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Dietary Fat and the Risk of Colorectal Cancer[1]

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ABSTRACT

The relationship between the intake of dietary fat and subsequent colorectal cancer during a 15-year follow-up was investigated in 7074 men of Japanese ancestry, 45 to 68 years old and living in Hawaii. Data on fat intake were obtained by 24-hr recall records at base-line examination. We found a statistically significant, negative association between colon cancer and the intake of saturated fat, whether assessed on the basis of g per day or as a percentage of the caloric intake. There was a similar association with total fat intake when expressed in terms of percentage of total calories. The strongest negative relationship was found in cancer of the right colon. In contrast, rectal cancer showed a weakly positive relationship to the intake of saturated fat when assessed on the basis of percentage of caloric intake.

INTRODUCTION

It is a popular view that persons consuming large amounts of fat are at high risk for large bowel cancer. Support for this concept originated from geographic correlation studies which showed that diets of populations at high risk for colon cancer contained a larger proportion of fat, particularly animal fat, than do the diets of populations at low risk for this tumor (31). Animal experiments have supported this hypothesis, but studies of diet in relation to human colon cancer have yielded inconsistent results. Of 6 case-control studies, 4 (4, 12, 15, 22) have supported but 2 (11, 21) did not support a positive association of beef or fat intake.

Case-control studies pose several methodological problems, when they are applied to the investigation of diet and cancer. Cancer induction can be a long-term process, and it is uncertain if the dietary information from a patient with confirmed cancer accurately represents the consumption patterns that existed prior to tumor development. Case-control studies are also faced with the problem of potential interviewer bias and patient awareness of their condition that may influence dietary histories. Moreover, the controls in such studies must be carefully identified so that they are comparable to cases in their chance of exposure to pertinent dietary factors. Differences in the selection of controls in past case-control studies may alone account for some of the divergent results that have been reported.

The present report attempts to avoid some of these problems with a prospective study of diet and large bowel cancer in a large defined population of Hawaii Japanese men assembled in 1965.

MATERIALS AND METHODS

The Japan-Hawaii Cancer Study is a prospective epidemiological investigation of cancer in a cohort of men of Japanese ancestry born in the years 1900 through 1919 and living on the island of Oahu in 1965 (30). Of the 11,148 eligible men located by updating World War II Selective Service files, 8,006 (72%) participated in the initial examination which was carried out during the years 1965 to 1968. The following subjects were excluded from the study: 292 prevalence cases of CHD[3]; 68 prevalence cases of cancer; and 92 incidence cases of cancer that could not be confirmed by tissue diagnosis. This yielded a remainder of 7,554 study subjects. Only 3 of the 292 prevalence CHD men subsequently developed large bowel cancer after examination; hence, removal of this group should have a negligible effect upon the results of the dietary analyses of men with these cancers. Elimination of an additional 480 men whose 24-hr diet recall was considered atypical (see below), left 7,074 eligible men.

Ascertainment of incident cancers was conducted by a daily review of the hospital discharge records for the island of Oahu. A computer linkage file was established with the Hawaii Tumor Registry to reduce the possibility of missing incidence cases during the study period. Only about 2% of the study subjects had moved from Oahu since their examination, so that the surveillance system identified virtually all of the newly diagnosed cancers in the cohort. Each case was confirmed by histological examination. Cancers of the colon and rectum were limited to epithelial tumors at these sites. Subsites of tumors in the large bowel were identified as follows: cecum and ascending colon (International Classification of Diseases, Eighth Revision, code 153.0); transverse and descending colon (153.1, 153.2); sigmoid colon (153.3); recto sigmoid (154.0); and rectum (154.1). The rectosigmoid segment was defined as being from 8 to 12 cm above the dentate line. Rectal and rectosigmoid tumors were combined for calculations of adjusted means for "rectum" cases.

The dietary information used for the present report was obtained at the initial examination. A 24-hr diet recall interview was administered by dietitians to each study subject. Food models and serving utensils were used to illustrate portion sizes. Food composition values were compiled from the best available sources to calculate the individual intakes of calories and nutrients (28). Dietary data were validated by a 7-day diet record administered to a subsample of 329 men examined 2 years later. The method has been used by most other large scale dietary studies, such as those carried out by the U. S. Department of Agriculture in 1965 (7), the Ten State Nutrition Survey of 1968 to 1970 (27) and the First Health and Nutrition Examination Survey, 1971–1972 (24). A subsample of 480 men were excluded from the study on the basis of "atypical" intake, *e.g.*, holidays and special occasion meals, etc. Total fat intake and the intake of saturated fat are highly correlated. In the interest of saving space, we have therefore restricted the comparison of the RR of colorectal cancer to only one of these, saturated fat.

