Emerging and Widening Colorectal Carcinoma Disparities Between Blacks and Whites in the United States (1975-2002)

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Abstract

Background: **Colorectal carcinoma (CRC) is the fourth most common cancer diagnosed and the second most common cause of cancer death in the U.S. Incidence and mortality rates have decreased since the mid-1980s, although more among Whites than Blacks.**
Methods: **To determine if these racial differences were changing over time, we examined CRC rates in the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program (1975-2002). Rates were stratified by gender, race, anatomic subsite, historic stage, and grade.**
Results: **CRC rates were higher among men than women and higher among Blacks than Whites, with Black men having the highest rates during the latter years. Prior to the mid-1980s, male CRC rates were actually higher among Whites than Blacks; after which there was ethnic crossover with Black rates higher than White rates, and the gaps are widening. Proximal and transverse CRCs were more common and rectal cancers were less common among Blacks than Whites. Over time, rates for localized and regional stages increased among Blacks and decreased among Whites. Rates for distant stages declined for both racial groups, although less among Blacks. Black-to-White rate ratio for distant stage was ~1.30. Notably, Blacks compared with Whites had lower grade tumors, despite higher stages and mortality rates.**
Conclusions: **CRC racial disparities have emerged and widened for three decades. These temporal trends probably reflect complicated racial differences between screening practice patterns and etiologic factors.** (Cancer Epidemiol Biomarkers Prev 2006;15(4):792–7)

Introduction

Colorectal carcinoma (CRC) is the fourth most common cancer diagnosed and the second most common cause of cancer death in the U.S., accounting for an estimated 145,290 new cancer cases and 56,290 deaths in the year 2005 (1). Similar rankings are reported in most western countries (2). In the U.S., overall CRC mortality rates have decreased since the 1970s and incidence rates have declined since the mid-1980s, probably due to earlier detection and intervention over time (3-5).

However, these encouraging overall national trends overshadow gender and racial disparities (3, 4, 6-12), possibly reflecting a complex mixture of screening and/or etiologic factors. For example, women compared to men have lower CRC incidence rates (13), perhaps due to decreased awareness and screening for CRC (14, 15), although protective factors such as hormone replacement therapy cannot be excluded (16). On the other hand, Blacks compared with Whites have higher incidence rates (13) despite less CRC screening (14) and less aggressive tumor characteristics (10, 12). To further examine these somewhat paradoxical epidemiologic patterns, we studied CRC incidence by gender, race, patient age-at-diagnosis, anatomic subsite, stage, and grade for CRC cases in the Surveillance, Epidemiology, and End Results (SEER) database (1975-2002).

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Materials and Methods

Materials. We used the National Cancer Institute's SEER Cancer Incidence Public-Use Database (November 2004 submission; ref. 17). Although the SEER program began in 1973, 1975 marks the earliest date that all nine original tumor registries were contributing, and represents the start of the most complete data on Blacks. For this analysis, there were n = 323,888 White and Black cases of CRC diagnosed during the years 1975 to 2002 from SEER's original registries, including Atlanta (Metropolitan), Connecticut, Detroit (Metropolitan), Hawaii, Iowa, New Mexico, San Francisco-Oakland Standard Metropolitan Statistical Area, Seattle (Puget Sound), and Utah.

Statistical Methods. Age-adjusted incidence rates (2000 U.S. standard population) were calculated using SEER*Stat 6.1.4 and expressed per 100,000 person-years (or persons per year). Mortality data according to the underlying cause of death were supplied by the National Center for Health Statistics (http://www.cdc.gov/nchs/). Gender and racial rate ratios (RR) were calculated to express relative risks. Differences in rates and RRs were tested for significance at the 95% confidence level, as previously described (18, 19). Standard errors (SE) are included in Table 1 for rates. Most of the RRs in Table 1 were statistically significant at the 95% confidence level; all nonsignificant RRs are in shown in boldface.

