrogen levels: high oestrone levels in Japanese-American women despite low weight. Br J Cancer 2000;82:1867–70.
31. Sodergard R, Backstrom T, Shanbhag V, Carstensen H. Calculation of free and bound fractions of testosterone and estradiol-17 beta to human plasma proteins at body temperature. J Steroid Biochem 1982;16:801–10.
32. Rinaldi S, Dechaud H, Toniolo P, Kaaks R. Reliability and validity of direct radioimmunoassays for measurement of postmenopausal serum androgens and estrogens. IARC Sci Publ 2002;156:323–5.
33. Riboli E, Norat T. Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk. Am J Clin Nutr 2003;78(suppl): 559S–69S.
34. Gandini S, Merzenich H, Robertson C, Boyle P. Meta-analysis of studies on breast cancer risk and diet: the role of fruit and vegetable consumption and the intake of associated micronutrients. Eur J Cancer 2000;36:636–46.
35. Trichopoulou A, Katsouyanni K, Stuver S, et al. Consumption of olive oil and specific food groups in relation to breast cancer risk in Greece. J Natl Cancer Inst 1995;87:110–6.
36. Wu AH, Koh WP, Wang R, Lee HP, Yu MC. Soy intake and breast cancer risk in Singapore Chinese Health Study. Br J Cancer 2008;99: 196–200.
37. Wu AH, Yu MC, Tseng CC, Pike MC. Epidemiology of soy exposures and breast cancer risk. Br J Cancer 2008;98:9–14.
38. Boyd NF, Stone J, Vogt KN, Connelly BS, Martin LJ, Minkin S. Dietary fat and breast cancer risk revisited: a meta-analysis of the published literature. Br J Cancer 2003;89:1672–85.
39. Taylor EF, Burley VJ, Greenwood DC, Cade JE. Meat consumption and risk of breast cancer in the UK Women's Cohort Study. Br J Cancer 2007;96:1139–46.
40. Gnagnarella P, Gandini S, La Vecchia C, Maisonneuve P. Glycemic index, glycemic load, and cancer risk: a meta-analysis. Am J Clin Nutr 2008;87:1793–801.
41. Romieu I, Lazcano-Ponce E, Sanchez-Zamorano LM, Willett W, Hernandez-Avila M. Carbohydrates and the risk of breast cancer among Mexican women. Cancer Epidemiol Biomarkers Prev 2004;13:1283–9.
42. Fung TT, Hu FB, Barbieri RL, Willett WC, Hankinson SE. Dietary patterns, the Alternate Healthy Eating Index and plasma sex hormone concentrations in postmenopausal women. Int J Cancer 2007;121:803–9.
43. Berrino F, Bellati C, Secreto G, et al. Reducing bioavailable sex hormones through a comprehensive change in diet: the diet and androgens (DIANA) randomized trial. Cancer Epidemiol Biomarkers Prev 2001; 10:25–33.
44. Newcomb PA, Klein R, Klein BE, et al. Association of dietary and life-style factors with sex hormones in postmenopausal women. Epidemiology 1995;6:318–21.
45. Setiawan VW, Haiman CA, Stanczyk FZ, Le Marchand L, Henderson BE. Racial/ethnic differences in postmenopausal endogenous hormones: the multiethnic cohort study. Cancer Epidemiol Biomarkers Prev 2006; 15:1849–55.
46. Wu AH, Pike MC, Stram DO. Meta-analysis: dietary fat intake, serum estrogen levels, and the risk of breast cancer. J Natl Cancer Inst 1999;91: 529–34.
47. Verkasalo PK, Thomas HV, Appleby PN, Davey GK, Key TJ. Circulating levels of sex hormones and their relation to risk factors for breast cancer: a cross-sectional study in 1092 pre- and postmenopausal women (United Kingdom). Cancer Causes Control 2001;12:47–59.
48. Aubertin-Leheudre M, Gorbach S, Woods M, Dwyer JT, Goldin B, Adlercreutz H. Fat/fiber intakes and sex hormones in healthy premenopausal women in USA. J Steroid Biochem Mol Biol 2008;112: 32–9.

49. Carruba G, Granata OM, Pala V, et al. A traditional Mediterranean diet decreases endogenous estrogens in healthy postmenopausal women. Nutr Cancer 2006;56:253–9.

