Research Article

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Dietary Patterns and the Risk of Colorectal Adenomas: the Black Women's Health Study

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Abstract

Background: Colorectal adenomas are benign lesions that may be precursors to colorectal cancer. No studies of African American women have investigated dietary patterns and the risk of developing colorectal adenomas. We examined data from the Black Women's Health Study to determine whether dietary patterns are associated with the risk of developing colorectal adenomas.

Methods: This is a prospective cohort study of 59,000 participants followed biennially since 1995. During 155,414 person-years of follow-up from 1997 to 2007 among women who had had at least one screening colonoscopy, 620 incident cases of colorectal adenomas were identified. By using Cox regression models, we obtained incidence rate ratios (IRR) for colorectal adenoma in relation to quintiles of each of two dietary patterns, adjusting for other colorectal adenoma risk factors.

Results: Two dietary patterns, Western and prudent, were utilized to assess the association between dietary intake and adenoma risk. The highest quintile of prudent diet, relative to the lowest quintile, was significantly associated with 34% lower colorectal adenoma risk overall (IRR = 0.66; 95% CI, 0.50–0.88; $P_{trend} < 0.01$). Higher scores on the Western pattern were associated with a higher risk of developing colorectal adenoma (IRR = 1.42; 95% CI, 1.09–1.85 for the highest quintile relative to the lowest; $P_{trend} = 0.01$).

Conclusion: Our findings suggest that African American women may be able to reduce their risk of developing colorectal adenomas by following a prudent dietary pattern and avoiding a more Western pattern.

Impact: A dietary modification could have a strong impact in colorectal adenoma prevention in African American women. *Cancer Epidemiol Biomarkers Prev; 20(5); 818–25. ©2011 AACR.*

Introduction

Colorectal cancer is the third leading cause of cancer incidence and death in the United States (1). An estimated 16,520 new cases of colorectal cancer were expected to be diagnosed among African Americans in 2009, and cancer at this site is the third most frequently diagnosed for African American women after lung and breast cancer (1). Colorectal cancer is the third leading cause of cancer death among African Americans, and it is estimated that African Americans are as much as 28% more likely to die of the disease than whites. Colorectal adenomas are recognized as precursors of colorectal cancer (2).

It is not clear why colorectal cancer incidence rates are higher among African Americans, but dietary factors may play a role (3). Certain dietary factors are thought to inhibit or promote the development and progression of colorectal adenomas and cancer (4, 5). There is epidemiologic evidence supporting a strong inverse association of fruits and vegetables against colorectal adenomas (6, 7). Other studies have found no significant association between fruits and vegetables and adenomas or colorectal polyps (8–10). Conversely, dietary fat and red meat have been associated with a higher risk of developing colorectal cancer and adenomas (11–15). Several studies have assessed dietary patterns in relation to incidence of colorectal adenoma or cancer. The prudent dietary pattern, characterized by higher intake of vegetables, fruits, whole grains, fish, and poultry, has been associated in some studies with a lower risk of developing colorectal cancer (16) or adenomas (17–19) but not in others (20–22). The Western dietary pattern, characterized by high fat dairy, meat, eggs, butter and fries, sweets, soda, and snacks, has been associated in some studies with a

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higher risk of developing colon cancer (16) or adenomas (23) but not in others (22). Survey data indicate that African Americans are less likely to eat a diet high in fruits, vegetables, and other components of a prudent dietary pattern than white Americans (24, 25).

A few studies have investigated the association between diet and colorectal cancer in African Americans. A study in North Carolina of older subjects examined dietary patterns in relation to colon cancer risk in 636 cases and 1,042 controls, of which 64 cases and 92 controls were African Americans (15). They found that the fruit and vegetable pattern was significantly inversely associated with colon cancer risk only in whites (OR = 0.4; 95% CI, 0.3–0.6). In a study of 99 African American colorectal cancer cases and 280 matched controls, Dales and colleagues (26) found that colon cancer cases were more likely to report high consumption of saturated fat-rich foods and less frequent consumption of foods high in dietary fiber; these findings were not statistically significant. In a case–control study by Satia-Abouta and colleagues (27) of 613 cases among African Americans, saturated fat was associated with a 2-fold higher colon cancer risk only in models that did not adjust for energy whereas dietary fiber was significantly associated with a lower risk regardless of whether adjustment was made for total energy.