Incidence and mortality trends were plotted on a log-linear scale by seven 4-year time periods (1975-1978, 1979-1982, 1983-1986, 1987-1990, 1991-1994, 1995-1998, and 1999-2002), as previously described (20). SEER's Joinpoint regression program was used to identify changes in secular trend (21). In brief, Joinpoint is a public-use statistical software for the analyses of trends to determine whether apparent changes in trend data are statistically significant. The software takes the annual rate data and fits the simplest Joinpoint (knots or nodes) that the data will allow. The user can choose the number of Joinpoints as well as the significance

Table 1. Colorectal cancer incidence by race, gender, age group, anatomic subsite, stage, grade, and histology for 9 SEER areas (1975-2002)

Variable													Male/Female		Black/White	
Total	White males			White females			Black males			Black females			White	Black	Male	Female
	Number	Rate	SE	Number	Rate	SE	Number	Rate	SE	Number	Rate	SE	RR	RR	RR	RR
	147,732	70.80	0.20	148,810	51.20	0.10	12,971	72.70	0.70	14,368	56.60	0.50	1.38	1.28	1.03	1.11
Age at diagnosis (y)																
<20	50	0.06	0.01	57	0.08	0.01	12	0.10	0.03	5	0.04	0.02	0.83	2.38	1.51	0.53
20-24	126	0.61	0.05	109	0.54	0.05	17	0.55	0.13	20	0.61	0.14	1.12	0.91	0.91	1.12
25-29	314	1.41	0.08	264	1.23	0.08	57	1.85	0.25	73	2.10	0.25	1.15	0.88	1.31	1.70
30-34	733	3.28	0.12	627	2.88	0.12	107	3.64	0.35	139	4.15	0.35	1.14	0.88	1.11	1.44
35-39	1,351	6.53	0.18	1,184	5.82	0.17	243	9.47	0.61	269	9.15	0.56	1.12	1.04	1.45	1.57
40-44	2,404	13.03	0.27	2,200	11.92	0.25	410	18.84	0.93	411	16.42	0.81	1.09	1.15	1.45	1.38
45-49	4,374	27.18	0.41	3,871	23.88	0.38	653	37.00	1.45	669	32.78	1.27	1.14	1.13	1.36	1.37
50-54	7,916	55.74	0.63	6,422	44.05	0.55	1,048	72.85	2.25	1,043	62.12	1.92	1.27	1.17	1.31	1.41
55-59	12,667	102.91	0.91	9,688	74.82	0.76	1,394	120.15	3.22	1,367	99.52	2.69	1.38	1.21	1.17	1.33
60-64	18,132	171.35	1.27	13,674	117.98	1.01	1,838	194.46	4.54	1,663	142.59	3.50	1.45	1.36	1.13	1.21
65-69	23,176	260.74	1.71	18,461	173.85	1.28	1,967	252.48	5.69	2,048	201.25	4.45	1.50	1.25	**0.97**	1.16
70-74	25,081	356.86	2.25	22,627	243.64	1.62	1,907	348.62	7.98	2,077	263.12	5.77	1.46	1.32	**0.98**	1.08
75-79	22,929	456.81	3.02	24,736	323.71	2.06	1,649	447.40	11.02	1,935	327.96	7.46	1.41	1.36	**0.98**	1.01
80+	28,486	573.35	3.43	44,890	427.40	2.02	1,669	525.25	12.97	2,649	398.53	7.74	1.34	1.32	0.92	0.93
Anatomic site																
Proximal	35,347	17.52	0.10	45,163	15.35	0.07	3,533	20.49	0.37	4,491	17.94	0.27	1.14	1.14	1.17	1.17
Transverse	18,002	8.84	0.07	20,234	6.91	0.05	1,946	11.18	0.27	2,167	8.53	0.19	1.28	1.31	1.26	1.24
Distal	43,048	20.32	0.10	40,045	13.91	0.07	3,574	19.93	0.36	3,977	15.53	0.25	1.46	1.28	**0.98**	1.12
Rectum	46,504	21.62	0.10	37,304	12.99	0.07	3,365	17.68	0.33	3,095	11.93	0.22	1.66	1.48	0.82	0.92
Other/unknown	4,838	2.50	0.04	6,064	2.02	0.03	553	3.40	0.16	638	2.65	0.11	1.24	1.28	1.36	1.31
Historic stage																
Localized	57,083	27.20	0.12	54,575	18.78	0.08	4,383	24.57	0.40	4,803	18.91	0.28	1.45	1.30	0.90	**1.01**
Regional	53,213	25.28	0.11	55,870	19.28	0.08	4,434	24.13	0.39	5,041	19.61	0.28	1.31	1.23	0.95	**1.02**
Distant	28,051	13.27	0.08	27,334	9.46	0.06	3,144	17.50	0.34	3,332	12.95	0.23	1.40	1.35	1.32	1.37
Other/unknown	9,392	5.05	0.05	11,031	3.65	0.04	1,010	6.47	0.22	1,192	5.11	0.15	1.38	1.27	1.28	1.40
Grade																
Low	91,279	43.64	0.15	86,906	29.97	0.10	7,957	44.52	0.54	8,826	34.69	0.38	1.46	1.28	**1.02**	1.16
High	22,026	10.46	0.07	25,878	8.90	0.06	1,551	8.37	0.23	1,760	6.79	0.16	1.18	1.23	0.80	0.76
Other/unknown	34,434	16.70	0.09	36,026	12.30	0.07	3,463	19.79	0.36	3,782	15.11	0.25	1.36	1.31	1.19	1.23
Histology																
Nonmucinous	133,710	64.11	0.18	132,755	45.65	0.13	11,634	65.38	0.65	12,864	50.73	0.45	1.40	1.29	**1.02**	1.11
Mucinous	13,793	6.58	0.06	15,901	5.46	0.04	1,312	7.20	0.22	1,473	5.74	0.15	1.21	1.25	1.09	**1.05**
Other/unknown	236	0.10	0.01	154	0.06	0.00	25	0.10	0.02	736	0.11	0.02	1.87	(0.87)	0.96	2.07