The relationship between nutrient intake and cancer incidence was first evaluated by comparing the age-adjusted means of dietary intake of men who developed cancer and those who remained free of cancer during the 15 to 18-year follow-up period. Age adjustment was done using one-way unbalanced analyses of covariance (8) with age as the covariate. When comparing age-adjusted means across cancer sites (or colorectal cancer subsites), the Bonferroni inequality was used to preserve the simultaneous multiple comparisons significance level at 5%.

The RR of colon cancer associated with fat intake was estimated from multiple logistic regression models (2), while adjusting for age at examination. Similar analyses for rectal cancer used 2 covariates, age and

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[3] The abbreviations used are: CHD, coronary heart disease; RR, relative risk.

alcohol intake in 24 hr, since alcohol is associated with rectal cancer risk (23) as well as with fat intake. Each fat intake variable was categorized into quintiles based on the (noncancer) controls only. The lowest fat intake quintile was chosen as the referent group. Using a (0, 1) indicator variable for each of the remaining quintiles, the RR for cancer adjusted for covariates could be calculated. This procedure also yields (approximate) covariate-adjusted confidence limits for the RR. Tests for linear trend in the logit of risk (25) were obtained from logistic models using the continuous, ungrouped fat variable, and relevant covariates. As noted by Jain *et al.* (15), we have not attempted multivariate analyses with more than one fat-related nutrient variables due to the high interrelations between them and the ensuing colinearity problems.

RESULTS

The age distribution of controls and men with cancers of the colon, the rectum, and all other sites combined is shown in Table 1. The 655 new cancers were ascertained from the first examination until July 1983.

The age-adjusted mean intake of selected nutrients, by cancer site, is shown in Table 2. When expressed in terms of g per day, the only dietary item significantly associated with bowel cancer was alcohol, and this association was only noted for rectal carcinoma. When assessed on the basis of percentage of total calories, a statistically significant negative association was demonstrated between colon cancer and total and saturated fat intake.

The relative risks of colon and rectal cancer by quintile of total and saturated fat were then calculated as shown in Tables 3 and 4. These indicate a statistically significant negative association between colon cancer risk and the intake of saturated fat, whether expressed in g per day (Table 3) or as a percentage of calories (Table 4). There was also a negative association between colon cancer risk and total fat intake, but this was statistically significant only when expressed in percentage of total calories. In contrast, a positive association was demonstrated for saturated fat and rectal cancer, but none of the associations was statistically significant. Table 5 presents the adjusted means of dietary lipids for subsites within the colon and rectum. These indicate lower levels of total and saturated fat intake for the right and sigmoid colon. These differences achieve statistical signifi-

Table 1

Age-specific cancer incidence rates by site

Age at examination (yr)	Colon		Rectum		Other cancers		Controls (no.)	All (no.)
	No.	Rate	No.	Rate	No.	Rate		
45–49	12	7.4	3	1.8	63	38.6	1553	1631
50–54	30	12.0	15	6.0	133	53.3	2319	2497
55–59	24	16.9	19	13.4	118	83.1	1259	1420
60–68	40	26.2	22	14.4	176	115.3	1288	1526
All	106	14.9	59	8.3	490	69.3	6419	7074

[a] Rates per 1000 men cumulative over the 15 to 18 years of study follow-up.

Table 2

Age-adjusted mean daily intake of selected nutrients by cancer site

Age adjustment and tests for differences in means was done by analysis of covariance methods.

