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Racial Disparities in Incidence of Young-onset Colorectal Cancer and Patient Survival

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Abstract

Background & Aims: Increasing rates of young-onset colorectal cancer (CRC) have attracted substantial research and media attention, but we know little about racial disparities among younger adults with CRC. We examined racial disparities in young-onset CRC by comparing CRC incidence and relative survival among younger (age <50 years) adults, in 2 time periods.

Methods: Using data from the Surveillance, Epidemiology, and End Results program of cancer registries, we estimated CRC incidence rates (per 100,000 persons ages 20 – 54 years) from 1992 through 2014, for different time periods (1992–1996 vs 2010–2014) and races (white vs black). Relative survival was calculated as the ratio of observed survival to expected survival in a comparable, cancer-free population.

Results: From 1992–1996 to 2010–2014, CRC incidence increased from 7.5/100,000 to 11.0/100,000 in white individuals and from 11.7/100,000 to 12.7/100,000 in black individuals. The increase in rectal cancer was larger in whites (from 2.7/100,000 to 4.5/100,000) than in blacks (from 3.4/100,000 to 4.0/100,000); in the 2010–2014 time period, black and whites had similar rates of rectal cancer. Compared to whites, blacks had smaller increases in relative survival with proximal colon cancer but larger increases in survival with rectal cancer (from 55.3% to 70.8%).

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Conception and design: Murphy, Wallace, Baron
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Data analysis and interpretation: All
Manuscript writing: Murphy
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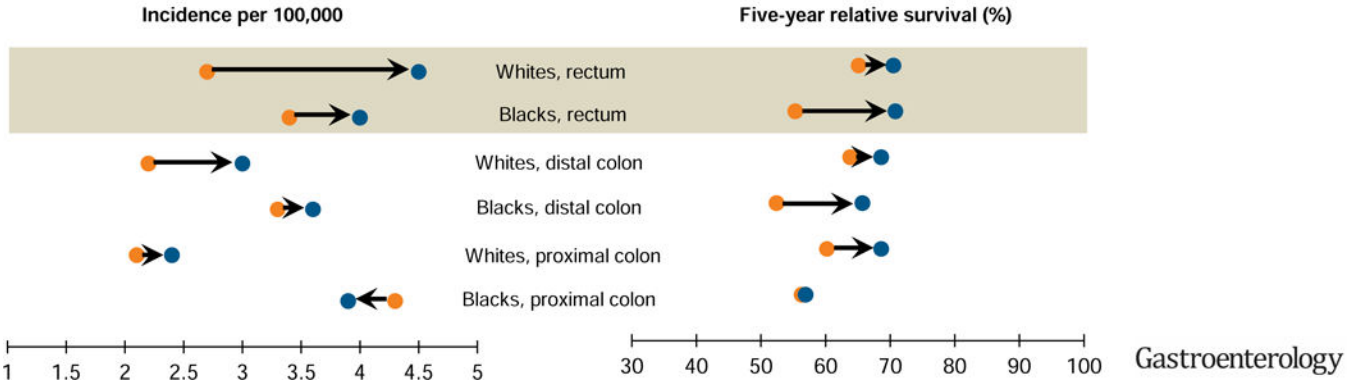
Conclusion: In an analysis of the Surveillance, Epidemiology, and End Results database, we found racial disparities in incidence of young-onset CRC and patient survival for cancer of the colon, but minimal difference for rectal cancer. Well-documented and recent increases in young-onset CRC have been largely due to increases in rectal cancer, especially in whites.

Graphical Abstract

From 1992–1996 [●] to 2010–2014 [●], increases in young-onset colorectal cancer have been largely due to increases in rectal cancer, especially in whites. In the most time recent period, blacks and whites have similar incidence and survival of rectal cancer, a sharp contrast to the striking disparities in colon cancer.



Lay Summary

The incidence of colorectal cancer is increasing in younger adults (age <50 years). The factor that has made the largest contribution to the increase in CRC among younger adults has been increasing rates of rectal cancer, which is more prominent among whites. Our findings provide clues for understanding reasons why incidence has increased.