NOTE: Rate, incidence rate per 100,000 person-years were age-adjusted to the 2000 U.S. standard population; SE, standard error; RR, rate ratio (most RRs were statistically significant at the 95% confidence level; nonsignificant RRs shown in boldface); nonmucinous and mucinous adenocarcinoma.

level. Age-specific incidence rates by 5-year age increments were plotted on a log-log scale (22, 23).

SEER's historical staging system defined localized disease as limited to the colorectum, regional disease as limited to nearby lymph nodes or other organs, and distant disease as systemic metastases. Anatomic subsites and histopathologic subtypes were defined using the third edition of the International Classification of Diseases for Oncology (24). Anatomic subsites were the proximal colon, which included the cecum, appendix, and ascending colon. The transverse colon included the hepatic flexure, transverse colon, and splenic flexure. The distal colon included the descending colon and sigmoid colon. The rectum included the rectosigmoid junction and rectum. Grade 1 (well differentiated) and grade 2 (moderately differentiated) were combined into a single ''low'' grade category. ''High'' grade was defined as grade 3 (poorly differentiated) and grade 4 (undifferentiated). Histologic subtypes were classified as mucinous adenocarcinoma (International Classification of Diseases for Oncology-3, 8480-8481), including signet ring tumors (International Classification of Diseases for Oncology-3, 8490), and nonmucinous containing all other adenocarcinomas (10, 24, 25).

Results

There were 160,703 White and Black male cases and n = 163,178 White and Black female cases with CRC in SEER's 9 Registry Database (Table 1). Age-adjusted incidence rates ranged from a low of 51.2 per 100,000 person-years among White women to a high of 72.7 per 100,000 person-years among Black men. CRC incidence rates were higher among males than females in all instances except among Blacks between the ages 20 to 24, 25 to 29, and 30 to 34 years. Notably, male-to-female RRs increased from proximal to rectal subsites among Whites (RRs increased from 1.14 to 1.66) as well as among Blacks (RRs increased from 1.14 to 1.48). The most predominant histologic type was nonmucinous adenocarcinoma, comprising ~90% of all CRCs for all gender and racial groups.

Blacks compared with Whites were more likely to have younger ages at diagnosis, proximal or transverse CRCs, distant SEER historic stage, and lower tumor grade. For example, Black-to-White RRs peaked at ages 35 to 39 years among males and females (RR, 1.45 and 1.57), respectively. Black-to-White RRs were 1.17 for proximal and about 1.25 for transverse CRCs among both men and women. Distant stage was >30% more likely among Blacks than Whites (RR, 1.32 and 1.37) among males and females, respectively.

