

Research Article

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Contribution of Screening and Survival Differences to Racial Disparities in Colorectal Cancer Rates

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Abstract

Background: Considerable disparities exist in colorectal cancer (CRC) incidence and mortality rates between blacks and whites in the United States. We estimated how much of these disparities could be explained by differences in CRC screening and stage-specific relative CRC survival.

Methods: We used the MISCAN-Colon microsimulation model to estimate CRC incidence and mortality rates in blacks, aged 50 years and older, from 1975 to 2007 assuming they had: (i) the same trends in screening rates as whites instead of observed screening rates (incidence and mortality); (ii) the same trends in stage-specific relative CRC survival rates as whites instead of observed (mortality only); and (iii) a combination of both. The racial disparities in CRC incidence and mortality rates attributable to differences in screening and/or stage-specific relative CRC survival were then calculated by comparing rates from these scenarios to the observed black rates.

Results: Differences in screening accounted for 42% of disparity in CRC incidence and 19% of disparity in CRC mortality between blacks and whites. Thirty-six percent of the disparity in CRC mortality could be attributed to differences in stage-specific relative CRC survival. Together screening and survival explained a little more than 50% of the disparity in CRC mortality between blacks and whites.

Conclusion: Differences in screening and relative CRC survival are responsible for a considerable proportion of the observed disparities in CRC incidence and mortality rates between blacks and whites.

Impact: Enabling blacks to achieve equal access to care as whites could substantially reduce the racial disparities in CRC burden. *Cancer Epidemiol Biomarkers Prev*; 21(5); 728–36. ©2012 AACR.

Introduction

Colorectal cancer (CRC) is the second leading cause of cancer death in the United States. In 2010, 142,570 people were estimated to be newly diagnosed with CRC and 51,370 were estimated to die of the disease (1). Although age-adjusted CRC incidence and mortality rates have been falling for white men and women since the late 1970s to early 1980s, the decreases began later and were slower in black men and women (2). Before 1980, CRC

incidence and mortality rates were lower in blacks than in whites (2). It is therefore unlikely that the disparity in CRC rates is the result of biological differences. Differences in social economic status and the resulting differential access to screening and treatment are more likely candidates (3).

Screening has been estimated to be the most important driver of the observed decline in CRC incidence and mortality (4). Although dissemination of CRC screening continues to be suboptimal in both whites and blacks despite recommendations of the U.S. Preventive Services Task Force (5) and the U.S. Multi-Society Task Force on Colorectal Cancer (6), dissemination is considerably lower for blacks than for whites (7). According to the 2008 National Health Interview Survey, 51.4% of blacks aged 50 years and older reported having either a fecal occult blood test (FOBT) within the past year, a sigmoidoscopy in the past 5 years, and/or colonoscopy in the past 10 years, compared with 57.0% among whites (7). In earlier years, the disparity in screening uptake was even bigger.

Treatment has been estimated to be another important driver of the observed decrease in CRC mortality (4). Several studies have shown that blacks are treated less frequently with chemotherapy and radiation therapy than whites (8–10), although randomized controlled trials

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showed that equal treatment leads to equal outcomes (11, 12). This treatment disparity has contributed to the poorer CRC survival in blacks than whites, even after correcting for potential confounding factors (13–15).

These differences in screening uptake and survival are both assumed to have contributed to the observed disparity in CRC incidence and mortality between blacks and whites (3). Differences in lifestyle factors known to influence CRC risk, such as obesity, smoking, and physical activity (16), have also likely contributed to the observed disparity in CRC incidence and mortality rates. However, although differences in screening and stage-specific relative CRC survival are mostly the result of differential access to care and may be mitigated by enabling equal access, lifestyle factors such as dietary pattern and leisure time physical activity, might be more difficult to modify. In this analysis, we therefore focused on access to care: we determined to what extent equal access to care could reduce observed disparities in CRC incidence and mortality rates between blacks and whites by estimating how much of these disparities can be explained by differences in CRC screening and stage-specific relative CRC survival.

Methods

We estimated the contribution of differences in CRC screening and stage-specific relative CRC survival between blacks and whites to disparities in CRC rates using the MISCAN-Colon microsimulation model (17, 18) of the Cancer Intervention and Surveillance Modeling Network (CISNET).

