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Preface

Colorectal cancer is the third most commonly diagnosed cancer and the third leading cause of cancer death in both men and women in the US. The American Cancer Society estimates that 136,830 people will be diagnosed with colorectal cancer and 50,310 people will die from the disease in 2014. The majority of these cancers and deaths could be prevented by applying existing knowledge about cancer prevention, increasing the use of recommended screening tests, and ensuring that all patients receive timely, standard treatment. In the past decade, there has been unprecedented progress in reducing colorectal cancer incidence and death rates in the US, largely due to the prevention and early detection of colorectal cancer through screening. However, in 2010 only 59% of people age 50 or older, for whom screening is recommended, reported having received colorectal cancer testing consistent with current guidelines.¹

Screening has the potential to prevent colorectal cancer because it can detect precancerous growths, called polyps, in the colon and rectum. Although most polyps will not become cancerous, removing them can prevent cancer from occurring. Furthermore, regular screening increases the likelihood that colorectal cancers that do develop will be detected at an early stage, when they are more likely to be cured, treatment is less extensive, and recovery is faster. In addition to following recommended screening guidelines, people can reduce their risk of developing or dying from colorectal cancer by maintaining a healthy body weight; engaging in regular physical activity; eating a healthy, well-balanced diet; limiting alcohol consumption; and not smoking.

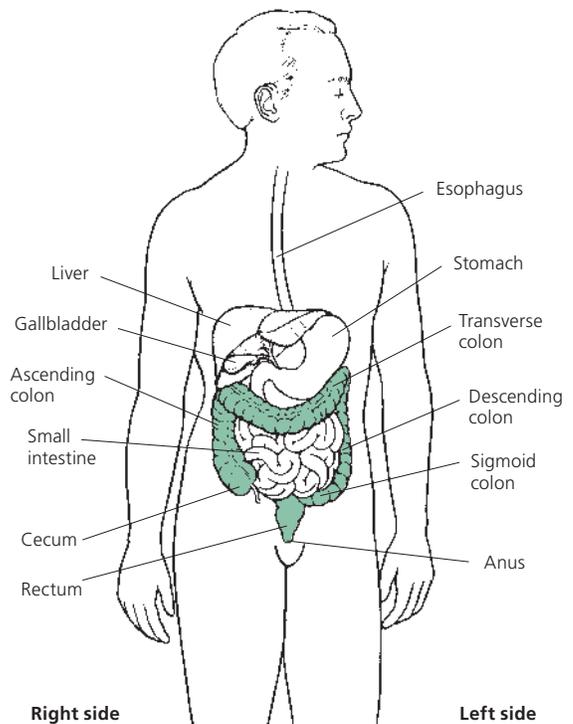
The American Cancer Society has identified colorectal cancer as a major priority because the application of existing knowledge has such great potential to prevent cancer, diminish suffering, and save lives. This fourth edition of *Colorectal Cancer Facts & Figures* is part of the Society's effort to motivate the public and medical communities to prevent the tragic and avoidable suffering caused by colorectal cancer. It is intended to provide basic information about colorectal cancer to the general public, the media, and health professionals. More detailed information on many topics related to colorectal cancer is available on the American Cancer Society's Web site at cancer.org.

Colorectal Cancer Basic Facts

What is colorectal cancer?

Colorectal cancer develops in the colon or the rectum, also known as the large intestine (Figure 1). The colon and rectum are parts of the digestive system, also called the gastrointestinal (GI) system. The digestive system processes food for energy and rids the body of solid waste (fecal matter or stool). After food is chewed and swallowed, it travels through the esophagus to the stomach. There it is partially broken down and sent to the small intestine, where digestion continues and most of the nutrients are absorbed. Cancer develops much less often in the small intestine than in the colon or rectum (colorectum). The small intestine joins the large intestine in the lower right abdomen. The small and large intestine are sometimes called the small and large bowel. The first and longest part of the large intestine is the colon, a muscular tube about 5 feet long. Water and mineral nutrients are absorbed from the food matter in the colon. Waste (feces) left from this process passes into the rectum, the final 6 inches of the large intestine, and is then expelled from the anus.

Figure 1. Anatomy of the Colon and Rectum



The colon has 4 sections:

- The first section is called the ascending colon; it begins with a pouch called the cecum, where undigested food is received from the small intestine, and extends upward on the right side of the abdomen.
- The second section is called the transverse colon because it crosses the body from the right to the left side.
- The third section is called the descending colon because it descends on the left side.
- The fourth section is called the sigmoid colon because of its “S” shape; the sigmoid colon joins the rectum, which connects to the anus.

The ascending and transverse sections are collectively referred to as the proximal colon, while the descending and sigmoid colon are referred to as the distal colon. Colorectal cancers have different characteristics based on their location within the colon or rectum.² For example, tumors in the proximal, or right, colon are more common among women and older patients whereas distal, or left-sided, tumors are more common among men and younger patients.^{3,4}

Colorectal cancer usually develops slowly, over a period of 10 to 20 years.⁵ Most begin as a noncancerous growth called a polyp that develops on the inner lining of the colon or rectum.⁶ The most common kind of polyp is called an adenomatous polyp or adenoma. Adenomas arise from glandular cells, which produce mucus to lubricate the colorectum. An estimated one-third to one-half of all individuals will eventually develop one or more adenomas.^{7,8} Although all adenomas have the capacity to become cancerous, fewer than 10% are estimated to progress to invasive cancer.^{9,10} The likelihood that an adenoma will evolve into cancer increases as it becomes larger.¹¹ Cancer that develops in glandular cells is called adenocarcinoma. Most colorectal cancers (approximately 96%) are adenocarcinomas.¹²

What are the stages of colorectal cancer?

Once cancer forms in the inner lining of the large intestine, it can grow into the wall of the colon or rectum (Figure 2). Cancer that has grown into the wall can also penetrate blood or lymph vessels. (Lymph vessels are thin channels that carry away cellular waste and fluid.) Cancer cells typically spread first into nearby lymph nodes, which are bean-shaped structures that help fight infections. Cancerous cells can also be carried in blood vessels to the liver or

lungs, or can spread into the pelvis and abdominal cavity to other organs and tissues, such as the peritoneum (membrane lining the abdomen) and ovary. The spread of cancer cells to distant parts of the body is called metastasis.

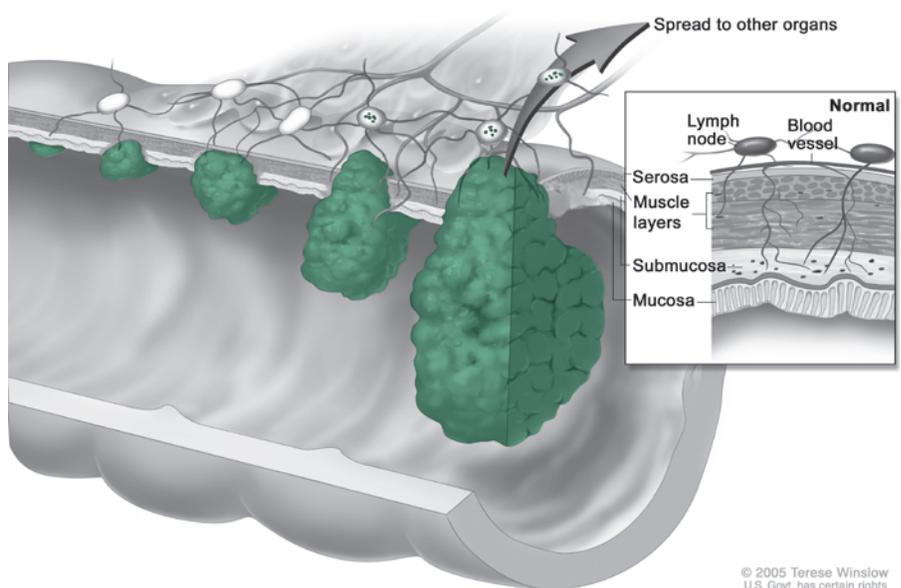
The extent to which cancer has spread at the time of diagnosis is described as its stage. Staging is essential in determining the choices for treatment and in assessing prognosis (prediction of disease outcome). More than one system is used for the staging of cancer. The two most common staging systems are the TNM system, typically used in clinical settings, and the Surveillance, Epidemiology, and End Results (SEER) summary staging system, used for descriptive and statistical analysis of tumor registry data. In this document, we will describe colorectal cancer stages using the SEER summary staging system:

- **In situ:** Cancers that have not yet begun to invade the wall of the colon or rectum; these preinvasive lesions are not included in the cancer statistics provided in this report.
- **Local:** Cancers that have grown into the wall of the colon or rectum, but have not extended through the wall to invade nearby tissues
- **Regional:** Cancers that have spread through the wall of the colon or rectum and have invaded nearby tissue, or that have spread to nearby lymph nodes
- **Distant:** Cancers that have spread to other parts of the body, such as the liver or lung

What are the symptoms of colorectal cancer?

Early colorectal cancer often has no symptoms, which is why screening is so important. As a tumor grows, it may bleed or

Figure 2. Colorectal Cancer Growth



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obstruct the intestine (Figure 2). See your doctor if you have any of these warning signs:

- Bleeding from the rectum
- Blood in the stool or in the toilet after having a bowel movement
- Dark or black stools
- A change in the shape of the stool (e.g., more narrow than usual)
- Cramping or discomfort in the lower abdomen
- An urge to have a bowel movement when the bowel is empty
- Constipation or diarrhea that lasts for more than a few days
- Decreased appetite
- Unintentional weight loss

In some cases, blood loss from the cancer leads to anemia (low number of red blood cells), causing symptoms such as weakness and excessive fatigue. Timely evaluation of symptoms consistent with colorectal cancer is essential, even for adults younger than age 50, among whom colorectal cancer incidence is rare, but increasing, and for whom screening is not recommended.

How many cases and deaths are estimated to occur in 2014?

Colorectal cancer will be diagnosed in about 71,830 men and 65,000 women in the US in 2014. While a similar number of men and women will be diagnosed with colon cancer (about 48,400), more men than women will be diagnosed with rectal cancer,

23,380 versus 16,620, respectively. An estimated 26,270 men and 24,040 women will die from colorectal cancer in 2014.

How many people alive today have been diagnosed with colorectal cancer?

As of January 1, 2012, there were almost 1.2 million Americans alive with a history of colorectal cancer.¹³ Some of these people were cancer-free, while others still had evidence of cancer and may have been undergoing treatment.

Who gets colorectal cancer?

Approximately 5%, or 1 in 20, Americans will be diagnosed with cancer of the colon or rectum in their lifetime.¹⁴

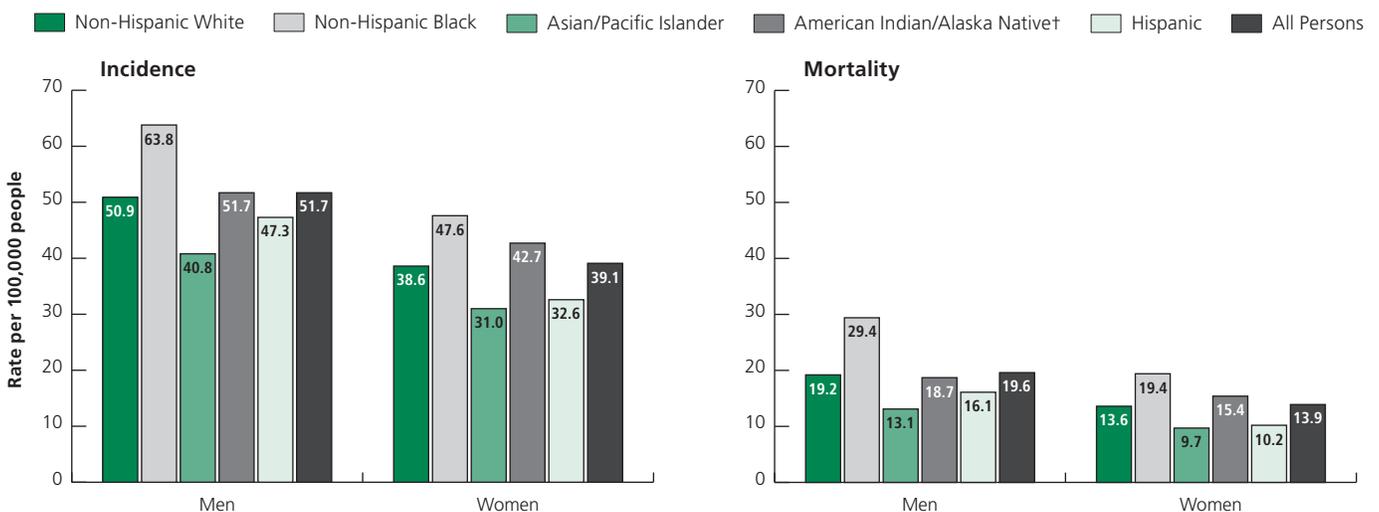
Age

Incidence and death rates for colorectal cancer increase with age. Overall, 90% of new cases and 93% of deaths occur in people 50 and older.^{15, 16} The median age at colon cancer diagnosis, 69 in men and 73 in women, is older than the median age at rectal cancer diagnosis, which is 63 in men and 65 in women.¹⁷

Sex

Overall, colorectal cancer incidence and mortality rates are about 30% to 40% higher in men than in women (Table 1, page 4). The reasons for this are not completely understood, but likely reflect complex interactions between gender-related differences in exposure to hormones and risk factors.¹⁸ Gender differences in risk patterns may also help explain why a larger proportion of tumors in women are located in the proximal colon, 45% versus 36% in men.¹⁶

Figure 3. Colorectal Cancer Incidence and Mortality Rates* by Race/Ethnicity and Sex, 2006-2010



*Rates are per 100,000, and age adjusted to the 2000 US standard population. †Statistics based on data from Contract Health Service Delivery Area (CHSDA) counties. Source: Incidence - Copeland et al.¹⁹ Mortality - Howlader et al.¹⁷

American Cancer Society, Surveillance Research, 2014

Table 1. Colorectal Cancer Incidence and Mortality Rates* by Race/Ethnicity and State, 2006-2010

