Incidence of Colorectal Cancer in Relation to Glycemic Index and Load in a Cohort of Women

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Abstract

Background: **Dietary glycemic index (GI) and glycemic load (GL) affect circulating insulin concentrations. Elevated circulating insulin concentrations can increase insulin-like growth factor-1, and both of these hormones may have growth-promoting effects within the colorectum.**
Methods: **We examined associations of GI and GL with colorectal cancer (CRC) among participants in the Iowa Women's Health Study (*n* = 35,197; ages 55-69 years at baseline in 1986). Over 15 years of follow-up, we identified 757 cases of colon cancer and 209 cases of rectal cancer (954 CRC cases).**
Results: **Overall, neither GI nor GL were significantly associated with incident CRC. However, among obese women (baseline body mass index ≥30 kg/m^2) CRC incidence was increased in the highest versus lowest quintiles of GI (relative risk, 1.66; 95% confidence intervals, 1.13-2.43; *P* for trend = 0.02) and GL (relative risk, 1.79; 95% confidence intervals, 1.19-2.70; *P* for trend < 0.01). This pattern of increased risk for obese women with high GI or GL tended to hold for both colon cancer and rectal cancer, and for nondiabetic women as well. No statistically significant associations were observed between GI or GL and CRC among subjects whose baseline body mass index was <30 kg/m^2.**
Conclusion: **Our findings suggest that high GI or GL are not major CRC risk factors among older women in general, but may increase CRC risk among women who are obese.** (Cancer Epidemiol Biomarkers Prev 2006;15(5):892–6)

Introduction

Obesity, greater energy consumption and low physical activity are putative risk factors for colorectal cancer (CRC; ref. 1). These risk factors modulate circulating levels of insulin (2), which may represent a uniform mechanism of carcinogenesis. By analogy, diets that produce high postprandial blood glucose and insulin responses might also increase CRC risk. However, previous studies disagree on the relationship between dietary glycemic measures and CRC risk (3-9).

Two indices of postprandial blood glucose response that are of epidemiologic interest are glycemic index (GI) and glycemic load (GL). GI is a ranking of carbohydrate-containing foods based on their postprandial glycemic effect (10). Foods with a high GI contain carbohydrates that are absorbed by the body quickly, which leads to a more rapid increase in blood glucose levels relative to foods with a lower GI. This is influenced by many things including the type of carbohydrates and other nutrients in the food and how the food has been processed. To calculate the GI of a specific food, the glycemic response is compared with the response of a control food (white bread or glucose) which is assigned a value of 100 (7). Whereas GI compares equal quantities of carbohydrate, GL is a measurement which also accounts for the quantity of carbohydrate consumed (7). Thus, GL quantifies the overall glycemic effect of a portion of food.

Because GI and GL have a direct effect on circulating insulin levels (7), we tested the hypothesis that they are positively associated with CRC incidence in a prospective cohort study of women. We also tested whether GL and GI interact with obesity, as previously suggested (4, 9).

Materials and Methods

A detailed description of the Iowa Women's Health Study has been published elsewhere (11). Briefly, in January of 1986, 41,836 Iowa women ages 55 to 69 years completed mailed questionnaires. The average age of respondents was 61.7 years; 99% were Caucasian.

Participants reported their current height and weight (12), which was used to calculate body mass index (BMI, kg/m^2). A friend measured waist (1 inch above the umbilicus) and hip (widest point) circumferences. These were used to calculate the waist/hip ratio. Three levels of physical activity (low, medium, and high) were created by asking three questions about the frequencies of moderate and vigorous activities. Participants were identified as having prevalent diabetes if they reported ever being told by a doctor that they had sugar diabetes (diabetes mellitus) or if they indicated current use of insulin or ''pills for sugar diabetes (or to lower blood sugar).''