The MISCAN-Colon model

Appendix 1 describes the MISCAN model. Briefly, the model simulates the life histories of a large population of individuals from birth to death and has a natural history component that tracks the progression of underlying colorectal disease in the absence of screening. As each simulated individual ages, there is a chance that one or more adenomas may develop depending on age, sex, race,

and individual risk. Adenomas can progress from small (1–5 mm) to medium (6–9 mm) to large (≥ 10 mm) size and some may eventually become malignant. A preclinical cancer (i.e., not detected) has a chance of progressing through stages I to IV and may be detected by symptoms at any stage. With screening, adenomas and preclinical cancers may be detected depending on the sensitivity of the test for that lesion and, for endoscopic tests, whether the lesion is within reach of the endoscope.

For each age and sex, the natural history model outcomes were calibrated to prescreening data from autopsy studies (19–29) and clinical incidence data from the Surveillance, Epidemiology, and End Results (SEER) Program before the introduction of screening (1975–1979; ref. 30). The model uses race- and sex-specific all-cause mortality estimates from the U.S. life tables. Trends in race- and sex-specific relative survival following CRC diagnosis from 1975 to 2003 were obtained from SEER (30; Appendix 2). Estimates for screening uptake over time by age, sex, and race were obtained from 7 waves of the National Health Interview Survey (31) (Table 1). We assumed no screening before 1978. For years for which no data were available, we linearly extrapolated trends from years for which data were available. The assumptions for the sensitivity and specificity of screening tests were based on a literature review (32). We assumed that colonoscopy reached the cecum in 98% of subjects. For sigmoidoscopy, we assumed that 80% of examinations reached the junction of the sigmoid and descending colon and 40% reached the beginning of the splenic flexure (33, 34). We assumed that 1 in 10,000 colonoscopies led to a fatal complication.

The validity of the model has previously been tested using the data from several large randomized screening and surveillance studies, such as the CoCap sigmoidoscopy study (35), the Minnesota Colon Cancer Control Study (35), and the National Polyp Study (36). In addition, the model was able to reproduce the observed CRC incidence and mortality trends in the United States while

Table 1. Age-standardized estimates from 7 waves of the National Health Interview Survey (31) for the percentage of the population aged 50 years and older ever having had a CRC test, having had home-based FOBT in the past 2 years or endoscopy in the past 10 years, by survey year, sex, and race

Population subgroup	% Ever had CRC test							% FOBT within past 2 years ^a						% Endoscopy in past 10 years ^a			
	1987	1992	1998	2000	2003	2005	2008	1992	1998	2000	2003	2005	2008	2000	2003	2005	2008
White men	35.9	45.7	52.9	54.4	58.1	60.4	66.0	15.6	19.6	22.5	22.0	18.3	16.0	37.1	42.7	47.4	55.3
White women	35.7	43.9	47.1	54.8	55.3	60.9	64.1	16.9	19.2	24.9	21.0	18.0	15.3	32.0	36.9	43.9	49.7
Black men	18.4	37.2	39.8	49.1	51.9	48.7	56.3	7.8	13.9	17.7	22.4	15.5	13.8	37.3	38.0	36.2	47.1
Black women	19.5	36.3	34.1	43.3	48.4	51.3	56.6	11.3	12.0	20.5	20.4	16.4	14.4	22.8	30.4	37.9	47.0

NOTE: Although we present age-standardized estimates in this table, age-specific estimates served as inputs for the model.

^aNo questions were asked about FOBT use in the past 2 years in the first NHIS wave of 1987. No questions were asked about endoscopy use in the past 10 years in the NHIS waves of 1987, 1992, and 1998.

accounting for secular trends in risk factor prevalence, screening practice, and chemotherapy treatment (37).

Study population. We used the model to simulate the U.S. population from 1975 to 2007 for black men and women. We restricted our analysis to the simulated population aged 50 years and older, because this is the group for whom screening is recommended (5, 6) and incidence and mortality in this group accounts for almost 90% of total CRC incidence and mortality (38).

CRC screening and survival runs. We used the simulation model to estimate annual age-standardized CRC incidence and mortality rates in black men and women ages 50 years and older for each year from 1975 to 2007 assuming trends in:

1. CRC screening and stage-specific relative CRC survival rates as observed for blacks (run 1);
2. CRC screening rates as observed for whites and in stage-specific relative CRC survival rates as observed for blacks (run 2);
3. CRC screening rates as observed for blacks and in stage-specific relative survival rates as observed for whites (run 3);
4. CRC screening and stage-specific relative CRC survival rates as observed for whites (run 4).

