

Increase in Incidence of Colorectal Cancer Among Young Men and Women in the United States

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Abstract

The recent, accelerated decline in colorectal cancer incidence rates has largely been attributed to an increase in screening rates among adults 50 years and older. We used data from 13 Surveillance, Epidemiology, and End Results cancer registries to report on colorectal cancer incidence trends from 1992 through 2005 among adults under age 50 years, for whom screening is not recommended for persons at average risk, by sex, race/ethnicity, age, stage at diagnosis, and anatomic subsite. Overall, incidence rates of colorectal cancer per 100,000 young individuals (ages 20-49 years) increased 1.5% per year in men and 1.6% per year in women from 1992 to 2005. Among non-Hispanic Whites, rates increased for

both men and women in each 10-year age grouping (20-29, 30-39, and 40-49 years) and for every stage of diagnosis. The increase in incidence among non-Hispanic Whites was predominantly driven by rectal cancer, for which there was an average increase of 3.5% per year in men and 2.9% per year in women over the 13-year study interval. In contrast to the overall decreasing trend in colorectal cancer incidence in the United States, rates are increasing among men and women under age 50 years. Further studies are necessary to elucidate causes for this trend and identify potential prevention and early detection strategies. (Cancer Epidemiol Biomarkers Prev 2009;18(6):1695-8)

Introduction

Overall incidence rates for colorectal cancer (CRC) in the United States have been generally declining since the mid-1980s (1, 2). In the most recent time period, the rate of decline has accelerated; since 1998, CRC incidence rates have decreased 2.8% per year in men and 2.2% per year in women (1). These rapid decreases have been largely attributed to an increase in CRC screening, particularly colonoscopy, among individuals ages 50 years and older (3, 4). Screening for CRC can reduce incidence by preventing cancer occurrence through the detection and removal of precancerous polyps (5, 6). Recent incidence trends among adults younger than 50 years, for whom CRC screening is not recommended for those at average risk, have not been analyzed, though a previous study limited to ages 20 to 39 years found an increase in incidence from 1973 to 1999 for all races combined (7). We report on trends in CRC incidence rates between 1992 and 2005 among young adults (ages 20 to 49 years) by sex, race/ethnicity, age, stage at diagnosis, and anatomic subsite.

Materials and Methods

We obtained invasive CRC cases diagnosed from 1992 through 2005 from the 13 oldest Surveillance, Epidemiology,

and End Results (SEER) registries, which provide population-based incidence data for the 5 major racial/ethnic populations (8). The states, metropolitan areas, and other registries that comprise the SEER 13 database, which covers ~14% of the U.S. population, are Atlanta, Connecticut, Detroit, rural Georgia, Hawaii, Iowa, Los Angeles, New Mexico, San Francisco-Oakland, San Jose-Monterey, Seattle-Puget Sound, Utah, and the Alaska Native Tumor Registry. We calculated annual, age-adjusted incidence rates (using the 2000 U.S. standard population) of CRC per 100,000 individuals ages 20 to 49 y by sex and race/ethnicity using SEER*Stat software version 6.4.4 (8, 9). We then examined the annual percent change (APC) in rates from 1992 to 2005 using the Joinpoint Regression Program, which fits a series of joined straight lines on a logarithmic scale to the trends in annual age-standardized rates (10). For illustrative purposes, we also plotted age-adjusted CRC incidence rates averaged over four time intervals (1992-1995, 1996-1998, 1999-2001, and 2002-2005) during the study period. Incidence rates for American Indians and Alaska Natives are not presented in this report due to sparse data.

The size of the incidence data set for non-Hispanic Whites was large enough to allow further analyses using the same analytic methods described above by 10-year age group (20-29, 30-39, and 40-49), stage at diagnosis, and anatomic subsite. Stage at diagnosis was coded according to SEER Summary Stage guidelines as local, regional, distant, and unstaged (8, 11). The anatomic site of each tumor was subdivided into three groupings according to the *International Classification of Diseases for Oncology*, 3rd edition (ICD-03): proximal colon (C18.0, C18.2-C18.5), distal colon (C18.6-C18.7), and rectum

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Table 1. CRC incidence trends among young adults (20 to 49 y) by sex and race/ethnicity, 1992 to 2005