Because adenomas are viewed as intermediate stages in the development of colorectal cancer, preventing the growth of adenomas will prevent colorectal cancer (28, 29). Our aim in this study was to identify major dietary patterns in a cohort of African American women and examine their associations with the incidence of colorectal adenomas.

Materials 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, a total of 59,000 women aged 21 to 69 years enrolled by responding to health questionnaires mailed to subscribers of Essence magazine, members of several African American professional associations, and friends of early respondents. 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 years to update information on reproductive history and other exposures and identify new occurrences of serious illnesses such as breast cancer. Follow-up of the baseline cohort has averaged more than 80% over 6 completed cycles of follow-up. Approval for the study was obtained from Boston University Institutional Review Board, and written consent was obtained from all participants.

Case ascertainment

Participants were asked about a list of diseases and the date of first diagnosis on the baseline and follow-up questionnaires. In 1999, a question on "colon or rectal polyps" was added to the list of illnesses. Pathology reports are sought for each participant reporting colorectal polyps and are reviewed to determine whether an adenoma was found. To date, in follow-up through 2007, there are 643 incident colorectal adenomas confirmed by medical records.

Assessment of dietary intake

A food frequency questionnaire (FFQ) based on the short National Cancer Institute's FFQ developed by Block and colleagues (30) was used to collect information on dietary intake (31). The FFQ assessed average food intake over the previous year and was administered in 1995 and 2001. Participants were asked how often (frequency) and how much (serving size) they consumed of each food. For frequency of consumption, the choice was from 9 frequency categories, ranging from "never or less than 1 per month" to "2 or more per day" for each food. For serving size in 1995, the options were small (0.5 times medium serving), medium, or large (1.5 times medium serving); a supersize option (2 times a medium serving) was added to the 2001 FFQ. The amount of weekly intake for each food item was calculated by multiplying the frequency of intake by the serving size. Total energy intake was calculated by summing intake from all foods. A validation study of BWHS participants showed good correlations between nutrients assessed by the 1995 FFQ, 24-hour dietary recalls, and food records over a 1-year period (31).

Assessment of other variables

Data on cigarette smoking, alcohol intake, weight, height, menopausal status, and female hormone use were collected on the baseline questionnaire and, except for height, updated on follow-up questionnaires every 2 years. Body mass index (BMI), a measure of obesity, was calculated as weight (in kilograms) divided by height (in meters squared). In the 1997, 1999, and 2001 questionnaires, participants provided information on the number of hours spent each week on vigorous exercise such as basketball, swimming, running, and aerobics. Information on education was obtained in 1995 and on family history of colorectal cancer (in a mother, father, or sibling) in 1999. Women were classified as premenopausal if they were still menstruating and as postmenopausal if they had a natural menopause (no periods for at least a year) or bilateral oophorectomy. Women who had a hysterectomy without removal of both ovaries were classified as premenopausal if their current age was less than the 10th percentile of age at natural menopause (age 43 years), as postmenopausal if their age was greater than the 90th percentile of age at natural menopause in the cohort (age 56 years), and as unknown menopausal status if age was 43 to 56 years.

Statistical analysis

For the present analysis, women were included if they completed the 1995 FFQ; had missing data on no more than 10 questions on the FFQ; had a total energy intake in the range of 500 to 3,800 kcal per day; had completed the 1997 follow-up questionnaire; reported colonoscopy or sigmodoscopy in 2003, 2005, or 2007; had not reported a diagnosis of cancer on the baseline or 1997 follow-up questionnaire; and had not reported a diagnosis of colorectal polyps on the baseline or 1997 follow-up questionnaire. There were 16,405 such women included in the present analyses, of whom 620 developed an incident colorectal adenoma during the course of follow-up.

Food items in our data were grouped into 33 food groups for both the 1995 and 2001 FFQs (32). Any food item, including alcohol, that did not fit into any of the groups was left as an individual food item. The food groups were adjusted for energy by dividing the intake of each food group/item by total energy and multiplying by 1,000 (33). Principal factor analysis was used to derive food patterns (food clusters) based on the 33 food groups. PROC FACTOR in SAS version 9.2 with option METHOD = PRINCIPAL was used. The function ROTATE = VARIMAX was used for the 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 (34). A factor loading an absolute value of 0.30 or greater was used to identify the primary factor on which the items are loaded. Other factor loading cutoffs have been used; for instance, Sieri and colleagues (35) used a loading of 0.25 or greater, and Velie and colleagues (36) used loadings of 0.2 or greater. For each participant, we calculated factor scores for each dietary pattern. 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.