	Nutrient																	
	Protein							Fat						Carbohydrate			Alcohol	
	wt (g)				% of cal			wt (g)			% of cal							
	Cal	Total	Animal	Vegetable	Total	Animal	Vegetable	Total	Saturated	Unsaturated	Total	Saturated	Unsaturated	wt (g)	% of cal	Dietary cholesterol (mg)	wt (g)	% of cal
Controls (6419)[a]	2298	95.1	71.3	23.8	16.7	12.5	4.3	86.5	60.1	26.4	33.5	23.2	10.3	263.4	46.4	551.7	12.3	3.4
Colon (106)	2248	92.9	69.5	23.4	16.4	12.2	4.2	79.9	53.7	26.2	31.3[b]	20.9[b]	10.3	263.9	47.5	492.2	14.7	4.8
Rectum (59)	2384	94.3	70.7	23.6	16.3	12.3	4.1	88.4	64.5	23.9	33.7	24.8	8.9	265.4	44.3	572.9	21.8[b]	5.8[b]
Other cancers (489)	2293	93.2	70.1	23.1	16.5	12.3	4.1	82.6	58.1	24.5	32.1[b]	22.6	9.5[b]	264.9	46.8	554.5	16.5[b]	4.6[b]

[a] Numbers in parentheses, number of men.
[b] Mean is significantly different from control mean ($p < 0.05$).

Table 3

Adjusted relative risks of colon and rectal cancer by quintile of saturated fat intake

Saturated fat intake (g/day)	No. of men			Colon cancer		Rectal cancer	
	Controls	Colon	Rectal	RR[a]	95% confidence interval	RR[b]	95% confidence interval
>84.5	1276	15	11	0.49	0.27, 0.91	1.51	0.65, 3.54
64.2–84.5	1284	14	14	0.44	0.24, 0.82	1.78	0.80, 3.94
49.0–64.1	1284	16	12	0.46	0.25, 0.82	1.30	0.58, 2.92
33.1–48.9	1286	21	10	0.59	0.34, 1.00	1.02	0.43, 2.37
<33.1	1289	40	12	1.00		1.00	
Total	6419	106	59	$p^c = 0.035$		$p^c = 0.222$	

[a] Adjusted for age at time of examination using logistic regression model.
[b] Adjusted for age at time of examination and ethanol intake (g per day) using logistic regression model.
[c] Test for linear trend in the values of $\log_e$ [risk/(1 − risk)], known as the logit of risk.

Table 4

Adjusted RRs of colon and rectal cancer by quintile of percentage of calories from saturated fat

% of cal from saturated fat	No. of men: Controls	Colon	Rectal	Colon cancer: RR[a]	95% confidence interval	Rectal cancer: RR[b]	95% confidence interval
>31.36	1280	13	13	0.44	0.23, 0.83	2.30	0.94, 5.64
25.66–31.36	1283	19	13	0.61	0.35, 1.08	2.08	0.86, 5.01
20.66–25.65	1285	22	12	0.70	0.41, 1.21	1.83	0.75, 4.46
15.00–20.65	1283	17	12	0.51	0.28, 0.91	1.60	0.66, 3.87
<15.00	1288	35	9	1.00		1.00	
Total	6419	106	59	$p^c = 0.014$		$p^c = 0.081$	

[a] Adjusted for age at time of examination using logistic regression model.
[b] Adjusted for age at time of examination and ethanol intake (g per day) using logistic regression model.
[c] Test for linear trend in the values of $\log_e$ [risk/(1 − risk)], known as the logit of risk.

Table 5

Adjusted mean daily intake of fat and other dietary variables by colorectal cancer subsite

Adjustment for age at time of examination and ethanol intake (g/day) was done by analysis of covariance.

	Dietary variables: Fat wt (g) Total	Saturated	Unsaturated	Fat % of cal Total	Saturated	Unsaturated	Dietary cholesterol (mg)	Total cal
Right colon (153.0)[a] (24)[b]	76.3	50.7	25.6	28.4[c]	18.2[c]	10.3	485	2362
Transverse, left colon (153.1, 153.2) (15)	98.8	70.9	27.9	31.7	23.6	8.1	629	2655
Sigmoid colon (153.3) (66)	78.3	51.7	26.5	32.5	21.5	11.0	466	2120
Rectosigmoid (154.0) (26)	95.8	67.3	28.5	37.3	26.3	11.0	616	2365
Rectum (154.1) (33)	83.7	63.1	20.6	32.5	24.7	7.8	530	2299
Controls (6419)	86.9	60.3	26.5	33.6	23.3	10.3	553	2305

[a] Numbers in parentheses, International Classification of Diseases Eighth Revision codes.
[b] Numbers in parentheses, number of men.
[c] Mean is significantly different from control mean ($p < 0.05$).