Keywords

SEER database; neoplasm; young adult; African American

Introduction

Despite overall reductions in colorectal cancer (CRC) incidence and mortality in the U.S.,[1] blacks continue to experience a greater CRC burden than whites.[2] CRC disproportionately affects blacks, and racial disparities persist across several important outcomes: higher incidence[3] and mortality[4] and worse CRC survival.[5]

Well-known, recent increases in CRC incidence have occurred in younger (ages <50 years) adults.[3,6–8] Starting in the early 1990s, incidence rates have increased among younger adults from 8.6 per 100,000 in 1992 to 12.5 per 100,000 in 2015, an overall 45% increase.[9] Increasing rates of young-onset CRC have attracted substantial research and media attention, but we know little about racial disparities in this population. Few studies[6,10–12] have examined racial differences in incidence by anatomic subsite (colon vs. rectum), or the corresponding implications for survival, which may mask important differences in disease

burden and etiology between whites and blacks. Examining racial disparities in both incidence and survival provides important insight concerning differences in risk factors, access to care, and treatment effectiveness.

To better understand CRC disparities between younger whites vs. blacks, we examined racial differences in CRC incidence in younger (age <50 years) adults during 1992 – 2014. We also examined differences in relative survival.

Methods

We derived CRC incidence using Surveillance, Epidemiology, and End Results (SEER) program of cancer registries during 1992 – 2014. The SEER 13 registries cover approximately 15% of the U.S. population and include Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, Utah, Los Angeles, San Jose-Monterrey, and Alaska Native Tumor Registry. Age-adjusted incidence (using the 2000 standard population) were obtained using SEER*Stat version 8.3.5 as rates per 100,000 persons. Corresponding 95% confidence intervals were calculated as modified gamma intervals.[13]

We estimated incidence across two time periods (1992–96 vs. 2010–14) and by race (black vs. white), anatomic subsite, and stage at diagnosis. We selected the time period 1992–96 for a baseline comparison because this was when increases in CRC incidence were first observed in younger populations.[3] Anatomic subsite included proximal colon (cecum, ascending colon, hepatic flexure, and transverse colon), distal colon (splenic flexure, descending colon, and sigmoid colon), and rectum (rectosigmoid junction and rectum). SEER defines stage at diagnosis using historic summary staging: local disease is confined to the large bowel, regional is limited to nearby lymph nodes or other organs, and distant is systemic metastasis.

We calculated relative survival using the Ederer II method[14] as the ratio of observed survival to expected survival in a comparable, cancer-free population. Relative survival provides a measure of excess mortality experienced by cancer patients without requiring cause of death information.[15] Expected survival was calculated from population life tables of the U.S. general population and adjusted across strata of age (15–44, 45–54 years), sex, and race. To illustrate trends, we estimated five-year relative survival in whites and blacks (all ages 20–49 years), contrasting time period (1992–96 vs. 2010–14), anatomic subsite, and stage at diagnosis.

Results

Among whites and blacks ages 20 – 49 years, 31,859 incident cases of CRC were diagnosed in whites, and 5,203 in blacks during 1992 – 2014. Overall incidence was 9.2 per 100,000 and 12.2 per 100,000 among whites and blacks, respectively.

Incidence

From 1992–96 to 2010–14, there were marked differences in incidence trends between whites and blacks. In whites, we observed a large increase in CRC incidence (from 7.5 to 11.0 per 100,000 or 47% relative increase), but in blacks, the increase was small (from 11.7 to 12.7 per 100,000) (Table 1). The incidence of proximal and distal colon cancer increased in whites but remained stable or decreased in blacks (Figure 1). Whites and blacks both experienced increases in rates of rectal cancer; however, this was more prominent in whites. Of the 3.5 per 100,000 absolute increase in CRC incidence among whites, more than half was from increases in rectal cancer. By 2010–14, whites had higher rates of rectal cancer than blacks.

For whites and blacks, incidence rates of local and distant disease increased similarly between 1992–96 and 2010–14 (Figure 2). For example, among whites, the rates of local disease increased from 2.5 to 3.8 per 100,000, accounting for nearly half of the overall increase in CRC incidence (Table 1). Rates of regional disease increased in whites but slightly decreased (from 4.6 to 4.3 per 100,000) in blacks. Despite larger increases in whites over the study period, the absolute incidence of CRC remained consistently higher across all stages in blacks, primarily driven by higher incidence of proximal colon cancer.

We observed a similar pattern in analyses stratified by sex, though women generally had lower incidence than men (results not shown).