Person-years of follow-up were computed for each participant as the amount of time since 1997, which was baseline for the present analyses, until the first of 1 of the following events occurred: colorectal adenoma or colorectal cancer diagnosis, loss to follow-up, death, and end of the 10-year follow-up period (these coincide with the times that observations were censored). PROC PHREG was used to fit questionnaire cycle-stratified Cox regression models to determine the multivariate adjusted incidence rate ratios (IRR) with the corresponding 95% CIs) for the association between the dietary patterns and breast cancer incidence. Quintiles of the 2 dietary pattern scores were assessed and the lowest quintile was used as the reference category. The 1995 dietary pattern score was updated in 2001 when the FFQ was again administered. The variables included in the Cox regression were age (continuous), BMI (continuous), education ($\leq$12, 13–15, $\geq$16 years), family history of colorectal cancer (yes/no), vigorous physical activity (none, $\leq$2, >2 h/wk), total energy intake (quintiles), menopausal status (pre-/postmenopausal), smoking history (nonsmoker, former smoker, and current smoker), alcohol intake (0, 1–3, $\geq$4 drinks per week), aspirin use (yes/no), and menopausal female hormone use (ever/never). These covariates were selected on the basis of known or potential associations with colorectal adenomas or cancer risk. In this analysis, age, BMI, alcohol intake, aspirin use, vigorous physical activity, smoking history, menopausal status, and female hormone use were handled as time-varying covariates and updated biennially. Further analyses were done within strata of age (younger than 50 years, and 50 years and older) and aspirin use (yes/no). 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 analysis was conducted using SAS statistical software (Version 9.2; SAS Institute, Inc.).

Results

Two dietary patterns, Western and prudent, identified previously in the BWHS (32), were utilized in this study. The Western dietary pattern was characterized by high loading on refined grains, high fat dairy, meat and processed meat, eggs, margarine, butter and mayonnaise, fries, sweets, soda, and snacks. The prudent dietary pattern was characterized by higher intakes of cruciferous and other vegetables, fruits, whole grains, cereals, beans, low fat dairy, fish, and poultry.

Table 1 shows the baseline distributions of selected colorectal adenomas risk factors by quintiles of dietary pattern score. Women who scored high on the Western pattern weighed more, were less educated, were more often smokers and aspirin users, were less often postmenopausal, and exercised less than those who scored low on this pattern. Women with high prudent scores were older, more educated, less likely to smoke, more often aspirin users and postmenopausal, and had higher levels of vigorous physical activity than those who scored low on this pattern. All trend tests for the variables in Table 1 except for family history were significant at $P < 0.05$,

Age-adjusted mean nutrient intakes were assessed across quintiles of the Western and prudent dietary pattern scores (Table 2). Women in the highest quintile of the Western dietary pattern had higher intakes of cholesterol, total fat, and saturated fat and lower intake of carbohydrate, β-carotene, and folate than women in lower quintiles, whereas women in the highest quintile for the prudent dietary pattern had lower intake of total energy, cholesterol, carbohydrate, fat, and saturated fat and higher intakes of β-carotene, fiber, and folate than women in lower quintiles. All trend tests across quintiles of dietary patterns were significant at $P < 0.05$.

Table 1. Baseline characteristics by quintiles of 1995 dietary patterns

Variable	Western			Prudent		
	Q_1	Q_3	Q_5	Q_1	Q_3	Q_5
Age, mean (SD), y	48.5 (9.2)	46.8 (8.9)	45.3 (9.0)	44.0 (8.5)	46.8 (8.8)	49.9 (9.0)
BMI, mean (SD), kg/m^2	27.8 (5.8)	28.9 (6.2)	29.5 (6.7)	29.3 (7.1)	28.9 (6.2)	28.1 (5.7)
Education, %						
≤12 y	12.4	16.9	26.1	20.5	17.0	17.9
13–15 y	24.8	32.7	37.9	31.4	33.1	30.8
≥16 y	62.8	50.5	36.0	48.2	48.3	51.0
Current smokers, %	7.6	14.5	22.7	16.9	15.2	12.8
Alcohol (≥4 drinks/wk), %	6.6	9.5	15.6	6.0	10.5	14.4
Aspirin user, %	16.9	17.3	19.4	15.6	16.6	20.1
Family history of colorectal cancer, %	2.8	2.4	2.6	2.4	3.0	3.0
Postmenopausal, %	37.0	31.8	28.6	22.5	31.2	43.0
Vigorous physical activity (>2 h/wk), %	25.5	15.8	10.1	11.4	17.0	22.8

NOTE: Baseline for the present analyses was 1997; $P_{trend} < 0.05$ across quintiles for all variables except family history.