A Harvard food frequency questionnaire (13) was included to determine each subject's usual dietary intake over the past year. Participants were asked to report their average consumption, over the past year, of 127 food items, including 29 vegetables; 15 fruits; 13 dairy foods; 14 meat, poultry, seafood, or egg items; 17 breads, cereals, or starches (including 8 whole-grain items); 14 beverages (including 4 alcoholic beverages); and 25 sweets, baked goods, and miscellaneous items. The food frequency questionnaire used in the present study was found to account for 93% of the total energy intake in a validation study with 194 female nurses (14). The average dietary GI was calculated in the following manner (15):

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Table 1. Adjusted RRs of CRC in relation to quintiles of GI and GL, Iowa Women's Health Study (1986-2000)

Factor	Quintile					*P* for trend
	1	2	3	4	5	
CRC (*n* events = 954)						
GI	<81.0	81.0-84.1	84.2-86.5	86.6-89.3	>89.3	
n events	186	165	195	195	213	
RR, adjusted for age and energy	1.0	0.87	1.02	1.00	1.08	0.18
95% CI		0.70-1.07	0.83-1.25	0.82-1.23	0.89-1.32	
RR, multiply-adjusted*	1.0	0.86	1.03	1.04	1.08	0.15
95% CI		0.69-1.06	0.84-1.26	0.84-1.27	0.88-1.32	
GL	<146	147-163	164-176	177-192	>193	
n events	175	197	190	203	189	
RR, adjusted for age and energy	1.0	1.05	0.98	1.04	0.98	0.86
95% CI		0.85-1.29	0.80-1.21	0.85-1.28	0.80-1.21	
RR, multiply-adjusted*	1.0	1.05	1.04	1.12	1.09	0.33
95% CI		0.85-1.30	0.84-1.29	0.91-1.38	0.88-1.35	
GL by BMI						
BMI (<25 kg/m^2)						
n events	70	52	58	71	72	
RR, multiply-adjusted†	1.0	0.72	0.83	1.03	0.94	0.57
95% CI		0.50-1.05	0.58-1.19	0.73-1.45	0.66-1.33	
BMI (25-30 kg/m^2)						
n events	73	64	85	72	68	
RR, multiply-adjusted†	1.0	0.79	1.09	0.90	0.85	0.59
95% CI		0.56-1.11	0.79-1.50	0.65-1.25	0.60-1.18	
BMI (≥30 kg/m^2)						
n events	43	49	52	52	73	
RR, multiply-adjusted†	1.0	1.19	1.20	1.24	1.66	0.02
95% CI		0.78-1.79	0.80-1.81	0.83-1.86	1.13-2.43	
BMI (≥30 kg/m^2), nondiabetics						
n events	35	40	44	45	64	
RR, multiply-adjusted	1.0	1.17	1.22	1.30	1.82	<0.01
95% CI		0.74-1.85	0.78-1.92	0.83-2.02	1.20-2.76	
GL by BMI						
BMI (<25 kg/m^2)						
n events	60	69	60	75	59	
RR, multiply-adjusted†	1.0	1.12	0.94	1.09	0.79	0.25
95% CI		0.78-1.61	0.65-1.37	0.76-1.56	0.54-1.16	
BMI (25-30 kg/m^2)						
n events	75	79	71	63	74	
RR, multiply-adjusted†	1.0	0.92	0.83	0.78	0.99	0.63
95% CI		0.66-1.28	0.60-1.17	0.55-1.11	0.71-1.37	
BMI (≥30 kg/m^2)						
n events	40	49	59	65	56	
RR, multiply-adjusted†	1.0	1.17	1.56	1.78	1.79	<0.01
95% CI		0.77-1.79	1.03-2.35	1.19-2.66	1.19-2.70	
BMI (≥30 kg/m^2), nondiabetics						
n events	27	42	49	60	50	
RR, multiply-adjusted	1.0	1.47	1.85	2.37	2.24	<0.01
95% CI		0.91-2.40	1.15-3.00	1.49-3.77	1.39-3.60	

*Adjusted for age (continuous), energy (quintiles), activity level (high, medium, low), multivitamin use (yes, no), diabetes (yes, no), smoking (pack-years), BMI (quintiles), and waist/hip ratio (quintiles).
†Adjusted for all characteristics listed above, except adjusted for BMI (continuous) within BMI quintiles.

$$\Sigma[(\text{servings of food per day}) \times (\text{carbohydrate content of food}) \times (\text{GI})] / \text{total carbohydrate in diet}$$

The average GL was calculated as follows:

$$\Sigma[(\text{servings of food per day}) \times (\text{carbohydrate content of food}) \times (\text{GI})]$$

GI and GL values were energy-adjusted by the residual method.

