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Fat, Fiber, Meat and the Risk of Colorectal Adenomas

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OBJECTIVE: The aim of this study was to determine the relationship between fat, fiber, and meat intake, and risk of colorectal adenoma recurrence.

METHODS: We determined adenoma recurrence and dietary intake for 1,520 participants in two randomized trials: The Antioxidant Polyp Prevention Study and Calcium Polyp Prevention Study. Subjects underwent baseline colonoscopy with removal of all adenomas, and dietary intake was estimated with a validated semiquantitative food frequency questionnaire. Follow-up colonoscopy was performed 1 and 4 yr later. Pooled risk ratios for adenoma recurrence were obtained by generalized linear regression, with adjustment for age, sex, clinical center, treatment category, study, and duration of observation.

RESULTS: In the total colorectum, fiber intake was weakly and nonsignificantly associated with a risk for all adenomas (RR quartile 4 *vs* quartile 1 = 0.85, 95% CI 0.69–1.05) and advanced adenomas (RR = 0.88, 95% CI 0.54–1.44). Associations were stronger for adenomas in the proximal colon (RR = 0.73, 95% CI 0.56–0.97) and some fiber subtypes (fruit and vegetable, grain). There was no association between fat or total red meat intake and risk of adenoma or advanced adenoma recurrence. However, when considering other meats, risk (quartile 4 *vs* quartile 1) for advanced adenoma was increased for processed meat (RR = 1.75, 95% CI 1.02–2.99) and decreased for chicken (RR = 0.61, 95% CI 0.38–0.98).

CONCLUSION: The inverse associations between fiber intake and risk of adenoma recurrence we observed are weak, and not statistically significant. Our data indicate that intake of specific meats may have different effects on risk.

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INTRODUCTION

Diet has long been studied as an important and modifiable exposure in the genesis of colorectal cancer (CRC) and numerous studies have been performed to better define the relationship between intake of macronutrients and the risk of colorectal neoplasia. Results of recent large cohort studies looking at the relationship of fiber intake and CRC have been inconsistent (1–3). While an extensive review determined that the literature "supports" an inverse association between dietary fiber intake and CRC risk (4), prospective intervention studies of fiber and colorectal adenomas (a CRC precursor) have not shown a reduction in risk (5–7). Findings regarding intake of fat are similarly inconsistent, with some large cohort studies suggesting a positive association between fat intake and colon carcinogenesis (8) and others (9–11) not.

The reason for the lack of consistency across these studies is not clear. It is possible that differences in study design or endpoint (cancer *vs* adenomas) account for the varying findings. Alternatively, the true effect size of dietary intake of fat and fiber may be small or the various associations observed may be chance findings. Finally, various studies have used different instruments to measure diet, possibly leading to some variation in results. To explore further the role of dietary fiber and fat and the development of colorectal adenomas, we used data from patients enrolled in two large randomized controlled trials (12, 13) and examined the independent associations of dietary intake of macronutrients and specific foods with adenoma development. We reasoned that the large sample size and the careful follow-up would permit us to determine associations between diet and adenoma incidence.

METHODS

Details of study design and data collection have been reported for each study (12, 13). Both were multicentered randomized double blind clinical trials, conducted with almost identical protocols (though different agents). To be eligible for either study, patients had to have a history of at least one large bowel adenoma excised within the 3 months before entry, and, on the basis of a colonoscopy, confirmed free of further polyps in the entire large bowel. Major exclusion criteria included a history of invasive CRC, or conditions that might be worsened by the study agents. In both studies, complete colonoscopic follow-up was planned for 1 yr and 4 yr after the qualifying examination, and the predetermined primary endpoint was the recurrence of adenomas during the 3-yr period between the year 1 and year 4 colonoscopy (*i.e.*, incident polyps).

The Antioxidant Polyp Prevention Study (13) randomized subjects to β-carotene (25 mg) or placebo and a combination of vitamin C and vitamin E (1 g and 400 mg, respectively) or placebo. A total of 864 subjects were randomly assigned to the four treatment arms. Of these, 709 subjects had two follow-up colonoscopies and complete dietary information and are included in this analysis.

The Calcium Polyp Prevention Study (12), was a clinical trial of calcium supplementation (1.2 g elemental calcium as carbonate). A total of 930 subjects were randomized. 811 subjects with two follow-up examinations and complete dietary information were included in this analysis.