During 155,414 person-years of follow-up from 1997 to 2007, a total of 620 incident colorectal adenomas were identified. As shown in Table 3, higher prudent diet score was associated with a lower risk of developing colorectal adenomas (with an IRR for fifth quintile relative to first quintile of IRR = 0.66; 95% CI, 0.50–0.88), and the P for trend across quintiles was less than 0.01. High Western diet score was associated with an increased risk of developing colorectal adenoma: for women in the fifth quintile relative to those in the first quintile (IRR = 1.42; 95% CI, 1.09–1.85) and the P value for trend across the quintiles was 0.01.

Stratifying on age (Table 4), the Western dietary pattern was significantly associated with an increased risk of developing colorectal adenomas in older (age ≥50 years) women (IRR = 1.53; 95% CI, 1.12–2.08) for quintile 5 relative to quintile 1 and $P_{trend} < 0.01$. The prudent pattern was also associated with a significant reduction in colorectal adenomas risk among older women (for quintile 5 relative to quintile 1, IRR = 0.58; 95% CI, 0.41–0.80; $P_{trend} < 0.01$). Neither pattern was significantly associated with the risk of developing adenomas among younger (age < 50 years) women. However, tests for interaction between dietary patterns and age were not significant (likelihood ratio tests for interaction: $P = 0.74$ between prudent pattern and age; $P = 0.88$ between Western pattern and age).

Stratifying on aspirin use (Table 5), the Western dietary pattern was associated with an increased risk of developing colorectal adenomas in aspirin users ($P_{trend} < 0.01$ across quintiles) and nonusers ($P_{trend} = 0.13$). The prudent pattern was associated with a reduction in colorectal adenoma risk among aspirin users ($P_{trend} = 0.04$) and nonusers ($P_{trend} = 0.03$). Tests for interaction between dietary patterns and aspirin use were not significant (likelihood ratio tests for interaction: $P = 0.69$ between

Table 2. Age-adjusted mean nutrient consumption by quintiles of dietary patterns

Variable	Western			Prudent		
	Q_1	Q_3	Q_5	Q_1	Q_3	Q_5
Energy, kcal	1,555	1,559	1,528	1,878	1,541	1,250
Cholesterol, mg	181	223	289	248	237	189
Protein, g	65	57	62	62	62	56
Total carbohydrates, g	217	199	166	237	189	167
Total fat, g	48	58	65	75	58	39
Total saturated fat, g	14	18	20	23	18	11
β-Carotene, μg	4,882	4,181	3,886	2,790	4,292	6,135
Fiber, g/d	14	11	9	9	11	14
Folate, μg	282	231	196	194	234	273

NOTE: $P_{trend} < 0.05$ across quintiles for all variables.

Table 3. Multivariate adjusted rate ratios for colorectal adenomas according to the 2 major dietary patterns

	Quintile	Cases	Person-years	Adjusted IRR (95% CI)[a]
Western	1	114	31,138	1.00
	2	120	31,109	1.11 (0.85–1.44)
	3	130	31,047	1.26 (0.97–1.63)
	4	119	31,047	1.20 (0.91–1.56)
	5	137	31,048	1.42 (1.09–1.85)
				$P_{trend} = 0.01$
Prudent	1	126	31,086	1.00
	2	126	31,102	0.93 (0.72–1.19)
	3	122	31,096	0.82 (0.63–1.07)
	4	131	31,045	0.82 (0.63–1.06)
	5	115	31,085	0.66 (0.50–0.88)
				$P_{trend} < 0.01$

[a]Adjusted for age, BMI, education, family history of colorectal cancer, vigorous physical activity, total energy intake, menopausal status, alcohol consumption, aspirin use, smoking status, and female hormone use.

