Influence of Family History of Colorectal Cancer on Health Behavior and Performance of Early Detection Procedures: The SUN Project

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PURPOSE: The aim of this study is to explore the relationship between family history of colorectal cancer and both health behavior and screening procedures in a population cohort.
METHODS: This study is a cross-sectional analysis of 15,169 participants belonging to a prospective cohort study (the SUN Project) based on two self-reported questionnaires: one of them related to lifestyle and the other a semiquantitative food frequency questionnaire. We explored the influence of family history of colorectal cancer in lifestyles (consumption of alcohol, weight, and diet) and medical management behaviors (screening of chronic diseases).
RESULTS: People with family history of colorectal cancer increased their number of colorectal cancer screening tests (adjusted odds ratio for fecal occult blood test: 1.98, 95% confidence interval: 1.48–2.65; and adjusted odds ratio for colonoscopy/sigmoidoscopy: 3.42, 2.69–4.36); nevertheless, health behavior changes in diet of relatives of colorectal cancer patients were undetectable.
CONCLUSIONS: We show that individuals with a family history of colorectal cancer increase their compliance with screening tests, although they exhibit no better health-related behaviors than people without family history of colorectal cancer. Further prospective studies are required to confirm these results and to identify tools to empower the subjects to change their risk profile.
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KEY WORDS: Colorectal Cancer, Family History, Healthy Behavior, Screening.

INTRODUCTION

Colorectal cancer (CRC) is currently the second leading type of cancer and one of the most common causes of cancer-related death worldwide among women and men (1). There were more than 610,000 deaths caused by CRC in 2008 worldwide (2). In Spain in 2006, it was responsible for 7703 deaths in men and 5631 in women (3).

Family history is a risk factor for CRC. It is estimated that 20% of all CRC cases are attributable to familial aggregation (4). Combinations of genetic and environmental factors are thought to play a role in the clustering of familial CRC (5). Evidence indicates that early detection reduces the rates of morbidity and mortality of CRC (6), and a variety of screening modes are endorsed by evidence-based guidelines (7, 8). Moreover, several behavioral factors such as exercise (9), obesity (10), alcohol consumption (11), and diet (12) have been suggested to modify the risk for this cancer.

The relationship between family history of cancer and changes in health behaviors has not been studied in CRC; however, some studies developed in women with family history of breast cancer shown contradictory results (13, 14). Lemon and colleagues (13) study showed an improvement in healthy habits (increase in physical activity, increase in fruit and vegetable consumption, decrease in fat consumption) in women with a first-degree relative with breast cancer when comparing health habits just after the diagnosis with health habits 6 months later; it should be noted that no control group (i.e., women without a first-degree relative with breast cancer) was included in their study. However, Madlensky and colleagues (14) classified women in three risk groups for breast cancer: highest risk, moderate risk, and average risk; they found no differences in lifestyle, although women at highest risk (i.e., those related with at least two individuals with breast cancer, at least one of which is a first-degree relative) were more likely to have had a mammography before the age of 40.

Regarding medical management behaviors, such as screening, most studies reported that people with family history of CRC have a clear increase in screening practices (15–17); nevertheless, in some studies authors suggested

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Selected Abbreviations and Acronyms

BMI = body mass index
CI = confidence interval
CRC = colorectal cancer
EKG = electrocardiogram
FOBT = fecal occult blood test
OR = odds ratio
SUN project = *Seguimiento Universidad de Navarra* project

that screening rates among people with family history of CRC are suboptimal (18).

It can be hypothesized that people with family history of CRC could change their health behavior; this change would be cancer-site specific (i.e., an increase in CRC screening and higher diet-fiber intake) or unspecific (i.e., more medical tests or improving health behavior, despite whether they are related with CRC). The main aim of this study is to explore the relationship between family history of CRC and health behavior and screening procedures in a population cohort.

METHODS

The SUN (Seguimiento Universidad de Navarra) project is a prospective cohort study of Spanish university graduates with the aim of establishing the association between several sociodemographic, nutritional, and lifestyle characteristics and the occurrence of different diseases (19). The SUN project was designed in 1998 in the Universidad de Navarra with the collaboration of some investigators from the Harvard School of Public Health and adapted the methods previously used by large cohorts such as Nurses' Health Study (20) and Health Professionals Follow-up Study (21). The response rate in our cohort is of 22%. However, our main concern during recruitment is to identify highly motivated participants. The very long baseline questionnaire helps us to identify them; with these faithful participants, we can obtain a retention rate of 89%, a basic element for a very high internal validity, and therefore, for external validity.

Recruitment is ongoing because this is a dynamic cohort study; at recruitment, participants are asked to complete a baseline self-administered questionnaire to record information on demographic data, personal and family medical history, screening tests performed, and lifestyles. A semi-quantitative questionnaire (22) previously validated in Spain (23) was used to assess food frequency. In this paper, a cross-sectional analysis of baseline data of this cohort was performed. Patients having a diagnosis of cardiovascular disease or cancer at recruitment were excluded from this analysis. Family history of CRC was dichotomized (with and without family history of CRC). A subject was considered to have family history of CRC if at least one of his/her first-degree relatives had been diagnosed with CRC.