	Non-Hispanic White				Non-Hispanic Black				Hispanic			
	Incidence		Mortality		Incidence		Mortality		Incidence		Mortality	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Alabama	55.3	38.5	20.5	13.5	68.0	48.2	33.0	21.2	25.5	24.9	†	†
Alaska	46.1	39.9	16.4	13.3	†	†	†	†	†	†	†	†
Arizona	40.7	32.0	17.1	12.1	44.9	37.8	26.5	13.8	41.7	29.2	18.0	10.8
Arkansas ^{‡,§}	54.2	39.0	22.0	14.5	70.6	53.0	32.3	24.6	43.5	†	†	†
California	49.9	38.4	17.9	13.5	62.9	51.1	27.0	20.5	45.0	30.7	15.8	9.4
Colorado	41.8	32.6	16.3	12.2	50.1	39.9	23.8	15.9	54.7	36.2	20.0	13.5
Connecticut	50.2	38.2	16.2	12.0	60.7	45.8	21.2	14.8	58.5	43.5	9.3	11.4
Delaware	53.5	39.5	21.2	13.1	58.4	42.2	18.7	15.4	†	†	†	†
Dist. of Columbia [¶]	30.5	29.4	11.0	13.2	62.6	51.2	30.4	22.0	34.6	†	†	†
Florida	46.2	35.7	17.7	12.6	55.7	41.9	25.1	17.3	53.0	37.4	17.5	11.8
Georgia	49.9	36.0	18.5	12.4	64.0	47.7	29.0	19.0	33.5	24.0	8.6	4.7
Hawaii	50.2	35.2	16.5	12.0	†	†	†	†	45.9	43.8	17.8	†
Idaho	43.3	34.5	16.5	12.5	†	†	†	†	32.9	25.1	†	†
Illinois	58.3	42.8	21.3	14.8	75.7	53.2	32.5	22.2	41.3	31.2	10.7	9.2
Indiana	54.1	40.9	21.0	14.2	65.9	51.3	30.1	20.2	33.8	34.6	10.9	9.2
Iowa	57.1	44.4	20.1	15.2	64.5	53.0	37.2	23.4	33.1	†	†	†
Kansas	54.2	38.5	20.3	13.0	73.0	54.8	32.1	20.2	49.2	43.3	10.9	11.4
Kentucky	63.7	45.6	23.3	16.0	74.4	54.0	30.1	22.7	32.2	24.2	†	†
Louisiana	60.0	41.1	21.6	14.2	75.4	53.8	34.1	20.6	38.6	31.5	†	9.9
Maine	51.4	41.2	20.2	13.8	†	†	†	†	†	†	†	†
Maryland	46.7	35.4	19.0	12.7	53.6	41.8	29.4	18.6	28.2	26.9	9.9	7.7
Massachusetts [#]	49.7	38.7	18.9	13.5	46.7	36.2	21.2	14.8	-	-	11.6	10.5
Michigan	48.7	37.7	18.5	13.7	64.6	48.1	27.9	18.7	44.3	29.2	19.0	11.8
Minnesota ^{**}	-	-	17.5	12.6	-	-	20.8	9.5	-	-	†	†
Mississippi	57.7	40.0	21.6	14.4	75.5	55.6	35.7	22.0	†	†	†	†
Missouri	54.0	39.6	20.7	14.2	76.0	52.0	32.2	20.7	36.7	35.3	†	†
Montana	49.6	37.3	16.2	13.4	†	†	†	†	†	†	†	†
Nebraska	57.6	43.9	21.6	14.8	76.6	58.1	34.1	29.0	45.0	30.9	†	†
Nevada ^{††}	53.3	39.1	22.1	15.5	54.7	48.2	23.3	21.1	42.8	31.0	14.9	10.2
New Hampshire	46.6	37.8	18.2	13.3	†	†	†	†	†	†	†	†
New Jersey	55.2	41.5	21.3	14.9	62.9	48.4	29.5	20.9	49.0	34.5	14.6	10.2
New Mexico	42.0	32.9	18.1	12.1	†	42.4	†	†	48.3	35.0	21.0	13.0
New York	52.4	41.0	18.8	13.6	59.7	44.6	25.3	17.0	54.6	35.5	16.5	11.4
North Carolina	49.4	35.5	18.1	12.2	62.8	45.2	28.8	18.0	27.8	22.9	9	†
North Dakota [¶]	58.9	41.2	21.0	13.3	†	†	†	†	†	†	†	†
Ohio ^{††}	54.3	41.3	21.2	15.0	63.6	45.4	30.5	18.5	48.2	33.9	12.6	8.6
Oklahoma	51.6	38.1	21.7	13.9	60.6	48.5	33.6	19.3	50.6	39.7	16.5	9.6
Oregon	44.5	36.4	18.4	13.4	57.5	41.2	†	†	40.1	26.4	12.6	7.8
Pennsylvania	55.4	42.3	21.0	14.9	62.2	46.2	32.4	18.3	55.0	33.3	15.9	6.6
Rhode Island	51.6	41.3	18.9	13.2	37.9	42.8	†	†	36.1	25.9	†	†
South Carolina [¶]	48.2	36.0	18.0	12.6	60.3	44.3	29.5	19.6	34.2	31.9	†	†
South Dakota	55.7	41.1	19.5	13.5	†	†	†	†	†	†	†	†
Tennessee	52.3	39.0	20.4	14.4	65.1	51.4	36.7	23.0	25.8	17.5	†	†
Texas	50.0	35.5	19.3	13.0	68.5	49.4	33.1	21.6	49.1	30.8	18.1	10.2
Utah	39.4	31.1	14.0	10.8	†	†	†	†	48.5	35.6	18.7	†
Vermont	45.1	38.4	17.3	14.7	†	†	†	†	†	†	†	†
Virginia [‡]	45.2	35.6	18.1	13.3	59.3	43.7	28.4	18.6	35.3	33.0	11.8	7.6
Washington	45.9	36.1	17.2	12.7	54.0	39.6	24.0	17.7	30.3	24.5	12.3	6.9
West Virginia	58.0	44.0	23.4	16.1	69.9	46.5	35.6	21.2	†	†	†	†
Wisconsin	47.2	36.6	17.9	12.6	71.3	49.1	26.5	20.9	34.9	31.9	†	†
Wyoming	46.5	37.8	18.3	14.5	†	†	†	†	56.8	†	†	†
US	50.9	38.6	19.2	13.6	63.8	47.6	29.4	19.4	47.3	32.6	16.1	10.2

*Rates are per 100,000 and age adjusted to the 2000 US standard population. †Statistic not displayed due to fewer than 25 cases or deaths. ‡This state's data are not included in US combined rates because they did not meet high-quality standards for one or more years during 2006-2010 according to NAACCR. §Rates are based on incidence data for 2006-2008. ¶Mortality rates for this state are not exclusive of Hispanic origin due to incomplete ethnicity data. #Information on Hispanic origin is not available for incidence data. **This state's registry did not submit 2006-2010 cancer incidence data to NAACCR. †† Rates are based on incidence data for 2006-2009.

Source: Incidence - North American Association of Central Cancer Registries (NAACCR), 2013. Mortality - National Center for Health Statistics, Centers for Disease Control and Prevention, 2013.

Race/ethnicity

Colorectal cancer rates are highest in black men and women and lowest in Asian/Pacific Islander (API) men and women (Figure 3, page 3).¹⁹ During 2006-2010, colorectal cancer incidence rates in blacks were about 25% higher than those in whites and about 50% higher than those in APIs. A larger disparity exists for colorectal cancer mortality, for which rates in blacks are about 50% higher than in whites and double those in APIs.

It is important to recognize that although cancer statistics are generally reported for broad racial and ethnic categories, the burden of colorectal cancer also varies greatly within these racial/ethnic groups. For example, American Indians/Alaska Natives (AI/ANs) living in Alaska have more than double the incidence rate of those living in New Mexico, 85.7 (per 100,000) versus 31.2, respectively.²⁰

Colorectal Cancer Occurrence

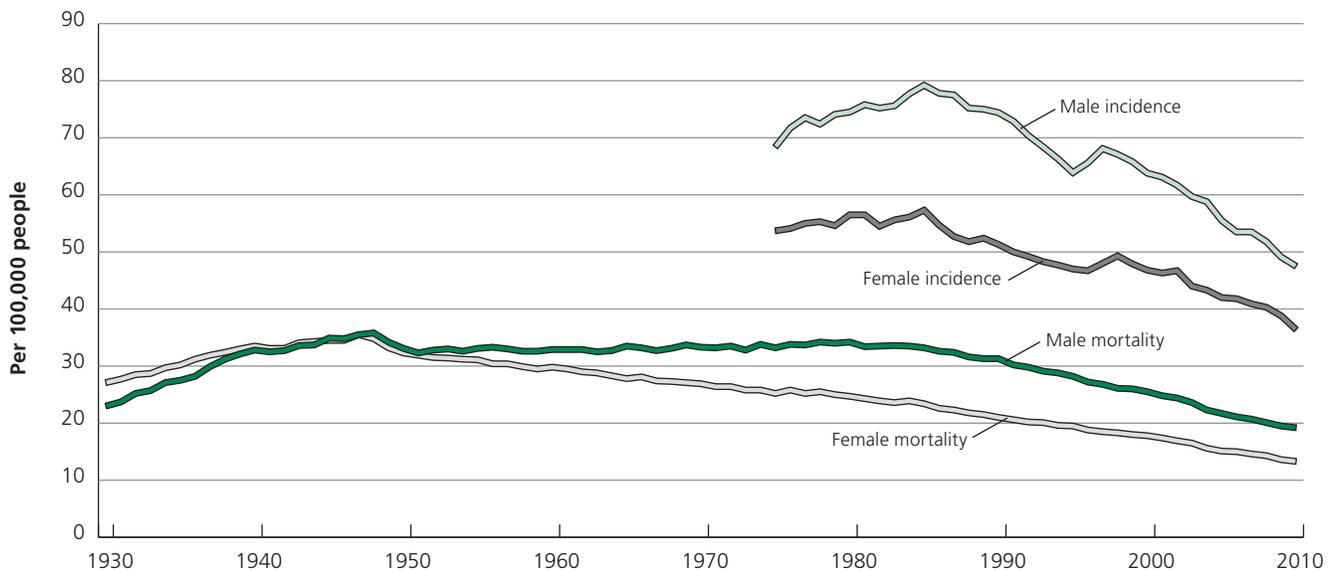
Changes over time

Incidence

Colorectal cancer incidence rates increased from 1975 through the mid-1980s, but have since decreased with the exception of a slight, unexplained bump in rates between 1996 and 1998 (Figure 4). Declines have accelerated during the past few years, such that from 2008 to 2010, incidence rates decreased by more than 4% per year in both men and women.¹⁷ The large declines over the past decade have largely been attributed to the detection and removal of precancerous polyps as a result of increased colorectal cancer screening.²¹

Figure 5 (page 6) presents trends in incidence rates by race and ethnicity. In the 1970s and 1980s, incidence rates were generally higher in white than black men and were similar in white and black women. However, since the late 1980s, rates have consistently been higher in blacks, among whom the downturn in incidence began later and was slower. This crossover likely reflects a combination of greater access to and utilization of colorectal cancer screening tests among whites, as well as racial differences in trends for colorectal cancer risk factors.²² Over the past decade of data (2001-2010), incidence rates declined a minimum of 1% per year among men and women of every major racial/ethnic group except AI/AN men, among whom rates were relatively stable.²³

Figure 4. Trends in Colorectal Cancer Incidence and Death Rates by Sex, US, 1930-2010



Rates were age adjusted to the 2000 US standard population. Incidence rates were adjusted for delays in reporting. Due to changes in International Classification of Diseases (ICD) coding for mortality, numerator information has changed over time.

Source: Incidence - Surveillance, Epidemiology, and End Results (SEER) Program, SEER 9 registries, National Cancer Institute, 2013.

Mortality - US Mortality Volumes 1930 to 1959, US Mortality Data 1960-2010, National Center for Health Statistics, Centers for Disease Control and Prevention, 2013.

American Cancer Society, Surveillance Research, 2014

Trends in colorectal cancer incidence rates also vary by age. Rates are declining among adults age 50 and older, but are increasing among those younger than 50 (Figure 6). This increase appears to be confined to cancers arising in the distal colon and rectum.²⁴ Reasons for this trend are unknown, but may reflect increased obesity prevalence and/or unfavorable dietary patterns in children and young adults.²⁵

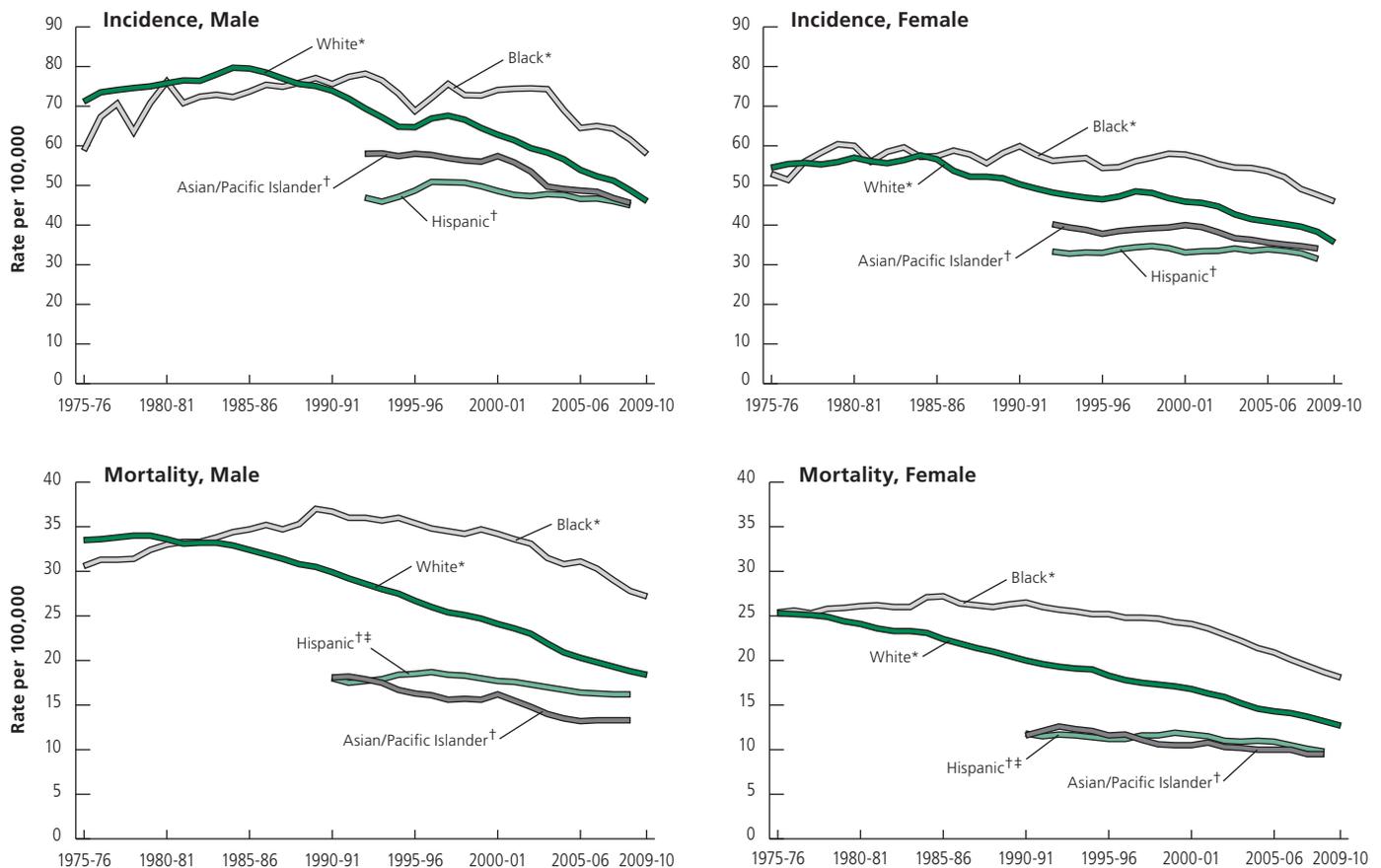
Mortality

Colorectal cancer death rates have been decreasing since 1980 in men and since 1947 in women (Figure 4, page 5). Declines since 1975 have been attributed to improvements in treatment (12%), changing patterns in colorectal cancer risk factors (35%), and screening (53%).²¹ Similar to incidence patterns, mortality rates declined most rapidly in the past decade. From 2001 to 2010, rates decreased by about 3% per year in both men and women, compared to declines of about 2% per year in the 1990s.¹⁷

Over the past three decades, there has been an increasing divergence in the mortality rates of blacks and whites (Figure 5). Prior to 1980, colorectal cancer mortality rates were lower in black than white men and similar among women of both races. However, the steep declines that began in whites in the early 1980s did not begin in blacks until the late 1990s. One study estimated that about half of this disparity can be attributed to the combined effect of less screening and lower stage-specific survival rates among blacks.²⁶ Although mortality rates in blacks remain substantially higher than those in whites, the gap has begun to shrink in recent years. From 2006 to 2010, annual declines in mortality rates were similar among black and white men (2.6% versus 2.5%) and slightly larger among black women than white women (3.3% versus 3.0%).¹⁷

From 2001 to 2010, colorectal cancer death rates decreased among men and women in every major racial/ethnic group except AI/ANs, among whom rates were stable.²³ The largest

Figure 5. Trends in Colorectal Cancer Incidence and Mortality Rates by Race/Ethnicity and Sex, 1975-2010



Trends for American Indians/Alaska Natives are not included due to sparse data. Rates are per 100,000 and age adjusted to the 2000 US standard population. *Rates are two-year moving averages. †Rates are three-year moving averages. ‡Rates exclude deaths from Connecticut, District of Columbia, Louisiana, Maine, Maryland, Minnesota, Mississippi, New Hampshire, New York, North Dakota, Oklahoma, South Carolina, Vermont, and Virginia due to incomplete ethnicity data.

Source: Incidence - Surveillance, Epidemiology, and End Results (SEER) Program, National Cancer Institute. Mortality - National Center for Health Statistics, Centers for Disease Control and Prevention, 2013.

American Cancer Society, Surveillance Research, 2014

declines (about 3% per year) occurred among white men and women and black women, while the smallest declines (about 1.5% per year) occurred among Hispanic men and API women.

Geographic differences in colorectal cancer rates

Colorectal cancer rates in the US vary widely by geographic area. Factors that contribute to this disparity include regional variations in risk factors and access to screening and treatment, which are influenced by socioeconomic factors, legislative policies, and proximity to medical services. The geographic pattern of colorectal cancer has changed dramatically over the past several decades. In contrast to the 1950s and 1960s, when rates were highest in the Northeast and lowest in the Southeast, rates are currently highest in the Midwest and mid-South and lowest in the Northeast.^{27, 28}

Table 1 (page 4) shows colorectal cancer incidence and death rates per 100,000 people by state and race/ethnicity. Although gender patterns are similar across states, there is much larger variation in incidence rates among men than women. For example, among both white men and women, rates are lowest in the District of Columbia and highest in Kentucky; however, in men rates range from 30.5 to 63.7, whereas in women the range is 29.4 to 45.6.

Geographic patterns of colorectal cancer mortality are generally similar for blacks and whites based on states for which there are a sufficient number of deaths to calculate rates (Figure 7, page 8). However, as previously noted, rates are substantially higher among blacks. For example, the highest age-adjusted state mortality rates for black men are more than 50% higher than those for white men.