Race/ethnicity		n	Line segment 1	
			Year	APC*
All races combined	Men	10,913	1992-2005	1.5 [†]
	Women	9,733	1992-2005	1.6 [†]
Non-Hispanic White	Men	6,748	1992-2005	2.0 [†]
	Women	5,626	1992-2005	2.2 [†]
Non-Hispanic Black	Men	1,409	1992-2005	-0.2
	Women	1,456	1992-2005	-0.6
Hispanic	Men	1,307	1992-2005	2.7 [†]
	Women	1,250	1992-2005	1.1
Asian American/Pacific Islander	Men	1,284	1992-2005	1.2
	Women	1,239	1992-2005	0.6

NOTE: Trends were analyzed by Joinpoint Regression Program, Version 3.0, with a maximum of three joinpoints (i.e., four line segments).

*APC based on incidence rates age-adjusted to the 2000 US standard population.

[†]The APC is significantly different from zero ($P < 0.05$).

(C19.9, C20.9; ref. 12). For comparison purposes, we also analyzed the annual percent change in CRC incidence rates among non-Hispanic Whites ages 50 y and older by stage and anatomic subsite.

Results

Overall incidence rates of CRC per 100,000 young adults (ages 20-49 y) increased 1.5% per year in men and 1.6% per year in women from 1992 to 2005. Specifically, incidence rates increased significantly among young non-Hispanic Whites, by 2.0% per year in men and 2.2% per year in women, and among Hispanic men, by 2.7% per year (Table 1; Supplementary Figure).

Among non-Hispanic Whites, incidence rates increased within each 10-year age grouping (20-29, 30-39, and 40-49) and for each stage of diagnosis in both men and women, though the increase in women for regional stage disease was not statistically significant (Table 2; Fig. 1). Notably, the largest annual percent increase in CRC inci-

dence was in the youngest age group (20-29 years), by 5.2% per year in men and 5.6% per year in women. Analysis by anatomic subsite showed significant increases in cancers of the distal colon and rectum in both men and women. On average, rectal cancer incidence rates increased 3.5% per year in men and 2.9% per year in women over the 13-year study interval. Although the incidence of rectal cancer seems to have leveled off in women since 1999 to 2001, rates in men continued to increase through 2002 to 2005 (Fig. 1). In marked contrast, among non-Hispanic White men and women ages 50 years and older, CRC incidence rates decreased by a minimum of 1.8% annually for every stage of diagnosis and a minimum of 2.7% annually for each anatomic subsite in the most recent time period (Supplementary Table).

Discussion

Our study found that in sharp contrast to the overall declining rates of CRC in the United States, incidence rates

Table 2. CRC incidence trends among young (20-49 y) non-Hispanic Whites by sex, age, stage at diagnosis, and anatomic subsite, 1992 to 2005

			n	Line segment 1		Line segment 2	
				Year	APC*	Year	APC*
Age	Men	20-29	249	1992-2005	5.2 [†]		
		30-39	1,419	1992-2005	3.0 [†]		
		40-49	5,080	1992-2005	1.5 [†]		
	Women	20-29	240	1992-2005	5.6 [†]		
		30-39	1,125	1992-2005	2.0 [†]		
		40-49	4,261	1992-2005	2.1 [†]		
Stage	Men	Local	2,345	1992-2005	2.5 [†]		
		Regional	2,626	1992-2005	2.0 [†]		
		Distant	1,554	1992-2005	1.8 [†]		
	Women	Unstaged	223	1992-2005	-3.1		
		Local	2,091	1992-2005	3.5 [†]		
		Regional	2,139	1992-1995	-4.6	1995-2005	1.7
Subsite	Men	Distant	1,266	1992-2005	3.7 [†]		
		Unstaged	130	1992-2005	-4.0		
		Proximal colon	2,054	1992-2005	0.0		
	Women	Distal colon	1,609	1992-2005	1.5 [†]		
		Rectum	2,609	1992-2005	3.5 [†]		
		Proximal colon	1,548	1992-2005	0.8		
		Distal colon	1,619	1992-2005	2.3 [†]		
		Rectum	2,065	1992-2005	2.9 [†]		

NOTE: Trends were analyzed by Joinpoint Regression Program, Version 3.0, with a maximum of three joinpoints (i.e., four line segments).