We collected data on the following tests: colonoscopy/sigmoidoscopy, fecal occult blood test (FOBT), medical check-up, electrocardiogram, chest X-ray, dental check-up, eye pressure test, Papanicolaou test, mammography, rectal digital test, transrectal ultrasound, and prostate-specific antigen, performed without diagnosis intention (i.e., without suggestive symptoms of disease); these variables were grouped as once or never. Body mass index (BMI) was grouped as normal (18.5–24.9 kg/m^2, overweight (25.0–29.9 kg/m^2), or obesity ($\geqslant$30.0 kg/m^2). Alcohol beverage consumption was assessed at baseline in 5 of the 136 items of the food frequency questionnaire, with 6 additional questions referring to the pattern of alcohol consumption. From this information, trained dietitians derived the total alcohol intake by using the latest available food-composition tables for Spain.

The association among family history of CRC and performing screening tests or lifestyle behaviors was assessed using odds ratios (OR) and 95% confidence interval (CIs); any nutrient distribution was categorized in quartiles, and the first quartile was used as reference; reported recent changes in health habits were coded as 1 (change to healthy behavior) or 0 (no change to healthy behavior); therefore, an OR greater than 1 indicates greater compliance with healthy behavior. In this regard, "recent changes" refers to the completion of questionnaire, despite when CRC appeared in relatives. Adjustment for confounders was performed with the use of unconditional logistic regression. All models were adjusted for age (years), sex, obesity (yes/no), and alcohol consumption (continuous). Because CRC can be perceived as threatening as people age, we repeat all analyses in people 43 years of age or older (i.e., people over the third quartile of age). The Benjamini-Hochberg method was used to correct p values for multiple comparisons (24). All analyses were performed with Stata 10/SE software (Stata Corporation, College Station, TX).

RESULTS

Population Description

A total of 20,579 subjects answered the recruitment questionnaire by December 31, 2010. A total of 4917 participants were excluded because of previous history of cardiovascular disease and 743 because of previous history of cancer of any localization (250 of them reported cardiovascular disease too); therefore, 15,169 subjects were included in this analysis. They were predominantly women (66%) and young adults (median: 33 years, interquartile range: 26–43 years; Table 1). There were 576 subjects (3.8%) who reported familial antecedents of CRC. People

TABLE 1. Description of the 15,164 subjects included in the study according to the family history of CRC

Variable	Category	Whole cohort	Family history of CRC	No family history of CRC	p value
Body mass index, kg/m^2		23.0 ± 3.3	23.7 ± 3.2	22.9 ± 3.3	<.001
	Normal weight (18.5–24.9)	11,571 (76.7%)	395 (68.7%)	11,176 (77.0%)	<.001
	Overweight (25.0–29.9)	3075 (20.4%)	166 (28.9%)	2909 (20.0%)	
	Obesity (⩾30)	445 (2.9%)	14 (2.4%)	431 (3.0%)	
Age, years		35.2 ± 10.8	43.4 ± 11.0	34.9 ± 10.7	<.001
	<45	12,157 (80.2%)	313 (54.3%)	11,844 (81.2%)	<.001
	45–64	2827 (18.7%)	242 (42.0%)	2585 (17.7%)	
	⩾65	178 (1.1%)	21 (3.7%)	157 (1.1%)	
Alcohol consumption, g/day		6.0 ± 9.3	6.9 ± 9.9	6.0 ± 9.3	.01
	First tertile (0–1.5 g/day)	5378 (35.5%)	188 (32.6%)	5190 (35.6%)	.06
	Second tertile (1.5–6.8 g/day)	5214 (34.4%)	189 (32.8%)	5025 (34.5%)	
	Third tertile (⩾6.8 g/day)	4572 (30.1%)	199 (34.6%)	4373 (30.0%)	
Tobacco smoking	Never smoker	7758 (49.5%)	883 (48.5%)	6875 (49.7%)	.06
	Current smoker	3612 (23.1%)	402 (22.1%)	3210 (23.2%)	
	Former smoker	3904 (24.9%)	496 (27.3%)	3408 (24.6%)	
Gender: women		10,047 (66.3%)	335 (58.2%)	9712 (66.6%)	<.001
Family history of CRC	Total		576 (3.8)		
	Father		366*		
	Mother		189*		
	Brother/Sister		41*		

CRC = colorectal cancer.
*Total adds more than 576 because 20 people reported more than one CRC in his/her first-degree relatives.

with previous history of CRC were older (43 years vs. 35 years), less frequently women (58.2% vs. 66.6%), had a greater BMI (23.7 kg/m^2 vs. 22.9 kg/m^2), and they drank more alcohol (6.9 g/day vs. 6.0 g/day; Table 1).