In both trials, dietary patterns were assessed at baseline with a self-administered semiquantitative food frequency questionnaire, developed by the National Cancer Institute (14). This instrument previously has been validated by others (15–17). The survey requested information regarding usual diet over the prior year and included 100 food items (plus open-ended questions for frequently eaten, unlisted foods). Overall, 1,794 subjects were randomized into the two studies and 98% (n = 1,765) returned the baseline dietary questionnaire. Of 1,765 questionnaires received, 48 were excluded because the participant indicated eating less than 3 foods/day, skipped greater than 50 foods on the grid or had a calculated total caloric intake greater than 5,000 kcal or less than 500 kcal. Nutrient intake was estimated using software developed by the National Cancer Institute in connection with the questionnaire (18).

Statistical Analyses

Overdispersed generalized linear models for the Poisson distribution as an approximation to the binomial family were used to compute relative risks of at least one adenoma. Primary analyses were directed at major macronutrients (total and saturated fat, protein), cholesterol and fiber. Given the heterogenous nature of fiber, we also examined fiber by subtype (fruit and vegetable, grain, bean). Finally, we examined specific types of meat intake (poultry, fish, processed meat) as well as well as a ratio of red meat to chicken and fish, as suggested by Willett *et al.* (8).

Models were adjusted for age, sex, clinical center, treatment category, study, and the duration of the observation period. Additional models controlling for alcohol intake, tobacco use, and BMI were also run but yielded essentially identical results and are not presented here. In nutrient analyses, we computed quartiles of the residuals of the regression of the logarithm of the nutrient on the logarithm of kilocalories and used the logarithm of caloric intake to adjust for energy intake (19). The p values for trend were obtained using the regression coefficient estimates for continuous residuals adjusted for the log of caloric intake.

The primary analysis examined the risk of at least one adenoma recurrence in the entire large bowel. Secondary analyses addressed the occurrence of advanced adenomas which were defined as tubulovillous adenomas (25–74% villous component), villous adenomas (75% or more villous), those containing advanced dysplasia or invasive cancer, or adenomas with an estimated diameter equal to or larger than 1 cm (as assessed by the endoscopist). Analyses examining the relationship of nutrient intake to proximal lesions (those in the cecum, ascending colon, hepatic flexure, and transverse colon, n = 159 in the antioxidant study and n = 195 in the calcium study) were also performed.

To assess whether study (*i.e.*, antioxidant *vs* calcium) modified the association between nutrient intake and adenoma risk, we used product interaction terms and the Wald test. We found no evidence of significant effect modification, and so we present only the combined analysis controlling for study as a covariate. Analyses among placebo subjects yielded results similar to those including all subjects with adjustment for treatment; only the latter are presented here.

RESULTS

The baseline characteristics of the study populations (*i.e.*, from the two trials) were generally similar (Table 1). The median age was about 61 yr at study entry and approximately 35% of subjects developed incident adenomas during the main risk period (*i.e.*, year 1 to year 4) of the study. In the antioxidant study, females accounted for 21.6% of subjects, *versus* 28% in the subsequent calcium trial, the result of an effort to enhance female recruitment.

No association was found between fat intake and adenoma risk (Table 2). When considering those in the highest quartile of total fat intake to the lowest, the RR for all adenomas was 0.98 (95% CI 0.81–1.19) and for advanced adenomas 1.12 (95% CI 0.69–1.82). Saturated fat and cholesterol intake were separately analyzed, but there was no association between increasing intake and adenoma recurrence. Findings in the proximal colon paralleled those for the total colorectum. Caloric intake was not clearly associated with the risk for all adenomas (RR for quartile 4 *vs* quartile 1 = 1.12, 95% CI 0.91–1.38) or advanced adenomas (1.40, 95% CI 0.85–2.32) in the trials (Table 2).