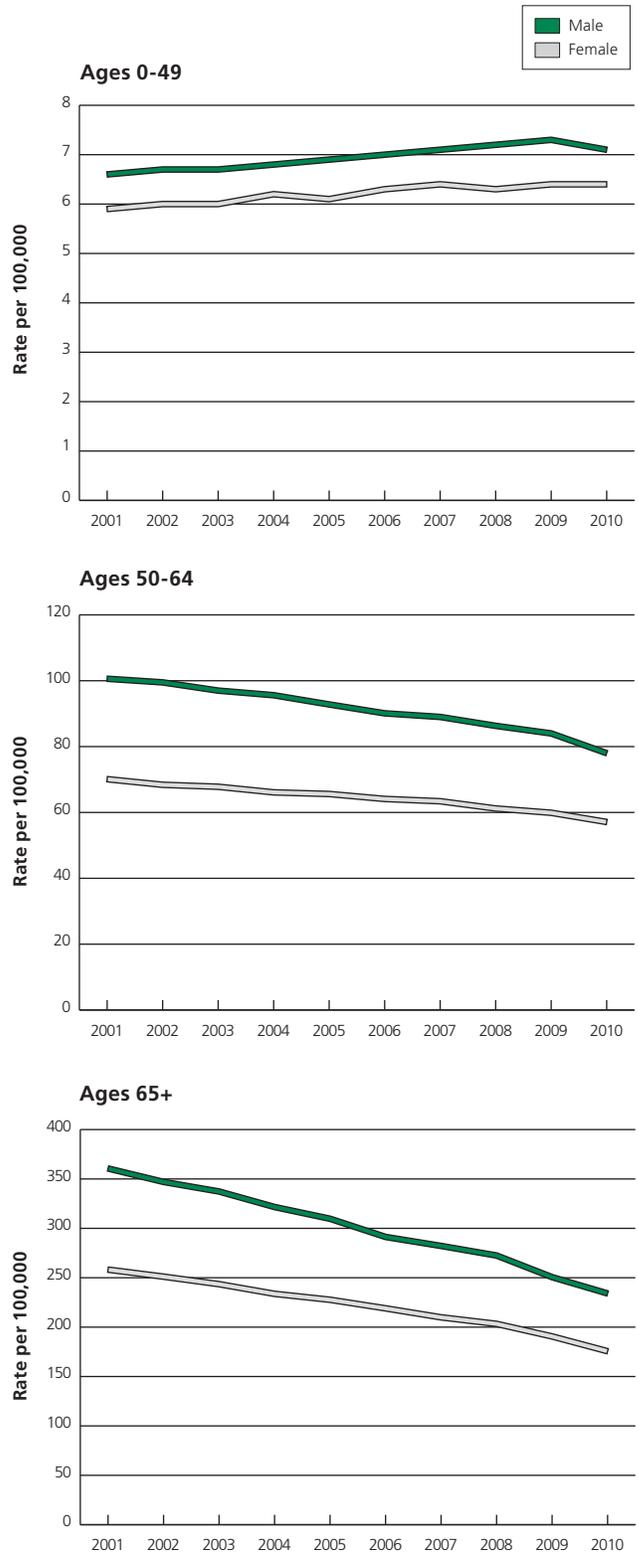
Stage distribution and cancer survival

The relative survival rate for colorectal cancer is 65% at 5 years following diagnosis and 58% at 10 years.¹⁷ Only 40% of colorectal cancer patients are diagnosed with localized-stage disease, for which the 5-year survival rate is 90%; survival declines to 70% and 13% for patients diagnosed with regional and distant stages, respectively.

Non-Hispanic whites are the most likely of all racial/ethnic groups to be diagnosed with colorectal cancer at a localized stage, when treatment is most successful (Figure 8, page 9). APIs are the most likely to survive 5 years after a colorectal cancer diagnosis (Figure 9, page 9).

Factors that contribute to disparities in colorectal cancer survival include differences in access to early detection tests, receipt of timely and high-quality treatment, and the prevalence of comorbidities (other illnesses).^{23, 29-31} Many studies have found that colorectal cancer patients who are black are less

Figure 6. Colorectal Cancer Incidence Trends by Age and Sex, 2001-2010



Rates are age adjusted to the 2000 US standard population.

Source: North American Association of Central Cancer Registries (NAACCR), 2013.

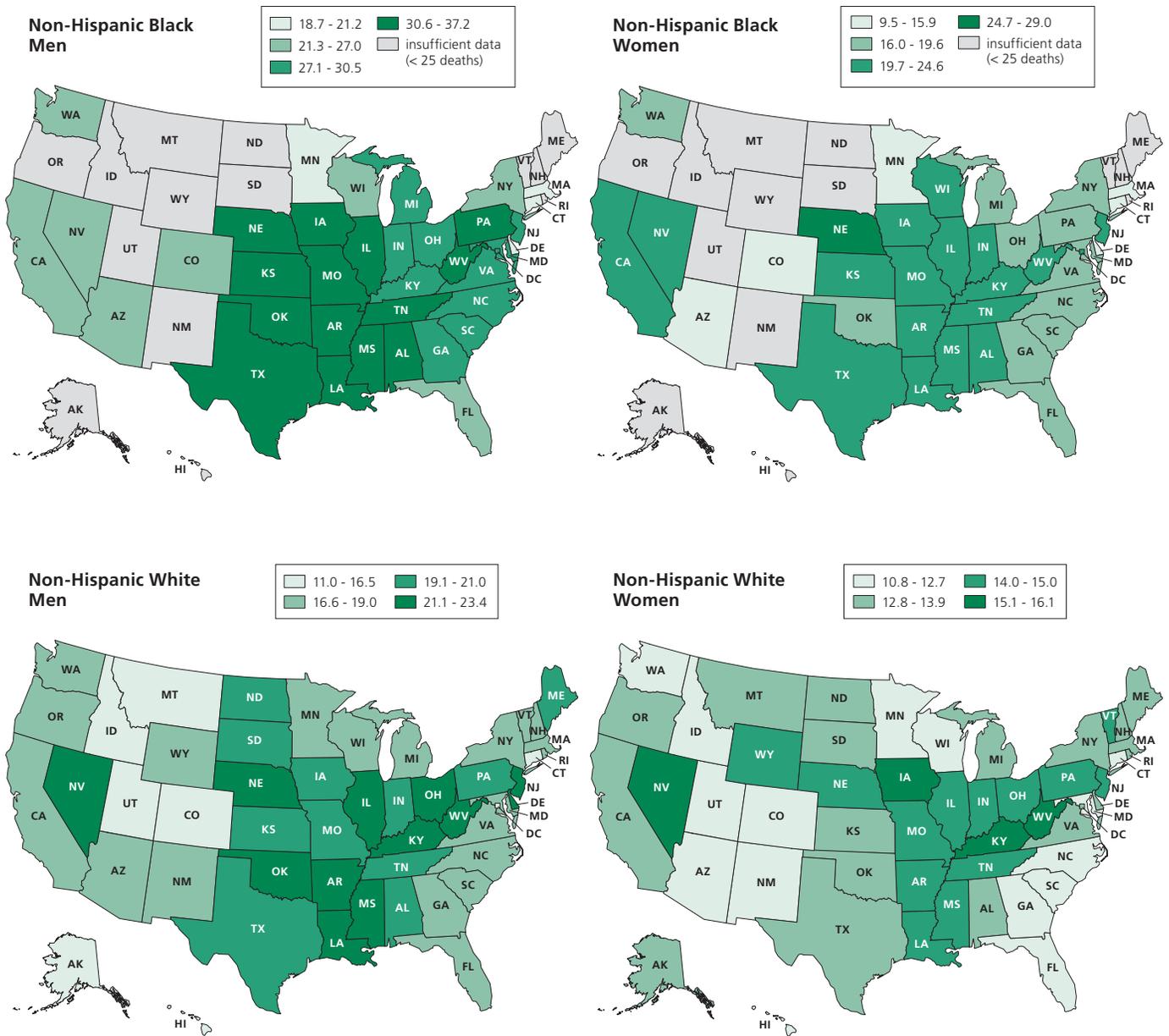
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likely than other patients to receive appropriate surgery, adjuvant chemotherapy, and radiation treatments.³²⁻³⁶ Importantly, compared to whites, black patients who receive chemotherapy experience a similar survival benefit with fewer negative side effects.^{37, 38} Survival differences largely disappear when cancer treatment and clinical care are comparable for similarly staged disease.²⁹ Survival disparities are largely driven by socioeco-

nommic inequalities and are evident within as well as between racial and ethnic groups. For example, blacks who are privately insured are 46% more likely to survive five years after a colorectal cancer diagnosis than blacks who are uninsured.³⁹

Since the mid-1970s, the 5-year relative survival rate has increased from 51% to 65% for colon cancer and from 48% to

Figure 7. Colorectal Cancer Death Rates* by State, Race, and Sex, US, 2006-2010

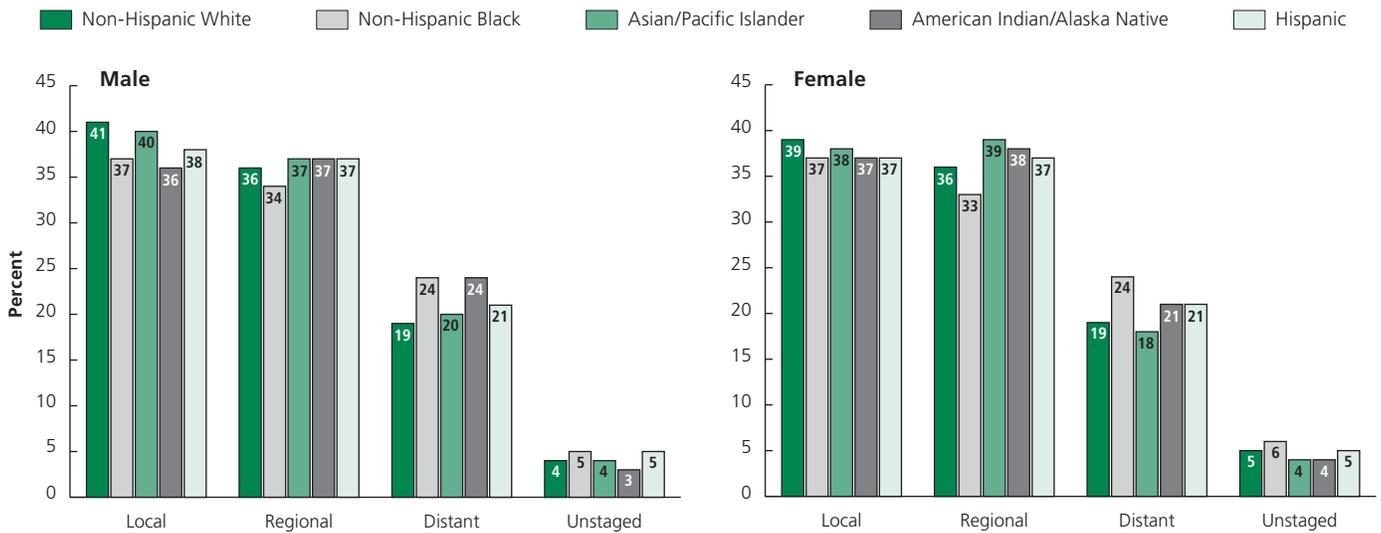


*Per 100,000 and age adjusted to the 2000 US standard population.
Source: National Center for Health Statistics, Centers for Disease Control and Prevention, 2013.

68% for rectal cancer.¹⁷ Rectal cancer is diagnosed at a localized stage more often than colon cancer (44% versus 38%, respectively), which probably contributes to the higher overall survival for rectal cancer. The largest improvement in 5-year survival has been for regional-stage disease, from 55% to 73% for colon can-

cer and from 45% to 69% for rectal cancer. This is likely due to the significant progress in treatment for these patients, namely 5-fluorouracil-based chemotherapy following surgery, which was recommended by a National Institutes of Health expert panel in 1990 for stage III cancers.^{40, 41}

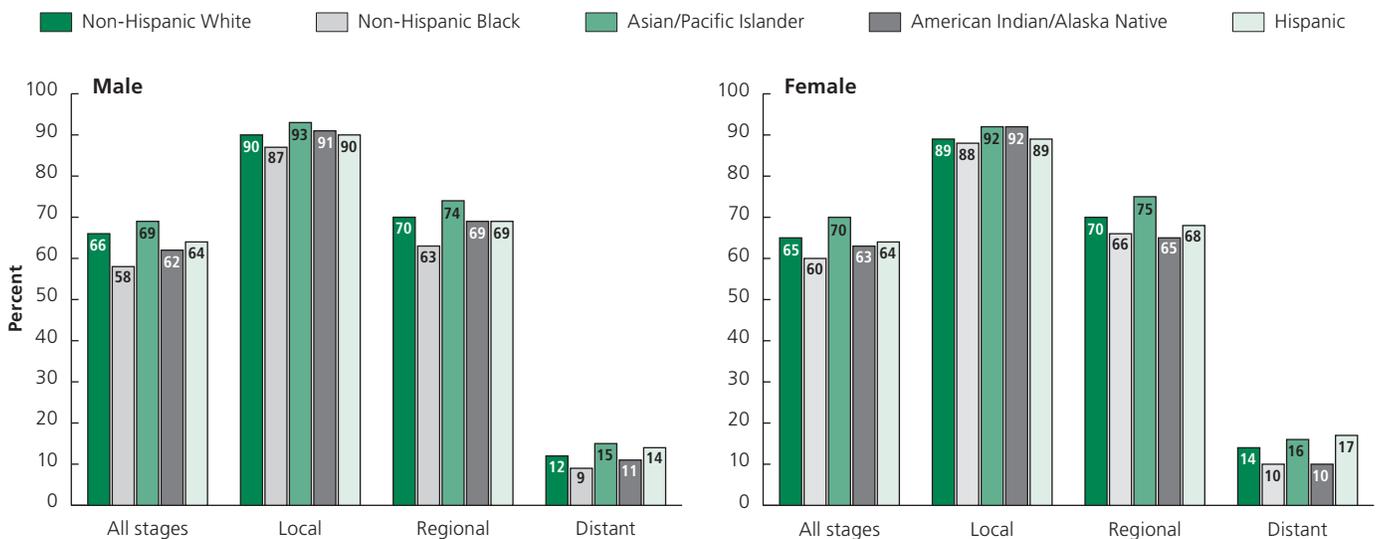
Figure 8. Colorectal Cancer Stage Distribution (%) by Race/Ethnicity, 2003-2009



Source: Surveillance, Epidemiology, and End Results (SEER) Program, SEER 18 registries, National Cancer Institute, 2013.

American Cancer Society, Surveillance Research, 2014

Figure 9. Five-year Colorectal Cancer-specific Survival* by Stage and Race/Ethnicity, 2003-2009



*The probability of not dying from colorectal cancer within 5 years of diagnosis. Patients were followed through 2010.

Source: Surveillance, Epidemiology, and End Results (SEER) Program, SEER 18 registries, National Cancer Institute, 2013.