Table 4. Multivariate adjusted rate ratios for colorectal adenomas according to age and 2 major dietary patterns

	Quintile	Cases	Person-years	Adjusted IRR (95% CI)[a]
Age <50 y				
Western	1	24	11,872	1.00
	2	36	13,552	1.21 (0.71–2.03)
	3	36	14,335	1.16 (0.68–1.97)
	4	42	15,515	1.22 (0.73–2.05)
	5	44	16,300	1.21 (0.71–2.03)
				$P_{trend} = 0.56$
Prudent	1	46	18,332	1.00
	2	46	16,643	1.10 (0.72–1.66)
	3	39	14,244	1.11 (0.71–1.73)
	4	28	12,486	0.87 (0.53–1.42)
	5	23	9,868	0.93 (0.54–1.58)
				$P_{trend} = 0.55$
Age ≥50 y				
Western	1	90	19,266	1.00
	2	84	17,557	1.07 (0.79–1.45)
	3	94	16,737	1.29 (0.96–1.74)
	4	77	15,532	1.17 (0.85–1.60)
	5	93	14,748	1.53 (1.12–2.08)
				$P_{trend} < 0.01$
Prudent	1	80	12,754	1.00
	2	80	14,459	0.85 (0.61–1.16)
	3	83	16,852	0.72 (0.52–0.99)
	4	103	18,559	0.77 (0.56–1.05)
	5	92	21,216	0.58 (0.41–0.80)
				$P_{trend} < 0.01$

[a]Adjusted for BMI, education, family history of colorectal cancer, vigorous physical activity, total energy intake, menopausal status, alcohol consumption, aspirin use, smoking status, and female hormone use.

Table 5. Multivariate adjusted rate ratios for colorectal adenomas according to aspirin use and 2 major dietary patterns

	Quintile	Cases	Person-years	Adjusted IRR (95% CI)[a]
Nonaspirin user				
Western	1	88	24,841	1.00
	2	100	25,034	1.16 (0.87–1.55)
	3	101	25,091	1.22 (0.90–1.63)
	4	91	24,849	1.14 (0.84–1.54)
	5	103	24,805	1.32 (0.97–1.78)
				$P_{trend} = 0.13$
Prudent	1	104	25,944	1.00
	2	96	25,370	0.89 (0.67–1.19)
	3	95	25,014	0.83 (0.62–1.11)
	4	99	24,343	0.83 (0.62–1.11)
	5	89	23,949	0.71 (0.51–0.96)
				$P_{trend} = 0.03$
Aspirin user				
Western	1	24	5,893	1.00
	2	20	5,647	0.97 (0.52–1.77)
	3	28	5,627	1.29 (0.83–1.56)
	4	27	5,812	1.17 (0.82–2.58)
	5	34	5,803	2.07 (1.18–3.63)
				$P_{trend} < 0.01$
Prudent	1	20	4,790	1.00
	2	30	5,363	1.23 (0.69–2.18)
	3	27	5,616	0.95 (0.52–1.73)
	4	31	6,244	0.84 (0.46–1.52)
	5	25	6,768	0.61 (0.32–1.15)
				$P_{trend} = 0.04$

[a]Adjusted for age, BMI, education, family history of colorectal cancer, vigorous physical activity, total energy intake, menopausal status, alcohol consumption, smoking status, and female hormone use.

prudent pattern and aspirin use; $P = 0.57$ between Western pattern and aspirin use).

Discussion

In this study, we utilized dietary patterns, Western and prudent, identified previously in the BWHS (32), that are consistent with patterns given in other published studies (16, 37). The 2 patterns were stable over the 2 questionnaire cycles, 1995 and 2001, in terms of the constituent food groups. This stability among food groups is in agreement with other longitudinal studies on dietary patterns (37, 38).

We found a strong inverse association between prudent dietary pattern and colorectal adenoma risk. A number of studies have also associated the intake of these foods with a decreased risk of developing colorectal adenomas (17–19, 21, 39, 40). We also found a positive association of Western dietary pattern with the risk of developing colorectal adenoma. Some studies have found positive associations of colorectal cancer or adenoma with Western pattern or dietary pattern similar to Western (11, 16, 21, 23, 41–43), whereas others have found no association between dietary patterns and colon cancer (20, 22, 40, 44–46). In our study, the association with Western pattern seemed stronger in older women, but a test for interaction by age was not statistically significant. Kesse and colleagues (23) found that high-risk adenomas were positively associated with the Western pattern in older women and not in younger women.