Analysis

Expected CRC incidence and mortality rates. Using the results of the above runs, we then calculated 3 sets of expected incidence and mortality rates: (i) expected annual CRC incidence and mortality rates if blacks would have had the same screening pattern as whites; (ii) expected annual CRC mortality rates if blacks would have had the same stage-specific relative CRC survival pattern as whites; and (iii) expected annual CRC mortality rates if blacks would have had the same screening and stage-specific relative CRC survival pattern as whites.

The expected CRC incidence and mortality rates for blacks, assuming that they had patterns of screening similar to those in whites from 1975 to 2007, were estimated by applying the percent difference in age-adjusted incidence (mortality) rate in each year between runs 1 and 2 to the observed CRC incidence (mortality) rate for blacks in that year. The expected CRC mortality rates for blacks, assuming they had the same stage-specific relative CRC survival as whites, were estimated by applying the percent difference in mortality rate each year between runs 1 and 3 to the observed CRC mortality rate for blacks. Finally, the expected CRC mortality rates for blacks, assuming they had the same patterns of screening and stage-specific relative CRC survival as whites, were estimated by applying the percent difference in mortality rate each year between runs 1 and 4 to the observed CRC mortality rate for blacks.

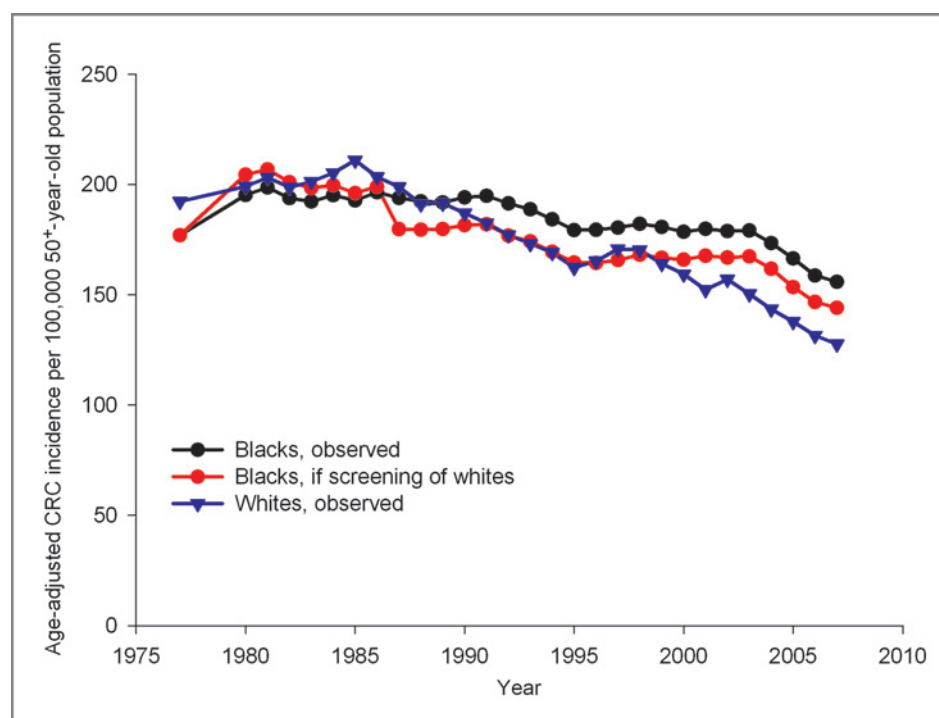
Calculation of disparities and proportion of disparities explained by screening and survival. We calculated observed and expected disparities in CRC incidence and mortality as the absolute difference in age-standardized

rates [the recommended measure for pairwise comparisons (39)] in 2007, the most recent year for which both incidence and mortality data are available from SEER. The observed disparity was calculated as the absolute difference in CRC incidence (mortality) rates between blacks and whites (formula 1, appendix 3). The expected disparity (in 2007) if blacks would have had the same screening as whites was calculated as the absolute difference between the expected CRC incidence (mortality) rate in blacks if they had had white trends in screening rates and the observed CRC incidence (mortality) in whites (formula 2, appendix 3). Similarly, the expected disparity in CRC mortality if blacks would have had the same stage-specific relative CRC survival as whites was calculated as the absolute difference between the expected CRC mortality rate in blacks if they had white trends in relative survival rates and the observed CRC incidence (mortality) in whites (formula 3, appendix 3). Finally, the expected disparity in CRC mortality if blacks would have had the same CRC screening and stage-specific relative CRC survival as whites was calculated as the absolute difference between the expected CRC mortality rate in blacks if they had white trends in screening and relative survival rates and the observed CRC incidence (mortality) in whites (formula 4, appendix 3).