American Cancer Society, Surveillance Research, 2014

Colorectal Cancer Risk Factors

There are many known factors that increase or decrease the risk of colorectal cancer (Table 2); some of these factors are modifiable while others are not. Nonmodifiable risk factors include a personal or family history of colorectal cancer or adenomatous polyps and a personal history of chronic inflammatory bowel disease. The American Cancer Society and other organizations recommend that some people at increased risk for colorectal cancer because of these conditions begin screening at an earlier age. (For more information on recommended colorectal cancer screening for individuals with increased risk, please see page 16.) Epidemiologic studies have also identified many modifiable risk factors for colorectal cancer. These include physical inactivity, obesity, high consumption of red and/or processed meats, smoking, and moderate-to-heavy alcohol consumption.⁴²

Heredity and family history

People with a first-degree relative (parent, sibling, or offspring) who has had colorectal cancer have 2 to 3 times the risk of developing the disease compared to individuals with no family history. If the relative was diagnosed at a young age or if there is more than one affected relative, risk increases to 3 to 6 times that of the general population.^{43, 44} About 20% of all colorectal cancer patients have a close relative who was diagnosed with the disease.⁴⁵ A family history of colorectal cancer is associated with better disease survival, perhaps due to increased awareness and earlier detection.⁴⁶

About 5% of patients with colorectal cancer have a well-defined genetic syndrome that causes the disease.⁴⁵ The most common of these is Lynch syndrome (also known as hereditary nonpolyposis colorectal cancer). Approximately 1 in 35 colorectal cancer patients has Lynch syndrome.⁴⁷ Although individuals with Lynch syndrome are predisposed to many types of cancer (e.g., endometrial, stomach, and ovarian), risk of colorectal cancer is highest.^{48, 49} A study of colorectal cancer in 147 Lynch syndrome families in the US found lifetime risks of 66% in men and 43% in women. The median age at diagnosis was 42 and 47, respectively, compared to 67 and 71, respectively, in the general population.⁴⁸ There is growing interest in improving methods for identifying Lynch syndrome among colorectal cancer patients in order to increase opportunities for cancer prevention.⁵⁰⁻⁵² In addition to prevention through screening, there is evidence to support chemoprevention among these high-risk patients.⁵³ A randomized clinical trial recently demonstrated 63% fewer colon cancers among Lynch syndrome patients who took daily aspirin (600 mg).⁵⁴

Familial adenomatous polyposis (FAP) is the second most common predisposing genetic syndrome, and is characterized by the development of hundreds to thousands of colorectal polyps in affected individuals.⁵⁵ Without intervention, the lifetime risk of colorectal cancer approaches 100% by age 40.⁵⁶ Although accurate identification of families with a history of colorectal cancer and/or a predisposing genetic abnormality is necessary so testing can begin at an early age, studies have shown that documentation of family cancer history in medical records is lacking in half of primary care patients.^{57, 58}

Personal medical history

People with a personal history of colorectal cancer are more likely to develop a subsequent cancer in the colon or rectum. A younger age at diagnosis is associated with higher risk.⁵⁹ The magnitude of the risk also varies by the anatomic location of the primary tumor.⁶⁰

A history of adenomatous polyps also increases the risk of colorectal cancer. This is especially true if the polyps were large or if there was more than one.⁸ A family history of adenomas appears to increase risk, though more research is needed in this area.⁶¹

People who have chronic inflammatory bowel disease, a condition in which the colon is inflamed over a long period of time, have a higher risk of developing colorectal cancer that increases with the extent and duration of disease.⁶² The most common forms of inflammatory bowel disease are ulcerative colitis and Crohn disease. It is estimated that 18% of patients with a 30-year history of ulcerative colitis will develop colorectal cancer.⁶³ However, there is some evidence that cancer risk in these patients may be lower in recent years due to improved disease management (through the use of medications to control inflammation) and the use of screening to detect premalignant lesions.⁶⁴⁻⁶⁶

Many studies have found that patients with diabetes have an increased risk of colorectal cancer.⁶⁷⁻⁶⁸ Though adult onset (Type 2) diabetes (the most common type) and colorectal cancer share similar risk factors, including obesity and a sedentary lifestyle, this association remains even after accounting for physical activity, body mass index, and waist circumference.⁶⁹ Studies suggest that the relationship may be stronger in men than in women.^{68, 70} A growing body of research indicates that some diabetic medications independently affect colorectal cancer risk.⁷¹ In general, colorectal cancer patients with diabetes appear to have slightly poorer survival than non-diabetic patients.⁷²

Behavioral risk factors

Physical inactivity

One of the most consistently reported behavioral factors related to colon cancer risk is physical activity. A recent review of the scientific literature found that the most physically active people

have a 25% lower risk of colon cancer than the least active people.⁷³ Conversely, colorectal cancer patients who are less active have a higher risk of colorectal cancer death than those who are more active.⁷⁴ In addition, epidemiologic studies find that:

- The more physically active people are, the lower their risk of colon cancer.
- Both recreational and occupational physical activity decrease risk.⁷⁵
- Sedentary people who become active later in life may reduce their risk.⁷⁶

Based on these findings, as well as the numerous other health benefits of regular physical activity, the American Cancer Society and the Centers for Disease Control and Prevention recommend engaging in at least 150 minutes of moderate-intensity activity or 75 minutes of vigorous-intensity activity each week (or a combination of these), preferably spread throughout the week. In 2012, only about half of US adults met these physical activity guidelines.⁷⁷

Overweight and obesity

Being overweight or obese is associated with a higher risk of colorectal cancer in men and colon cancer in women, with stronger associations more consistently observed in men than in women.⁷⁸ Overweight and obesity increase risk of colorectal cancer independent of physical activity.⁷⁹ Abdominal obesity (measured by waist size) appears to be a more important risk factor than overall obesity in both men and women.^{80, 81} The prevalence of obesity among US adults increased from 19% in 1997 to 29% in 2012.⁷⁷

Diet

Geographic differences in colorectal cancer rates and temporal changes in risk among immigrant populations suggest that diet and lifestyle strongly influence the occurrence of colorectal cancer. Although research is still accumulating on the role of specific dietary elements on colorectal cancer risk, current evidence indicates that:

- High consumption of red and/or processed meat increases the risk of both colon and rectal cancer.⁸² The reasons for this association remain unclear, but may be related to carcinogens (cancer-causing substances) that form when red meat is cooked at a high temperature for a long period of time and/or nitrite additives for food preservation.⁸³
- Intake of dietary fiber, cereal fiber, and whole grains is associated with a reduced risk of colorectal cancer.⁸⁴ Specifically, for every 10 grams of daily fiber consumption there is a 10% reduction in cancer risk.
- Moderate daily fruit and vegetable intake is slightly protective against colon (but not rectal) cancer compared to low consumption; very high consumption appears to add little additional benefit.^{85, 86}

Table 2. Summary of Selected Risk Factors for Colorectal Cancer

	Relative Risk*
Factors that increase risk:	
Heredity and Medical History	
Family history	
1 first-degree relative ⁴³	2.2
more than 1 relative ⁴³	4.0
relative with diagnosis before age 45 ⁴⁴	3.9
Inflammatory bowel disease ^{† 62}	
Crohn disease (colon)	2.6
Ulcerative colitis	
colon	2.8
rectum	1.9
Diabetes ⁴²	1.2
Behavioral factors⁴²	
Alcohol consumption (heavy vs. nondrinkers)	1.6
Obesity	1.2
Red meat consumption	1.2
Processed meat consumption	1.2
Smoking (current vs. never)	1.2
Factors that decrease risk:	
Physical activity (colon) ⁷³	0.7
Dairy consumption ⁸⁷	0.8
Fruit consumption ⁸⁵	0.9
Vegetable consumption ⁸⁵	0.9
Total dietary fiber (10 g/day) ⁸⁴	0.9

*Relative risk compares the risk of disease among people with a particular "exposure" to the risk among people without that exposure. Relative risk for dietary factors compares the highest with the lowest consumption. If the relative risk is more than 1.0, then risk is higher among exposed than unexposed persons. Relative risks less than 1.0 indicate a protective effect.

†Several recent, small studies indicate that current risk may be lower due to improvements in treatment and the use of colonoscopy screening to detect precancerous lesions.

- Higher consumption of total dairy products, milk, and calcium decreases the risk of developing colorectal cancer.⁸⁷ This protective effect appears to be irrespective of milk fat content.⁸⁸
- Higher blood levels of vitamin D are associated with slightly lower risk of developing colorectal cancer compared to low blood levels.⁸⁹
- Dietary folate intake appears to decrease colorectal cancer risk.⁹⁰ There is some evidence that folic acid (the form of folate used in supplements and fortification) promotes cancer growth, leading to the hypothesis that increased folate levels among Americans as a result of mandatory fortification of enriched flour and cereals in 1998 was responsible for the unexplained uptick in colorectal cancer incidence rates in the late 1990s (Figure 4, page 5).^{91, 92} However, a recent analysis of data from the American Cancer Society Cancer Prevention Study-II confirmed the inverse association between total dietary folate and colorectal cancer reported in previous

Reduce your risk of colorectal cancer.

1. Get screened regularly.
2. Maintain a healthy weight throughout life.
3. Adopt a physically active lifestyle.
4. Consume a healthy diet with an emphasis on plant sources; specifically:
 - Choose foods and beverages in amounts that help achieve and maintain a healthy weight.
 - Limit consumption of red and processed meat.
 - Eat at least 2½ cups of vegetables and fruits each day.
 - Choose whole grains instead of refined grain products.
5. If you drink alcoholic beverages, limit consumption.
6. Consume the recommended levels of calcium, primarily through food sources
7. Avoid tobacco products.

studies and found no evidence that fortification or supplementation increased cancer risk.⁹³

Thus, research suggests that following the Society's dietary recommendations, which include limiting consumption of red and processed meats; eating a variety of vegetables and fruits each day; and choosing whole grains instead of refined grain products, will help reduce the risk of developing colorectal cancer. Consuming the recommended levels of calcium may also help lower risk.

Smoking

In November 2009, the International Agency for Research on Cancer reported that there is sufficient evidence to conclude that tobacco smoking causes colorectal cancer.⁹⁴ The association appears to be stronger for rectal than for colon cancer and for particular molecular subtypes of colorectal cancer.⁹⁵⁻⁹⁸ It is

thought that early studies failed to detect this association because of a particularly long latency period – at least three to four decades – between tobacco exposure and colorectal cancer diagnosis.

Alcohol

Colorectal cancer has been linked to moderate and heavy alcohol use.^{99, 100} People who have a lifetime average of 2 to 4 alcoholic drinks per day have a 23% higher risk of colorectal cancer than those who consume less than 1 drink per day.¹⁰⁰

Medications

There is extensive evidence that long-term, regular use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) lowers risk of colorectal cancer.¹⁰¹⁻¹⁰⁴ The American Cancer Society does not currently recommend use of these drugs for cancer prevention in the general population because of the potential side effects of gastrointestinal bleeding from aspirin and other traditional NSAIDs or heart attack from selective COX-2 inhibitors (a type of NSAID commonly used to treat arthritis). However, people who are already taking NSAIDs for other medical conditions may have a lower risk of colorectal cancer as a side benefit.

There is evidence that women who use postmenopausal hormones have lower rates of colorectal cancer than those who do not.^{105, 106} Decreased risk is especially evident in women with long-term hormone use, though risk returns to that of nonusers within three years of cessation.^{107, 108} However, use of postmenopausal hormones increases risk for breast and other cancers, as well as cardiovascular disease, so it is not recommended for the prevention of colorectal cancer.¹⁰⁵ Studies suggest that oral contraceptive use may also be associated with a slightly decreased risk.¹⁰⁹ Recent studies suggest that oral bisphosphonates, which are used to treat and prevent osteoporosis, may also reduce risk.¹¹⁰

At present, the American Cancer Society does not recommend any medications or supplements to prevent colorectal cancer because of uncertainties about effectiveness, appropriate dosage, and potential toxicity.

Colorectal Cancer Screening

The slow course of growth from precancerous polyp to invasive cancer provides a unique opportunity for the prevention and early detection of colorectal cancer.⁵ Screening can prevent cancer through the detection and removal of precancerous growths, as well as detect cancer at an early stage, when treatment is more successful. As a result, screening reduces colorectal cancer mortality both by decreasing the incidence of disease and by increasing the likelihood of survival.

Recommended options for colorectal cancer screening

In 2008, the American Cancer Society collaborated with the American College of Radiology and the US Multi-Society Task Force on Colorectal Cancer (a consortium representing the American College of Gastroenterology, the American Society of Gastrointestinal Endoscopy, the American Gastroenterological

Association, and representation from the American College of Physicians) to publish consensus guidelines for colorectal cancer screening.¹¹¹ The leadership of these organizations believed that a single set of jointly developed and promoted recommendations would highlight their importance and promote evidence-based practice. The guidelines draw a distinction between screening tests that primarily detect cancer (stool tests) and those that are more likely to detect both cancer and precancerous growths (structural exams that visualize part or all of the large bowel, such as flexible sigmoidoscopy and colonoscopy). The recommendations emphasize that cancer prevention should be the primary goal of colorectal cancer screening. To achieve this goal, exams that are designed to detect both early cancer and precancerous polyps should be encouraged if resources are available and patients are willing to undergo an invasive test. The higher likelihood of polyp detection with the use of these tests substantially increases opportunities for polyp removal and colorectal cancer prevention.

The following options are recommended for colorectal cancer screening in men and women age 50 and older at average risk (summarized in Table 3, page 14):

Tests that are more likely to detect both adenomatous polyps and cancer

Flexible sigmoidoscopy: A slender, flexible, hollow, lighted tube is inserted through the rectum into the colon by a trained examiner. The sigmoidoscope is about 2 feet long (60 cm) and provides a visual examination of the rectum and lower one-third of the colon (sigmoid colon).¹¹¹ Simple bowel cleansing, usually with enemas, is necessary to prepare the colon, and the procedure is typically performed without sedation. If there is a polyp or tumor present, the patient is referred for a colonoscopy so that the entire colon can be examined.

Analysis of data from clinical trials, in which participants are invited to screening (but don't necessarily comply), indicates that sigmoidoscopy is associated with a 21% reduction in colorectal cancer incidence and a 26% reduction in colorectal cancer mortality.¹¹² Cohort studies based on patient self-reported screening history find larger benefits (e.g., 41% reduction in colorectal cancer mortality).¹¹³ A randomized clinical trial in the United Kingdom reported that among participants who completed a single sigmoidoscopy screening between the ages of 55 and 64, colorectal cancer incidence was reduced by 33% and mortality by 43%.¹¹⁴

Colonoscopy: Like sigmoidoscopy, this procedure allows for direct visual examination of the colon and rectum. A colonoscope is similar to a sigmoidoscope, but is a much longer, more complex instrument, allowing visualization of the entire colon and removal of polyps. Before undergoing a colonoscopy, patients are instructed to take special laxative agents to completely cleanse the colon. Sedation is usually provided during

the examination to minimize discomfort.¹¹¹ If a polyp is found, it may be removed during the procedure.

Studies show that colonoscopy is the most sensitive method for the detection of colorectal cancer or adenomatous polyps.¹¹⁵ A recent analysis of data from the National Polyp Study found that patients who had adenomas removed during colonoscopy (with follow-up colonoscopy at one or three years) had a 53% lower risk of death from colorectal cancer than the general population.¹¹⁶ Colonoscopy also has the longest rescreening interval of all forms of testing; if normal, the exam does not need to be repeated for 10 years in average-risk patients.

However, colonoscopy does have limitations. The procedure misses approximately 20% of all adenomas and 10% of large (5 mm or larger) or advanced adenomas.¹¹⁷ Colonoscopy also has a higher risk of complications compared to other screening tests, including bowel tears and bleeding, especially when a polyp is removed.¹¹¹

Barium enema with air contrast: Use of this procedure, which is also called double-contrast barium enema (DCBE), has become very uncommon due to the increased availability of colonoscopy, changing patient and physician preferences, a limited number of radiologists adequately trained to perform the procedure, and lower insurance reimbursement. Barium sulfate is introduced into a cleansed colon through the rectum to partially fill and open the colon. Air is then introduced to expand the colon and increase the quality of x-rays that are taken. This method is less sensitive than colonoscopy for visualizing small polyps or cancers. If a polyp or other abnormality is seen, the patient should be referred for a colonoscopy so that the colon can be examined further.

Computed tomographic colonography (CTC): Also referred to as virtual colonoscopy, this imaging procedure was introduced in the 1990s and results in detailed, cross-sectional, 2- or 3-dimensional views of the entire colon and rectum with the use of a special x-ray machine linked to a computer.¹¹¹ Although a full bowel cleansing is necessary for a successful examination, sedation is not required. A small, flexible tube is inserted into the rectum in order to allow air or carbon dioxide to open the colon; then the patient passes through the CT scanner, which creates multiple images of the interior colon. CTC is less invasive than other structural exams, requires no recovery time, and typically takes approximately 10 to 15 minutes to complete. Patients with polyps of significant size (larger than 5 mm) or other abnormal results are referred for colonoscopy, optimally on the same day in order to alleviate the necessity of a second bowel preparation. Studies have shown that the performance of CTC is similar to optical colonoscopy for the detection of invasive cancer and polyps approximately 1 cm or larger in size.^{118, 119}

Table 3. Considerations When Deciding with Your Doctor Which Test Is Right for You:

Benefits	Performance & Complexity*	Limitations	Test Time Interval
Structural Exams			
Flexible Sigmoidoscopy			
<ul style="list-style-type: none"> Fairly quick Few complications Minimal bowel preparation Does not require sedation or a specialist 	<p>Performance: High for rectum & lower one-third of the colon</p> <p>Complexity: Intermediate</p>	<ul style="list-style-type: none"> Views only one-third of colon Cannot remove large polyps Small risk of infection or bowel tear Slightly more effective when combined with annual fecal occult blood testing Colonoscopy still needed if abnormalities are detected Limited availability 	5 years
Colonoscopy			
<ul style="list-style-type: none"> Examines entire colon Can biopsy and remove polyps Can diagnose other diseases Required for abnormal results from all other tests 	<p>Performance: Highest</p> <p>Complexity: Highest</p>	<ul style="list-style-type: none"> Full bowel preparation needed Can be expensive Sedation of some kind usually needed, necessitating a chaperone to return home Patient may miss a day of work. Highest risk of bowel tears or infections compared with other tests 	10 years
Double-contrast Barium Enema			
<ul style="list-style-type: none"> Can usually view entire colon Few complications No sedation needed 	<p>Performance: High (for large polyps)</p> <p>Complexity: High</p>	<ul style="list-style-type: none"> Full bowel preparation needed Some false positive test results Cannot remove polyps or perform biopsies Exposure to low-dose radiation Colonoscopy necessary if abnormalities are detected Very limited availability 	5 years
Computed Tomographic Colonography			
<ul style="list-style-type: none"> Examines entire colon Fairly quick Few complications No sedation needed Noninvasive 	<p>Performance: High (for large polyps)</p> <p>Complexity: Intermediate</p>	<ul style="list-style-type: none"> Full bowel preparation needed Cannot remove polyps or perform biopsies Exposure to low-dose radiation Colonoscopy necessary if abnormalities are detected Not covered by all insurance plans 	5 years
Stool Tests (Low-sensitivity stool tests, such as single-sample FOBT done in the doctor's office or toilet bowl tests, are not recommended)			
High-Sensitivity Guaiac-based Fecal Occult Blood Test (FOBT)			
<ul style="list-style-type: none"> No bowel preparation Sampling is done at home Low cost Noninvasive 	<p>Performance: Intermediate for cancer</p> <p>Complexity: Low</p>	<ul style="list-style-type: none"> Requires multiple stool samples Will miss most polyps May produce false-positive test results Pre-test dietary limitations Slightly more effective when combined with a flexible sigmoidoscopy every five years Colonoscopy necessary if abnormalities are detected 	Annual
Fecal Immunochemical Test (FIT)			
<ul style="list-style-type: none"> No bowel preparation Sampling is done at home Low cost Noninvasive 	<p>Performance: Intermediate for cancer</p> <p>Complexity: Low</p>	<ul style="list-style-type: none"> Requires multiple stool samples Will miss most polyps May produce false-positive test results Slightly more effective when combined with a flexible sigmoidoscopy every five years Colonoscopy necessary if abnormalities are detected 	Annual
Stool DNA Test			
<ul style="list-style-type: none"> No bowel preparation Sampling is done at home Requires only a single stool sample Noninvasive 	<p>Performance: Intermediate for cancer</p> <p>Complexity: Low</p>	<ul style="list-style-type: none"> Will miss most polyps High cost compared to other stool tests New technology with uncertain interval between testing Colonoscopy necessary if abnormalities are detected 	Uncertain

*Complexity involves patient preparation, inconvenience, facilities and equipment needed, and patient discomfort.