Temporal Trends for All CRC Cases. Using SEER's Joinpoint regression program and Cancer Statistics Review (13, 21), 1985 was a pivotal year or change point for CRC with overall incidence rates beginning to decrease. For example, incidence rates increased 6%, from 61.9 to 65.5 per 100,000 person-years during the time periods 1975 to 1978 and 1983 to 1986 (Fig. 1A); whereas rates fell 18% from

65.5 to 53.8 per 100,000 person-years during the time periods 1983 to 1986 and 1999 to 2002. Overall mortality rates fell during the entire study period (Fig. 1A).

Prior to the mid-1980s, total CRC incidence rates were highest among White males (Fig. 2A). Rates then began to decline among White men but not among Black men. Around the year 1990, incidence rates crossed among Black and White men, after which, rates were higher among Blacks than Whites. Incidence trends among women were similar to men, although the ethnic crossover occurred much earlier for women than men.

From 1975 to 2002, CRC mortality rates decreased among Whites but increased then stabilized among Blacks (Fig. 2A). Prior to the late 1970s, mortality rates were actually lower among Black men than Whites, after which there was crossover. Mortality rates were never lower among Black women compared with White women.

Temporal Trends by Anatomic Subsite. Approximately equal numbers of CRCs developed in the proximal (27.3%), distal (28.0%), and rectal (27.9%) anatomic subsites, whereas only 13.1% developed in the transverse colon (Table 1). Incidence rates overall decreased for all anatomic subsites except for the proximal colon (Fig. 1B).

Proximal CRC rates were similar among Blacks and Whites during the 1970s, then increased among Blacks but were mostly stable among Whites (Fig. 2B). Transverse CRC rates were relatively stable among Blacks but fell among Whites (Fig. 2C). Distal CRC rates decreased among both Blacks and Whites, although less among Blacks than Whites (Fig. 2D). During the early years, rectal CRC rates were lower among Blacks than Whites but declined more rapidly among Whites than Blacks, narrowing racial differences (Fig. 2E).

Temporal Trends by SEER Historic Stage. Nearly three-fourth's of all CRC cases were classified as localized (37.3%) or regional (36.6%), whereas 19.1% were distant SEER stage (Table 1). Coinciding with the 1985 change point for CRC overall (Fig. 1A), rates for localized and regional stages increased and then crossed (Fig. 1C), as regional rates declined earlier and further than localized rates. Prior to the mid-1980s, overall rates for localized stages were lower than rates for regional stages after which rates were higher for localized than regional stage, suggestive of earlier detection over time (Fig. 1C).

Racial disparities worsened for all stages during the study period (Fig. 2F-H). For example, among all gender and racial groups, rates increased for localized and regional stages during the early years, and then declined among Whites but not among Blacks. Rates for distant stages decreased among Whites, declined modestly among Black women, and were basically stable among Black men.

Temporal Trends by Tumor Grade. Rates for tumor grade must be interpreted with caution due to the relatively large amounts of unknown data (Fig. 1D; Table 1), ranging from 23% for White males to 27% for Black males during the study period 1975 to 2002. Over time, the proportion with unknown grade declined for all cases (Fig. 1D), as well as for all gender and racial groups from ~40% during the earliest time period (1978-1978) to ~15% to 20% during the latest time interval (1999-2002). Rates for low-grade lesions increased rapidly prior to the mid-1980s, probably in part due to a shift from unknown to low grade, and then plateaued (Fig. 1D). High-grade tumors remained fairly steady, showing a slight increase in the latter years.

Among both Blacks and Whites, rates for low grade tumors increased rapidly prior to the mid-1980s, then plateaued among Blacks and decreased modestly among Whites (Fig. 2I). Rates for high-grade lesions decreased among Blacks during the early years then increased (Fig. 2J). Rates for high-grade tumors remained fairly constant among Whites over time, and have been consistently higher among Whites than Blacks.

Age Distribution at Diagnosis. Age-specific RRs among Blacks compared with Whites peaked at ages 35 to 44 years (Table 1). Notably, incidence rates increased steadily with age irrespective of race, gender, anatomic subsite, SEER historic stage, and tumor grade (Fig. 3).