Table 1. Description of Study Cohort and Baseline Dietary Information

	Antioxidant Polyp Prevention Study (n = 709)	Calcium Polyp Prevention Study (n = 811)	Combined (n = 1,520)
Mean age (SD)	61.1 (8.3)	60.9 (8.9)	61.0 (8.6)
Male, n (%)	556 (78.4)	584 (72%)	1,140 (75%)
With recurrent adenoma, n	260	280	540
With recurrent advanced adenoma, n	61	74	135
Mean intake per day (SD)			
Energy (kcal)	1,970 (758)	2,021 (749)	1,997 (753)
Total fat (g)	87 (43)	87 (41)	87 (42)
Saturated fat (g)	30 (16)	29 (15)	26 (15)
Protein (g)	76 (29)	77 (31)	77 (30)
Total dietary fiber (g)	14.4 (7.3)	16.6 (8.0)	16 (8)
Mean servings per week (SD)			
Red meat	3.7 (3.0)	3.3 (2.6)	3.5 (2.8)
Red meat : White meat ratio	3.4 (4.8)	3.2 (4.9)	3.3 (4.9)

There was an inverse association between total dietary fiber and risk of adenomas that was not statistically significant overall (p value for trend = 0.07) (Table 2). For all adenomas, the RR for quartile 4 *versus* quartile 1 was 0.85 (95% CI 0.69–1.05); for advanced adenoma the RR was 0.88 (95% CI 0.54–1.44). The relationship was stronger when only adenomas found in the proximal colon were considered: the RR for developing one or more proximal adenomas was 0.73 (95% CI 0.55–0.97) when comparing those with the highest quartile of fiber intake to the lowest. In the complementary

Table 2. Relative Risks for All Adenomas and Advanced Adenomas by Nutrient Intake

Nutrient (in Quartile of Intake)*		n	Cases, n	RR (95% CI) for Adenoma[†]		Cases, n[a]	RR (95% CI) for Advanced Adenoma[†]	
Kilocalories	1	380	119	1.0	Reference	27	1.0	Reference
	2	380	131	1.06	(0.86, 1.30)	34	1.31	(0.80, 2.14)
	3	380	153	1.22	(1.00, 1.49)	39	1.49	(0.92, 2.42)
	4	380	137	1.12	(0.91, 1.38)	35	1.40	(0.85, 2.32)
	p for trend[‡]				0.62			0.27
Total fat	1	380	143	1.0	Reference	30	1.0	Reference
	2	380	110	0.78	(0.64, 0.95)	31	1.04	(0.65, 1.68)
	3	380	155	1.11	(0.92, 1.33)	44	1.59	(1.02, 2.47)
	4	380	132	0.98	(0.81, 1.19)	30	1.12	(0.69, 1.82)
	p for trend				0.59			0.07
Saturated fat	1	380	130	1.0	Reference	27	1.0	Reference
	2	380	120	0.93	(0.76, 1.14)	32	1.22	(0.75, 1.98)
	3	380	152	1.19	(0.98, 1.44)	43	1.74	(1.09, 2.75)
	4	380	138	1.11	(0.91, 1.36)	33	1.43	(0.87, 2.33)
	p for trend				0.41			0.12
Cholesterol	1	380	129	1.0	Reference	35	1.0	Reference
	2	380	134	1.08	(0.89, 1.32)	35	1.08	(0.69, 1.69)
	3	380	143	1.15	(0.95, 1.40)	33	1.06	(0.67, 1.68)
	4	380	129	1.05	(0.86, 1.29)	32	1.01	(0.63, 1.62)
	p for trend				0.23			0.81
Protein	1	380	144	1.0	Reference	44	1.0	Reference
	2	380	128	0.89	(0.74, 1.09)	29	0.66	(0.42, 1.03)
	3	380	126	0.88	(0.72, 1.07)	31	0.73	(0.47, 1.13)
	4	380	142	1.01	(0.84, 1.23)	31	0.76	(0.48, 1.18)
	p for trend				0.76			0.32
Total dietary fiber	1	380	134	1.0	Reference	30	1.0	Reference
	2	380	153	1.10	(0.91, 1.33)	39	1.18	(0.75, 1.86)
	3	380	128	0.90	(0.74, 1.10)	32	0.93	(0.58, 1.52)
	4	380	125	0.85	(0.69, 1.05)	34	0.88	(0.54, 1.44)
	p for trend				0.07			0.53

n[a] = number with advanced adenomas.

*Assuming a 2,000 calorie diet, the mean intake of micronutrient in grams ± standard deviation across quartiles (Q) ranged for total fat from 41.8 ± 9.6 (Q1) to 144.3 ± 34.2 (Q4), saturated fat from 13.3 ± 3.4 (Q1) to 49.8 ± 12.9 (Q4), protein from 44.0 ± 8.7 (Q1) to 116.6 ± 25.5 (Q4), and total fiber from 7.92 ± 1.8 (Q1) to 25.75 ± 7.6 (Q4).