Familial Antecedents of CRC and Performance of Diagnostic Tests

Table 2 displays the numbers of people performing screening tests without clinical symptoms (i.e., tests intended to early diagnosis, irrespective of whether they were usually recommended). People with previous history of CRC received FOBT twice as frequently and colonoscopy three times as frequently as people without such antecedent (adjusted OR 1.98, 95% CI 1.48–2.65 for FOBT and 3.42, 95% CI 2.69–4.36 for colonoscopy); these results did not differ by gender. Women with previous history of CRC performed mammography or Papanicolaou test more frequently, but these differences disappeared when we adjusted for age, alcohol consumption, and obesity; however, men with previous history of CRC performed screening tests related with prostate cancer more frequently than men without familial antecedents (digital rectal examination: adjusted OR 1.73, 95% CI 1.21–2.47; transrectal ultrasound: adjusted OR 1.84, 95% CI 1.30–2.61; prostatic specific antigen: adjusted OR 1.50, 95% CI 1.01–2.24); only transrectal

TABLE 2. Relationship between family history of CRC and performance of diagnosis tests without clinical symptomatology

Test	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	Crude OR (95% CI)	Adjusted OR (95% CI)*	Adjusted p value*	BH p value
Medical check-up	396	10,068	0.99 (0.82–1.19)	1.10 (0.90–1.33)	.35	.95
Electrocardiogram	356	7522	1.52 (1.28–1.82)	1.09 (0.90–1.31)	.39	.95
Chest X-ray	296	5897	1.56 (1.32–1.85)	1.08 (0.90–1.29)	.44	.95
Fecal occult blood test	61	654	2.52 (1.88–3.34)	1.98 (1.48–2.65)	<.001	<.001
Colonoscopy/sigmoidoscopy	119	679	5.34 (4.26–6.64)	3.42 (2.69–4.36)	<.001	<.001
Dental check-up	527	13,332	1.02 (0.75–1.40)	0.92 (0.66–1.27)	.61	.95
Eye pressure test	214	4526	1.31 (1.10–1.57)	0.96 (0.80–1.16)	.69	.95
Papanicolaou test	280 (n = 335)	6405 (n = 9717)	2.63 (1.96–3.59)	1.08 (0.77–1.51)†	.66†	.95
Mammography	189 (n = 335)	2893 (n = 9717)	3.05 (2.44–3.83)	1.11 (0.82–1.48)†	.50†	.95
Rectal digital exam	64 (n = 241)	572 (n = 4881)	2.72 (1.99–3.70)	1.73 (1.21–2.47)†	.003†	.13
Transrectal ultrasound	60 (n = 241)	563 (n = 4881)	2.54 (1.84–3.47)	1.84 (1.30–2.61)†	.001†	.05
Prostate-specific antigen	52 (n = 241)	452 (n = 4881)	2.70 (1.91–3.74)	1.50 (1.01–2.24)†	.04†	.40

BH p value = Benjamini-Hochberg corrected for multiple comparisons; CI = confidence interval; CRC = colorectal cancer; OR = odds ratio.
*Adjusted for age, gender, obesity, and alcohol consumption.
†Adjusted for age, obesity, and alcohol consumption.

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ultrasound remains significant after we adjusted for multiple comparisons. There were no other differences in performing screening tests (Table 2). In the subgroup of people older than 43 years of age, only FOBT and colonoscopy remain statistically significant (OR 2.27, 95% CI 1.60–3.23, $p < .001$ for FOBT; OR 3.15, 95% CI 2.32–4.27, $p < .001$ for colonoscopy).

Familial Antecedents of CRC and Healthy Habits

Differences in the average consumption of nutrients in subjects with and without familial antecedents of CRC are shown in Table 3. There are small differences in total fat, saturated fat acids, greater alcohol consumption, and more fiber consumption, but all differences were nonsignificant after we adjusted for multiple comparisons. To further analyze the relationship between nutrient consumption and familial antecedents of CRC, we categorized each nutrient in quartiles of consumption, and we estimated its association with familial antecedents via logistic regression adjusting for age, gender, alcohol consumption and obesity; most odds ratios were near 1, and no single association remained significant after adjusting p-values for multiple comparisons (Table 4–Appendix).

Finally, we assessed the association between the familial antecedents of CRC and the change in health habits. We found no changes in the consumption of fiber, fruit, vegetables, fish, or specific nutrients according to familial antecedents of CRC (Table 5). No single nutrient or reported change in health habits reach statistical signification in the subgroup with higher age (i.e., people older than 43 years of age). All results remain substantially unchanged when we adjusted

TABLE 3. Description of food category and nutrient intake among the study sample: comparison of means between patients with and without familial antecedents of colorectal cancer