In this sample from Iowa, the energy-adjusted reliability coefficients reported for total carbohydrate and crude fiber were 0.66 and 0.82, respectively, based on two administrations of the food frequency (16). The correlations of the carbohydrate and crude fiber values from the food frequency with five 24-hour dietary recalls were 0.45 and 0.24, respectively (16).

Cohort Follow-up. Cancer incidence and deaths in Iowa from 1986 to 2000 were ascertained by computer linkage with the State Health Registry of Iowa, which includes a Surveillance, Epidemiology, and End Results cancer registry. Colon and rectal cancers were identified using International Classification of Diseases for Oncology (second edition) codes 18.0 to 18.9 and 19.9 and 20.9, respectively. Address changes, deaths outside of Iowa, and nonfatal, noncancer end points were identified by follow-up questionnaires mailed to the participants in 1987, 1989, 1992, and 1997, and by the National Death Index.

Data Analysis. Our hypothesis was that baseline dietary GI and GL would be positively associated with incident CRC. We excluded women who had a prevalent cancer (*n* = 3,830); women who indicated implausible energy intake levels (<600 or >5,000 calories per day) or who left ≥30 missing items in the food frequency questionnaire (*n* = 3,102), and women who had a CRC of nontypical morphology (*n* = 19). These exclusions left 35,197 women for analysis.

Table 2. Adjusted RRs of colon and rectal cancers in relation to quintiles of GI and GL, Iowa Women's Health Study (1986-2000)

Factor	Quintile					*P* for trend
	1	2	3	4	5	
Colon cancer (*n* events = 757)						
GI by BMI						
BMI (<25 kg/m^2)						
n events	54	36	40	56	64	
RR, multiply-adjusted*	1.0	0.63	0.70	1.03	1.03	0.23
95% CI		0.41-0.99	0.46-1.08	0.70-1.52	0.70-1.51	
BMI (25-30 kg/m^2)						
n events	58	47	68	59	59	
RR, multiply-adjusted*	1.0	0.71	1.07	0.92	0.93	0.82
95% CI		0.48-1.06	0.75-1.54	0.63-1.33	0.64-1.34	
BMI (≥30 kg/m^2)						
n events	39	39	42	37	59	
RR, multiply-adjusted†	1.0	1.06	1.07	0.92	1.45	0.21
95% CI		0.68-1.66	0.69-1.67	0.58-1.45	0.96-2.19	
BMI (≥30 kg/m^2), nondiabetics						
n events	32	31	37	32	52	
RR, multiply-adjusted	1.0	1.01	1.12	1.00	1.60	0.07
95% CI		0.62-1.66	0.69-1.80	0.61-1.64	1.02-2.51	
GL by BMI						
BMI (<25 kg/m^2)						
n events	44	53	49	60	44	
RR, multiply-adjusted†	1.0	1.09	0.98	1.14	0.74	0.26
95% CI		0.71-1.65	0.64-1.50	0.76-1.72	0.47-1.14	
BMI (25-30 kg/m^2)						
n events	58	66	58	46	63	
RR, multiply-adjusted†	1.0	0.96	0.85	0.72	1.10	0.81
95% CI		0.66-1.39	0.58-1.25	0.48-1.08	0.76-1.58	
BMI (≥30 kg/m^2)						
n events	32	39	50	52	43	
RR, multiply-adjusted†	1.0	1.14	1.54	1.72	1.68	<0.01
95% CI		0.71-1.83	0.97-2.44	1.09-2.70	1.06-2.67	
BMI (≥30 kg/m^2), nondiabetics						
n events	21	34	42	49	38	
RR, multiply-adjusted	1.0	1.50	2.02	2.39	2.12	<0.01
95% CI		0.87-2.60	1.18-3.45	1.42-4.03	1.24-3.64	
Rectal cancer (*n* events = 209)						
GI by BMI						
BMI (<25 kg/m^2)						
n events	16	16	20	15	9	
RR, multiply-adjusted†	1.0	1.02	1.13	1.00	0.52	0.16
95% CI		0.50-2.09	0.56-2.28	0.48-2.06	0.22-1.23	
BMI (25-30 kg/m^2)						
n events	16	18	19	15	9	
RR, multiply-adjusted†	1.0	1.08	1.11	0.75	0.58	0.12
95% CI		0.53-2.16	0.55-2.24	0.35-1.62	0.25-1.33	
BMI (≥30 kg/m^2)						
n events	4	10	10	17	15	
RR, multiply-adjusted†	1.0	2.38	2.43	3.84	3.34	0.02
95% CI		0.73-7.77	0.76-7.79	1.27-11.62	1.09-10.20	
BMI (≥30 kg/m^2), nondiabetics						
n events	3	9	7	15	13	
RR, multiply-adjusted	1.0	2.77	2.26	5.07	4.22	0.01
95% CI		0.73-10.5	0.58-8.78	1.46-17.6	1.19-14.9	
GL by BMI						
BMI (<25 kg/m^2)						
n events	16	17	12	15	16	
RR, multiply-adjusted†	1.0	1.14	0.73	0.87	0.88	0.54
95% CI		0.55-2.35	0.32-1.63	0.41-1.88	0.41-1.86	
BMI (25-30 kg/m^2)						
n events	18	15	15	18	11	
RR, multiply-adjusted†	1.0	0.71	0.71	0.95	0.66	0.54
95% CI		0.34-1.50	0.33-1.50	0.47-1.92	0.30-1.44	
BMI (≥30 kg/m^2)						
n events	8	10	12	13	13	
RR, multiply-adjusted†	1.0	1.26	1.17	1.99	2.23	0.04
95% CI		0.49-3.24	0.43-3.17	0.81-4.89	0.91-5.45	
BMI (≥30 kg/m^2), nondiabetics						
n events	6	8	10	11	12	
RR, multiply-adjusted	1.0	1.33	1.71	2.20	2.62	0.03
95% CI		0.46-3.88	0.60-4.89	0.80-6.06	0.97-7.09	