The prudent diet is characterized by a high intake of fruits, vegetables, and grains. Fruits and vegetables are rich in phytochemicals and antioxidant nutrients, such as the isothiocyanates and folate, which can prevent oxidative DNA damage and enhance DNA repair (37). In addition, the prudent diet is high in dietary fiber, which has been shown to reduce gastrointestinal transit time, increase bulk, and the binding of carcinogens (5, 47). The Western dietary pattern, especially red meat, may be linked to increased colorectal adenoma risk because of its effect on colorectal carcinogenesis (48, 49). Part of the biologic argument is that red meat contains high levels of heterocyclic amines and other mutagens that occur when cooking at high

temperatures. The formation of these substances may result in DNA modification that may promote mutation if not repaired (50).

Our study has a number of strengths. We used data from an FFQ that has been validated in our population (31). By using the dietary pattern approach versus analyzing the nutrient content of single foods, we increased the likelihood of capturing nutrient interactions and greater variation in dietary components because all foods made weighted contributions to the factor score (51). From the large number of variables in our study, we could control for known and suspected confounders in the relationship between dietary patterns and colorectal adenomas. The prospective design of our study reduced the opportunity for differential dietary recall to bias our findings. The use of 2 time point measures of dietary intake reduced intraindividual variation, leading to more precise effect estimates.

However, our study has some limitations as well. Factors analysis includes subjective judgments in determining the number of patterns, labeling the patterns, and interpreting these patterns (52). In addition, misclassification of dietary intake is a well-known problem of FFQs (53, 54) and likely would have tended to bias the risk estimates toward the null.

In summary, African American women are at high risk of developing colorectal cancer and there is a paucity of research that examined dietary factors and colorectal adenoma risk. Our results suggest that a prudent dietary pattern may reduce the incidence of colorectal adenomas and Western pattern may increase the incidence.

Disclosure of Potential Conflicts of Interest

The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Cancer Institute or NIH.

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References

1. American Cancer Society. Cancer Facts & Figures. American Cancer Society, ACS; 2009.
2. Cotton S, Sharp L, Little J. The adenoma-carcinoma sequence and prospects for the prevention of colorectal neoplasia. Crit Rev Oncog 1996;7:293–342.
3. Agrawal S, Bhupinderjit A, Bhutani MS, Boardman L, Nguyen C, Romero Y, et al. Colorectal cancer in African Americans. Am J Gastroenterol 2005;100:515–23; discussion 514.
4. Peipins LA, Sandler RS. Epidemiology of colorectal adenomas. Epidemiol Rev 1994;16:273–97.
5. Lipkin M, Reddy B, Newmark H, Lamprecht SA. Dietary factors in human colorectal cancer. Annu Rev Nutr 1999;19:545–86.
6. Michels KB, Giovannucci E, Chan AT, Singhania R, Fuchs CS, Willett WC, et al. Fruit and vegetable consumption and colorectal adenomas in the Nurses' Health Study. Cancer Res 2006;66:3942–53.