Variable	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	p value	BH p value*
Carbohydrates, g/day	273.6 ± 116.5	279.9 ± 121.5	.22	.88
Proteins, g/day	111.0 ± 38.8	112.7 ± 41.9	.33	.95
Lipids, g/day	101.8 ± 44.3	106.1 ± 46.1	.03	.36
Saturated fatty acids, g/day	34.7 ± 17.2	36.5 ± 17.8	.02	.34
Monounsaturated fatty acids, g/day	43.6 ± 19.6	45.1 ± 20.3	.08	.54
Poliunsaturated fatty acids, g/day	14.6 ± 7.6	15.3 ± 8.7	.07	.51
Alcohol, g/day	6.9 ± 9.9	6.0 ± 9.3	.02	.34
Total energy, Kcal/day	2503 ± 928	2567 ± 969	.12	.65
Vitamin C, mg/day	305.6 ± 188.9	295.0 ± 184.2	.18	.80
Fiber from vegetables, g/day	11.5 ± 8.7	10.8 ± 8.0	.02	.34
Fiber from fruits, g/day	6.7 ± 5.4	6.3 ± 5.6	.14	.67
Fiber from cereals, g/day	3.6 ± 3.2	3.6 ± 3.3	.89	.96
Fiber from legumes, g/day	4.00 ± 3.64	4.01 ± 4.47	.96	.97
Fiber from others, g/day	4.17 ± 3.74	4.18 ± 4.84	.94	.96
Vegetables, g/day	570.1 ± 380.1	545.8 ± 392.1	.14	.67
Fruits, g/day	378.9 ± 325.5	362.5 ± 349.9	.27	.95
Nonfat dairy products, g/day	232.1 ± 276.4	226.8 ± 259.7	.63	.95
Olive oil, g/day	15.8 ± 14.5	16.1 ± 14.5	.56	.95
Unsaturated fats, g/day	85.7 ± 40.2	89.7 ± 42.5	.03	.36
ω-3 fatty acids, g/day	2.90 ± 1.52	2.81 ± 1.58	.20	.86
Vitamin A, (μg/day)	2204 ± 1705	2076 ± 1663	.07	.51
Folic acid, (μg/day)	445.4 ± 243.9	430.5 ± 220.0	.11	.64
Vitamin B1, mg/day	1.95 ± 0.79	1.97 ± 0.79	.65	.95
Vitamin B2, mg/day	2.36 ± 0.95	2.39 ± 0.95	.41	.95
Vitamin B3, mg/day	44.5 ± 15.8	44.9 ± 17.3	.59	.95
Vitamin B6, mg/day	2.96 ± 1.27	2.91 ± 1.25	.39	.95
Vitamin B12, μg/day	9.87 ± 5.32	10.01 ± 5.85	.56	.95
Vitamin D, μg/day	3.97 ± 0.12	3.86 ± 2.87	.37	.95
Vitamin E, mg/day	7.47 ± 4.59	7.66 ± 4.95	.38	.95
Fast food, g/day	21.7 ± 31.4	24.3 ± 26.4	.02	.34
Red meat, g/day	80.2 ± 57.0	82.0 ± 54.5	.44	.95
Sugar, g/day	15.8 ± 17.2	17.3 ± 18.0	.05	.47
Sodium, mg/day	3468 ± 2292	3699 ± 3116	.08	.54
Sodium from salt, mg/day	4074 ± 2383	4331 ± 3202	.06	.50
Potassium, mg/day	5128 ± 2146	5074 ± 2185	.56	.95

BH p value = Benjamini-Hochberg corrected for multiple comparisons; CRC = colorectal cancer.
*Benjamini-Hochberg corrected for multiple comparisons.

TABLE 5. Relationship between reporting recent changes in health habits and family history of CRC

Variable	Category	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	Adjusted OR*	Adjusted p value*	BH p value†
Try to eat a lot of fiber	Yes	365	8463	1.06 (0.88–1.28)	.55	.95
	No	205	5878	1	–	
Try to eat a lot of fruit	Yes	423	9745	1.09 (0.89–1.33)	.43	.95
	No	148	4675	1	–	
Try to eat a lot of vegetables	Yes	474	11,556	1.03 (0.81–1.31)	.83	.96
	No	96	2851	1	–	
Try to eat a lot of fish	Yes	369	8337	1.17 (0.97–1.42)	.10	.62
	No	196	6022	1	–	
To adhere to a special diet	Yes	51	918	1.27 (0.92–1.77)	.15	.7
	No	515	13,319	1	–	
Try to reduce consumption of butter	Yes	423	9735	1.12 (0.91–1.38)	.30	.95
	No	140	4448	1	–	
Try to reduce consumption of fat	Yes	450	11,033	0.93 (0.74–1.17)	.53	.95
	No	109	3179	1	–	
Try to reduce consumption of meat	Yes	227	4699	1.13 (0.93–1.36)	.22	.88
	No	334	9496	1	–	
Try to reduce consumption of salt	Yes	257	6005	1.08 (0.90–1.30)	.41	.95
	No	302	8166	1	–	
Try to reduce consumption of sugar	Yes	152	4051	0.95 (0.77–1.17)	.62	.95
	No	401	10,088	1	–	
Try to reduce consumption of sweets	Yes	368	8858	1.04 (0.86–1.26)	.67	.95
	No	186	5285	1	–	

BH p value = Benjamini-Hochberg corrected for multiple comparisons; CI = confidence interval; CRC = colorectal cancer; OR = odds ratio.
*Adjusted for age, gender, obesity, and alcohol consumption.
†Benjamini-Hochberg corrected for multiple comparisons.

for BMI in three categories (normal weight, overweight, and obesity) or when models were not adjusted for BMI.