[†]Adjusted for age, sex, clinical center, treatment category, study, and the duration of the observation period.

[‡]See text for details on calculation of *p*.

analysis, there was no association between recurrence of distal lesions (including rectum) and fiber intake (RR for quartile 4 *vs* quartile 1 = 1.00, 95% CI 0.73–1.37).

When individual types of fiber were analyzed (Table 3), adenoma recurrence was less frequent in those with higher intakes of fiber from fruit and vegetables (RR for quartile 4 *vs* quartile 1 = 0.86, 95% CI 0.70–1.06) and grains (RR for quartile 4 *vs* quartile 1 = 0.82, 95% CI 0.68–0.99). Again, when considering only proximal adenoma recurrence, associations were stronger for those with highest intakes of fruit and vegetables (RR = 0.77, 95% CI 0.58–1.01) and grain (RR = 0.75, 95% CI 0.58–0.97) compared to those with the lowest intakes. There was no association between bean fiber intake and adenoma recurrence when considering the total colorectum (Table 3).

Overall, there was no clear association of red meat or fish intake with adenoma development (Table 4). Patients with the highest intake of processed meats when compared to the lowest quartile had a nonsignificantly increased risk of any adenoma (RR = 1.15, 95% CI 0.92–1.43) and a significant increase in risk for advanced adenomas (RR = 1.75, 95% CI 1.02–2.99). Intake of chicken was associated with a decreased risk for any adenoma (RR = 0.79, 95% CI 0.64–0.96) and for advanced adenomas (RR = 0.61, 95% CI 0.38–0.98).

A more general measure of the pattern of meat consumption is the ratio of intake of red and processed meat to that of chicken and fish. Utilizing this ratio, there was a suggestion of an increased risk of any adenoma (RR = 1.19, 95% CI 0.96–1.47) and advanced adenoma (RR = 1.52, 95% CI 0.92–2.54) for those with the higher ratios of intake, but neither result reached statistical significance.

DISCUSSION

In these two cohorts of closely followed subjects, there were some suggestions of an inverse association of vegetable and cereal fiber intake with risk of adenomas, specifically for adenomas arising in the proximal colon. Intake of dietary fat was unrelated to risk. While greater intake of chicken was associated with a reduction in adenoma formation, and processed meats were related to an increased risk, more general measures of red and white meat intake were not significantly associated with the outcome.

The results of prior observational studies of fiber intake and colorectal neoplasia have been inconsistent. For example, some case-control studies have shown a reduction in adenomas in those with a high-fiber diet (20, 21), but a large case-control study using data pooled from a number of colonoscopy-based adenoma studies found no association of fiber intake with either adenomas or advanced adenomas (22). Similarly, cohort studies of this topic have reached differing conclusions. In the Health Professionals' Follow-Up Study (23) total dietary fiber intake was inversely associated with distal adenoma risk (RR for highest *vs* lowest quintile = 0.36, 95% CI 0.22–0.66). However, in the Nurses Health Study (2) (a cohort of 88,757 women), there was no evidence of a protective benefit from any type of fiber on incident distal colon adenomas. More recently, an evaluation of adenoma prevalence in the Prostate, Lung, Colorectal, and Ovarian Cancer (PLCO) Screening Trial found that participants in the highest quintile of dietary fiber intake had a 27% lower risk of adenomas than those in the lowest quintile of intake (3). When cancer has been used as the outcome of interest, findings have also been inconclusive with some evidence supporting a protective role for fiber (1) and others finding no effect (2, 24).

Our results may not be directly comparable to those obtained in the other cohort studies (2, 3, 23), since to be enrolled in our trials all subjects had to have a history of adenoma. We thus studied a high-risk population for adenoma recurrence, rather than the average-risk population studied in the cohorts. Also, all of our study population was examined for adenomas