Relative survival

For both racial groups combined, five-year relative survival improved from 61.5% in 1992–96 to 67.7% in 2010–14 (Table 2). However, similar to incidence trends, relative survival differed for whites and blacks in the colon versus rectum. Compared to whites, blacks experienced smaller increases in relative survival of proximal colon cancer and much greater improvements in relative survival of rectal cancer (from 55.3 to 70.8%). As a result, by 2010–14, the survival disparity between blacks and whites had increased for cancers in the proximal colon but essentially disappeared for rectal cancer (Figure 3).

Differences in relative survival by stage remained approximately the same over time, and blacks had worse stage-specific survival than whites throughout the study period (Table 2). For example, relative survival of regional stage disease increased from 68.1 to 80.9% among whites, compared to an increase from 57.5 to 72.1% among blacks (Figure 4).

This pattern persisted in analyses stratified by sex, and women had slightly higher relative survival (results not shown).

Discussion

Well-documented, recent increases in young-onset CRC have largely manifested as increases in rectal cancer, particularly among whites. Increases in colon cancer were small by comparison – and seen only in whites. Thus, the dominant factor driving increases in CRC among younger adults has been increasing rates of rectal cancer, more prominent among whites. Similarly, survival increased for both blacks and whites, but this was more evident

for rectal cancer. Blacks and whites now have similar rectal cancer incidence and relative survival, a sharp contrast to the marked disparities in proximal and distal colon cancer. We also observed larger increases in the incidence of local and distant (vs. regional) disease in both blacks and whites, with particularly notable increases in local disease among whites.

Our findings point to differences in the etiology of rectal (vs. colon) cancer that may explain the increase in incidence among younger adults. Factors more strongly associated with rectal cancer and similar in prevalence by race/ethnicity likely play a role. For example, dysbiosis-related factors, such as antibiotic use[16–18] and periodontal disease,[19] seem to be associated with higher risk of rectal cancer, mediated by their effect on microbial diversity and composition. Differences in pH levels across the entire colorectum may differentially influence susceptibility to environmental risk factors and create more favorable conditions for bacterial dysbiosis.[20] Another possibility is the influence of tobacco and heavy alcohol use – these risk factors are differentially associated with colon vs. rectal cancer.[21–25] Familial risk (both family history and genetic syndromes),[20] obesity,[26–28] and physical activity[27,29] also appear less influential in development of rectal cancer. Growing evidence supports etiologic differences in colon and rectal cancer, but researchers have not yet examined whether these risk factors also act differently in blacks and whites.

Prior studies have generally focused on the increasing number of late-stage diagnoses among younger adults,[6,8] but our results show a larger increase in local as opposed to distant disease. The increases in local disease suggest two possible underlying phenomena: 1) diagnostic factors,[7] for example, screening with colonoscopy at age 40 or 45, which may facilitate earlier detection of small tumors; and 2) slow-growing tumors, which may be more amenable to detection at an early stage and more common in younger adults than previously thought. Although we know little about indications for colonoscopy in younger adults, or differences in receipt by race, our prior work shows increases in colonoscopy use that parallel incidence of young-onset CRC,[7] which may explain increasing rates of local disease. Meanwhile, others have suggested unrecognized symptoms,[30] such as rectal bleeding,[31] contribute to delays in presentation and increasing rates of late-stage disease.

Prior studies have also shown racial differences in colon cancer survival are concentrated in younger (vs. older) adults,[5,32–35] even after adjusting for confounders, such as treatment receipt.[5,32–36] Because screening has not been recommended in this younger population, the racial disparities in relative survival that we and others have observed are unlikely to be the result of differential uptake of screening. Consequently, our findings raise a number of questions concerning the contribution of treatment[37,38] and therapeutic response[39,40] to racial differences in relative survival, which may also differ for colon and rectal cancers. We observed no difference in relative survival between blacks and whites with rectal cancer diagnosed in 2010–14, suggesting that equal receipt of guideline-recommended therapies[41] has played a prominent role in achieving health equity for rectal cancer outcomes. Multimodality therapy (i.e., neoadjuvant chemoradiation followed by surgery) for locally advanced rectal cancer reduces risk of recurrence and improves survival[42] and became standard in the early 2000s. Others have similarly reported improvements in overall and disease-free survival for rectal cancer patients between 2001 and 2012, noting that this is likely due to preoperative radiation therapy and chemotherapy.[43]