7. Miller PE, Lesko SM, Muscat JE, Lazarus P, Hartman TJ. Dietary patterns and colorectal adenoma and cancer risk: a review of the epidemiological evidence. Nutr Cancer 2010;62:413–24.
8. Smith-Warner SA, Elmer PJ, Fosdick L, Randall B, Bostick RM, Grandits G, et al. Fruits, vegetables, and adenomatous polyps: the Minnesota Cancer Prevention Research Unit case-control study. Am J Epidemiol 2002;155:1104–13.
9. Breuer-Katschinski B, Nemes K, Marr A, Rump B, Leiendecker B, Breuer N, et al. Colorectal adenomas and diet: a case-control study. Colorectal Adenoma Study Group. Dig Dis Sci 2001;46:86–95.
10. Mathew A, Peters U, Chatterjee N, Kulldorff M, Sinha R. Fat, fiber, fruits, vegetables, and risk of colorectal adenomas. Int J Cancer 2004;108:287–92.
11. Giovannucci E. Modifiable risk factors for colon cancer. Gastroenterol Clin North Am 2002;31:925–43.
12. Hawk ET, Limburg PJ, Viner JL. Epidemiology and prevention of colorectal cancer. Surg Clin North Am 2002;82:905–41.
13. Martinez ME. Primary prevention of colorectal cancer: lifestyle, nutrition, exercise. Recent Results Cancer Res 2005;166:177–211.
14. Miller PE, Lazarus P, Lesko SM, Muscat JE, Harper G, Cross AJ, et al. Diet index-based and empirically derived dietary patterns are associated with colorectal cancer risk. J Nutr 2010;140:1267–73.
15. Satia JA, Tseng M, Galanko JA, Martin C, Sandler RS. Dietary patterns and colon cancer risk in Whites and African Americans in the North Carolina Colon Cancer Study. Nutr Cancer 2009;61:179–93.
16. Fung T, Hu FB, Fuchs C, Giovannucci E, Hunter DJ, Stampfer MJ, et al. Major dietary patterns and the risk of colorectal cancer in women. Arch Intern Med 2003;163:309–14.
17. Peipins LA, Sandler RS. Epidemiology of colorectal adenomas. Epidemiol Rev 1994;16:273–97.
18. Giovannucci E, Stampfer MJ, Colditz G, Rimm EB, Willett WC. Relationship of diet to risk of colorectal adenoma in men. J Natl Cancer Inst 1992;84:91–8.
19. Neugut AI, Jacobson JS, De VI. Epidemiology of colorectal adenomatous polyps. Cancer Epidemiol Biomarkers Prev 1993;2:159–76.
20. Slattery ML, Boucher KM, Caan BJ, Potter JD, Ma KN. Eating patterns and risk of colon cancer. Am J Epidemiol 1998;148:4–16.
21. Wu K, Hu FB, Fuchs C, Rimm EB, Willett WC, Giovannucci E, et al. Dietary patterns and risk of colon cancer and adenoma in a cohort of men (United States). Cancer Causes Control 2004;15:853–62.
22. Dixon LB, Balder HF, Virtanen MJ, Rashidkhani B, Männistö S, Krogh V, et al. Dietary patterns associated with colon and rectal cancer: results from the Dietary Patterns and Cancer (DIETSCAN) Project. Am J Clin Nutr 2004;80:1003–11.
23. Kesse E, Clavel-Chapelon F, Boutron-Ruault MC. Dietary patterns and risk of colorectal tumors: a cohort of French women of the National Education System (E3N). Am J Epidemiol 2006;164:1085–93.
24. Kant AK, Graubard BI. Ethnicity is an independent correlate of biomarkers of micronutrient intake and status in American adults. J Nutr 2007;137:2456–63.
25. Fulgoni V, III, Nicholls J, Reed A, Buckley R, Kafer K, Huth P, et al. Dairy consumption and related nutrient intake in African-American adults and children in the United States: continuing survey of food intakes by individuals 1994–1996, 1998, and the National Health And