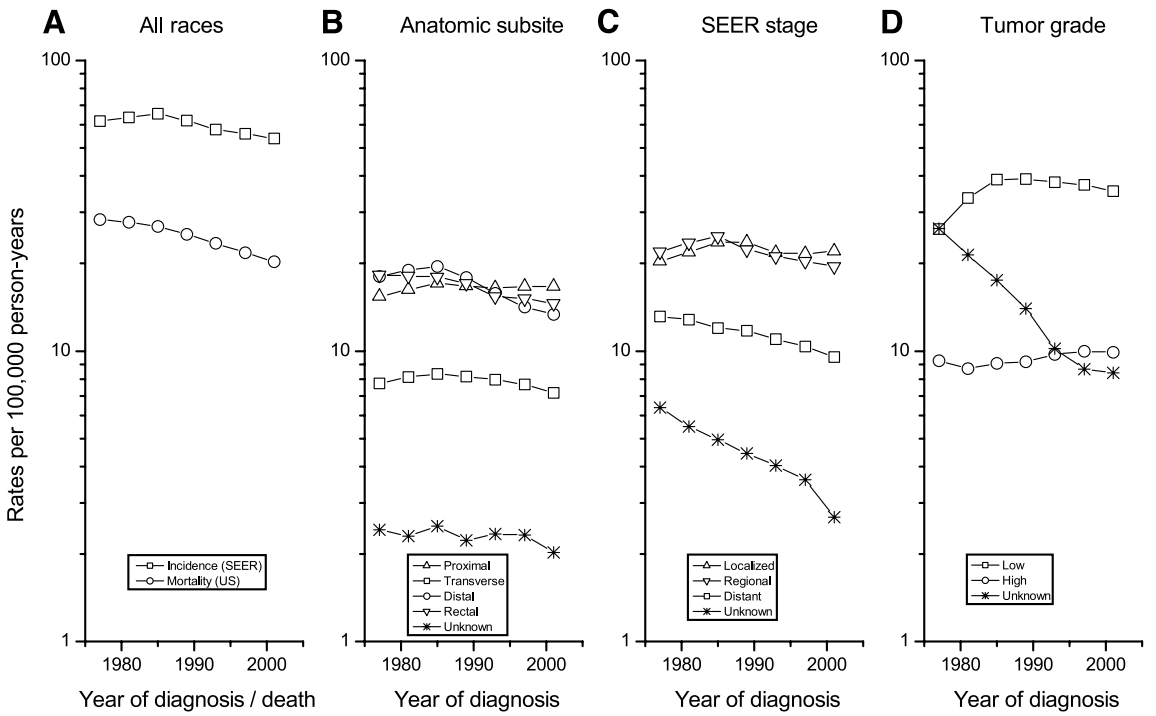

Figure 1. Age-adjusted colorectal cancer incidence and mortality rates per 100,000 person-years (2000 U.S. standard) for all races combined, and incidence by anatomic subsite, SEER historic stage, and tumor grade (1975-1978 to 1999-2002).