We also found that improvements in proximal colon cancer survival in blacks lagged far behind whites. The fact that survival disparities by race persist only in colon cancer may reflect differences in tumor biology and treatment response. Blacks are more frequently diagnosed with microsatellite stable tumors in the proximal colon, and patients with proximal colon cancer tend to have higher mortality and poorer outcomes.[44] This is particularly true for those exhibiting low microsatellite instability[45] or with advanced disease.[46–49] Further, growing evidence suggests combination chemotherapies and targeted therapies (e.g., anti-VEGF or anti-EGFR) are less effective in treating proximal colon cancer,[50,51] and tumors in blacks respond poorly to treatment – even when administered at similar rates as whites.[39,40] Clinical characteristics (e.g., obesity, diabetes),[52–55] mutation profiles,[56,57] or differences in immune response may modify therapeutic effectiveness and contribute to poorer outcomes among blacks. Compared to whites, blacks have a greater burden of prognostic factors associated with inflammation, which may contribute to treatment resistance and tumor progression.[58,59] Better understanding factors driving treatment response may reveal additional insight concerning racial disparities in colon cancer survival.

We acknowledge limitations inherent in cancer registry data. These data may not be uniformly accurate for all population subgroups, including racial/ethnic minorities, or geographic regions, and we had incomplete data on stage and anatomic subsite. However, prior studies on the quality of SEER data show near complete case ascertainment (98%),[60] no differences in case ascertainment by race,[61] and excellent agreement between SEER records and self-reported race.[62] Sparse data precluded relative survival estimates by 5-year age groups. Relative survival ignores cause of death information, and instead, differences in relative survival reflect differences in cancer rather competing causes of death.[15] Finally, although our findings raise the possibility that racial disparities in relative survival may be due to receipt of treatment and tumor biology, cancer registries do not systematically collect molecular characteristics or chemotherapy-based treatment regimens.

In summary, our findings emphasize the importance of distinguishing rectal vs. colon cancer while studying the increasing incidence of young-onset CRC and racial disparities in this age group. Although others have reported racial differences in mortality rates of young-onset CRC,[11] mortality reflects both incidence and case fatality. We examined incidence and relative survival as markers of population burden, treatment receipt and effectiveness, and tumor biology. Moving forward, identifying etiologic differences in colon and rectal cancer, and how risk factors may act differently in black and whites, will advance our understanding of young-onset CRC.

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Abbreviations:

CRC	colorectal cancer

SEER Surveillance, Epidemiology and End Results

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What You Need to Know

Background: We investigated racial disparities (black vs white patients) in incidence of young-onset CRC and survival for different time periods (1992–1996 vs 2010–2014).

Findings: In an analysis of the Surveillance, Epidemiology, and End Results database, we found that the incidence of CRC increased from 7.5/100,000 to 11.0/100,000 in white individuals and from 11.7/100,000 to 12.7/100,000 in black individuals. In the 2010–2014 time period, the incidence of rectal cancer incidence did not differ significantly between races.

Implications for patient care: There are racial disparities in incidence of young-onset colon cancer and patient survival, but no significant differences between races in incidence of rectal cancer or survival