Table 3. Relative Risks of All Adenomas and Advanced Adenomas by Fiber Type

Nutrient (in Quartile of Intake)		n	Cases, n	RR (95% CI) for Adenomas*		Cases, n[a]	RR (95% CI) for Advanced Adenomas*	
Fruit and vegetable fiber	1	380	135	1.0	Reference	29	1.0	Reference
	2	380	138	0.98	(0.81, 1.19)	31	0.95	(0.59, 1.55)
	3	380	141	0.97	(0.79, 1.17)	45	1.32	(0.84, 2.09)
	4	380	126	0.86	(0.70, 1.06)	30	0.82	(0.49, 1.36)
	p for trend†				0.18			0.58
Grain fiber	1	380	150	1.0	Reference	33	1.0	Reference
	2	380	134	0.86	(0.71, 1.05)	40	1.16	(0.75, 1.81)
	3	380	128	0.85	(0.70, 1.03)	31	0.94	(0.59, 1.50)
	4	380	128	0.82	(0.68, 0.99)	31	0.88	(0.55, 1.41)
	p for trend				0.07			0.63
Bean fiber	1	380	136	1.0	Reference	26	1.0	Reference
	2	379	117	0.89	(0.72, 1.09)	26	1.07	(0.64, 1.80)
	3	379	142	1.05	(0.85, 1.29)	46	1.83	(1.12, 3.00)
	4	379	145	1.07	(0.87, 1.33)	37	1.51	(0.89, 2.57)
	p for trend				0.33			0.03

n[a] = number with advanced adenomas.
*Adjusted for age, sex, clinical center, treatment category, study, and the duration of the observation period.
†See text for details on calculation of *p*.

Table 4. Relative Risks for All Adenomas and Advanced Adenomas by Meat Intake

Nutrient (in Quartile of Intake)		n	Cases, n	RR (95% CI) for Adenoma*		Cases, n[a]	RR (95% CI) for Advanced Adenoma*	
Red meat	1	411	154	1.0	Reference	39	1.0	Reference
	2	370	123	0.88	(0.73, 1.08)	30	0.91	(0.57, 1.43)
	3	365	129	0.98	(0.80, 1.20)	38	1.19	(0.76, 1.87)
	4	373	133	0.97	(0.78, 1.21)	28		(0.49, 1.42)
	p for trend[†]				0.76		0.84	0.51
Processed meat	1	390	132	1.0	Reference	25	1.0	Reference
	2	381	132	0.99	(0.81, 1.21)	39	1.65	(1.02. 2.67)
	3	385	129	0.95	(0.78, 1.17)	33	1.41	(0.84, 2.35)
	4	363	146	1.15	(0.92, 1.43)	38	1.75	(1.02, 2.99)
	p for trend				0.71			0.58
Fish	1	403	147	1.0	Reference	35	1.0	Reference
	2	371	126	0.91	(0.75, 1.11)	31	0.98	(0.62, 1.55)
	3	371	123	0.87	(0.72, 1.06)	32	0.98	(0.61, 1.56)
	4	374	143	1.00	(0.82, 1.22)	37	1.15	(0.72, 1.84)
	p for trend				0.12			0.64
Chicken	1	404	158	1.0	Reference	44	1.0	Reference
	2	375	117	0.80	(0.66, 0.97)	33	0.79	(0.51, 1.22)
	3	366	148	1.03	(0.85, 1.23)	30	0.70	(0.44, 1.09)
	4	372	115	0.79	(0.64, 0.96)	28	0.61	(0.38, 0.98)
	p for trend				0.19			0.01
Red meat to white meat	1	380	121	1.0	Reference	27	1.0	Reference
	2	379	132	1.08	(0.88, 1.32)	37	1.40	(0.87, 2.25)
	3	381	145	1.17	(0.96, 1.44)	34	1.30	(.79, 2.14)
	4	377	140	1.19	(0.96, 1.47)	37	1.52	(0.92, 2.54)
	p for trend				0.08			0.16

n[a] = number with advanced adenomas.
*Adjusted for age, sex, clinical center, treatment category, study, and the duration of the observation period.
[†]See text for details on calculation of *p*.

with complete colonoscopy, so factors related to screening and endoscopy use should not affect our results.

We noted possibly differing effects of fiber between the proximal and distal colon, with a reduction in risk confined to the proximal colon. The local microenvironment is known to vary across the length of the colon, which may explain the variation in association that we observed. For example, differences between colonic regions in pH (25), bacterial composition (26), and apoptotic index (27) have all been described and these factors may modulate fibers action on the colon. Others have reported finding differential effects of fiber on adenoma formation across the colon. For example, two prior studies (3, 28) found beneficial effects of fiber on adenoma formation in the distal colon, but not the rectum. Both of these studies were based on outcome assessment with flexible sigmoidoscopy, and so no specific data on exclusively proximal lesions are available. A large prospective evaluation of fiber intake and CRC also observed a differential effect across the bowel (1). Like the adenoma studies, no effect of fiber intake on rectal cancer was noted. The protective benefit of fiber intake in that study was marginally stronger in the distal colon as compared to the proximal colon.