*Adjusted for age (continuous), energy (quintiles), activity level (high, medium, low), multivitamin use (yes, no), diabetes (yes, no), smoking (pack-years), BMI (quintiles), and waist/hip ratio (quintiles).
†Adjusted for all characteristics listed above, except adjusted for BMI (continuous) within BMI quintiles.

Age- and energy-adjusted prevalences of risk factors were compared by quintiles of GI and GL using analysis of covariance. Age-adjusted incidence rates and relative risks (RR) of colorectal, colon, and rectal cancers in relation to baseline characteristics, along with their 95% confidence intervals (CI), were calculated using Poisson regression. Multivariately adjusted RRs and their 95% CI as well as *P* values for trend (using an ordinal variable to designate the quintiles) were computed using proportional hazards regression. Potential confounders were identified by first examining each characteristic's relation to the exposure (GI/GL) and outcome (colorectal, colon, or rectal cancer). Those that seemed to be related to both the exposure and the outcome were added to the proportional hazards model. They were retained in the model as confounders if they changed the GI/GL regression coefficient by >10%. Multiplicative interactions were tested using cross-product terms.

Results

The mean ± SD baseline value for the energy-adjusted average GI of the baseline diets was 85.1 ± 5.5, and for the energy-adjusted GL was 169.6 ± 32.4. Because of the large sample size, GI and GL were correlated statistically significantly, but often weakly, with most baseline characteristics analyzed (Web Table S1).

Over 15 years of follow-up, we identified 757 incident cases of colon cancer and 209 cases of rectal cancer (954 CRC cases). As shown in Web Table S2, age-adjusted incidence of CRC from 1986 to 2000 was associated positively with baseline BMI, waist/hip ratio, history of diabetes mellitus, pack-years of smoking, and height, and was associated inversely with estrogen use, physical activity level, fruit, dairy, whole-grain consumption, energy intake, fiber (insoluble and soluble), carbohydrates, calcium, folate, multivitamin use, vitamin D, vitamin A, and vitamin E. No association was seen with alcohol use, vegetable intake, starch, caffeine, β-carotene, fructose, glucose, or sucrose intake.