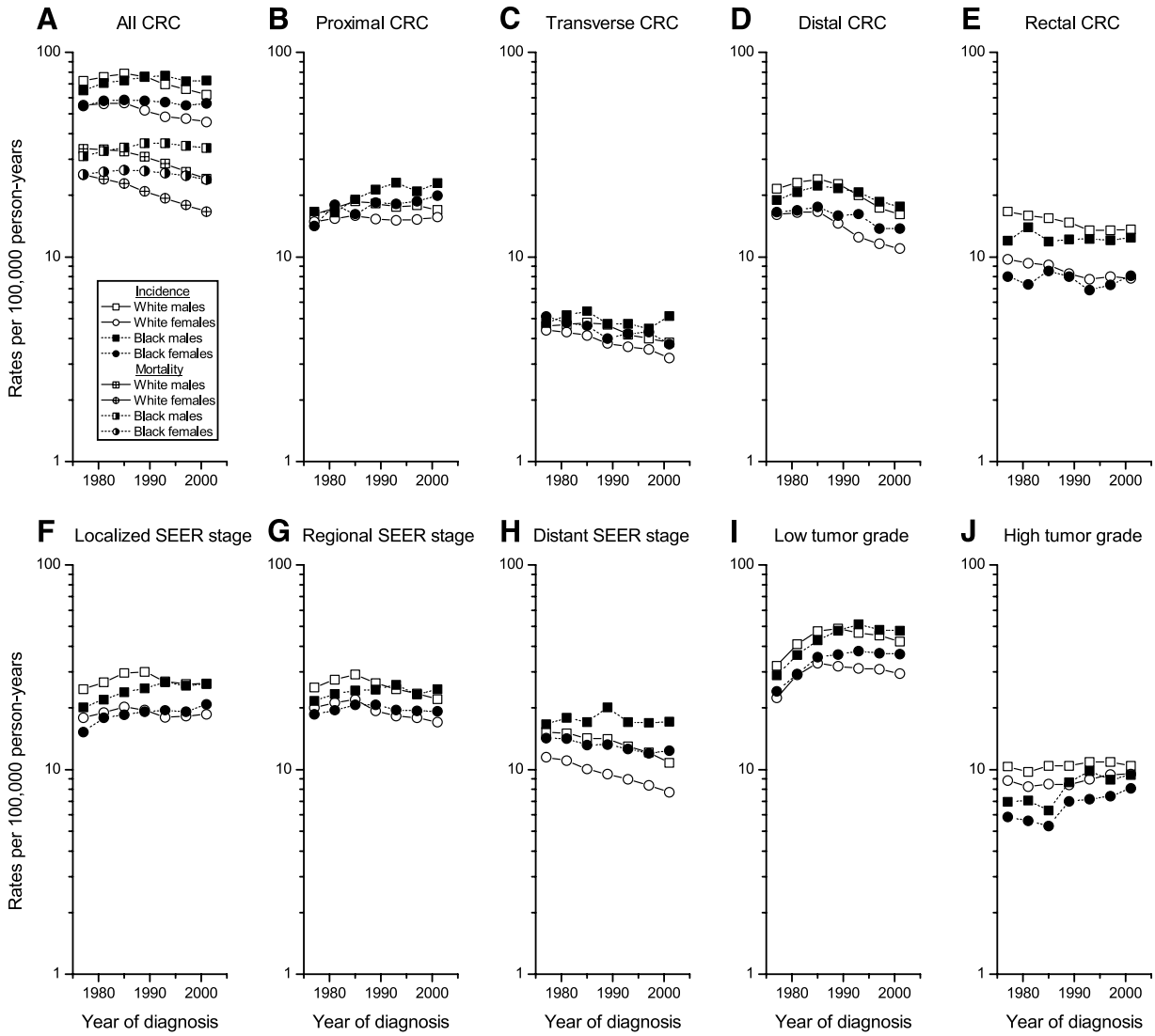

Figure 2. Age-adjusted colorectal cancer incidence and mortality rates per 100,000 person-years (2000 U.S. standard) for all cases by gender and race, anatomic subsite, SEER historic stage, and tumor grade (1975-1978 to 1999-2002).

Discussion

These results suggest emerging and widening CRC racial disparities. Moreover, men compared with women had higher CRC rates at all ages, with Black men having the highest rates of all. These rate patterns most probably reflected complicated interactions between *screening* and *etiologic* factors, as suggested by stage- and subsite-specific temporal trends. Notably, Black-to-White incidence and mortality trends were worse at the end than at the beginning of the study period during the years 1975 to 2002.

Screening Factors. The decline in CRC rates in the mid-1980s has been attributed to widespread CRC screening, due to increased public awareness following President Ronald Reagan's CRC diagnosis in July 1985 (26). After that time, there was a population-based crossover for localized and regional stages, consistent with improved detection, and intervention over time (3, 4). This stage-specific crossover occurred sooner for men than women and sooner for Whites than Blacks.