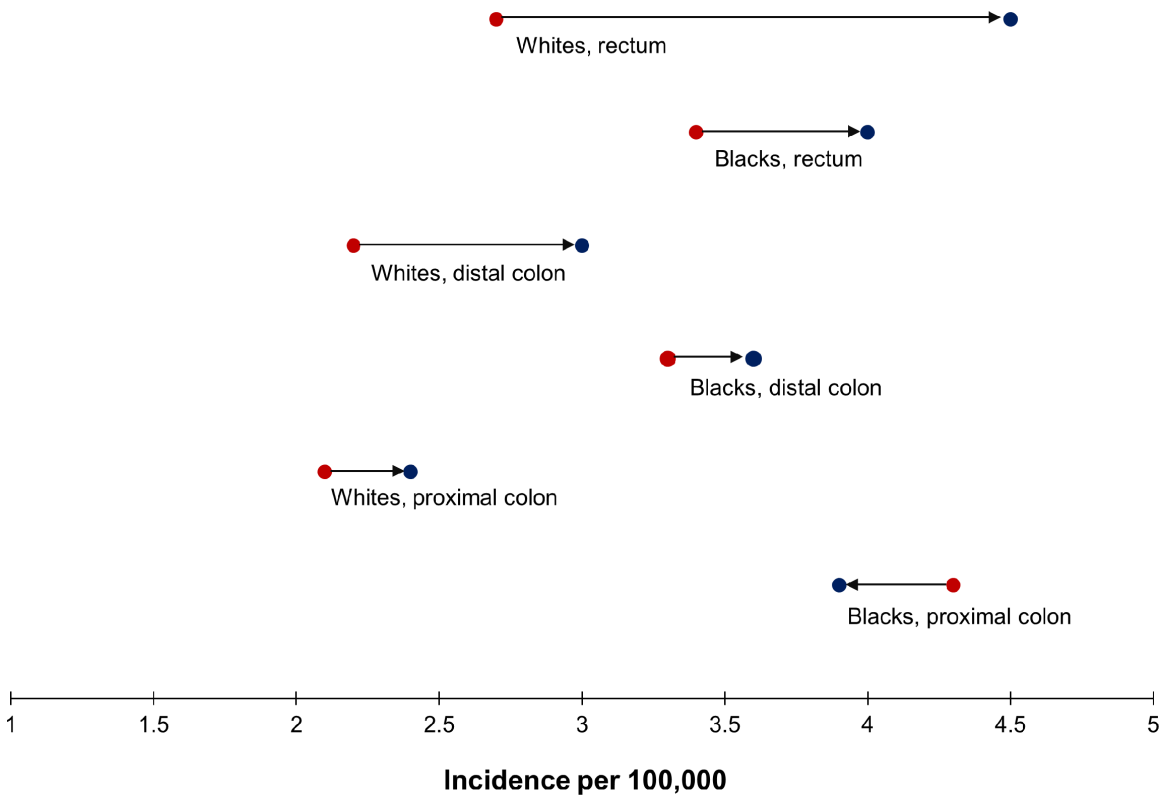

Figure 1.

Incidence (rate per 100,000) of colorectal cancer (ages 20–54 years) by anatomic subsite and race, SEER 13, shown as the rate over the period 1992–96 [●] and over the period 2010–14 [●] : 1992–96 ●→● 2010–14