*APC based on incidence rates age-adjusted to the 2000 US standard population.

[†]The APC is significantly different from zero ($P < 0.05$).

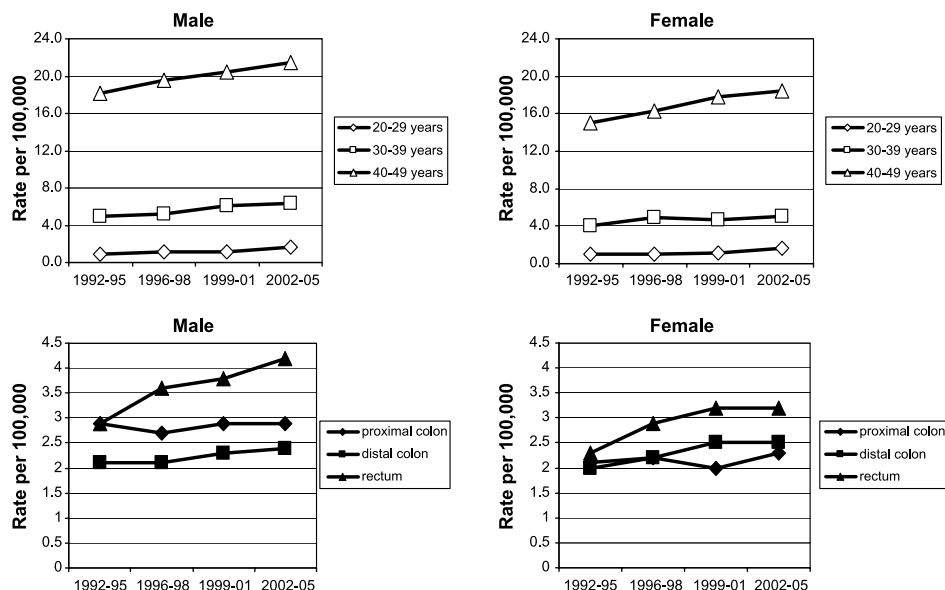


Figure 1. CRC incidence trends among young non-Hispanic White adults (20-49 y) by age and anatomic subsite, 1992 to 2005.

among adults younger than age 50 years are increasing due to an increase in left-sided tumors, particularly in the rectum. These findings are generally consistent with two previous studies that analyzed CRC trends using SEER databases (3, 7). O'Connell et al. (7) reported an increase in incidence rates in ages 20 to 39 years for both colon and rectal cancers during 1973 to 1999; however, this analysis was limited by the exclusion of 40 to 49 year-olds, who represent 73% of CRC patients under age 50 years, and the inability to examine trends by race/ethnicity and to include the most recent 6 years of data, during which the accelerated decline in overall CRC incidence rates occurred. Cress et al. (3) documented an increase in incidence rates in ages 0 to 49 years in rectal cancer, but not colon cancer, in all races combined during 1992 to 2001.

Obesity is a major risk factor for CRC in men and, to a lesser extent, for colon cancer in women (13). However, there is accumulating evidence that obesity confers a stronger risk of CRC in premenopausal, compared with postmenopausal, women (14-16). In the past three decades, the prevalence of obesity has increased markedly among individuals of all ages and racial/ethnic groups in the United States (17-19), which may have contributed to the overall increase in CRC incidence rates among young adults. However, CRC incidence rates among non-Hispanic Whites substantially increased for left-sided tumors (distal and rectal) but not for right-sided tumors (proximal). It is unknown whether the mechanism through which adiposity induces tumor development and the latency period from exposure to disease occurrence differs by anatomic subsite. In tandem with obesity trends, type 2 diabetes, also an established risk factor for CRC (20), has increased dramatically in the United States (21, 22), and may have likewise contributed to the observed increase in CRC incidence in young adults.