APPENDIX A

Mediterranean Diet Score

Vegetables: spinach, Brussels sprouts, cauliflower, lettuce, green or bell peppers, celery, green beans, sprouts, corn, other green vegetables, other vegetables, coleslaw, broccoli, cabbage, bok choy, carrots, yellow-orange winter squash, tomatoes

Legumes: dried beans, chili with beans, bean burritos, refried beans, baked beans, other dried beans, fresh green soybeans, dried soybeans, fresh tofu, dried or pressed tofu

Fruit and nuts: orange, grapefruit or pomelo, apples, pineapple, banana, peach, apricots, prunes, tangerines, papayas, cantaloupe, watermelon, strawberries, other fresh or canned fruit, peanuts or other nuts

Cereals: bran or high-fiber cereals, other dry, cold cereal, cooked cereals, dark bread

Fish: raw fish, fried fish, all other fish cooked, canned tuna, other canned fish, salted and dried fish, fried shrimp, steamed fish, shellfish

Total carbohydrates: oriental noodles; rice gruel; white, brown, fried, Mexican, or Spanish rice; Inari sushi; other sushi or barazushi; fried noodles, such as chow mein; pasta or noodles with tomato sauce, with meat, cheese; pasta salad or somen salad; macaroni or potato salad; French-fried, mashed, scalloped, baked, or boiled white potato; white bread; other breads, including mixed grain, rolls, buns, bagels, or English muffins; blueberry muffin; pancakes

Dairy products: whole, low-fat, nonfat, or chocolate milk; milk-based drinks; yogurt; cottage cheese; low-fat cheese; other cheese; ice cream; ice milk

Meat: fast-food hamburger, hamburger patties, beef steak, pork chops, ham, bacon, luncheon, hot dogs, liver, chicken wings, other fried chicken, roasted chicken or duck, turkey

Alcohol: Hard liquor; white, pink, or red wine; regular or draft beer, light beer

APPENDIX B

Factor loadings of ≥0.30 based on principal components analysis of average daily intake of 174 food and beverage items

Food	Factor 1: Western meat/starch	Factor 2: ethnic meat/starch	Factor 3: vegetables/soy
Lettuce	0.48	—	—
Pasta with meat	0.48	—	—
Beef taco	0.47	—	—
Fried potatoes	0.44	—	—
Baked potatoes	0.43	—	—
Salad dressing	0.42	—	—
Pizza	0.41	—	—
Mashed potatoes	0.41	—	—
Enchilada	0.41	—	—
Beef burrito	0.40	—	—
Meat balls	0.40	—	—
Fruit pie	0.40	—	—
Hamburger	0.38	—	—
Pancake	0.37	—	—
Bagels	0.37	—	—
Cheese	0.36	—	—
Popcorn	0.36	—	—
Chili	0.35	—	—
Chips	0.35	—	—
Mayonnaise on bread	0.35	—	—
Sour cream potatoes	0.35	—	—
Refried beans	0.34	—	—
Luncheon meats	0.34	—	—
Cookies	0.33	—	—
Other sushi	0.33	—	—
Butter on potatoes	0.32	—	—
Bacon	0.32	—	—
Turkey	0.32	—	—
Pasta with tomato, no meat	0.31	—	—
Low-fat cheese	0.30	—	—
Sinigang (pork/fish vegetable soup)	—	0.62	—
Liver	—	0.59	—
Pork spareribs	—	0.54	—
Vegetable oil on liver	—	0.52	—
Rice	—	0.51	—

(Continued)

APPENDIX B (*Continued*)

Food	Factor 1: Western meat/starch	Factor 2: ethnic meat/starch	Factor 3: vegetables/soy
Filipino sweets	—	0.47	—
Vegetable oil on pork	—	0.45	—
Pineapple	—	0.45	—
Salted/dried fish	—	0.43	—
Fried/Spanish rice	—	0.43	—
Fried shellfish	—	0.43	—
Papayas/mangoes	—	0.41	—
Chicken wings	—	0.39	—
Fried noodles	—	0.38	—
Yellow orange winter squash	—	0.39	—
Fried dim sum	—	0.38	—
Diningding (pork/fish vegetable stew)	—	0.38	—
Hot dogs	—	0.36	—
Rolls	—	0.36	—
Taro or poi	—	0.33	—
Stew with poultry/fish	—	0.33	—
Other canned fish	—	0.33	—
Decaffeinated regular soda	—	0.32	—
Vegetable oil on beef	—	0.32	—
Beef, lamb, veal	—	0.31	—
Stew with red meat	—	0.30	—
Fried chicken	—	0.30	—
Green beans/peas	—	—	0.51
Carrots	—	—	0.50
Other green vegetables	—	—	0.49
Cabbage	—	—	0.48
Bean sprouts	—	—	0.48
Green peppers	—	—	0.46
Bok choy	—	—	0.46
Fresh tofu	—	—	0.45
Leafy greens	—	—	0.45
Broccoli	—	—	0.42
Cauliflower	—	—	0.41
Tomatoes	—	—	0.40
Celery	—	—	0.39
Fresh soybeans, cooked	—	—	0.38
Soybean milk	—	—	0.36
Watermelon	—	—	0.36
Stir-fried tofu and vegetables	—	—	0.34
Oranges	—	—	0.33
Apples	—	—	0.33
Yogurt	—	—	0.32