The proportion of disparity in CRC incidence (mortality) in 2007 that was explained by screening was then calculated as the difference between the observed disparity in CRC incidence (mortality) rates and the expected disparity if blacks had white patterns of screening, expressed as the percentage of the observed disparity in CRC incidence (mortality) rates (formula 5, appendix 3). The proportion of disparity in CRC mortality explained by CRC survival was calculated as the difference between the observed disparity in CRC mortality rates and the disparity if blacks had white patterns of stage-specific relative CRC survival (formula 6, appendix 3). The total proportion of disparity that could be explained by screening and survival was calculated as the difference between the observed disparity in CRC mortality rates and the expected disparity if blacks had white patterns of CRC screening and stage-specific relative CRC survival expressed as the percentage of the observed disparity in CRC mortality rates (formula 7, appendix 3).

Sensitivity analyses. We conducted 3 sensitivity analyses. First, we conducted an analysis in which blacks not only received less screening but also lower quality screening, assuming 25% lower adenoma detection rates with endoscopy. We then reestimated the proportion of disparity explained by differences in screening assuming white screening adherence and quality. Second, we explored the robustness of our results to the assumption that equal access to care resulted in the same stage-specific relative CRC survival for blacks and whites by assuming that 25% of the difference in relative survival between blacks and whites could not be taken away with equal access to care. Finally, we evaluated the impact on mortality disparity if equal access to care not only resulted in

Figure 1. Age-standardized CRC incidence rates in the 50⁺ year-old population from 1975 to 2007, as observed for whites and blacks, and as expected for blacks if they would have had the same screening pattern as whites. (The rates for blacks have been smoothed by taking 3-yearly averages for each datapoint).



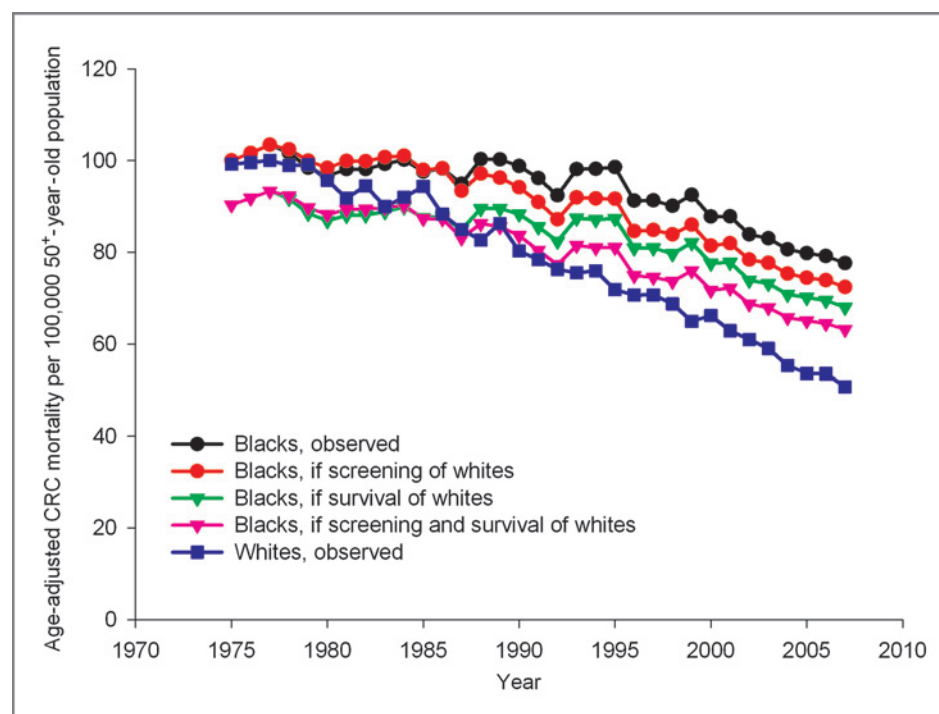
the same stage-specific relative CRC survival for blacks as for whites, but also in the same stage distribution.