Tests that are primarily effective at detecting cancer

Although high-sensitivity stool tests will detect some precancerous polyps, the potential for prevention is both limited and incidental and cannot be the primary goal of screening with these tests. Modeling studies show that annual screening with high-sensitivity stool tests results in a mortality benefit comparable to structural exams (e.g., colonoscopy, sigmoidoscopy, or CTC), though adherence to yearly testing is a challenge in the community setting.¹²⁰⁻¹²²

Fecal occult blood test (FOBT): Cancerous tumors and some large polyps bleed intermittently into the intestine. This blood can be detected in stool by the FOBT kit, which is obtained from a health care provider for use at home. Bleeding from colorectal cancer may be intermittent or undetectable, so accurate test results require annual testing that consists of collecting 1 to 3 samples (depending on the product) from consecutive bowel movements.

There are two types of FOBT available – guaiac-based tests, which detect blood from any source (including meat in the diet), and immunochemical-based tests, which detect only human blood from the large bowel. While there are numerous guaiac-based tests available, the American Cancer Society recommends only high-sensitivity tests (e.g., Hemoccult Sensa, etc.) for colorectal cancer screening.¹²³ For guaiac-based FOBT (gFOBT), people are instructed to avoid nonsteroidal anti-inflammatory drugs and red meat for 3 days prior to the test because they can lead to false positive results. Vitamin C and large amounts of citrus juices should also be avoided because they can lead to false negative test results. Six samples from 3 consecutive bowel movements are collected by smearing the stool sample thinly on a special card.¹¹¹ The fecal immunochemical test (FIT) is more convenient because it does not require special dietary restrictions and usually requires the collection of fewer stool samples. A recent clinical trial comparing three different FOBT kits found that patients were more likely to comply with FIT than gFOBT testing.¹²⁴

Upon completing either of these tests, patients return the kit to their doctor or to a laboratory for evaluation. Patients who have a positive gFOBT or FIT are referred for a colonoscopy to rule out the presence of polyps or cancer. Recently reported data from a large clinical trial indicated that the regular use of FOBT reduced the risk of death from colorectal cancer by 32% after 30 years of follow up.¹²⁵ In addition, FOBT has been shown to decrease the incidence of colorectal cancer by 20% by detecting large precancerous polyps.¹²⁶ It is important to emphasize that the effectiveness of FOBT is dependent on repeated screenings over time.

Often during the course of an exam in a physician's office, a single stool sample is collected during a digital rectal exam and placed on an FOBT card for colorectal cancer screening.

Despite the lack of endorsement for this form of testing by any organization, and many specifically recommending against it, the in-office FOBT is still performed by as many as one-third of primary care physicians.¹²⁷ One national survey of physicians who reported performing FOBT found that one-quarter of practitioners used only the single-specimen in-office test and more than half (53%) used both the in-office and home tests.¹²⁸ The single-sample FOBT is not a recommended screening test for colorectal cancer because it performs poorly in its ability to detect the disease. In one large study, this form of testing missed 95% of precancerous polyps and cancers that were revealed by subsequent colonoscopy.¹²⁹

Table 4. Colorectal Cancer Screening (%) among Adults Age 50 and Older in the US, 2010

	FOBT*	Endoscopy†	Either FOBT or Endoscopy‡
Gender			
Men	9.0	57.4	60.2
Women	8.6	55.6	58.3
Age (years)			
50-64	8.0	52.3	55.2
65+	9.7	61.2	63.7
Race/Ethnicity			
White (non-Hispanic)	9.2	58.5	61.5
Black (non-Hispanic)	8.4	53.0	55.5
Asian§	6.9	44.5	45.9
American Indian/ Alaskan Native¶	6.1	46.5	48.1
Hispanic/Latino	5.6	45.3	47.0
Education (years)			
11 or fewer	5.8	42.1	43.9
12	6.8	51.9	54.2
13 to 15	11.0	59.5	63.1
16 or more	10.4	66.7	69.2
Health Insurance			
Yes	9.2	59.4	62.2
No	1.6	17.8	18.8
Immigration			
Born in US	9.2	58.0	60.9
Born in US Territory	4.7	53.3	55.6
In US less than 10 years	1.7	24.1	25.3
In US 10 years or more	6.5	46.5	48.4
Total	8.8	56.4	59.1

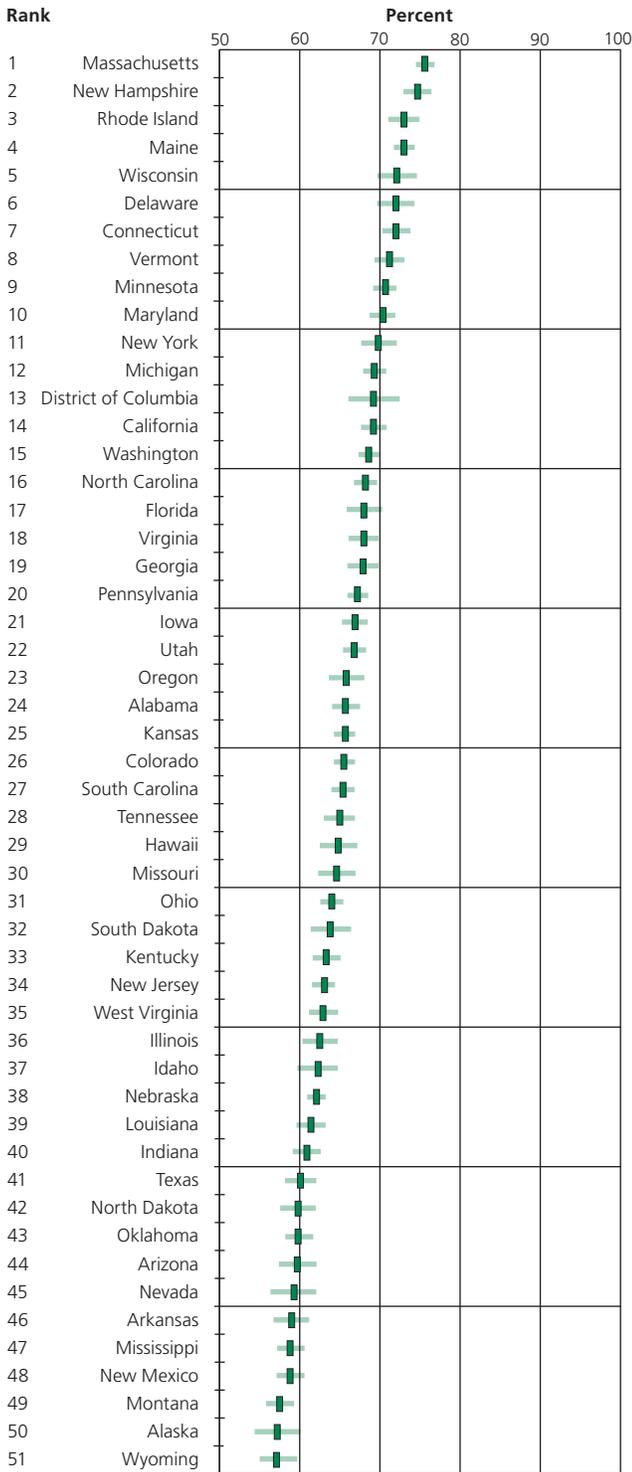
Percentages are age adjusted to the 2000 US standard population.

Note: The 2010 estimate for endoscopy and combined FOBT/endoscopy cannot be compared to estimates from 2008 and prior because of changes in questions assessing endoscopy use.

*A home fecal occult blood test within the past year. †A sigmoidoscopy within the past five years or a colonoscopy within the past 10 years. ‡Either a fecal occult blood test within the past year, sigmoidoscopy within the past five years, or a colonoscopy within the past 10 years. §Does not include Native Hawaiians or other Pacific Islanders. ¶Estimates should be interpreted with caution because of the small sample sizes.

Source: National Health Interview Survey Public Use Data File 2010, National Center for Health Statistics, Centers for Disease Control and Prevention.

Figure 10. Colorectal Cancer Screening* Prevalence among Adults Age 50 Years and Older by State, 2012



*Either a fecal occult blood test in the past year or a sigmoidoscopy or colonoscopy in the past 10 years.

Source: Behavioral Risk Factor Surveillance System Public Use Data Tapes 2012, National Center for Disease Prevention and Health Promotion, Centers for Disease Control and Prevention.

American Cancer Society, Surveillance Research, 2014

“Toilet bowl tests” are guaiac-based tests that are often promoted as a type of FOBT. They consist of strips of paper to be dropped into the toilet water with your stool and are sold in drugstores and other retail outlets. These tests have not been evaluated in the types of rigorous clinical studies done on the guaiac-based FOBT and the FIT and are not recommended for colorectal cancer screening by the American Cancer Society or any other major medical organization.

Stool DNA (sDNA) test: The stool DNA test approved for colorectal cancer screening in 2008 is no longer commercially available. A new test has undergone extensive study and may be evaluated for inclusion as a recommended testing option in the future. This method of screening is the result of increasing knowledge about the molecular properties of cancer. Cancerous tumors and large polyps shed cells into the large bowel that contain altered DNA that can be detected in stool samples. Patients with a positive test result would be referred for a colonoscopy.

Any of the above recommended options are useful in screening for colorectal cancer in average-risk adults. Each of these tests has strengths and limitations related to accuracy, potential for prevention, cost, and risks (Table 3, page 14). However, positive results from any test other than a colonoscopy should be followed with a colonoscopy for complete diagnostic evaluation. When choosing a screening test, patients should be given information about each test and should engage in a shared decision-making process with a health care professional based on the patient’s health and medical history. It is also important to solicit and acknowledge patient preferences regarding screening tests. For example, in 2012, blacks were more likely than whites to report FOBT use across all income and education levels.¹³⁰ A growing body of evidence demonstrates that offering patients different test options substantially increases adherence to screening recommendations.^{131, 132}

Screening for individuals at increased risk for colorectal cancer

Some people who are at increased risk of colorectal cancer because of family history or certain medical conditions (see page 10) should begin colorectal cancer screening before age 50. Colonoscopy is the recommended screening method for most individuals in these increased and high-risk groups. Recommendations regarding age to initiate screening and rescreening intervals may differ based on individual circumstances, so individuals with these risk factors should discuss screening with their health care provider. For additional information on colorectal cancer screening in high-risk individuals, see Levin et al.¹¹¹

Use of colorectal cancer screening

Despite the evidence supporting the effectiveness of colorectal cancer screening and the availability of a variety of screening

tests, only 59% of the US population age 50 and older is current for recommended testing.¹ Screening prevalence has been increasing modestly since 2000 exclusively due to an increase in colonoscopy testing.¹³³ Colonoscopy use almost tripled in the US during the past decade, from 19% in 2000 to 55% in 2010.¹³⁴ Among adults 50 and older, 9% report screening with FOBT and 56% report an endoscopy (colonoscopy or sigmoidoscopy) within the recommended time intervals (Table 4, page 15). (Endoscopy prevalence includes tests performed for both screening and diagnostic purposes.) Compared to the overall 50 and older population, screening prevalence is lower among adults younger than 65 and among those who are non-white, have fewer than 13 years of education, lack health insurance, and are recent immigrants. Women are slightly less likely than men to be current for screening.

Screening prevalence also varies by state:

- The percentage of adults 50 and older who report being current for screening ranges from 57% in Wyoming to 76% in Massachusetts (Figure 10; Table 5, page 18).
- Among the 29 states with adequate data on colorectal cancer screening in blacks, rates range from a low of 51% in Mississippi to a high of 76% in New York (Table 5, page 18).
- Massachusetts and New Hampshire are the only two states that meet the American Cancer Society's 2015 goal of 75% of all adults 50 and older being current for colorectal cancer screening.

Barriers to colorectal cancer screening

Several common themes have emerged from studies conducted to understand why rates of colorectal cancer screening remain low.

- Factors most strongly and consistently associated with inadequate colorectal cancer screening relate to cost and a general lack of access to health care, most often as a result of no health

insurance. Populations that are most likely to have lower screening rates include Hispanics, new immigrants, individuals born outside the US, and those with limited English language proficiency. These are also the groups that are least likely to be aware of the need for colorectal screening.^{135, 136}

- Inadequate communication by health care providers about the importance of screening is another major factor in screening underutilization. A physician's recommendation increases the likelihood of screening among both insured and uninsured individuals.^{135, 137, 138}
- Differences in patient and provider testing preferences impact screening rates. For example, physicians who discuss screening with their patients typically recommend colonoscopy; however, some patients prefer FOBT and are more likely to follow screening recommendations when presented with that option.^{131, 132, 139, 140}
- Individuals with the lowest educational attainment and income levels, among whom the colorectal cancer burden is the highest, have the lowest colorectal cancer screening rates, even among insured populations (Table 4, page 15).¹³⁵
- Personal barriers to screening include fear and embarrassment.^{135, 141}

Strategies to increase colorectal cancer screening

Clinicians and health care systems can play a major role in increasing the utilization and quality of screening for colorectal cancer through both patient- and provider-level initiatives. Implementing a diverse set of strategies, including the use of electronic health records to facilitate interventions, can maximize the positive impact on screening. Studies have shown that the following interventions increase colorectal cancer screening utilization.^{136, 142, 143}

Recent progress in policies and legislation related to colorectal cancer screening

On March 23, 2010, Congress passed and the president signed health care reform legislation, which included approximately 160 provisions that will meaningfully improve the health care system for cancer patients. Many of those provisions will help colorectal cancer patients and give greater access to colorectal cancer screening and treatment. For example:

- Ensure that individuals with a history of colorectal cancer are no longer denied coverage because of a pre-existing condition.
- Prohibit the sudden discontinuation of coverage because a patient is diagnosed with colorectal cancer or another health condition.
- Prohibit the use of annual dollar limits on coverage and lifetime limits that leave cancer patients without coverage.
- Require that all commercial health insurance plans cover colorectal cancer screening tests (fecal occult blood testing, sigmoidoscopy, or colonoscopy) for all adults beginning at age 50 and continuing until age 75.
- Ensure that colorectal cancer screening tests, except when a polyp is removed during a screening colonoscopy, are administered at no cost to patients in the Medicare program. (Patients can be charged a co-pay if a polyp is removed during a screening colonoscopy.)
- Create a national prevention and public health fund to expand and sustain national investment in prevention and public health programs, including health screenings

Table 5. Colorectal Cancer Screening* Prevalence among Adults Age 50 Years and Older by Race/Ethnicity and State, 2012