The delayed stage-specific shift among women compared with men was possibly due to gender-specific differential screening practices. Indeed, in the 2000 National Health Interview Survey (14), fewer women than men age >50 years had routine screening for colorectal cancer. Moreover, during the years 1987 to 2000, rates for colorectal endoscopy increased more rapidly among men than women then stabilized, whereas rates for colorectal endoscopy among women sustained a constant (although slower) increase (14). The greater initial surge in colorectal endoscopy screening among men might be attributed to a gender connection with Ronald Reagan. In contrast, when Katie Couric underwent her televised colonoscopy in the year 2000, colonoscopy rates increased mostly in women and younger individuals (27, 28). The continuous increase in colorectal endoscopy among women during the latter years may partly coincide with Ms. Couric's colorectal screening campaign. Admittedly, this explanation is speculative because there are many differences other than CRC screening factors between men and women, including differential hormonal exposures (16, 29-31).

The stage-specific shift among Blacks also occurred after the Ronald Reagan diagnosis. Additionally, rates for distant disease show a stable long-term trend among Blacks, suggesting that Blacks are still not being screened enough. Indeed, in the 2000 National Health Interview Survey (14), Black men and women were screened less than White men and women.

On the other hand, rates for proximal CRC seem to be rising among Blacks, and this trend cannot be attributed to decreased screening (7) because incidence rates would not increase with decreased screening and/or delayed detection. Additionally, if delayed detection were the sole determinant for CRC racial variations, Blacks compared with Whites should have older (not younger) ages at onset, as do women compared with men. Moreover, despite higher stages and increased mortality rates compared with Whites, Blacks tend to have less aggressive tumor characteristics even after adjusting for age, sex, geographic location, stage, socioeconomic status, body mass index, health care access, and utilization (10, 12). All in all, these many age-specific, subsite-specific, and pathologic-specific racial variations suggest that there may be some biological differences in carcinogenic risks and/or exposures among Blacks and Whites, which are not improving over time.

Etiologic Factors. Colorectal carcinogenesis is a prototypical long-term multistep process with each step corresponding to key genetic events (22, 23, 32, 33). Over a lifetime of accumulated carcinogenic exposures, genetic changes are mirrored by the pathologic evolution from the premalignant adenoma to invasive CRC. A conspicuous epidemiologic consequence of the adenoma-to-carcinoma sequence is a linear age-specific incidence rate curve on a log-log scale, as we observed in Fig. 3 for all anatomic sites, stages, and tumor grades among White males, White females, Black males, and Black females.

Similarly shaped age-specific rate curves for all gender and racial groups, subsites, stages, and grades imply similar carcinogenic processes for most CRCs. CRC disparities then may be more related to differential exposures (promotion) than to unique initiating genetic events. Indeed, migrant studies show a dominant etiologic role for promotional factors among three-fourth's of all CRCs (2, 15, 34-36). Western diet, physical inactivity, obesity, and central deposition of adiposity are consistent CRC risk factors (37, 38). Chronic hyperinsulinemia may be a common mechanistic pathway for many of these risk factors (37, 39-41).

For example, despite some variability among five studies (39, 42-45), summary risk estimates show a stronger association between type 2 diabetes mellitus and the proximal (odds ratio, 1.55; 1.39-1.64) than the distal colon (odds ratio, 1.25; 1.15-1.36; ref. 45). Given the consistently higher baseline prevalence for diabetes among Blacks than Whites, the link between diabetes and proximal CRC could possibly explain the higher Black-to-White RRs for proximal CRCs. In fact, the age-adjusted prevalence rates for diabetes were ~50% to 75% higher among Blacks compared with Whites during our study period (http://www.cdc.gov/diabetes/statistics/prev/national/menuraceethsexage.htm).

Subsite-specific etiologic heterogeneity also suggests that CRC may not be a single monolithic disease but rather a mixed carcinogenic process. Indeed, proximal and distal CRCs have different population-based characteristics (7-9), pathophysiologic features, and molecular signatures (46-49). These differences may reflect distinct biological characteristics acquired in