DISCUSSION

The main result in this study is that people with family history of CRC are more likely to receive screening for CRC with colonoscopy/sigmoidoscopy or FOBT. Nevertheless, they do not fulfill other preventive measures, such as controlling their weight, attempting to follow a healthier diet, or decreasing alcohol and tobacco consumption.

Individuals with family history of CRC are three times more likely to undergo colonoscopy/sigmoidoscopy and more than two times in the case of FOBT. Similar results have been reported by others (15–17). However, Bleiker and colleagues (18) observed that approximately one-fourth of those with family history of CRC deviated significantly from the recommended frequency of screening.

On the other hand, our study shows a lack of influence of family antecedents on behavioral changes. People with family history of CRC do not adopt protective behavioral patterns, such as weight control, more physical exercise, or a decrease in alcohol consumption. In this regard, other authors (25, 26) found no differences in alcohol consumption between women with and without a family history of breast cancer.

Regarding dietary patterns in our study, people with family history of CRC consume similar amounts of fiber, sweets, and fat than those without a family history of CRC, a result that contrasts with the widely discussed relationship between CRC and dietetic habits (12). We can speculate that people with family history easily comply with measures performed by health professionals but avoid complying with measures that require changes in their own conduct.

In summary, the general picture our results suggest is that people at high risk for CRC because of their familial history undergo more screening tests because physicians perform more tests in high-risk patients or because the patients themselves—being more aware of their history—demand more tests. However, they do not change their health behaviors to decrease their CRC risk; we have no data on whether these subjects are unaware of the relevance of changing diet and other behaviors or—being aware of it—they are unable to implement the healthy changes.

The consequence is the lack of an effective reduction in risk factor prevalence in high-risk subjects. Most guidelines on CRC prevention point at early diagnostic efforts via screening tests (27–30), whereas they pay scarce or no attention to the reduction of CRC risk by lowering a person's exposure to risk factors. According to our results, we suggest that guidelines on CRC prevention should also focus on reinforcing the subject knowledge on modifiable risk factors and their ability to change them; further prospective studies are needed to identify the effectiveness of clinician recommendations to modify patient behaviors.

Finally, several limitations of our work are to be noted: first, the reported results are derived from a cross-sectional analysis and do not allow cause and effect to be distinguished. Second, our study population is entirely composed of university graduates, and people with high level of education might overestimate their healthy lifestyle; because this overestimation would probably be as great in subjects with and without family history of CRC, it could cause a nondifferential classification bias. However, selecting university graduates presents the advantages that they understand mailed questionnaires (15) more easily and completely. In addition, high socioeconomic level is associated with healthier life styles (31); thus, it is possible that participants with family history of CRC in our study required less pronounced changes in diet that people of lower socioeconomic status with a family history of CRC, which can explain why diet changes were statistically nonsignificant in our sample. Nevertheless, it is noteworthy that no changes in diet were identified when we compared the first and forth quartiles in nutrient distribution (Table 4). Despite this, whether the lack of relationship between diet and family history of CRC we have found is specific of the high educational level in our sample or can be generalized requires further study. Third, we have not measured the time after CRC diagnosis of first-degree relative(s), which could influence both risk perceptions and lifestyle changes in these participants.

In conclusion, our study shows that individuals with family history of CRC demonstrate greater compliance with screening tests than those without family history, whereas no association can be found between family history of CRC and health-related behaviors. Further prospective studies are required in order to confirm these results and to identify tools to empower the subjects to change their risk profile.