State	All races combined			Non-Hispanic White			Non-Hispanic Black		
	Rank	%	± 95% CI	Rank	%	± 95% CI	Rank	%	± 95% CI
Massachusetts	1	75.6	1.2	1	76.9	1.2	13	66.1	6.2
New Hampshire	2	74.7	1.7	4	74.5	1.7	†	†	–
Rhode Island	3	73.0	2.0	2	75.2	1.9	†	†	–
Maine	4	73.0	1.3	7	73.6	1.4	†	†	–
Wisconsin	5	72.1	2.4	8	73.3	2.4	†	†	–
Delaware	6	72.0	2.2	10	72.1	2.3	4	69.8	6.9
Connecticut	7	72.0	1.7	5	73.9	1.7	17	64.1	8.0
Vermont	8	71.2	1.8	11	71.8	1.8	†	†	–
Minnesota	9	70.7	1.5	9	72.1	1.4	†	†	–
Maryland	10	70.4	1.6	12	71.4	1.7	3	70.8	3.7
New York	11	69.8	2.2	18	69.2	2.2	1	75.6	7.5
Michigan	12	69.3	1.5	15	70.5	1.6	16	64.5	5.8
District of Columbia	13	69.2	3.2	3	75.0	3.5	5	68.8	4.2
California	14	69.2	1.6	6	73.8	1.5	2	75.0	6.3
Washington	15	68.6	1.3	13	71.0	1.2	†	†	–
North Carolina	16	68.2	1.5	16	69.9	1.6	15	66.0	4.0
Florida	17	68.0	2.1	14	70.8	2.1	8	67.4	7.0
Virginia	18	68.0	1.8	17	69.5	1.9	9	67.2	5.1
Georgia	19	67.9	2.0	19	69.2	2.3	14	66.1	4.6
Pennsylvania	20	67.2	1.3	22	68.0	1.3	7	67.6	5.2
Iowa	21	66.9	1.6	25	67.4	1.6	†	†	–
Utah	22	66.8	1.5	20	68.8	1.5	†	†	–
Oregon	23	65.8	2.1	26	67.3	2.1	†	†	–
Alabama	24	65.7	1.7	30	65.8	2.0	10	67.1	3.8
Kansas	25	65.7	1.4	27	66.8	1.3	11	66.5	7.9
Colorado	26	65.5	1.4	23	67.6	1.5	†	†	–
South Carolina	27	65.4	1.5	24	67.5	1.8	24	59.9	3.6
Tennessee	28	65.0	2.0	28	66.3	2.0	25	57.8	6.8
Hawaii	29	64.8	2.3	21	68.3	3.3	†	†	–
Missouri	30	64.6	2.1	32	64.8	2.2	18	63.9	7.6
Ohio	31	64.0	1.5	36	63.8	1.5	12	66.2	5.6
South Dakota	32	63.8	2.5	31	64.9	2.5	†	†	–
Kentucky	33	63.3	1.7	37	63.8	1.8	20	60.9	8.2
New Jersey	34	63.1	1.5	34	64.4	1.6	19	63.0	4.7
West Virginia	35	62.9	1.9	40	63.0	1.9	†	†	–
Illinois	36	62.5	2.2	35	64.3	2.3	22	60.4	7.5
Idaho	37	62.3	2.5	39	63.1	2.6	†	†	–
Nebraska	38	62.1	1.2	38	63.5	1.2	26	56.9	9.3
Louisiana	39	61.4	1.9	33	64.7	2.2	28	55.2	4.0
Indiana	40	60.9	1.7	45	61.7	1.8	23	60.0	6.6
Texas	41	60.1	2.0	29	65.9	2.2	6	68.7	6.5
North Dakota	42	59.8	2.2	47	60.8	2.2	†	†	–
Oklahoma	43	59.8	1.7	46	60.9	1.9	21	60.8	8.0
Arizona	44	59.7	2.3	41	62.7	2.4	†	†	–
Nevada	45	59.3	2.9	44	62.4	3.0	†	†	–
Arkansas	46	59.0	2.2	48	59.9	2.4	27	56.9	7.0
Mississippi	47	58.8	1.8	42	62.6	2.1	29	50.9	3.6
New Mexico	48	58.8	1.7	43	62.5	2.0	†	†	–
Montana	49	57.5	1.7	49	58.7	1.8	†	†	–
Alaska	50	57.2	2.9	50	58.3	3.3	†	†	–
Wyoming	51	57.1	2.3	51	57.9	2.4	†	†	–

Note: CI = confidence interval, which is similar to a margin of error. *Either a fecal occult blood test in the past year or a sigmoidoscopy or colonoscopy in the past 10 years. † Sample size insufficient to provide a stable estimate.

Source: Behavioral Risk Factor Surveillance System Public Use Data Tapes 2012, National Center for Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. BRFSS 2012 data results should be considered baseline and are not directly comparable to previous years because of changes in weighting methodology and the addition of the cell phone sampling frame.

Patient-level interventions

- Eliminating structural barriers by providing FOBT cards and instructions for patients to use at home
- One-on-one comprehensive discussions with a health care provider or health educator about the importance of colorectal cancer screening, including a detailed explanation of the benefits and limitations of various testing options
- Mailed reminders to patients who are due for screening

Health care system-level interventions

- Implementation of centralized or office-based reminder systems to assist clinicians in counseling eligible patients about screening

- The use of patient navigators to help manage referrals, help patients navigate the health care system, and facilitate follow-up screening

One comprehensive resource that is available to aid primary care providers in improving patient screening rates is the online manual *How to Increase Colorectal Cancer Screening Rates in Practice: A Primary Care Clinician's Evidence-Based Toolbox and Guide*, produced by the American Cancer Society, Thomas Jefferson University, and the National Colorectal Cancer Roundtable, which is available at cancer.org/colonmd.

Colorectal Cancer Treatment

Treatment decisions are made by patients with their physicians after considering the best options available for the stage and location of the cancer, as well as the risks and benefits associated with each.

Colon cancer

Most people with colon cancer will have some type of surgery to remove the tumor. Adjuvant therapy (additional treatments after surgery) may also be used.

Carcinoma in situ

Carcinoma in situ is cancer that has not spread beyond the layer of cells in which it began. Surgery to remove the growth of abnormal cells may be accomplished by polypectomy (polyp removal) or local excision through the colonoscope. Resection of a segment of the colon may be necessary if the tumor is too large to be removed by local excision or if cancer cells are found at the edges of the polyp after it is removed.

Localized stage

Localized stage refers to invasive cancer that has penetrated the wall of the colon. Surgical resection to remove the cancer, together with a length of colon on either side of the tumor and nearby lymph nodes, is the standard treatment.

Regional stage

Regional stage includes cancers that have grown through the wall of the colon, as well as cancers that have spread to nearby lymph nodes. If the cancer has only grown through the wall of the colon but has not spread to nearby lymph nodes, surgical resection of the segment of colon containing the tumor may be the only treatment needed. If the cancer is likely to come back, because of its appearance under the microscope or because it

is growing into other tissues, radiation therapy and/or chemotherapy may also be recommended. If the cancer has spread to nearby lymph nodes, surgical resection of the segment of colon containing the tumor is the first treatment, usually followed by chemotherapy. Chemotherapy treatments based on the drug fluorouracil (5-FU) have been shown to improve survival in patients with stage III or high-risk stage II disease, primarily by reducing disease recurrence.¹⁴⁴ Radiation therapy may also be recommended if the cancer has grown into adjacent tissues.

Adjuvant (given after surgery) chemotherapy or radiation for colon cancer is as effective in patients age 70 and older (more than half of all patients) who are otherwise healthy as in younger patients, though certain drugs (i.e., oxaliplatin) may be avoided to limit toxicity. However, a recent study in California found that although chemotherapy reduced colon cancer mortality similarly across all age groups, individuals 75 and older were far less likely than younger patients to receive this treatment.¹⁴⁵

Distant stage

At this stage, the cancer has spread to distant organs and tissues, such as the liver, lungs, peritoneum (lining of the abdomen), or ovaries. When surgery is performed, the goal is usually to relieve or prevent blockage of the colon and to prevent other local complications. If there are only a few metastases to the liver or lungs, surgery to remove these, as well as the colon tumor, may be an option. Surgery is not recommended for all patients.

Chemotherapy, radiation, and biologically targeted therapies may be given alone or in combination to relieve symptoms and prolong survival. A number of targeted therapies have been approved in recent years by the US Food and Drug Administration (FDA) to treat metastatic colorectal cancer. Some of these drugs inhibit new blood vessel growth to the tumor by targeting a protein called vascular endothelial growth factor (VEGF). Oth-

ers interfere with cancer cell growth by targeting the epidermal growth factor receptor (EGFR) or other proteins. Tumors with certain genetic mutations do not benefit from treatment with some of these drugs.¹⁴⁶

Rectal cancer

Surgery is usually the main treatment for rectal cancer, with the exception of some patients with distant-stage disease. Additional treatments, such as chemotherapy and radiation, are often used before surgery (neoadjuvant therapy) and/or after surgery (adjuvant therapy) to reduce the risk of recurrence and metastasis. The chemotherapy drugs used in the treatment of rectal cancer are the same as those used for colon cancer.

Carcinoma in situ

Removing or destroying the growth of abnormal cells is all that is needed. Treatment options include polypectomy (polyp removal), local excision, or full-thickness rectal resection. This resection may be carried out through the anus. No further treatment is needed.

Localized stage

At this stage, the cancer has grown through the first layer of the rectum into deeper layers, but has not spread outside the rectal wall. Some small localized rectal cancers may be treated by removal through the anus, without an abdominal incision. For other cancers, depending on where they are located, surgery may involve removal of the cancer and some surrounding normal tissue through an abdominal incision. For cancers close to the anus, surgery may require removal of the anus and the sphincter muscle, so a permanent colostomy is required (see next section for information about colostomy). In most cases, no further treatment is needed unless the tumor tests positive for high-risk features. Patients who are not candidates for surgery may be treated with radiation therapy. This may mean endocavitary radiation therapy (radiation delivered through the anus) or brachytherapy (radioactive pellets placed next to or directly in the cancer). Radiation therapy alone has not been proven to be as effective as surgery in treating rectal cancer.

Regional stage

If the cancer has spread through the wall of the rectum into nearby tissue and/or lymph nodes, radiation and chemotherapy are often given together before surgery, with additional chemotherapy often given after surgery.

Distant stage

In this stage, the cancer has spread to distant organs and tissues, such as the liver or lung. In rare cases, the cancer can be successfully treated by removing all of the tumors with surgery, along with other treatments. Otherwise, surgery, chemotherapy, and/or radiation therapy are used to relieve, delay, or prevent symptoms and to prolong life.

Colostomy

When a section of the colon or rectum is removed, the surgeon can usually connect the healthy parts, allowing the patient to eliminate waste normally. However, sometimes reconnection is not possible immediately. In this case, the surgeon connects the colon to an opening (a stoma) that is made in the skin of the abdomen, allowing waste to leave the body. The surgical procedure to create an opening in the body for the elimination of waste is called an ostomy. When the stoma is connected to the colon it is called a colostomy; when the stoma is connected to the small intestine it is called an ileostomy. Usually a flat bag fits over the stoma, held in place by a special adhesive, to collect waste.

Most patients with colorectal cancer who require a colostomy need it only temporarily, until the colon or rectum heals from surgery. After healing takes place, usually in 6 to 8 weeks, the surgeon reconnects the ends of the colon and closes the stoma. A colostomy is necessary more often for rectal than for colon cancer patients, 26% versus 7%.¹⁴⁷

A person with an ostomy learns to care for it with help from doctors, nurses, and enterostomal therapists (health professionals trained to care for people with stomas). Often, if the surgery is expected to result in an ostomy, an enterostomal therapist will visit the patient before surgery to explain what to expect and how to care for the ostomy after surgery. They will also talk about lifestyle issues, including emotional, physical, and sexual concerns, and can provide information about resources and support groups.

Side effects of colorectal cancer treatment

Surgery

The time needed to heal after surgery is different for each person. Patients often have some pain for the first few days; however, this can usually be controlled with medication. It can take a few days to be able to eat normally again. Patients are monitored for signs of bleeding, infection, or other problems requiring immediate treatment. Side effects from surgery for colorectal cancer may include:

- Fatigue, possibly for an extended period
- Constipation or diarrhea
- A temporary or permanent colostomy
- Sexual dysfunction (e.g., erectile dysfunction in men) after more extensive operations for rectal cancer

Radiation therapy

Side effects of radiation therapy for colorectal cancer can include skin irritation, nausea, diarrhea, rectal irritation, bladder irritation, fatigue, or sexual problems. Rectal irritation can lead to the urge to defecate frequently and rectal bleeding, while bladder irritation can lead to urinary urgency, frequency, and pain.

Many of these side effects go away after treatments are completed, but some, like sexual problems and some degree of rectal and/or bladder irritation, may be permanent. These or other side effects should be discussed with a clinician because treatment options may be available.

Chemotherapy

The chemotherapy drugs most often used in the treatment of colorectal cancer are 5-fluorouracil (5-FU) capecitabine, oxaliplatin, and irinotecan. Chemotherapy drugs kill cancer cells, but also damage some normal cells. Side effects depend on the type and dosage of drugs and the length of treatment. General side effects from chemotherapy include:

- Fatigue
- Nausea and vomiting
- Diarrhea
- Loss of appetite
- Hair loss
- Swelling and rashes
- Mouth sores
- Numbness, tingling, or blistering of the hands and feet

Some patients may experience low blood cell counts because chemotherapy can damage the blood-producing cells of the bone marrow. This can increase the chance of infection (due to a shortage of white blood cells), bleeding or bruising after minor cuts or injuries (due to a shortage of blood platelets), or worsening fatigue (due to a shortage of red blood cells).

There are remedies for many of the temporary side effects of chemotherapy. For example, antiemetic drugs can prevent or reduce nausea and vomiting, and drugs known as growth factors can improve the levels of white blood cells. People receiving chemotherapy should talk with their doctor if they have any unrelieved side effects. Most side effects go away or lessen once treatment is stopped. For example, hair grows back after treatment ends, though it may look different.

Targeted therapy

Targeted therapy is a newer group of drugs developed as a result of a greater understanding of the molecular changes involved in cancer occurrence. These drugs target specific molecules involved in tumor growth and progression and usually have less severe side effects than chemotherapy drugs.

Epidermal growth factor receptor (EGFR) inhibitors

These drugs work by slowing or stopping cancer cell growth and may cause skin-related side effects, such as:

- Acne-like rash
- Dry skin
- Swelling or pain in the fingernails or toenails

Vascular endothelial growth factor (VEGF) inhibitors

These drugs work by preventing the formation of new blood vessels necessary for tumor growth. Side effects include:

- Problems with bleeding (e.g., nose bleeds, wound healing)
- High blood pressure
- Clots in the arteries or veins
- Kidney damage

What is the American Cancer Society Doing about Colorectal Cancer?

Research

Colorectal cancer is an active area of scientific research. Studies currently funded by the American Cancer Society span the cancer continuum from prevention and early detection to treatment and beyond. The Society is currently funding more than \$43 million in colorectal cancer research, with \$3.8 million awarded in fiscal year 2012. Below are just some examples of the projects in which intramural and extramural Society researchers are engaged.

Prevention and early detection

- Interventions to increase colorectal cancer screening within health systems and communities, including hard-to-reach, low-income populations

- Interventions aimed at lowering risk of colorectal cancer through improvement in diet and physical activity in minority populations
- Research on new screening tests that may be more accurate and/or more comfortable for patients than current options
- Research into the mechanisms underlying the association between obesity, physical activity, and colorectal cancer
- Monitoring and mitigating disparities in screening, treatment, and survivorship
- Evaluating the impact of health care reform on utilization of screening, diagnostic testing, treatment services, and health care costs

Cancer development

A large proportion of research is focused on understanding the cellular and molecular mechanisms underlying colorectal tumor development, which are currently poorly understood.

- Genetic research studying errors during cell division, which lead to abnormal cell growth and carcinogenesis (cancer development)
- Identification and study of certain natural substances in the body that appear to block cancer cell growth
- Monitoring disparities and emerging trends in incidence and mortality rates

Treatment

- Gene studies to determine optimal, individualized treatment for advanced colorectal cancer based on patient gene profile
- Evaluation of drugs that boost the immune system's reaction to colorectal cancer, as well as new combinations of chemotherapy drugs and the best ways to combine chemotherapy with radiation or immunotherapy
- Research on several new targeted therapies to increase the number of treatment options with fewer side effects
- Comparing strategies to personalize care using biomarkers

Behavior and survivorship

- Understanding patient, health care provider, and health systems factors that influence health behaviors, such as the adoption of evidence-based prevention, screening and treatment recommendations
- Identification of factors responsible for survival differences following a colorectal cancer diagnosis
- Interventions that reduce stress and fatigue, improve nutrition, and increase levels of physical activity during and after chemotherapy and radiation treatment
- Palliative care interventions to improve both the quality of care and the quality of life for survivors and their caregivers
- Decision support interventions using novel approaches to engage persons in the decision-making process regarding screening, treatment, and genetic testing

The Society's Approach through Health Systems

Improvements in the prevention, early detection, and treatment of colorectal cancer provide major, unrealized opportunities to save lives. Ultimately, prevention through changes in tobacco use, diet, physical activity, and body weight can have the largest impact on health in general, including reduced risk of colorectal cancer. In the near term, improvements in screening are more easily achieved. A substantial proportion of the 50,310 people expected to die of colorectal cancer in 2014 could have been

saved with recommended screening. Despite the potential to prevent colorectal cancer and reduce the risk of dying from the disease, too few Americans are getting tested according to the recommended guidelines.