Results

If blacks aged 50 years and older would have had the same screening pattern as observed in whites, their

expected CRC incidence rate per 100,000 in 2007 would have been 144.1, 7.6% lower than the observed rate for blacks in 2007 (Fig. 1). The expected CRC mortality rate would have been 72.4 per 100,000 (6.7% lower than observed, Fig. 2). If blacks would have had the same trend in stage-specific relative CRC survival as whites, CRC

Figure 2. Age-standardized CRC mortality rates in the 50⁺-year-old population from 1975 to 2007, as observed for whites and blacks, and as expected for blacks if they would have had the same screening pattern as whites, or the same survival pattern or a combination. (The rates for blacks have been smoothed by taking 3-yearly averages for each datapoint).



mortality would drop to 68.0 per 100,000 in 2007, 12.4% lower than the observed rate in 2007. With the same trend in screening and stage-specific relative CRC survival as whites, CRC mortality in blacks would have been 19.6% lower than observed for a rate of 63.2 per 100,000 in 2007.

The observed disparities in age-standardized CRC incidence and mortality rates (per 100,000) in 2007 between blacks and whites aged 50 years and older were 28.2 incident cases and 26.8 deaths, respectively (Table 2). With the same screening pattern for blacks as for whites the disparity in these rates would decrease to 16.4 incident cases and 21.6 deaths per 100,000. With the same stage-specific relative CRC survival for blacks as for whites, the disparity in CRC mortality rates would decrease to 17.2 deaths per 100,000. If blacks would both have the same screening and stage-specific relative CRC survival as whites, the disparity in CRC mortality would further decrease to 12.4 deaths per 100,000. As such, differences in CRC screening explained 42% of the observed disparity in CRC incidence between blacks and whites and 19% of disparity in CRC mortality. Stage-specific relative CRC survival differences explained 36% of the disparity in CRC mortality. Together differences in screening and survival explained 54% of disparity in CRC mortality.

Results for men and women separately were similar (Table 2), with the exception of the percent of disparity in CRC incidence explained by screening. For men, 51% of the observed disparity in CRC incidence between blacks and whites could be explained by screening, while for women this was 32%.

Sensitivity analysis

If blacks not only receive less screening but also lower quality screening, the percent of disparity in CRC incidence and mortality explained by screening increased to 68% and 34%, respectively (Table 3). If 25% of difference in stage-specific relative CRC survival between blacks and whites cannot be removed through equal access to care, only 28% of the disparity in CRC mortality could be attributed to differences in treatment. Finally, if equal access to care not only results in the same stage-specific relative CRC survival in blacks as in whites but also in the same stage distribution, 60% of the disparity in CRC mortality was attributable to differences in access to care.

Discussion

This study shows that more than 40% of disparity in CRC incidence and approximately 20% of disparity in CRC mortality between blacks and whites can be explained by differences in screening uptake. Approximately 35% of the disparity in CRC mortality can be explained by differences in stage-specific relative CRC survival. Together, differences in screening and relative survival explain a little more than 50% of disparity in CRC mortality between blacks and whites.

Table 2. Base case analysis: disparities in CRC incidence and mortality between blacks and whites aged 50 years and older, as observed in 2007 and as expected if blacks had the same CRC screening and stage-specific relative CRC survival patterns as whites

Sex	CRC incidence disparity ^a			CRC mortality disparity ^a			
	Observed	Expected with same screening	% Explained by screening	Observed	Expected with same screening	% Explained by screening	% Explained by screening and survival ^b
Male	30.0	14.8	51%	35.3	28.7	19%	51%
Female	29.8	20.3	32%	23.2	18.9	19%	53%
Total	28.2	16.4	42%	26.8	21.6	19%	54%

^aPer 100,000 50⁺-year-old population. Disparity is defined as the absolute difference in rates in 2007. For example, the observed disparity in CRC incidence (28.2 per 100,000 50⁺-year-old population) is calculated as the observed CRC incidence rate in blacks in 2007 (155.9 final datapoint of black line in Fig. 1) minus the observed CRC incidence rate in whites in 2007 (127.7; final datapoint of red line in Fig. 1).

^bBecause of synergy in effects, separate effects of screening and survival do not add up.