Table 6

Adjusted RRs of colon and rectal cancer by tertile of saturated fat intake and by time interval from examination to diagnosis

Entries are the RRs adjusted for the same covariates as in Tables 3 and 4.

Saturated fat intake	No. of controls	RR: Colon cancer time interval <5 yr	5–10 yr	10+ yr	Rectal cancer time interval <5 yr	5–10 yr	10+ yr
g/day							
≥70.0	2146	0.31 (2)[a]	0.38 (5)	0.87 (19)	1.47 (7)	1.21 (8)	4.28[b] (8)
44.0–69.9	2136	1.25 (9)	0.48 (7)	0.49[b] (12)	0.95 (5)	0.81 (7)	1.72 (4)
<44.0	2137	1.00 (8)	1.00 (16)	1.00 (28)	1.00 (6)	1.00 (11)	1.00 (3)
p^c		0.114	0.05	0.54	0.70	0.79	0.02
% cal							
≥27.31	2139	0.68 (6)	0.43 (5)	0.76 (16)	0.75 (4)	2.28 (12)	2.05 (5)
18.95–27.30	2140	0.33 (3)	0.84 (10)	0.87 (19)	1.19 (7)	1.22 (7)	2.12 (6)
<18.95	2140	1.00 (10)	1.00 (13)	1.00 (24)	1.00 (7)	1.00 (7)	1.00 (4)
p^c		0.28	0.06	0.17	0.82	0.11	0.11

[a] Numbers in parentheses, number of cases.
[b] Relative risk is significantly different from unity, at $p < 0.05$.
[c] Test for linear trend in the logit of risk.

cance only in the right colon and only when expressed in terms of percentage of total calories.

The influence of the time interval between examination and the diagnosis of colonic and rectal cancer in respect to the relative risk estimates is shown in Table 6. With saturated fat in g per day, the negative association with colon cancer risk (Table 3) is shown to persist throughout the entire follow-up period, although it is most obvious for cases diagnosed 5 to 10 years after examination. The weakly positive association with rectal cancer risk is primarily due to cases diagnosed 10 or more years after examination. The result is essentially the same for saturated fat as a percentage of calories, although here the weak positive association with rectal cancer risk becomes evident as soon as 5 years after examination. Few of these risk relationships had a significant linear trend, although the power to detect that is of course reduced when analyzing subsets of the data.

DISCUSSION

The Hawaii Japanese experience the same rates of colorectal carcinoma as do U. S. Whites (29). These rates represent a 3-fold increase over those of indigenous Japanese (29). The rise in frequency of colon cancer parallels a rise in the frequency of CHD among Hawaii Japanese (9). If CHD and colorectal cancer each stem from an excess of dietary fat, one might expect this to be supported in prospective studies. A comparison of diets consumed by men in Hiroshima, Japan (low risk for CHD and colorectal cancer) with Japanese men in Honolulu (high risk for CHD and colorectal cancer) does show a 2-fold increase in dietary fat in Hawaii (28). A report on the impact of diet upon CHD in Hawaii showed that fat intake, when stated as percentage of total caloric intake, was significantly increased in Japanese men who subsequently developed myocardial infarction (32). On this basis, a positive association between fat intake and colon cancer was expected in our study of the same cohort of Hawaii Japanese men, but such was not the case.

An earlier analysis of this cohort (14) indicated that after 10 years of follow-up, examined men had a significantly lower risk of death from all causes, death from cancer, and incidence of stomach cancer than had unexamined men. This was not unexpected since other studies (3, 5, 6) have shown that study volunteers are healthier than nonvolunteers. There were minimal, statistically nonsignificant differences between examined and unexamined men in respect to the incidence of large bowel cancer, and these showed lower rates among examined men. The recruitment of healthier subjects into this study should not, in itself, bias the results of the investigation. The participation rate among potential cohort members was high (72%); hence, it is unlikely that any potential differential association of fat intake with colorectal cancer among nonparticipants could challenge the results of this study.

When analyzing fat intake, it is unclear whether one should base comparative studies upon consumption in g per day or on the basis of percentage of calories. We have presented our data in both forms. Studies of CHD have generally shown significant results only when fat intake was expressed in terms of percentage of calories. Since both measures have yielded similar results in this study, it probably would not have mattered which method was used.