To increase the number of people who get screened, the American Cancer Society has reached out to the public, health care professionals, and, through the American Cancer Society Cancer Action NetworkSM (ACS CAN, the nonprofit, nonpartisan advocacy affiliate of the American Cancer Society), legislators on the federal and state level. The Society also works in collaboration with health care system partners to increase the number of people who receive regular colorectal cancer screening. During National Colon Cancer Awareness Month every March, and throughout the year, the Society encourages regular colorectal cancer screening for people age 50 and older; encourages clinicians to proactively recommend regular screening to all age-appropriate patients; and advocates for laws that improve access to screening and treatment, as well as addressing the needs of the medically underserved. The key message to men and women age 50 and older is that screening is the most important step to help prevent colon cancer. People should talk to their doctors about when to start testing and which test is right for them.

To reach consumers with these messages, the Society:

- Uses national, regional, and local media to encourage consumers to talk with their doctors about colorectal cancer testing
- Uses online and social media channels to communicate with constituents and the public about the importance of colon cancer screening, while also establishing a dialogue and engaging feedback. The Society's Facebook pages and groups, Twitter feeds, YouTube channel, and other social media avenues are utilized daily to connect with our constituents and send mission-related messages.
- Encourages consumers to visit cancer.org/colon to learn more about colorectal cancer screening
- Builds collaborations within communities nationwide to reach specific populations

Health care professionals play a vital role in a patient's decision to get tested for colon cancer. Research shows patients are more likely to get screened if their doctor recommends it. To reach health care professionals with messages and information about the importance of talking to their patients about colon cancer screening, the Society:

- Encourages health care professionals to visit cancer.org/colonmd for tools and resources on how to talk to their patients about colorectal cancer testing and improve testing rates in their practice

- Builds collaborative relationships to facilitate regular communication between health care professionals and the patients they serve
- Collaborates with the Centers for Medicare & Medicaid Services (CMS) to develop messages targeted at health care professionals about the importance of colorectal screening and the availability of resources to help improve testing rates in their practice
- Collaborates with 53 quality improvement organizations to increase the number of colorectal cancer screenings and their documentation in electronic medical records systems
- Collaborates with CIGNA and United HealthGroup to disseminate reminder messages to more than 500,000 members to prompt participation in colorectal cancer screening

Advocacy

ACS CAN is involved in advocacy efforts at both the federal and state level that will increase access to quality colorectal cancer screening, treatment, and care for all adults. Listed below are some of the efforts the Society and ACS CAN are involved in:

- Implementing the provisions in the Patient Protection and Affordable Care Act, more commonly referred to as the Affordable Care Act or ACA. The reforms in the ACA, which was

signed into law in March 2010, represent a profound structural change in how insurance will operate and how consumers and patients will utilize the health insurance system. ACS CAN and the Society have a significant impact at the federal and state levels through our advocacy work, which will urge policy makers to implement the law to ensure that all Americans have access to evidence-based prevention, early detection, and treatment services critical to colorectal cancer patients.

- Supporting the work of the CDC's Colorectal Cancer Control Program (CRCCP), which currently provides funding to 25 states and four tribes across the US. The CRCCP's goal is to increase colorectal cancer screening rates among men and women age 50 and older to 80%. The program provides grants for both population-based education campaigns and to improve access to vital colorectal cancer screening tests and follow-up services for low-income, uninsured, and underinsured individuals between the ages of 50 and 64, as well as those under 50 who are at high risk of developing colorectal cancer.
- Advocating for passage of the Removing Barriers to Colorectal Cancer Screening Act of 2013, which will ease the financial burden of people living on a fixed income by allowing Medicare beneficiaries to receive screenings without coinsurance, even when a polyp is removed. This legislation would help increase screening rates and reduce the incidence of colorectal cancer.

The National Colorectal Roundtable

The National Colorectal Cancer Roundtable (NCCRT) is a coalition of more than 70 public, private, and voluntary organizations, led by the American Cancer Society and the Centers for Disease Control and Prevention, whose mission is to advance colorectal cancer control efforts by improving communication, coordination, and collaboration among health agencies, medical-professional organizations, and the public.

The ultimate goal of the Roundtable is to increase the use of colorectal cancer screening tests among the population for whom screening is recommended. It serves as a forum for communication and developing consensus in order to advance key initiatives that can address gaps and create opportunities to improve cancer screening. Once the Roundtable identifies a key issue, it leverages the talents of the members to conduct studies, create tools, and identify emerging issues that can advance colorectal cancer control efforts. While the Roundtable focuses on colorectal cancer control, many of the initiatives, tools, and evidence-based interventions developed by the coalition can easily be adapted to inform a broad array of cancer control activities.

Recent initiatives include:

- Partnering with Patient Centered Primary Care Collaborative to increase cancer screening in the patient centered medical home
- Collaborating with the National Association of Community Health Centers to implement strategies that increase colorectal cancer screening for the vulnerable populations served by these facilities
- Developing the signature guide: *How to Increase Colorectal Cancer Screening Rates in Practice: A Primary Care Clinician's Evidence-Based Toolbox and Guide*
- Promoting collaborative efforts to improve the quality of screening colonoscopy
- Developing a March Colorectal Cancer Awareness Month marketing kit
- Commissioning research to assess state-by-state Medicaid coverage of preventive services published in *Health Affairs*
- Developing a colorectal cancer evaluation tool kit that includes template evaluation materials in both English and Spanish and conducts evaluation training

In short, the National Colorectal Cancer Roundtable and its partners work together to unify and magnify efforts around colorectal cancer. In this way, it maximizes limited resources, pools talent, and strengthens the collective energy behind CDC strategic priorities for increasing colorectal cancer screening.



- Advocating for passage of the Colorectal Cancer Prevention, Early Detection, and Treatment Act, which will authorize the CRCCP so more states will have access to federal funding to help improve colorectal cancer screening rates. The legislation will also give states the option to provide full Medicaid benefits to uninsured, low-income men and women under age 65 who are identified by the CRCPP and are in need of treatment for colorectal cancer.
- Advocating for federal funding to strengthen and further expand the scope of the CDC's Colorectal Cancer Screening, Education, & Outreach Program to promote colorectal cancer screening nationwide, to identify and eliminate certain clinical and consumer barriers to screening, and to further reduce colorectal cancer incidence and mortality rates.

Sources of Statistics

New cancer cases. The estimated number of colorectal cancer cases in the US in 2014 was projected using a spatio-temporal model based on incidence data from 49 states and the District of Columbia for the years 1995 to 2010 that met the North American Association of Central Cancer Registries' (NAACCR) high-quality data standard for incidence. For more information on this method, please see Zhu et al.¹⁴⁸

Incidence rates. Incidence rates are defined as the number of people per 100,000 who are diagnosed with cancer during a given time period. Colorectal cancer incidence rates for the US were calculated using case data from the Surveillance, Epidemiology, and End Results (SEER) program of the National Cancer Institute, the National Program of Cancer Registries of the Centers for Disease Control and Prevention, and the North American Association of Central Cancer Registries (NAACCR), and population data collected by the US Census Bureau. Incidence rates were age adjusted to the 2000 US standard population and adjusted for delays in reporting when possible.

Estimated cancer deaths. The estimated number of colorectal cancer deaths in the US in 2014 was calculated by fitting the actual numbers of colorectal cancer deaths from 1995 through 2010 to a statistical model that forecasts the number of deaths in 2014. The actual numbers of deaths are obtained from the National Center for Health Statistics (NCHS) at the Centers for Disease Control and Prevention. For more information on this method, please see Chen et al.¹⁴⁹

Mortality rates. Mortality rates, or death rates, are defined as the number of people per 100,000 dying of a disease during a given year. Mortality rates are based on counts of cancer deaths compiled by NCHS and population data from the US Census Bureau. Death rates are age adjusted to the 2000 US standard population.

Survival. Both relative and cause-specific survival rates are presented. Currently, population-based survival rates are limited to those patients diagnosed in SEER cancer registry areas

between 1975 and 2009. Relative survival rates account for normal life expectancy (including events such as death from heart disease, accidents, and diseases of old age). Cause-specific survival rates are presented for Hispanics/Latinos, Asian/Pacific Islanders, and American Indians/Alaska Natives because reliable estimates of normal life expectancy have historically not been available for these groups. Cause-specific survival rates are the probability of not dying from colorectal cancer within a specified time period (usually 5 years) following a diagnosis.

Screening. The prevalence of colorectal cancer screening among US adults was obtained from the National Health Interview Survey (NHIS) 2010 data file, obtained from the National Center for Health Statistics, Centers for Disease Control and Prevention, released in 2011 (cdc.gov/nchs/nhis.htm). The NHIS is a centralized survey conducted by the US Census Bureau that is designed to provide national prevalence estimates on health characteristics such as cancer screening behaviors. Data are collected through in-person interviews.

Prevalence data for colorectal cancer screening by state are from the 2012 Behavioral Risk Factor Surveillance System (BRFSS) public use data tapes, obtained from the National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention (cdc.gov/nccdphp/brfss/). The BRFSS was designed to provide state prevalence estimates of health behaviors and is conducted by state health departments. The BRFSS is a telephone survey, so prevalence estimates are limited to those adults who have a cellular phone or who live in a household with a residential telephone line. Prevalence rates are age adjusted to the 2000 US standard population.

Important note about estimated cases and deaths. The projected numbers of new cancer cases and deaths for the current year are model based and may produce numbers that vary considerably from year to year. For this reason, we discourage the use of our estimates to track cancer trends. Age-standardized incidence and mortality rates are used to track cancer incidence and mortality trends.

References

1. National Center for Health Statistics, Division of Health Interview Statistics. National Health Interview Survey Public Use Data File 2010. Centers for Disease Control and Prevention. Hyattsville, MD., 2011.
2. Iacopetta B. Are there two sides to colorectal cancer? *Int J Cancer*. 2002;101: 403-408.
3. Matanoski G, Tao X, Almon L, Adade AA, Davies-Cole JO. Demographics and tumor characteristics of colorectal cancers in the United States, 1998-2001. *Cancer*. 2006;107: 1112-1120.
4. Nawa T, Kato J, Kawamoto H, et al. Differences between right- and left-sided colon cancer in patient characteristics, cancer morphology and histology. *J Gastroenterol Hepatol*. 2008;23: 418-423.
5. Winawer SJ, Zauber AG. The advanced adenoma as the primary target of screening. *Gastrointest Endosc Clin N Am*. 2002;12: 1-9, v.
6. Stryker SJ, Wolff BG, Culp CE, Libbe SD, Ilstrup DM, MacCarty RL. Natural history of untreated colonic polyps. *Gastroenterology*. 1987;93: 1009-1013.
7. Bond JH. Polyp guideline: diagnosis, treatment, and surveillance for patients with colorectal polyps. Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol*. 2000;95: 3053-3063.
8. Schatzkin A, Freedman LS, Dawsey SM, Lanza E. Interpreting precursor studies: what polyp trials tell us about large-bowel cancer. *J Natl Cancer Inst*. 1994;86: 1053-1057.
9. Levine JS, Ahnen DJ. Clinical practice. Adenomatous polyps of the colon. *N Engl J Med*. 2006;355: 2551-2557.
10. Risio M. The natural history of adenomas. *Best Pract Res Clin Gastroenterol*. 2010;24: 271-280.
11. Pickhardt PJ, Kim DH, Pooler BD, et al. Assessment of volumetric growth rates of small colorectal polyps with CT colonography: a longitudinal study of natural history. *Lancet Oncol*. 2013;14: 711-720.
12. Stewart SL, Wike JM, Kato I, Lewis DR, Michaud F. A population-based study of colorectal cancer histology in the United States, 1998-2001. *Cancer*. 2006;107: 1128-1141.
13. Siegel R, DeSantis C, Virgo K, et al. Cancer treatment and survivorship statistics, 2012. *CA Cancer J Clin*. 2012;62: 220-241.
14. National Cancer Institute. DevCan: Probability of Developing or Dying of Cancer Software, Version 6.7.0; Statistical Research and Applications Branch, National Cancer Institute, 2005.
15. Surveillance, Epidemiology, and End Results (SEER) Program SEER*Stat Database: Mortality – All COD, Aggregated With State, Total U.S. (1969-2010) <Katrina/Rita Population Adjustment>, National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2013. Underlying mortality data provided by NCHS 2013.
16. Surveillance, Epidemiology and End Results (SEER) Program SEER*Stat Database: NAACCR Incidence – CiNA Analytic File, 1995-2010, for Expanded Races, Custom File With County, ACS Facts and Figures projection Project, North American Association of Central Cancer Registries, 2013.
17. Howlader N, Noone AM, Krapcho M, et al. *SEER Cancer Statistics Review, 1975-2010*. Bethesda, MD: National Cancer Institute, 2013.
18. Murphy G, Devesa SS, Cross AJ, Inskip PD, McGlynn KA, Cook MB. Sex disparities in colorectal cancer incidence by anatomic subsite, race and age. *Int J Cancer*. 2010;128: 1668-1675.
19. Copeland G, Lake A, Firth R, et al. *Cancer in North America: 2006-2010. Volume One: Combined Cancer Incidence for the United States, Canada and North America*. Springfield, IL: North American Association of Central Cancer Registries, Inc., 2013.
20. Surveillance, Epidemiology and End Results Program. SEER*Stat Database: Incidence – SEER 18 Regs Public Use, Nov 2012 Sub (2000-2010) – Linked to County Attributes – Total US, 1969-2011 Counties. Bethesda, MD: National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, 2013.
21. Edwards BK, Ward E, Kohler BA, et al. Annual report to the nation on the status of cancer, 1975-2006, featuring colorectal cancer trends and impact of interventions (risk factors, screening, and treatment) to reduce future rates. *Cancer*. 2010;116: 544-573.
22. Irby K, Anderson WF, Henson DE, Devesa SS. Emerging and widening colorectal carcinoma disparities between Blacks and Whites in the United States (1975-2002). *Cancer Epidemiol Biomarkers Prev*. 2006;15: 792-797.
23. Edwards BK, Noone A, Boscoe F, et al. Annual report to the nation on the status of cancer, 1975-2010, featuring comorbidity prevalence and impact on survival among persons with lung, colorectal, breast or prostate cancer. *Cancer*. 2014;[published online ahead of print December 16, 2013].
24. Surveillance, Epidemiology and End Results Program. SEER*Stat Database: Incidence – SEER 13 Regs Public Use, Nov 2011 Sub (1992-2010) – Linked to County Attributes – Total US, 1969-2011 Counties. Bethesda, MD: National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch 2013.
25. Siegel RL, Jemal A, Ward EM. Increase in incidence of colorectal cancer among young men and women in the United States. *Cancer Epidemiol Biomarkers Prev*. 2009;18: 1695-1698.
26. Lansdorp-Vogelaar I, Kuntz KM, Knudsen AB, van Ballegooijen M, Zauber AG, Jemal A. Contribution of screening and survival differences to racial disparities in colorectal cancer rates. *Cancer Epidemiol Biomarkers Prev*. 2012;21: 728-736.
27. Blot WJ, Fraumeni JF, Jr., Stone BJ, McKay FW. Geographic patterns of large bowel cancer in the United States. *J Natl Cancer Inst*. 1976;57: 1225-1231.
28. Naishadham D, Lansdorp-Vogelaar I, Siegel R, Cokkinides V, Jemal A. State disparities in colorectal cancer mortality patterns in the United States. *Cancer Epidemiol Biomarkers Prev*. 2011;20: 1296-1302.
29. Bach PB, Schrag D, Brawley OW, Galaznik A, Yakren S, Begg CB. Survival of blacks and whites after a cancer diagnosis. *JAMA*. 2002;287: 2106-2113.
30. Le H, Ziogas A, Lipkin SM, Zell JA. Effects of socioeconomic status and treatment disparities in colorectal cancer survival. *Cancer Epidemiol Biomarkers Prev*. 2008;17: 1950-1962.
31. Ward E, Jemal A, Cokkinides V, et al. Cancer disparities by race/ethnicity and socioeconomic status. *CA Cancer J Clin*. 2004;54: 78-93.
32. Ayanian JZ, Zaslavsky AM, Fuchs CS, et al. Use of adjuvant chemotherapy and radiation therapy for colorectal cancer in a population-based cohort. *J Clin Oncol*. 2003;21: 1293-1300.
33. Butler EN, Chawla N, Lund J, Harlan LC, Warren JL, Yabroff KR. Patterns of colorectal cancer care in the United States and Canada: a systematic review. *J Natl Cancer Inst Monogr*. 2013;2013: 13-35.
34. Du XL, Fang S, Vernon SW, et al. Racial disparities and socioeconomic status in association with survival in a large population-based cohort of elderly patients with colon cancer. *Cancer*. 2007;110: 660-669.
35. Gross CP, Smith BD, Wolf E, Andersen M. Racial disparities in cancer therapy: did the gap narrow between 1992 and 2002? *Cancer*. 2008;112: 900-908.