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REFERENCES

1. Curado MP, Edwards B, Shin HR, Storm H, Ferlay J, Heanue M, et al. Cancer Incidence in Five Continents, Vol IX. Scientific Publications No 160. Lyon: IARC; 2008.
2. World Health Organization. Cancer. Fact sheet No 297. WHO. 2009. Available at: http://www.who.int/mediacentre/factsheets/fs297/en/.
3. Cabanes Domenech A, Pérez-Gómez B, Aragonés N, Pollán M, López-Abente G. La situación del Cáncer en España. 1975–2006. Madrid: Centro Nacional de Epidemiología. ISCIII; 2009.
4. Lynch HT, de la Chapelle A. Hereditary colorectal cancer. N Engl J Med. 2003;348:919–932.
5. Noe M, Schory P, Demierre MF, Babayan R. Increased cancer risk for individuals with a family history of prostate cancer, colorectal cancer, and melanoma and their associated screening recommendations and practices. Cancer Causes Control. 2008;19:1–12.
6. Mendel JS, Bond JH, Church TR, Snover DC, Bradley GM, Schuman LM, et al. Reducing mortality from colorectal cancer by screening for fecal occult blood. Minnesota Colon Cancer Control Study. N Engl J Med. 1993;328:1365–1371.
7. Levin B, Lieberman DA, McFarland B, Andrews KS, Brooks D, Bond J, et al. American Cancer Society Colorectal Cancer Advisory Group, US Multi-Society Task Force, American College of Radiology Colon Cancer Committee. Screening and surveillance for the early detection of colorectal cancer and adenomatous polyps 2008: a joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. Gastroenterology. 2008;134:1570–1595.
8. Rex DK, Johnson DA, Anderson JC, Schoenfeld PS, Burke CA, Inadomi JM. American College of Gastroenterology Guidelines for Colorectal Cancer Screening 2008. Am J Gastroenterol. 2009;104:739–750.
9. Harriss DJ, Atkinson G, Batterham A, George K, Cable NT, Reilly T, et al. Colorectal Cancer, Lifestyle, Exercise And Research Group. Lifestyle factors and colorectal cancer risk (2): a systematic review and meta-analysis of associations with leisure-time physical activity. Colorectal Dis. 2009;11:689–701.
10. Harriss DJ, Atkinson G, George K, Cable NT, Reilly T, Haboubi N, et al. C-CLEAR group. Lifestyle factors and colorectal cancer risk (1): systematic review and meta-analysis of associations with body mass index. Colorectal Dis. 2009;11:547–563.
11. Cho E, Smith-Warner SA, Ritz J, van den Brandt PA, Colditz GA, Folsom AR, et al. Alcohol intake and colorectal cancer: a pooled analysis of 8 cohort studies. Ann Intern Med. 2004;140:603–613.
12. Ryan-Harshman M, Aldoori W. Diet and colorectal cancer: review of the evidence. Can Fam Physician. 2007;53:1913–1920.
13. Lemon SC, Zapka JG, Clemow L. Health behaviour change among women with recent familial diagnosis of breast cancer. Prev Med. 2004;39:253–262.
14. Madlensky L, Vierkant RA, Vachon CM, Pankratz VS, Cerhan JR, Vadaparampil ST, et al. Preventive health behaviors and familial breast cancer. Cancer Epidemiol Biomarkers Prev. 2005;14:2340–2345.
15. Felsen CB, Piasecki A, Ferrante JM, Ohman-Strickland PA, Crabtree BF. Colorectal cancer screening among primary care patients: does risk affect screening behavior? J Community Health. 2011;36:605–611.
16. Longacre AV, Cramer LD, Gross CP. Screening colonoscopy use among individuals as higher colorectal cancer risk. J Clin Gastroenterol. 2006;40:490–496.
17. Manne S, Markowitz A, Winawer S. Correlates of colorectal cancer screening compliance and stage of adoption among siblings of individuals with early onset colorectal cancer. Health Psychol. 2002;21:3–15.
18. Bleiker EM, Menko FH, Taal BG. Screening behaviour of individuals at high risk for colorectal cancer. Gastroenterology. 2005;128:280–287.
19. Martínez-González MA, Sánchez-Villegas A, De Irala J, Martí A, Martínez JA. Mediterranean diet and stroke: objectives and design of the SUN project. Nutr Neurosci. 2002;5:65–73.
20. Lui S, Manson JE, Stamper MJ, Rexrode KM, Hu FB, Rimm EB, et al. Whole grain consumption and risk of ischemic stroke in women. JAMA. 2000;284:1534–1540.
21. Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D, Willett WC. Prospective study of major dietary patterns and risk of coronary heart disease in men. Am J Nutr. 2000;72:912–921.
22. Willet WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, et al. Reproducibility and validity of a semiquantitative food frequency questionnaire. Am J Epidemiol. 1985;122:51–65.

23. Martin-Moreno JM, Boyle P, Gorgojo L, Maisonneuve P, Fernandez-Rodriguez JC, Salvini S, et al. Development and validation of a food frequency questionnaire in Spain. Int J Epidemiol. 1993;22:512–519.
24. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. J Royal Stat Soc B. 1995;57:289–300.
25. Madlensky L, Flatt SW, Bardwell WA, Rock CL, Pierce JP, WHEL Study group. Is family history related to preventive health behaviors and medical management in breast cancer patients? Breast Cancer Res Treat. 2005;90:47–54.
26. Emmons KM, Kalkbrenner KJ, Klar N, Light T, Schneider KA, Garber JE. Behavioral risk factors among women presenting for genetic testing. Cancer Epidemiol Biomarkers Prev. 2000;9:89–94.
27. U.S. Preventive Services Task Force. Screening for colorectal cancer: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2008;149:627–637.
28. Kaiser Permanente Care Management Institute. Colorectal Cancer Screening Clinical Practice Guideline. Oakland (CA): Kaiser Permanente Care Management Institute; 2008.
29. Hereditary Colorectal CancerAmsterdam, The Netherlands: Association of Comprehensive Cancer Centres (ACCC); 2009.
30. Levin B, Lieberman DA, McFarland B, Smith RA, Brooks D, Andrews KS, et al. American Cancer Society Colorectal Cancer Advisory Group, US Multi-Society Task Force, American College of Radiology Colon Cancer Committee. Screening and surveillance for the early detection of colorectal cancer and adenomatous polyps, 2008: a joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. CA Cancer J Clin. 2008;58:130–160.
31. Alvarez-Dardet C, Montahud C, Ruiz MT. The widening social class gap of preventive health behaviours in Spain. Eur J Public Health. 2001;11: 225–226.