Table 3. Sensitivity analyses: percentage of disparities in CRC incidence and mortality between blacks and whites aged 50 years and older explained by screening and survival

Sensitivity analysis	CRC incidence disparity	CRC mortality disparity		
	% explained by screening	% explained by screening	% explained by survival	% explained by screening and survival ^a
Base case analysis	42	19	36	54
Lower quality endoscopy in blacks	68	34	36	66
25% remaining survival difference	42	19	28	46
Same stage distribution as whites	42	19	57	60

^aBecause of synergy in effects separate effects of screening and survival do not add up.

Although differences in screening uptake and survival explain approximately half of the racial disparities in CRC incidence and mortality, the other half cannot be explained by these factors. A closer look at the data reveals that this finding would have been anticipated. Where racial disparities in CRC incidence and mortality have been increasing over the years (3), the disparity in screening has been stable and even decreasing slightly (Table 1). The most recent estimates for colorectal screening uptake report a less than 5% difference in uptake between whites (59.8%) and blacks (55.0%) (40). If screening would have explained the vast majority of the difference in CRC incidence and mortality, we would have expected the same diverging trend for screening rates as for CRC rates. Moreover, part of the observed disparity, especially in males, is caused by an increase in CRC incidence and mortality rates in blacks over the period 1975 to 1980 (38), which was before the introduction or wide dissemination of screening modalities and/or adjuvant chemotherapies. Because this increase in CRC incidence and mortality cannot be a result of screening or survival, part of the disparity could never be explained by these factors.

Other factors that may have contributed to the racial disparity in CRC incidence and mortality rates are differences in susceptibility, quality of care, and lifestyle. The prevalence of polymorphisms associated with CRC risk has been shown to differ between whites and blacks (41). On the contrary, several studies have shown that most of black/white differences in CRC outcomes such as stage of disease at diagnosis or survival are no longer present when correcting for socioeconomic status, including health insurance status (8). Furthermore, a follow-up study of Prostate, Lung, Colorectal and Ovarian Cancer Screening Trial participants after a positive sigmoidoscopy revealed a lower uptake of diagnostic colonoscopies among blacks when compared with whites but little differences in the yield of colorectal neoplasia (42). Finally, CRC incidence and mortality in blacks were lower or comparable with whites in the 1970s (30), making it unlikely that susceptibility is an important driver of current disparities.

In our primary analysis, we only considered uptake of screening, assuming equal quality of screening among blacks and whites. Quality of endoscopy has been shown to be dependent on the skill of the endoscopist carrying out the procedure, with adenoma detection rates of high adenoma detectors being double that of low adenoma detectors (43–47). Adenoma detection rate is an independent predictor of the risk of interval CRC after screening colonoscopy (47). Physicians treating black patients are known to be less well trained and have less access to clinical resources than physicians treating white patients (48). Different quality of screening between blacks and whites is therefore not improbable. The sensitivity analysis indeed showed that if blacks received lower quality screening, a larger proportion of the CRC disparity would be explained by screening. We have eliminated disparities in quality of stage-specific treatments, by evaluating the impact of assuming the stage-specific relative CRC survival of whites for blacks. However, we have not eliminated potential disparities in timeliness of treatment. Blacks are known to present with more advanced stage of disease than whites (41). Although part of this difference may be explained by differences in screening uptake, timeliness of care seeking may also play an important role. In a sensitivity analysis assuming the same stage distribution for blacks as for whites (in the absence of screening), the proportion of disparity in CRC mortality explained by access to care indeed increased.

Known or unknown lifestyle factors are the most likely candidates to explain the remaining 34% to 46% (Table 3) of the disparity in CRC incidence and mortality that cannot be explained by screening and stage-specific relative CRC survival differences. Several lifestyle factors are known to be associated with CRC risk. Alcohol, smoking, obesity, and meat consumption increase the risk of CRC, whereas physical activity and postmenopausal hormone replacement therapy (in women) decrease risk (49). Smoking prevalence had been higher in black men than white men until the late nineties (50). Furthermore, obesity has been consistently higher in blacks than in whites since 1970, while rates of physical activity have consistently

been lower (50). We have not explicitly evaluated the effect of these established lifestyle factors. Instead, we decided to focus on the more actionable items of screening and survival because lifestyle factors might be more difficult to modify.