Consumption of red and processed meat has been shown to increase risk of cancers of the distal colon and rectum (23), whereas milk and calcium consumption have shown a protective effect against these subsites (24). Between the late 1970s and the mid-1990s, fast-food consumption in the United States increased 5-fold among

children (ages 2 to 17 y) and 3-fold among adults (ages 18 years and older) (25). A diet high in fast food is associated with both greater meat consumption (26) and reduced milk consumption (27). The average energy intake from hamburgers/cheeseburgers increased 30% from 1977-78 to 1994-96 (28); concurrently, the proportion of energy intake from milk decreased 42% among both adolescents (12-18 years) and young adults (19-29 years) (29). It is plausible that the emergence of unfavorable dietary patterns in children and young adults over the past three decades may have contributed to the increase in CRC among young adults observed in our study.

Other behavioral factors associated with an increased risk of CRC are alcohol intake (30) and smoking (31, 32). It is unlikely that trends in alcohol use explain the recent increase in CRC among young adults because there has been a decline in alcohol consumption in the United States since 1981, both overall and among high school students (33-35). Despite transient increases in smoking prevalence within some birth cohorts since 1964, tobacco exposure is unlikely to have played a role in the recent increase in CRC incidence in young adults because of the requisite length (minimum 30 years) of the induction period (32, 36).

The outcome of CRC treatment depends strongly on stage at diagnosis. Clinical practice guidelines suggest that patients with inflammatory bowel disease, polyposis syndromes, a known genetic predisposition, or a personal or family history of adenomatous polyps or CRC begin screening before age 50 years. Early recognition of CRC in patients under age 50 without these risk factors requires clinical awareness and aggressive pursuit of symptoms. A study of initial presentation of young onset CRC patients without established risk factors found that 86% were symptomatic at the time of diagnosis, with the most common symptoms of rectal bleeding (51%), abdominal pain (32%), and change in bowel habits (18%). The most common factors leading to diagnosis in asymptomatic patients were anemia (14%) and positive fecal occult blood test (7%) (37). Our findings of a recent increase in CRC among those under age 50 years suggests the importance of timely evaluation of the distal colorectum, at a minimum, in young

adults who present with symptoms consistent with possible underlying cancer.

The increasing incidence of CRC in young adults is in contrast with the rapidly declining incidence among older individuals. The disparate increase in left-sided CRC suggests that particular attention be given to studies to elucidate the behavioral and environmental risk factors responsible for this trend and potential prevention and early detection strategies.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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References