Nutrition Examination Survey 1999–2000. J Am Diet Assoc 2007; 107:256–64.
26. Dales LG, Friedman GD, Ury HK, Grossman S, Williams SR. A case-control study of relationships of diet and other traits to colorectal cancer in American blacks. Am J Epidemiol 1979;109:132–44.
27. Satia-Abouta J, Galanko JA, Potter JD, Ammerman A, Martin CF, Sandler RS, et al. Associations of total energy and macronutrients with colon cancer risk in African Americans and Whites: results from the North Carolina colon cancer study. Am J Epidemiol 2003;158: 951–62.
28. Schatzkin A, Freedman LS, Dawsey SM, Lanza E. Interpreting precursor studies: what polyp trials tell us about large-bowel cancer. J Natl Cancer Inst 1994;86:1053–7.
29. Lanza E, Yu B, Murphy G, Albert PS, Caan B, Marshall JR, et al. The polyp prevention trial continued follow-up study: no effect of a low-fat, high-fiber, high-fruit, and -vegetable diet on adenoma recurrence eight years after randomization. Cancer Epidemiol Biomarkers Prev 2007;16:1745–52.
30. Block G, Hartman AM, Naughton D. A reduced dietary questionnaire: development and validation. Epidemiology 1990;1:58–64.
31. Kumanyika SK, Mauger D, Mitchell DC, Phillips B, Smiciklas-Wright H, Palmer JR, et al. Relative validity of food frequency questionnaire nutrient estimates in the Black Women's Health Study. Ann Epidemiol 2003;13:111–8.
32. Agurs-Collins T, Rosenberg L, Makambi K, Palmer JR, ms-Campbell L. Dietary patterns and breast cancer risk in women participating in the Black Women's Health Study. Am J Clin Nutr 2009;90:621–8.
33. Reedy J, Wirfält E, Flood A, Mitrou PN, Krebs-Smith SM, Kipnis V, et al. Comparing 3 dietary pattern methods–cluster analysis, factor analysis, and index analysis–With colorectal cancer risk: The NIH-AARP Diet and Health Study. Am J Epidemiol 2010; 171:479–87.
34. Fung TT, Hu FB, Holmes MD, Rosner BA, Hunter DJ, Colditz GA, et al. Dietary patterns and the risk of postmenopausal breast cancer. Int J Cancer 2005;116:116–21.
35. Sieri S, Krogh V, Pala V, Muti P, Micheli A, Evangelista A, et al. Dietary patterns and risk of breast cancer in the ORDET cohort. Cancer Epidemiol Biomarkers Prev 2004;13:567–72.
36. Velie EM, Schairer C, Flood A, He JP, Khattree R, Schatzkin A, et al. Empirically derived dietary patterns and risk of postmenopausal breast cancer in a large prospective cohort study. Am J Clin Nutr 2005;82:1308–19.
37. Hu FB, Rimm E, Smith-Warner SA, Feskanich D, Stampfer MJ, Ascherio A, et al. Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. Am J Clin Nutr 1999;69:243–9.
38. Newby PK, Weismayer C, Akesson A, Tucker KL, Wolk A. Long-term stability of food patterns identified by use of factor analysis among Swedish women. J Nutr 2006;136:626–33.
39. Witte JS, Longnecker MP, Bird CL, Lee ER, Frankl HD, Haile RW, et al. Relation of vegetable, fruit, and grain consumption to colorectal adenomatous polyps. Am J Epidemiol 1996;144:1015–25.
40. Mizoue T, Yamaji T, Tabata S, Yamaguchi K, Shimizu E, Mineshita M, et al. Dietary patterns and colorectal adenomas in Japanese men: the Self-Defense Forces Health Study. Am J Epidemiol 2005;161:338–45.
41. Steinmetz KA, Potter JD. Food-group consumption and colon cancer in the Adelaide Case-Control Study. II. Meat, poultry, seafood, dairy foods and eggs. Int J Cancer 1993;53:720–7.
42. Shannon J, White E, Shattuck AL, Potter JD. Relationship of food groups and water intake to colon cancer risk. Cancer Epidemiol Biomarkers Prev 1996;5:495–502.
43. Kim MK, Sasaki S, Otani T, Tsugane S. Dietary patterns and subsequent colorectal cancer risk by subsite: a prospective cohort study. Int J Cancer 2005;115:790–8.
44. Butler LM, Wang R, Koh WP, Yu MC. Prospective study of dietary patterns and colorectal cancer among Singapore Chinese. Br J Cancer 2008;99:1511–6.
45. Randall E, Marshall JR, Brasure J, Graham S. Dietary patterns and colon cancer in western New York. Nutr Cancer 1992;18:265–76.
46. 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.
47. Potter JD, Slattery ML, Bostick RM, Gapstur SM. Colon cancer: a review of the epidemiology. Epidemiol Rev 1993;15:499–545.
48. Norat T, Lukanova A, Ferrari P, Riboli E. Meat consumption and colorectal cancer risk: dose-response meta-analysis of epidemiological studies. Int J Cancer 2002;98:241–56.
49. Norat T, Bingham S, Ferrari P, Slimani N, Jenab M, Mazuir M, et al. Meat, fish, and colorectal cancer risk: the European Prospective Investigation into cancer and nutrition. J Natl Cancer Inst 2005;97: 906–16.
50. Martínez ME, Jacobs ET, Ashbeck EL, Sinha R, Lance P, Alberts DS, et al. Meat intake, preparation methods, mutagens and colorectal adenoma recurrence. Carcinogenesis 2007;28:2019–27.
51. Flood A, Rastogi T, Wirfalt E, Mitrou PN, Reedy J, Subar AF, et al. Dietary patterns as identified by factor analysis and colorectal cancer among middle-aged Americans. Am J Clin Nutr 2008;88:176–84.
52. Martinez ME, McPherson RS, Levin B, Glober GA. A case-control study of dietary intake and other lifestyle risk factors for hyperplastic polyps. Gastroenterology 1997;113:423–9.
53. Magkos F, Manios Y, Babaroutsi E, Sidossis LS. Differences in the quantitative and qualitative performance of a calcium-specific food frequency questionnaire across age and sex. J Hum Nutr Diet 2006;19:331–42.
54. Byrne C, Ursin G, Ziegler RG. A comparison of food habit and food frequency data as predictors of breast cancer in the NHANES I/NHEFS cohort. J Nutr 1996;126:2757–64.