Five limitations are noteworthy. First, we assumed black–white differences in adenoma onset, CRC location and stage distribution, but not in types of cancers or tumor aggressiveness. This assumption is supported by the fact that CRC incidence and mortality used to be lower in blacks than in whites (30). Second, we did not incorporate risk factors into the model. As a result of that limitation and because of other potential exogenous factors, the simulated CRC incidence and mortality levels will not correspond with the observed. Instead, we assumed that the simulated relative benefit of white screening and stage-specific relative CRC survival patterns over black would be applicable to the observed CRC incidence and mortality. This approach mirrors a relative risk approach, in which it is assumed that the relative risk of, for example, screening is constant irrespective of the background incidence and mortality level. This assumption seems reasonable: all 3 randomized controlled trials on biennial guaiac FOBT screening found similar percent mortality reductions ranging from 15% to 21% despite being carried out in populations with a different background incidence level (51–53).

Third, screening uptake in the model was based upon estimated test rates from multiple waves of the National Health Interview Survey. These tests may have been carried out for other reasons than screening. Furthermore, the estimates from these surveys may be biased because they are based on self-report; the data are not longitudinal so we had to make assumptions for screening patterns within individuals in the model; the questions on CRC screening have changed from survey to survey; and the respondents may not be representative for the U.S. population as a whole. This last bias in particular may have influenced our results if the bias differs for whites versus blacks. If we have overestimated screening rates in blacks, the contribution of screening differences to the observed disparities in CRC rates may be higher than the estimated 40% to 50%.

Fourth, CRC incidence and mortality data for blacks are sparse. For this analysis, we therefore used 3-year pooled estimates for CRC incidence and mortality rates over time. However, even with this pooling, an interesting discrepancy exists between the racial disparities in CRC incidence and mortality for women. The absolute racial disparity in CRC incidence is larger than that for mortality, while the opposite would have been expected (as can be seen for men). As a result, the proportion of CRC incidence disparity that can be explained by screening is much smaller for women than for men, whereas the results for CRC mortality are similar. Fifth, we restricted our analysis to the simulated population aged 50 years and older, because this is the group for whom screening is recommended (5, 6) and

incidence and mortality in this group account for almost 90% of total CRC incidence and mortality (38). CRC incidence and mortality is disproportionately higher in blacks among people younger than 50 years. Racial disparities in survival could have played a role in this difference.

Finally, we have not explicitly considered racial differences in treatment but used racial differences in stage-specific relative CRC survival as a proxy. Data on use and quality of CRC treatment by race are sparse, especially for the population below 65 years of age. There is some information on chemotherapy use (9, 10, 41), but data on other types of treatment such as surgery and radiotherapy are hard to come by. If part of the racial differences in survival cannot be explained by differences in (quality of) treatment, we have overestimated the potential for reducing disparities in CRC mortality. A systematic review of cancer-specific survival differences between blacks and whites showed that only modest cancer-specific survival differences are evident for blacks and whites treated comparably for similar stage cancer. For CRC, no difference was found (54). Therefore, differences in cancer biology between racial groups are unlikely to be responsible for a substantial portion of the observed discrepancy in stage-specific relative CRC survival (54). We explored the impact of our assumption in a sensitivity analysis and found that the effect was limited.

Although differences in screening and stage-specific relative CRC survival do not explain all of the observed racial disparities in CRC incidence and mortality, they do explain roughly half. Measures should therefore be taken to eliminate the gaps in screening use and survival between blacks and whites. The NIH's State-of-the-Science conference has concluded that elimination of financial barriers should be the first priority area to enhance the use of CRC screening (55). The Affordable Care Act may be an important step toward this elimination. The Act aims to improve access to quality health care for all Americans (56). Furthermore, all new health plans must cover certain preventive services including CRC screening without charging a deductible, copay, or coinsurance. Several studies have shown that in situations with equal access to care, such as military medical centers, Department of Defense facilities, Medicare, Medicaid, or clinical trials, no differences in screening uptake or CRC treatments between blacks and whites exist (57–62).

In conclusion, this study shows that approximately half of the disparities in CRC incidence and mortality rates between blacks and whites can be explained by differences in screening and survival. Enabling blacks to achieve equal access to care as whites could therefore substantially reduce the racial disparities in CRC burden.

Disclosure of Potential Conflicts of Interest

The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; or

preparation, review, and approval of the manuscript. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Cancer Institute or the NIH. No potential conflicts of interest were disclosed.

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Development of methodology: I. Lansdorp-Vogelaar, K.M. Kuntz, A.B. Knudsen, M. van Ballegooijen, A.G. Zauber, A. Jemal.

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