The overall lack of an association between fiber intake and adenoma formation in our study is consistent with the results of randomized trials of fiber supplements (6, 7). The Arizona Wheat Bran Fiber trial (6) examined the effect of a cereal fiber supplement in the study of 1,429 subjects randomized to either high amounts (13.5 g per day) or low amounts (2 g per day) of a cereal fiber supplement. The multivariate adjusted odds ratio for recurrent adenoma in the high-fiber group as compared with the low-fiber group was 0.88 (95% CI 0.70–1.11). Another fiber supplement trial utilized ispaghula husk and found that the intervention group was at higher risk for subsequent adenoma development (RR = 1.67, 95% CI 1.01–2.76) (7).

We found little relationship between fat intake and adenoma development. Trials of the effect of either a low fat, high-fiber diet (5), or a low-fat diet (29), on adenoma risk have had negative findings, consistent with our results. Neither did we find significant risk associated with consuming more red meat in the diet.

While more general measures of meat intake were not associated with risk, we did find specific associations between certain types of meat intake and adenoma formation. Those with higher intakes of chicken were at decreased risk for adenomas and advanced adenomas. While generally speaking, studies examining poultry intake and CRC have shown no clear association, our result is consistent with that of two recent large cohort studies examining nutritional intake and CRC incidence. The first specifically demonstrated that increasing consumption of chicken was associated with a reduction in risk for CRC (hazard ratio, highest quartile 0.7, 95% CI 0.6–1.0) (30). The second looked at the long-term intake of poultry and fish and found a lower risk for both proximal (RR = 0.77, 95% CI 0.59–1.02) and distal (RR = 0.70, 95% CI 0.50–0.99) colon cancer for those in the highest tertile of intake (31). The mechanism by which poultry intake would independently reduce CRC incidence is not clearly delineated. Poultry is a minor source of the nutrients selenium and calcium that have been associated with a decreased risk for CRC.

Our finding of a possible increased risk for adenoma development with intake of processed meat was largely confined to development of advanced adenoma and was only seen among subjects in the highest quartile of intake. Two recent reviews of the literature noted an association between higher intakes of processed meats and CRC (32, 33). A large prospective cohort study from the Netherlands also found an increase risk for CRC associated with increasing processed meat intake (RR = 1.17, 95% CI 1.03–1.33) (11). Most recently, two large cohorts studies (one from Europe (34) and the other from the United States (31)) both found an increased risk for CRC in those with greater processed meat consumption. Meat processing often involves salting, smoking, or the addition of nitrates to preserve the food. Such processing may increase potentially carcinogenic *N*-nitroso compounds in the foods, providing biological plausibility for the increase in risk.

Our study has a number of strengths, including its relatively large size and the careful assessment of both diet and adenoma occurrence. We enrolled a diverse population of subjects from several centers and utilized complete colonoscopy both at baseline and follow-up. Therefore, the adenomas found represent incident lesions. An independent study pathologist confirmed all adenomas. A validated instrument was utilized to collect information on dietary exposures before outcome development, eliminating the potential for recall bias.

Limitations of our work include those inherent to most studies of dietary epidemiology. For example, for these analyses we used information on intake collected at a single point in time (study baseline), and a single assessment may not have reflected the long-term dietary patterns of our subjects. More generally, measurement error may be a problem in dietary assessments, possibly resulting in attenuated relative risk estimates and the findings could be distorted by dietary (or other) confounding factors that are either incorrectly measured or omitted.

In summary, we examined the effect of diet on incident adenoma development in subjects enrolled in two randomized chemoprevention trials. When considering the total colorectum, there was no strong benefit afforded by a high-fiber diet in this population on either adenoma or advanced adenoma formation. This result is consistent with randomized trials of fiber for adenoma chemoprevention. We also did not see a deleterious effect associated with a diet high in fat or red meat. We did find some evidence of an increased risk with the highest levels of intake of processed meat and a benefit from consumption of chicken. These findings are consistent with results from recent large cohort studies (30, 31, 34) examining the effect of meat intake on CRC incidence and supports suggestions (35) that individuals consider modifying intake of red and processed meats.

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