- Ries L, Melbert D, Krapcho M, et al., editors. SEER Cancer Statistics Review 1975-2005. Bethesda (MD): National Cancer Institute; 2008.
- Chu KC, Tarone RE, Chow WH, Hankey BF, Ries LAG. Temporal patterns in colorectal cancer incidence, survival, and mortality from 1950 through 1990. *J Natl Cancer Inst* 1994;86:997-1006.
- Cress RD, Morris C, Ellison GL, Goodman MT. Secular changes in colorectal cancer incidence by subsite, stage at diagnosis, and race/ethnicity, 1992-2001. *Cancer* 2006;107 Suppl 5:1142-52.
- Phillips KA, Liang SY, Ladabaum U, et al. Trends in colonoscopy for colorectal cancer screening. *Med Care* 2007;45:160-7.
- Mandel JS, Church TR, Bond JH, et al. The effect of fecal occult-blood screening on the incidence of colorectal cancer. *N Engl J Med* 2000;343:1603-7.
- Winawer SJ, Zauber AG, Ho MN, et al. Prevention of colorectal cancer by colonoscopic polypectomy. The National Polyp Study Workgroup. *N Engl J Med* 1993;329:1977-81.
- O'Connell JB, Maggard MA, Liu JH, et al. Rates of colon and rectal cancers are increasing in young adults. *Am Surg* 2003;69:866-72.
- SEER*Stat Database: Incidence - SEER 13 Regs Limited-Use, Nov 2007 Sub (1992-2005) - Linked To County Attributes - Total U.S., 1969-2005 Counties. In: Surveillance, Epidemiology, and End Results (SEER) Program, (<http://www.seer.cancer.gov>) National Cancer Institute DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2008, based on the November 2007 submission.
- Program SR. SEER*Stat software, (<http://www.seer.cancer.gov/seerstat>) In. 6.4.4 ed. Bethesda (MD): National Cancer Institute; 2008.
- Joinpoint Regression Program, Version 3.0. April 2005; Statistical Research and Applications Branch, National Cancer Institute.
- Young JL RS, Ries LAG, Fritz AG, Hurlbut AA, editors. SEER Summary Staging Manual - 2000: Codes and Coding Instructions. Bethesda (MD): National Cancer Institute, NIH Pub. No. 01-4969; 2001.
- Fritz A, Percy C, Jack A, et al., editors. International Classification of Diseases for Oncology. 3rd ed. World Health Organization; 2000.
- Larsson SC, Wolk A. Obesity and colon and rectal cancer risk: a meta-analysis of prospective studies. *Am J Clin Nutr* 2007;86:556-65.
- Terry PD, Miller AB, Rohan TE. Obesity and colorectal cancer risk in women. *Gut* 2002;51:191-4.
- Slattery ML, Ballard-Barbash R, Edwards S, Caan BJ, Potter JD. Body mass index and colon cancer: an evaluation of the modifying effects of estrogen (United States). *Cancer Causes Control* 2003;14:75-84.
- Reeves GK, Pirie K, Beral V, et al. Cancer incidence and mortality in relation to body mass index in the Million Women Study: cohort study. *BMJ* 2007;335:1134.
- Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics* 1998;101:497-504.
- Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes Relat Metab Disord* 1998;22:39-47.
- Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA* 2006;295:1549-55.
- Larsson SC, Orsini N, Wolk A. Diabetes mellitus and risk of colorectal cancer: a meta-analysis. *J Natl Cancer Inst* 2005;97:1679-87.
- Skyler JS, Oddo C. Diabetes trends in the USA. *Diabetes Metab Res Rev* 2002;18 Suppl 3:S21-6.
- Engelgau MM, Geiss LS, Saaddine JB, et al. The evolving diabetes burden in the United States. *Ann Intern Med* 2004;140:945-50.
- Larsson SC, Wolk A. Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies. *Int J Cancer* 2006;119:2657-64.
- Cho E, Smith-Warner SA, Spiegelman D, et al. Dairy foods, calcium, and colorectal cancer: a pooled analysis of 10 cohort studies. *J Natl Cancer Inst* 2004;96:1015-22.
- Guthrie JF, Lin BH, Frazao E. Role of food prepared away from home in the American diet, 1977-78 versus 1994-96: changes and consequences. *J Nutr Educ Behav* 2002;34:140-50.
- Pereira MA, Kartashov AI, Ebbeling CB, et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet* 2005;365:36-42.
- Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics* 2004;113:112-8.
- Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977-1998. *JAMA* 2003;289:450-3.
- Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in food locations and sources among adolescents and young adults. *Prev Med* 2002;35:107-13.
- Ferrari P, Jenab M, Norat T, et al. Lifetime and baseline alcohol intake and risk of colon and rectal cancers in the European prospective investigation into cancer and nutrition (EPIC). *Int J Cancer* 2007;121:2065-72.
- Paskett ED, Reeves KW, Rohan TE, et al. Association between cigarette smoking and colorectal cancer in the Women's Health Initiative. *J Natl Cancer Inst* 2007;99:1729-35.
- Botteri E, Iodice S, Bagnardi V, et al. Smoking and colorectal cancer: a meta-analysis. *JAMA* 2008;300:2765-78.
- Caetano R, Clark CL. Trends in alcohol consumption patterns among whites, blacks and Hispanics: 1984 and 1995. *J Stud Alcohol* 1998;59:659-68.
- Zhang Y, Guo X, Saitz R, et al. Secular trends in alcohol consumption over 50 years: the Framingham Study. *Am J Med* 2008;121:695-701.
- Trends in the Prevalence of Alcohol Use. In: Survey YRB, editor. National YRBS: 1991-2007: Centers for Disease Control; 2008.
- Giovannucci E. An updated review of the epidemiological evidence that cigarette smoking increases risk of colorectal cancer. *Cancer Epidemiol Biomarkers Prev* 2001;10:725-31.
- Dozois EJ, Boardman LA, Suwanthanna W, et al. Young-onset colorectal cancer in patients with no known genetic predisposition: can we increase early recognition and improve outcome? *Medicine (Baltimore)* 2008;87:259-63.