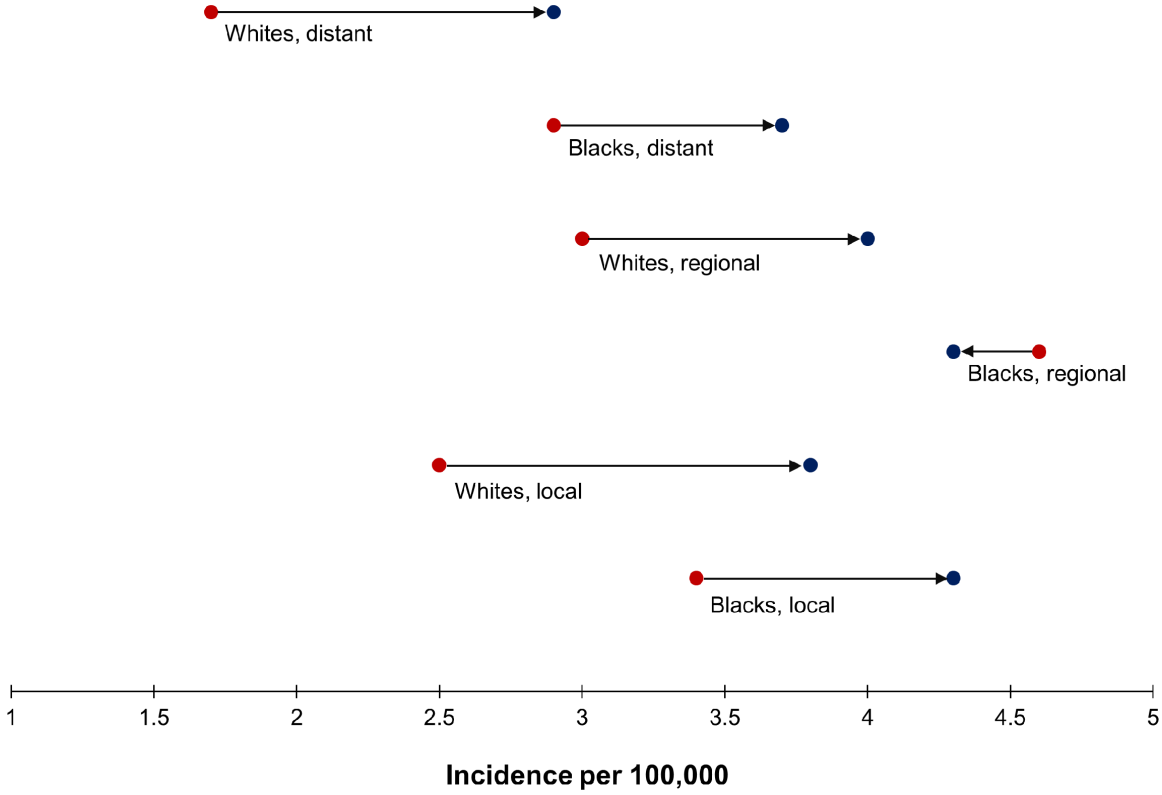

Figure 2.

Incidence (rate per 100,000) of colorectal cancer (ages 20–54 years) by stage at diagnosis and race, SEER 13, shown as the rate over the period 1992–96 [●] and over the period 2010–14 [●] : 1992–96 ●→● 2010–14

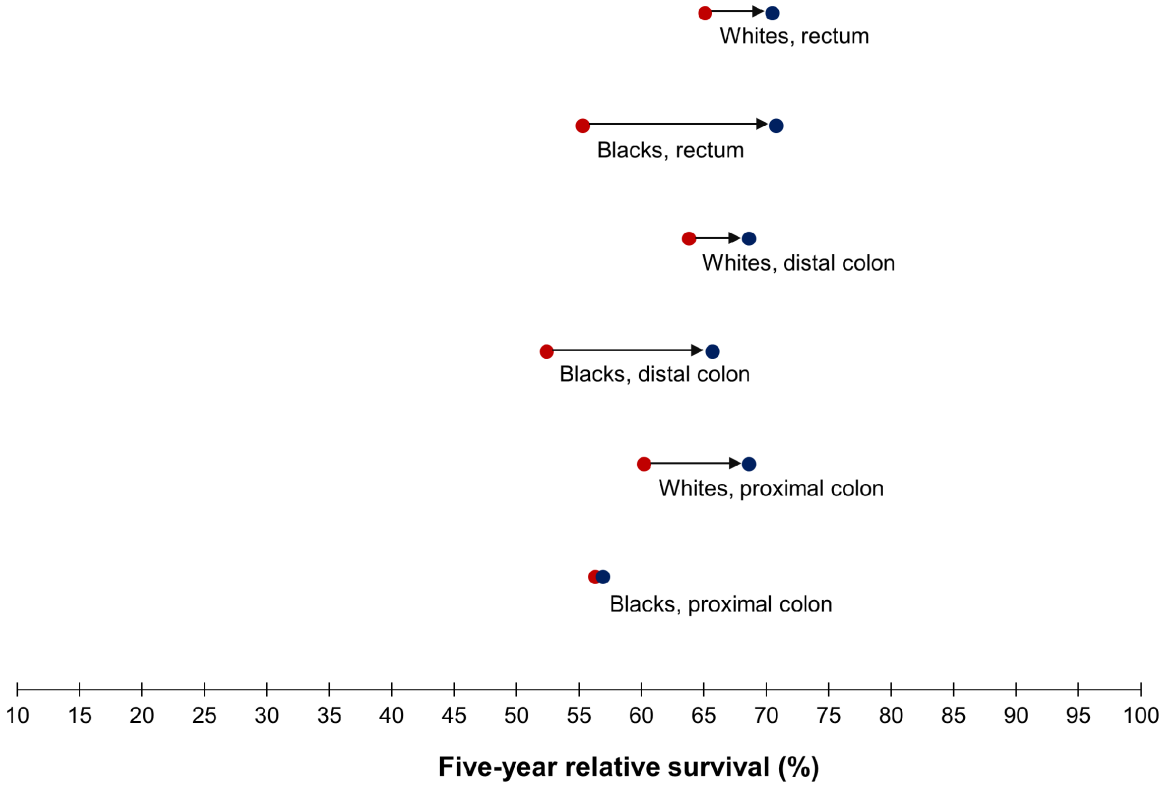

Figure 3.