When considering fat as a percentage of calories, the level of intake will be influenced by the intake of other nutrients. Alcohol may be important in this regard. Heavy drinkers have a lower percentage of calories from fat than do light drinkers or nondrinkers. Thus, an inverse relationship between percentage of calories as fat and cancer risk might actually reflect a positive relationship between alcohol consumption and cancer incidence. This is the probable cause for a negative relationship between fat intake and the sum of all other cancers. Included among these are cancers of the lung, esophagus, mouth, larynx, and liver, conditions which have been associated with alcohol intake. The colonic cancer patients and their controls consumed similar amounts of alcohol; hence, this problem does not apply for these tumors. In the case of the rectum, however, there is a distinctly higher level of alcohol consumption among patients than among controls (23). Adjustment for alcohol intake was therefore included in assessing the impact of fat intake on rectal cancer risk.

There is a weak correlation between the serum cholesterol level and the fat intake of men in this cohort (16), and we have demonstrated previously a negative association between serum cholesterol and colon cancer (26). The low serum cholesterol levels in these and other studies are believed to have been attributed to the presence of colon cancer at the time of examination, although they held true for men in our study who developed cancer from 5 to 10 years after examination. As in the cholesterol study,the time interval between examination and the diagnosis of cancer had little influence on the degree of negative association between colon cancer and saturated fat intake. The low cholesterol levels were found predominantly in subjects with right-sided colon cancer in the earlier study. The numbers of cases in the colon subsites were small, but the negative association of cancer of the right colon with fat intake in this study is in the same direction as the results of the serum cholesterol study (26), and it is accompanied by a weak inverse association with cancer of the sigmoid colon as well. These findings suggest that colon cancer and CHD occur in different subsets of the Hawaii Japanese population, a result that has been anticipated by Kinlen (17).

It is possible that the divergent results obtained by different studies of fat intake in relation to colon cancer risk have a methodological rather than a physiological basis. A case in point is a case-control study of colon cancer in Hawaii Japanese which indicated that patients consumed more meat (a nutrient highly correlated with fat) than did controls (12). These results obviously diverge from the present study, and the basis for this divergence is probably methodological. The dietary data in the present study were obtained by dietitians using the 24-hr recall method. This raises the question as to whether this method truly reflects individual dietary practice. An analysis of within-person variability of nutrient intake by members of the cohort has been reported by McGee *et al.* (20). The responses of cohort men to the 24-hr dietary recall were compared with those obtained in an average of a 7-day food diary record. The values obtained by the 2 methods were almost identical. As may have been expected, there was considerable variation in each individual's dietary intake from 1 day to another throughout the week. For this reason, the 24-hr recall is of limited value in characterizing the usual dietary intake of one person but is probably adequate when comparing large numbers of subjects, as was done in this study. The case-control study (12) measured current dietary practice, used hospital patients as controls rather than the community-based sample analyzed in our report, made estimates of the relative frequency of specific foods, assessed tumors in both sexes rather than in males alone, combined rectal and colonic tumors for analysis, and did not use trained dietitians for the purpose of conducting interviews. Bjelke's (1) prospective study of parallel cohorts in Norway and Minnesota comes closer to the present study in methodology and failed to show an association between meat and colon cancer.

Gordon *et al.* (10), in a paper dealing with 3 geographically separated longitudinal studies (including the Honolulu cohort at an earlier time) found that total fat and caloric intake were related significantly and inversely to total mortality. A later analysis of the Honolulu cohort summarized its mortality experience 10 years after examination (19). This showed a significant inverse relationship between fat intake and cancer mortality, stroke mortality, and total mortality. This inverse relationship obtained whether fat intake was stated in terms of g per day or as a percentage of total calories. Finally, it should be noted that weight gain from age 25 has been shown to be related to only one

cancer site, the colon.[4] It might be anticipated that body weight and caloric and fat intake should be well-correlated, whereas in the case of colon cancer, body weight diverges from the other 2 variables. This suggests that the balance between energy consumption and expenditure might be more important than the amounts or types of nutrients consumed. Although the biological basis for this hypothesis remains to be identified or proven, it is consistent with observed positive associations between colon cancer and 2 closely related socioeconomic factors, a high family income and sedentary occupation (13, 18).

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