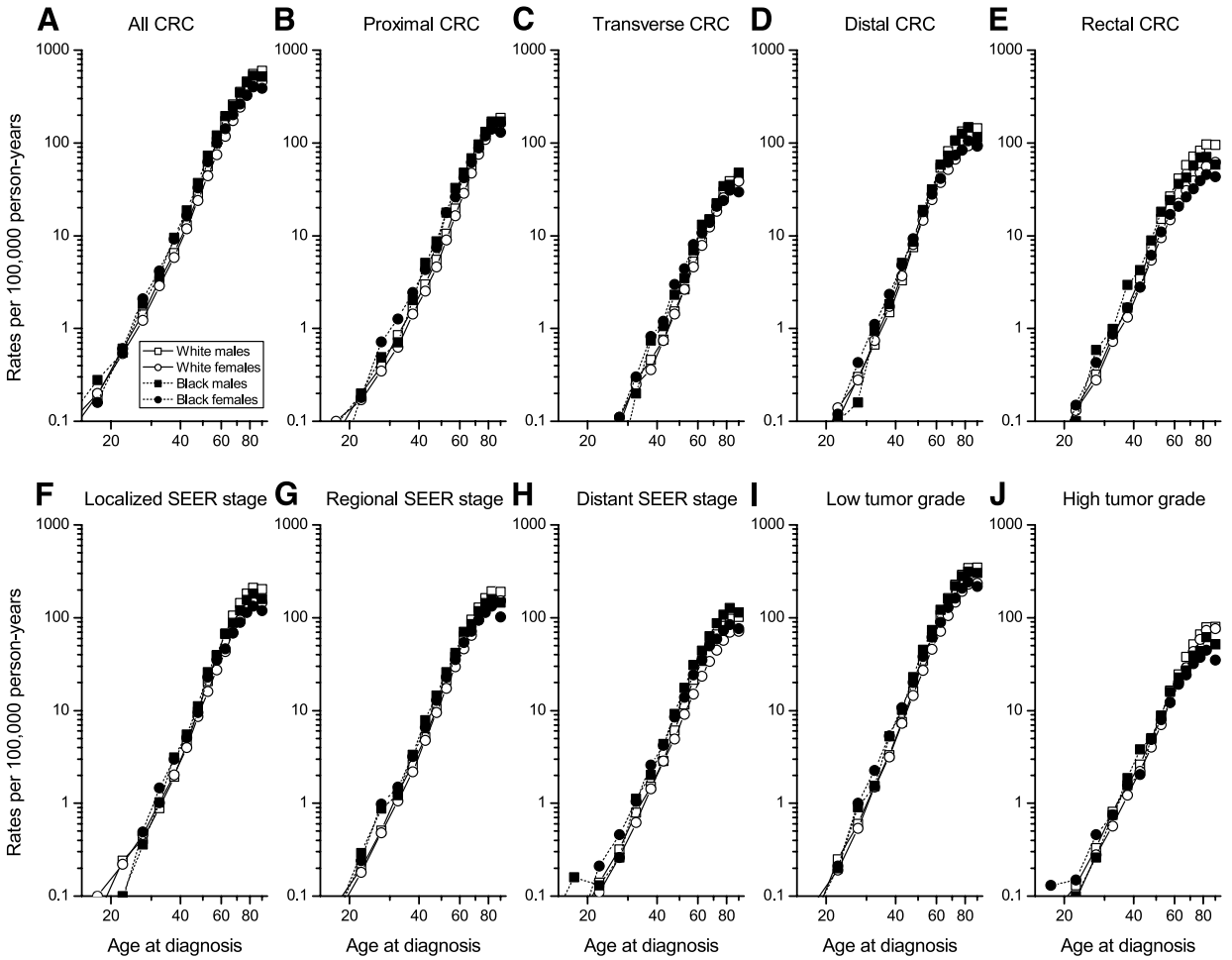

Figure 3. Age-specific colorectal cancer incidence rates by gender and race, anatomic subsite, SEER historic stage, and tumor grade (1975-2002).

embryonic or postnatal development (47, 48, 50) or differing exposure factors in different parts of the colorectum.

In sum, we observed emerging and widening stage- and subsite-specific CRC racial disparities, which cannot be entirely explained by screening practice patterns. Etiologic factors are also probably important. Clearly, additional research is required to better delineate the determinants for CRC racial disparities. However, it is also time for action (51). Racial cancer disparities have been documented for decades (52), and still CRC racial differences do not seem to be improving. This is especially unfortunate given that the long-term multistep cancer process for CRC (53), which provides ample time for effective early detection and intervention. At the very least, efforts should be increased to provide routine screening options for all at-risk populations as well as to ensure standard treatment for all persons with newly diagnosed CRC (54).

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