Five-year relative survival of colorectal cancer (ages 20–54 years) by anatomic subsite and race, SEER 13, shown as the rate over the period 1992–96 [●] and over the period 2010–14 [●] : 1992–96 ●→● 2010–14

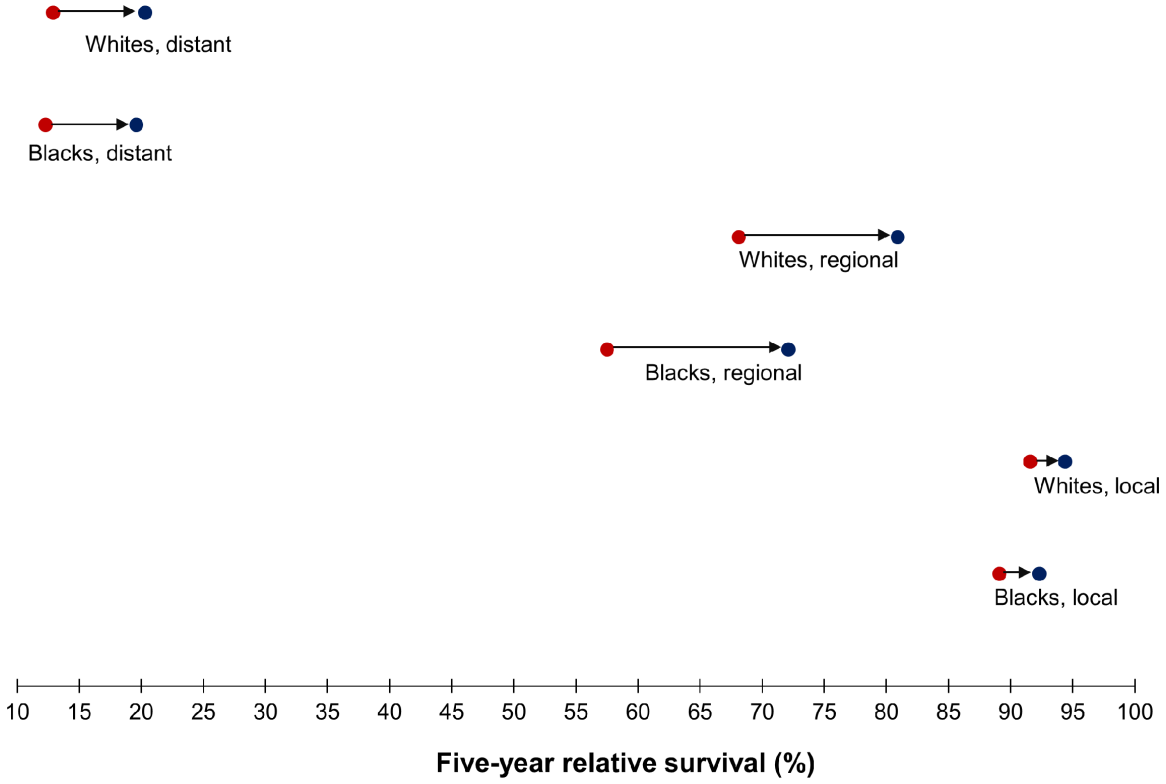

Figure 4.

Five-year relative survival of colorectal cancer (ages 20–54 years) by stage at diagnosis and race, SEER 13, shown as survival over the period 1992–96 [●] and over the period 2010–14 [●] : 1992–96 ●→● 2010–14

Table 1.

Age-adjusted[1] incidence (rate per 100,000) of colorectal cancer (age 20–49 years) by race and time period (1992–96 vs. 2010–14), overall and by anatomic subsite and stage at diagnosis, SEER 13, 1992—2014