Table 1 shows the adjusted RRs of CRC in relation to quintiles of GI and GL. When adjusted for age and energy, we found no statistically significant associations between either GI or GL and incident CRC. Adjustment for other risk factors did not appreciably alter the RR estimates. Adding other dietary variables (dairy, fruit, whole-grain, carbohydrates, soluble fiber, calcium, vitamin E, and vitamin D) to the risk model produced nearly identical results (data not shown). Separate analyses based on colon and rectal cancer subsites were similarly unremarkable. When we restricted these analyses to the 33,071 women free of diabetes at baseline, among whom 877 developed CRC, we still observed no association with GI or GL.

As shown in Table 1, analyses stratified by BMI (<25, 25-30, and ≥30 kg/m^2) showed that GI and GL were positively associated with CRC in the highest BMI category (*P* for interaction = 0.04 for GI and > 0.05 for GL). Colon and rectal cancers were then analyzed individually (Table 2). GL, but not GI, was positively associated with colon cancer in the highest BMI category, whereas GL and GI were both positively associated with rectal cancer in the highest BMI category. All of these associations in the high BMI stratum were somewhat stronger among nondiabetic women (Tables 1 and 2).

Discussion

In this prospective study, average dietary GI and GL were associated positively, in a graded fashion, with incidence of CRC among obese women. The multivariately-adjusted RR among obese women was 1.66 for the highest versus lowest quintile of GI, and 1.79 for the highest versus lowest quintile of GL. The association with GL in obese women held for both colon and rectal cancers. The association with GI in obese women held for rectal, but not colon cancer. All of these findings were slightly stronger after excluding women with baseline diabetes.

Three prior case-control studies (3-5) and two cohort studies (7, 9) have reported a positive association between GI/GL and CRC, but two cohort studies (6, 8) did not find an association. Similar to our finding, one of these previous studies found that elevated BMI (in men) or waist/hip ratio (in women) seemed to enhance CRC risk when the diet was high in GL (4). Another study found that the association between GL and CRC was slightly stronger among men with elevated BMI, but no such interaction existed among the women studied (9). Two previous studies have found that the associations between GL and CRC did not vary appreciably across strata of BMI (6, 8). Another study on colon cancer found a three-way interaction of colon cancer with physical activity, BMI and sucrose/dietary fiber ratio (3). Thus, several studies support a stronger association between GI/GL and CRC in obese subjects; however, this interaction is not universally seen, perhaps due to limits of statistical power, differences in study populations, or chance.

Obesity increases insulin resistance (17), which leads to high blood insulin levels. When compared with low GI foods, the consumption of foods with a high GI results in a higher increase in blood glucose levels leading to a higher increase in blood insulin levels (10). This combination could produce a condition of extremely high blood insulin levels; increased insulin has been shown to increase insulin-like growth factor-I, both of which could stimulate the proliferation of colorectal cells and inhibit apoptosis (18). The proliferation of colorectal cells in an environment with reduced apoptosis could potentially result in an accumulation of mutations leading to unregulated growth and cancer.

The main weakness of our analysis was that it was based on a single food frequency questionnaire. A single measure of diet is imprecise and a typically random error would lead to an underestimation of the true association. Although we examined a number of characteristics for potential confounding, additional confounding factors may have been missed (e.g., family history of CRC). Due to the small number of rectal cancer, separate analyses of rectal cancer and GI/GL had low power. Our study population comprised women who were 55 to 69 years old at baseline and were primarily Caucasian (99%); our findings may not hold true for men or other ethnic groups.

In summary, our analyses indicate that there is no overall association between diets with a high average GI/GL and CRC risk among nonobese, older women. However, women who are obese may experience increased CRC risks by consuming a high glycemic diet. Our data support recently revised diet and exercise guidelines (19), which recommend maintaining body weight within a healthy range, and consumption of whole-grains rather than refined grains as the preferred source of carbohydrates.

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