APPENDIX

TABLE 4. Relationship between health habits and familial antecedents of colorectal cancer (OR obtained via logistic regression)

Variable	Quartile	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	Adjusted OR*	Adjusted p value*	BH p value†
Carbohydrates	q1	153	3437	1	–	
	q2	153	3648	1.02 (0.80–1.30)	.87	.96
	q3	120	3788	0.69 (0.53–0.90)	.006	.20
	q4	150	3715	0.98 (0.77–1.25)	.87	.96
Proteins	q1	158	3493	1	–	
	q2	156	3590	1.05 (0.83–1.34)	.68	.95
	q3	123	3704	0.82 (0.63–1.06)	.13	.67
	q4	139	3801	0.94 (0.73–1.20)	.60	.95
Lipids	q1	158	3251	1	–	
	q2	149	3621	0.97 (0.76–1.24)	.79	.96
	q3	143	3803	1.01 (0.79–1.29)	.93	.96
	q4	126	3913	0.90 (0.69–1.17)	.42	.95
Saturated fats	q1	152	3171	1	–	
	q2	160	3610	1.09 (0.85–1.39)	.49	.95
	q3	141	3828	0.98 (0.76–1.26)	.87	.96
	q4	123	3979	0.89 (0.68–1.16)	.39	.95
Monounsaturated fatty acids	q1	144	3338	1	–	
	q2	155	3611	1.11 (0.87–1.43)	.40	.95
	q3	141	3776	1.09 (0.84–1.40)	.53	.95
	q4	136	3863	1.06 (0.82–1.37)	.67	.95
Polyunsaturated fatty acids	q1	140	3412	1	–	
	q2	174	3613	1.32 (1.03–1.69)	.03	.36
	q3	125	3723	1.01 (0.78–1.32)	.94	.96
	q4	137	3826	1.12 (0.87–1.46)	.38	.95
Alcohol	q1	164	4416	1	–	
	q2	113	3292	0.96 (0.74–1.25)	.78	.96
	q3	148	3665	1.15 (0.89–1.48)	.28	.95
	q4	151	3215	1.13 (0.79–1.62)	.49	.95
Total energy	q1	158	3373	1	–	
	q2	162	3664	1.01 (0.79–1.29)	.93	.96
	q3	115	3781	0.73 (0.57–0.96)	.02	.34
	q4	141	3770	0.94 (0.73–1.21)	.64	.95
Vitamin C	q1	137	3683	1	–	
	q2	151	3661	1.09 (0.85–1.40)	.50	.95
	q3	139	3602	0.98 (0.76–1.27)	.87	.96
	q4	149	3642	0.96 (0.74–1.24)	.75	.96

(*Continued*)

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TABLE 4. (Continued)

Variable	Quartile	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	Adjusted OR*	Adjusted p value*	BH p value†
Fiber from vegetables	q1	131	3660	1	–	
	q2	147	3740	1.11 (0.85–1.44)	.44	.95
	q3	136	3602	1.01 (0.78–1.32)	.93	.96
	q4	162	3586	1.16 (0.90–1.51)	.25	.91
Fiber from fruits	q1	127	3777	1	–	
	q2	141	3680	1.00 (0.77–1.30)	1.00	1.00
	q3	151	3611	0.99 (0.76–1.29)	.94	.96
	q4	157	3520	1.01 (0.78–1.31)	.91	.96
Fiber from cereals	q1	153	3729	1	–	
	q2	134	3638	0.94 (0.73–1.21)	.61	.95
	q3	147	3678	0.93 (0.73–1.19)	.58	.95
	q4	142	3543	0.97 (0.76–1.24)	.79	.96
Fiber from legumes	q1	146	3657	1	–	
	q2	151	3732	0.99 (0.77–1.26)	.92	.96
	q3	130	3576	0.93 (0.72–1.20)	.58	.95
	q4	149	3623	0.97 (0.75–1.24)	.79	.96
Fiber from other sources	q1	137	3629	1	–	
	q2	135	3729	0.99 (0.76–1.28)	.94	.96
	q3	145	3656	1.12 (0.87–1.45)	.37	.95
	q4	159	3574	1.15 (0.89–1.47)	.28	.95
Vegetables	q1	141	3707	1	–	
	q2	144	3677	1.02 (0.79–1.31)	.91	.96
	q3	131	3595	0.92 (0.70–1.19)	.51	.95
	q4	160	3609	1.05 (0.82–1.36)	.68	.95
Fruits	q1	125	3789	1	–	
	q2	141	3695	0.98 (0.76–1.28)	.91	.96
	q3	158	3563	1.05 (0.81–1.36)	.71	.96
	q4	152	3541	0.99 (0.76–1.28)	.91	.96
Nonfat dairy products	q1	151	3902	1	–	
	q2	167	3992	1.13 (0.88–1.44)	.34	.95
	q3	113	3152	1.04 (0.80–1.35)	.78	.96
	q4	145	3542	1.13 (0.87–1.45)	.36	.95
Olive oil	q1	163	3990	1	–	
	q2	161	4101	1.01 (0.80–1.29)	.90	.96
	q3	163	4204	1.04 (0.82–1.33)	.73	.96
	q4	89	2293	1.06 (0.80–1.41)	.69	.95
Nonolive oil fat	q1	158	3249	1	–	
	q2	148	3615	1.02 (0.79–1.30)	.90	.96
	q3	147	3781	1.04 (0.81–1.33)	.78	.96
	q4	123	3943	0.91 (0.70–1.19)	.49	.95
ω-3 fatty acids	q1	133	3657	1	–	
	q2	139	3712	1.12 (0.86–1.45)	.41	.95
	q3	144	3607	1.23 (0.95–1.58)	.12	.65
	q4	160	3612	1.31 (1.01–1.68)	.04	.40
Vitamin A	q1	128	3613	1	–	
	q2	154	3683	1.17 (0.90–1.52)	.23	.88
	q3	137	3663	1.08 (0.82–1.40)	.59	.95
	q4	157	3629	1.12 (0.86–1.46)	.39	.95
Folic acid	q1	148	3689	1	–	
	q2	146	3653	0.93 (0.73–1.20)	.59	.95
	q3	134	3661	0.85 (0.66–1.10)	.22	.88
	q4	148	3585	0.92 (0.71–1.18)	.51	.95
Vitamin B1	q1	157	3439	1	–	
	q2	147	3652	0.98 (0.77–1.26)	.89	.96
	q3	128	3742	0.82 (0.64–1.06)	.14	.67
	q4	144	3755	0.95 (0.74–1.22)	.67	.95