	White			Black			Overall		
	1992 – 96	2010 – 14	Rate Diff.[2]	1992 – 96	2010 – 14	Rate Diff.	1992 – 96	2010 – 14	Rate Diff.
Overall	7.5 ± 0.1	11.0 ± 0.1	3.5	11.7 ± 0.4	12.7 ± 0.4	1.0	7.9 ± 0.1	11.2 ± 0.1	3.3
Anatomic subsite									
Proximal colon	2.1 ± 0.1	2.4 ± 0.1	0.3	4.3 ± 0.2	3.9 ± 0.2	−0.4	2.4 ± 0.1	2.6 ± 0.1	0.2
Distal colon	2.2 ± 0.1	3.0 ± 0.1	0.8	3.3 ± 0.2	3.6 ± 0.2	0.3	2.3 ± 0.1	3.1 ± 0.1	0.8
Rectum	2.7 ± 0.1	4.5 ± 0.1	1.8	3.4 ± 0.2	4.0 ± 0.2	0.6	2.7 ± 0.1	4.4 ± 0.1	1.7
Appendix/unspecified	0.5 ± 0.0	1.1 ± 0.0	0.6	0.7 ± 0.1	1.1 ± 0.1	0.4	0.5 ± 0.0	1.1 ± 0.1	0.6
Stage at diagnosis									
Local	2.5 ± 0.1	3.8 ± 0.1	1.3	3.4 ± 0.2	4.3 ± 0.2	1.1	2.6 ± 0.1	3.8 ± 0.1	1.2
Regional	3.0 ± 0.1	4.0 ± 0.1	1.0	4.6 ± 0.2	4.3 ± 0.2	−0.3	3.2 ± 0.1	4.0 ± 0.1	0.8
Distant	1.7 ± 0.1	2.9 ± 0.1	1.2	2.9 ± 0.2	3.7 ± 0.2	0.8	1.8 ± 0.1	3.0 ± 0.1	1.2
Unstaged	0.3 ± 0.0	0.3 ± 0.1	0.0	0.7 ± 0.1	0.5 ± 0.1	−0.2	0.3 ± 0.0	0.3 ± 0.0	0.0

[1] Age-adjusted to the 2000. U.S. standard population

[2] Rate differences correspond to the incidence rate in 1992–96 subtracted from the incidence rate in 2010–14; a negative rate difference indicates decreasing incidence.

Table 2.

Five-year relative survival (including standard errors) of colorectal cancer (age 20 – 49 years) by race/ethnicity and time period (1992–96 vs. 2010–14), overall and by anatomic subsite and stage at diagnosis, SEER 13, 1992—2014

	White			Black			Overall		
	1992 – 96	**2010 – 14**	**Diff.**[1]	**1992 – 96**	**2010 – 14**	**Diff.**	**1992 – 95**	**2010 – 14**	**Diff.**
Overall	63.0 ± 0.8	68.6 ± 0.8	5.6	54.3 ± 1.4	62.8 ± 1.9	8.5	61.5 ± 0.7	67.7 ± 0.8	6.2
Anatomic subsite									
Proximal colon	60.2 ± 1.4	68.6 ± 1.5	8.4	56.3 ± 2.8	56.9 ± 3.2	0.6	60.2 ± 1.2	66.3 ± 1.4	6.1
Distal colon	63.8 ± 1.4	68.6 ± 1.5	4.8	52.4 ± 3.3	65.7 ± 3.4	13.3	62.0 ± 1.3	68.2 ± 1.4	6.2
Rectum	65.1 ± 1.3	70.5 ± 1.3	5.4	55.3 ± 3.2	70.8 ± 3.3	15.5	63.7 ± 1.2	70.5 ± 1.2	6.8
Appendix/unspecified	60.6 ± 3.1	67.8 ± 2.7	7.2	46.1 ± 7.1	46.1 ± 7.7	0.0	58.2 ± 2.9	64.9 ± 2.6	6.7
Stage at diagnosis									
Local	91.6 ± 0.8	94.4 ± 0.8	2.8	89.1 ± 2.2	92.3 ± 2.2	3.2	91.3 ± 0.8	94.1 ± 0.8	2.8
Regional	68.1 ± 1.2	80.9 ± 1.2	12.8	57.5 ± 2.8	72.1 ± 3.2	14.6	66.3 ± 1.1	79.7 ± 1.1	13.4
Distant	12.9 ± 1.1	20.3 ± 1.6	7.4	12.3 ± 2.2	19.6 ± 2.8	7.3	12.8 ± 1.0	20.4 ± 1.4	7.6
Unstaged	68.6 ± 3.8	56.9 ± 5.0	−11.7	49.2 ± 7.4	68.1 ± 9.5	18.9	64.1 ± 3.4	59.0 ± 4.4	−5.1

[1] Difference corresponds to relative survival in 1992–96 subtracted from related survival in 2010–14