(*Continued*)

TABLE 4. (Continued)

Variable	Quartile	Family history of CRC (n = 576)	No family history of CRC (n = 14,593)	Adjusted OR*	Adjusted p value*	BH p value†
Vitamin B2	q1	154	3440	1	–	
	q2	155	3544	1.10 (0.86–1.40)	.46	.95
	q3	124	3808	0.86 (0.66–1.11)	.24	.90
	q4	143	3796	1.00 (0.78–1.28)	1.00	1.00
Vitamin B3	q1	147	3531	1	–	
	q2	159	3662	1.03 (0.81–1.32)	.79	.96
	q3	127	3667	0.89 (0.69–1.15)	.36	.95
	q4	143	3728	0.96 (0.75–1.24)	.78	.96
Vitamin B6	q1	146	3635	1	–	
	q2	142	3717	0.95 (0.73–1.22)	.67	.95
	q3	139	3622	0.92 (0.72–1.19)	.54	.95
	q4	149	3614	0.98 (0.76–1.25)	.85	.96
Vitamin B12	q1	143	3551	1	–	
	q2	149	3761	1.07 (0.83–1.38)	.59	.95
	q3	146	3547	1.11 (0.86–1.43)	.42	.95
	q4	138	3729	1.03 (0.80–1.33)	.84	.96
Vitamin D	q1	140	3586	1	–	
	q2	150	3688	1.03 (0.80–1.32)	.84	.96
	q3	122	3773	0.92 (0.70–1.19)	.52	.95
	q4	164	3541	1.19 (0.93–1.51)	.17	.77
Vitamin E	q1	140	3492	1	–	
	q2	156	3661	1.27 (0.98–1.63)	.07	.51
	q3	144	3724	1.11 (0.86–1.44)	.42	.95
	q4	136	3711	1.10 (0.85–1.43)	.45	.95
Fast food	q1	167	3170	1	–	
	q2	165	4039	1.16 (0.91–1.48)	.23	.88
	q3	112	3445	1.09 (0.83–1.44)	.51	.95
	q4	132	3934	1.24 (0.95–1.62)	.11	.64
Red meat	q1	170	4016	1	–	
	q2	137	3349	1.03 (0.80–1.31)	.83	.96
	q3	146	3927	0.89 (0.70–1.13)	.34	.95
	q4	123	3296	0.99 (0.76–1.27)	.91	.96
Sugar	q1	168	3545	1	–	
	q2	152	3912	0.89 (0.70–1.12)	.32	.95
	q3	139	3831	0.77 (0.60–0.98)	.04	.40
	q4	117	3300	0.78 (0.60–1.01)	.06	.50
Sodium	q1	153	3384	1	–	
	q2	152	3653	1.06 (0.83–1.35)	.66	.95
	q3	138	3775	0.96 (0.75–1.24)	.78	.96
	q4	133	3776	1.03 (0.80–1.33)	.84	.96
Sodium from salt	q1	157	3336	1	–	
	q2	142	3666	0.94 (0.73–1.20)	.62	.95
	q3	147	3797	0.98 (0.76–1.26)	.86	.96
	q4	130	3789	0.95 (0.74–1.23)	.69	.95
Potassium	q1	147	3635	1	–	
	q2	145	3636	1.03 (0.80–1.33)	.80	.96
	q3	139	3694	0.94 (0.73–1.21)	.62	.95
	q4	145	3623	0.95 (0.73–1.22)	.67	.95

BH p value = Benjamini-Hochberg corrected for multiple comparisons; CI = confidence interval; CRC = colorectal cancer; OR = odds ratio; q = quartiles in the nutrient distribution.

*Adjusted for age, gender, obesity and alcohol consumption.

†Benjamini-Hochberg corrected for multiple comparisons.