36. Potosky AL, Harlan LC, Kaplan RS, Johnson KA, Lynch CF. Age, sex, and racial differences in the use of standard adjuvant therapy for colorectal cancer. *J Clin Oncol*. 2002;20: 1192-1202.
37. Haller DG, Catalano PJ, Macdonald JS, et al. Phase III study of fluorouracil, leucovorin, and levamisole in high-risk stage II and III colon cancer: final report of Intergroup 0089. *J Clin Oncol*. 2005;23: 8671-8678.
38. McCollum AD, Catalano PJ, Haller DG, et al. Outcomes and toxicity in african-american and caucasian patients in a randomized adjuvant chemotherapy trial for colon cancer. *J Natl Cancer Inst*. 2002;94: 1160-1167.
39. Ward E, Halpern M, Schrag N, et al. Association of insurance with cancer care utilization and outcomes. *CA Cancer J Clin*. 2008;58: 9-31.
40. NIH consensus conference. Adjuvant therapy for patients with colon and rectal cancer. *JAMA*. 1990;264: 1444-1450.
41. Moertel CG, Fleming TR, Macdonald JS, et al. Levamisole and fluorouracil for adjuvant therapy of resected colon carcinoma. *N Engl J Med*. 1990;322: 352-358.
42. Huxley RR, Ansary-Moghaddam A, Clifton P, Czernichow S, Parr CL, Woodward M. The impact of dietary and lifestyle risk factors on risk of colorectal cancer: a quantitative overview of the epidemiological evidence. *Int J Cancer*. 2009;125: 171-180.
43. Butterworth AS, Higgins JP, Pharoah P. Relative and absolute risk of colorectal cancer for individuals with a family history: a meta-analysis. *Eur J Cancer*. 2006;42: 216-227.
44. Johns LE, Houlston RS. A systematic review and meta-analysis of familial colorectal cancer risk. *Am J Gastroenterol*. 2001;96: 2992-3003.
45. Lynch HT, de la Chapelle A. Hereditary colorectal cancer. *N Engl J Med*. 2003;348: 919-932.
46. Morris EJ, Penegar S, Whitehouse LE, et al. A retrospective observational study of the relationship between family history and survival from colorectal cancer. *Br J Cancer*. 2013;108: 1502-1507.
47. Hampel H, Frankel WL, Martin E, et al. Feasibility of screening for Lynch syndrome among patients with colorectal cancer. *J Clin Oncol*. 2008;26: 5783-5788.
48. Stoffel E, Mukherjee B, Raymond VM, et al. Calculation of risk of colorectal and endometrial cancer among patients with Lynch syndrome. *Gastroenterology*. 2009;137: 1621-1627.
49. Win AK, Lindor NM, Young JP, et al. Risks of primary extracolonic cancers following colorectal cancer in lynch syndrome. *J Natl Cancer Inst*. 2012;104: 1363-1372.
50. Mishra N, Hall J. Identification of patients at risk for hereditary colorectal cancer. *Clin Colon Rectal Surg*. 2012;25: 67-82.
51. Moreira L, Balaguer F, Lindor N, et al. Identification of Lynch syndrome among patients with colorectal cancer. *JAMA*. 2012;308: 1555-1565.
52. Ward RL, Hicks S, Hawkins NJ. Population-based molecular screening for Lynch syndrome: implications for personalized medicine. *J Clin Oncol*. 2013;31: 2554-2562.
53. Burn J, Mathers JC, Bishop DT. Chemoprevention in Lynch syndrome. *Fam Cancer*. 2013.
54. Burn J, Gerdes AM, Macrae F, et al. Long-term effect of aspirin on cancer risk in carriers of hereditary colorectal cancer: an analysis from the CAPP2 randomised controlled trial. *Lancet*. 2011;378: 2081-2087.
55. Jaspersion KW, Tuohy TM, Neklason DW, Burt RW. Hereditary and familial colon cancer. *Gastroenterology*. 2010;138: 2044-2058.
56. Galiatsatos P, Foulkes WD. Familial adenomatous polyposis. *Am J Gastroenterol*. 2006;101: 385-398.
57. Murff HJ, Greevy RA, Syngal S. The comprehensiveness of family cancer history assessments in primary care. *Community Genet*. 2007;10: 174-180.
58. Volk LA, Staroselsky M, Newmark LP, et al. Do physicians take action on high risk family history information provided by patients outside of a clinic visit? *Stud Health Technol Inform*. 2007;129: 13-17.
59. Mysliwiec PA, Cronin KA, Schatzkin A. Chapter 5: New Malignancies Following Cancer of the Colon, Rectum, and Anus. *New Malignancies Among Cancer Survivors: SEER Cancer Registries, 1973-2000*. Bethesda, MD: National Cancer Institute, 2006.
60. Phipps AI, Chan AT, Ogino S. Anatomic subsite of primary colorectal cancer and subsequent risk and distribution of second cancers. *Cancer*. 2013;119: 3140-3147.
61. Imperiale TF, Ransohoff DF. Risk for colorectal cancer in persons with a family history of adenomatous polyps: a systematic review. *Ann Intern Med*. 2012;156: 703-709.
62. Bernstein CN, Blanchard JF, Kliever E, Wajda A. Cancer risk in patients with inflammatory bowel disease: a population-based study. *Cancer*. 2001;91: 854-862.
63. Eaden JA, Abrams KR, Mayberry JF. The risk of colorectal cancer in ulcerative colitis: a meta-analysis. *Gut*. 2001;48: 526-535.
64. Jess T, Simonsen J, Jorgensen KT, Pedersen BV, Nielsen NM, Frisch M. Decreasing risk of colorectal cancer in patients with inflammatory bowel disease over 30 years. *Gastroenterology*. 2012;143: 375-381 e371; quiz e313-374.
65. Nguyen GC, Bressler B. A tale of two cohorts: are we overestimating the risk of colorectal cancer in inflammatory bowel disease? *Gastroenterology*. 2012;143: 288-290.
66. Rubin DT. The changing face of colorectal cancer in inflammatory bowel disease: progress at last! *Gastroenterology*. 2006;130: 1350-1352.
67. Larsson SC, Orsini N, Wolk A. Diabetes mellitus and risk of colorectal cancer: a meta-analysis. *J Natl Cancer Inst*. 2005;97: 1679-1687.
68. Campbell PT, Deka A, Jacobs EJ, et al. Prospective Study Reveals Associations Between Colorectal Cancer and Type 2 Diabetes Mellitus or Insulin Use in Men. *Gastroenterology*. 2010;139: 1138-46.
69. Larsson SC, Giovannucci E, Wolk A. Diabetes and colorectal cancer incidence in the cohort of Swedish men. *Diabetes Care*. 2005;28: 1805-1807.
70. Luo W, Cao Y, Liao C, Gao F. Diabetes mellitus and the incidence and mortality of colorectal cancer: a meta-analysis of 24 cohort studies. *Colorectal Dis*. 2012;14: 1307-1312.
71. Singh S, Singh H, Singh PP, Murad MH, Limburg PJ. Anti-Diabetic Medications and the Risk of Colorectal Cancer in Patients with Diabetes Mellitus: A Systematic Review and Meta-Analysis. *Cancer Epidemiol Biomarkers Prev*. 2013;22: 2258-2268.
72. Mills KT, Bellows CF, Hoffman AE, Kelly TN, Gagliardi G. Diabetes Mellitus and Colorectal Cancer Prognosis: A Meta-analysis. *Dis Colon Rectum*. 2013;56: 1304-1319.
73. Boyle T, Keegel T, Bull F, Heyworth J, Fritschi L. Physical activity and risks of proximal and distal colon cancers: a systematic review and meta-analysis. *J Natl Cancer Inst*. 2012;104: 1548-1561.
74. Campbell PT, Patel AV, Newton CC, Jacobs EJ, Gapstur SM. Associations of recreational physical activity and leisure time spent sitting with colorectal cancer survival. *J Clin Oncol*. 2013;31: 876-885.
75. Samad AK, Taylor RS, Marshall T, Chapman MA. A meta-analysis of the association of physical activity with reduced risk of colorectal cancer. *Colorectal Dis*. 2005;7: 204-213.
76. Chao A, Connell CJ, Jacobs EJ, et al. Amount, type, and timing of recreational physical activity in relation to colon and rectal cancer in older adults: the Cancer Prevention Study II Nutrition Cohort. *Cancer Epidemiol Biomarkers Prev*. 2004;13: 2187-2195.

77. Centers for Disease Control and Prevention. Early Release of Selected Estimates Based on Data From the 2012 National Health Interview Survey: National Center for Health Statistics, 2013.
78. Renehan AG, Tyson M, Egger M, Heller RF, Zwahlen M. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. *Lancet*. 2008;371: 569-578.
79. Larsson SC, Wolk A. Obesity and colon and rectal cancer risk: a meta-analysis of prospective studies. *Am J Clin Nutr*. 2007;86: 556-565.
80. Aleksandrova K, Pischon T, Buijsse B, et al. Adult weight change and risk of colorectal cancer in the European Prospective Investigation into Cancer and Nutrition. *Eur J Cancer*. 2013;49: 3526-3536.
81. Bardou M, Barkun AN, Martel M. Obesity and colorectal cancer. *Gut*. 2013;62: 933-947.
82. Norat T, Bingham S, Ferrari P, et al. Meat, fish, and colorectal cancer risk: the European Prospective Investigation into cancer and nutrition. *J Natl Cancer Inst*. 2005;97: 906-916.
83. Cross AJ, Ferrucci LM, Risch A, et al. A large prospective study of meat consumption and colorectal cancer risk: an investigation of potential mechanisms underlying this association. *Cancer Res*. 2010;70: 2406-2414.
84. Aune D, Chan DS, Lau R, et al. Dietary fibre, whole grains, and risk of colorectal cancer: systematic review and dose-response meta-analysis of prospective studies. *BMJ*. 2011;343: d6617.
85. Aune D, Lau R, Chan DS, et al. Nonlinear reduction in risk for colorectal cancer by fruit and vegetable intake based on meta-analysis of prospective studies. *Gastroenterology*. 2011;141: 106-118.
86. Lee JE, Chan AT. Fruit, vegetables, and folate: cultivating the evidence for cancer prevention. *Gastroenterology*. 2011;141: 16-20.
87. Aune D, Lau R, Chan DS, et al. Dairy products and colorectal cancer risk: a systematic review and meta-analysis of cohort studies. *Ann Oncol*. 2012;23: 37-45.
88. Murphy N, Norat T, Ferrari P, et al. Consumption of Dairy Products and Colorectal Cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC). *PLoS One*. 2013;8: e72715.
89. Touvier M, Chan DS, Lau R, et al. Meta-analyses of vitamin D intake, 25-hydroxyvitamin D status, vitamin D receptor polymorphisms, and colorectal cancer risk. *Cancer Epidemiol Biomarkers Prev*. 2011;20: 1003-1016.
90. Sanjoaquin MA, Allen N, Couto E, Roddam AW, Key TJ. Folate intake and colorectal cancer risk: a meta-analytical approach. *Int J Cancer*. 2005;113: 825-828.
91. Cole BF, Baron JA, Sandler RS, et al. Folic acid for the prevention of colorectal adenomas: a randomized clinical trial. *JAMA*. 2007;297: 2351-2359.
92. Mason JB, Dickstein A, Jacques PF, et al. A temporal association between folic acid fortification and an increase in colorectal cancer rates may be illuminating important biological principles: a hypothesis. *Cancer Epidemiol Biomarkers Prev*. 2007;16: 1325-1329.
93. Stevens VL, McCullough ML, Sun J, Jacobs EJ, Campbell PT, Gapsur SM. High levels of folate from supplements and fortification are not associated with increased risk of colorectal cancer. *Gastroenterology*. 2011;141: 98-105, 105 e101.
94. Secretan B, Straif K, Baan R, et al. A review of human carcinogens – Part E: tobacco, areca nut, alcohol, coal smoke, and salted fish. *Lancet Oncol*. 2009;10: 1033-1034.
95. Liang PS, Chen TY, Giovannucci E. Cigarette smoking and colorectal cancer incidence and mortality: systematic review and meta-analysis. *Int J Cancer*. 2009;124: 2406-2415.
96. 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: 1729-1735.
97. Giovannucci E. An updated review of the epidemiological evidence that cigarette smoking increases risk of colorectal cancer. *Cancer Epidemiol Biomarkers Prev*. 2001;10: 725-731.
98. Limsui D, Vierkant RA, Tillmans LS, et al. Cigarette smoking and colorectal cancer risk by molecularly defined subtypes. *J Natl Cancer Inst*. 2010;102: 1012-1022.
99. Cho E, Smith-Warner SA, Ritz J, et al. Alcohol intake and colorectal cancer: a pooled analysis of 8 cohort studies. *Ann Intern Med*. 2004;140: 603-613.
100. 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: 2065-2072.
101. Flossmann E, Rothwell PM. Effect of aspirin on long-term risk of colorectal cancer: consistent evidence from randomised and observational studies. *Lancet*. 2007;369: 1603-1613.
102. Rothwell PM, Wilson M, Elwin CE, et al. Long-term effect of aspirin on colorectal cancer incidence and mortality: 20-year follow-up of five randomised trials. *Lancet*. 2010.
103. Chan AT, Giovannucci EL, Meyerhardt JA, Schernhammer ES, Curhan GC, Fuchs CS. Long-term use of aspirin and nonsteroidal anti-inflammatory drugs and risk of colorectal cancer. *JAMA*. 2005;294: 914-923.
104. Cook NR, Lee IM, Zhang SM, Moorthy MV, Buring JE. Alternate-day, low-dose aspirin and cancer risk: long-term observational follow-up of a randomized trial. *Ann Intern Med*. 2013;159: 77-85.
105. Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results From the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288: 321-333.
106. Chlebowski RT, Wactawski-Wende J, Ritenbaugh C, et al. Estrogen plus progestin and colorectal cancer in postmenopausal women. *N Engl J Med*. 2004;350: 991-1004.
107. Heiss G, Wallace R, Anderson GL, et al. Health risks and benefits 3 years after stopping randomized treatment with estrogen and progestin. *JAMA*. 2008;299: 1036-1045.
108. Hildebrand JS, Jacobs EJ, Campbell PT, et al. Colorectal cancer incidence and postmenopausal hormone use by type, recency, and duration in cancer prevention study II. *Cancer Epidemiol Biomarkers Prev*. 2009;18: 2835-2841.
109. Bosetti C, Bravi F, Negri E, La Vecchia C. Oral contraceptives and colorectal cancer risk: a systematic review and meta-analysis. *Hum Reprod Update*. 2009;15: 489-498.
110. Thosani N, Thosani SN, Kumar S, et al. Reduced risk of colorectal cancer with use of oral bisphosphonates: a systematic review and meta-analysis. *J Clin Oncol*. 2013;31: 623-630.
111. Levin B, Lieberman DA, McFarland B, et al. Screening and surveillance for the early detection of colorectal cancer and adenomatous Polyps, 2008: a joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. *CA Cancer J Clin*. 2008;58: 130-160.
112. Schoen RE, Pinsky PF, Weissfeld JL, et al. Colorectal-cancer incidence and mortality with screening flexible sigmoidoscopy. *N Engl J Med*. 2012;366: 2345-2357.
113. Nishihara R, Wu K, Lochhead P, et al. Long-term colorectal-cancer incidence and mortality after lower endoscopy. *N Engl J Med*. 2013;369: 1095-1105.

114. Atkin WS, Edwards R, Kralj-Hans I, et al. Once-only flexible sigmoidoscopy screening in prevention of colorectal cancer: a multicentre randomised controlled trial. *Lancet*. 2010;375: 1624-1633.
115. Rockey DC, Paulson E, Niedzwiecki D, et al. Analysis of air contrast barium enema, computed tomographic colonography, and colonoscopy: prospective comparison. *Lancet*. 2005;365: 305-311.
116. Zauber AG, Winawer SJ, O'Brien MJ, et al. Colonoscopic polypectomy and long-term prevention of colorectal-cancer deaths. *N Engl J Med*. 2012;366: 687-696.
117. Heresbach D, Barrioz T, Lapalus MG, et al. Miss rate for colorectal neoplastic polyps: a prospective multicenter study of back-to-back video colonoscopies. *Endoscopy*. 2008;40: 284-290.
118. Johnson CD, Chen MH, Toledano AY, et al. Accuracy of CT colonography for detection of large adenomas and cancers. *N Engl J Med*. 2008;359: 1207-1217.
119. Pickhardt PJ, Choi JR, Hwang I, et al. Computed tomographic virtual colonoscopy to screen for colorectal neoplasia in asymptomatic adults. *N Engl J Med*. 2003;349: 2191-2200.
120. Zauber AG, Lansdorp-Vogelaar I, Knudsen AB, Wilschut J, van Ballegooijen M, Kuntz KM. *Evaluating Test Strategies for Colorectal Cancer Screening—Age to Begin, Age to Stop, and Timing of Screening Intervals: A Decision Analysis of Colorectal Cancer Screening for the U.S. Preventive Services Task Force from the Cancer Intervention and Surveillance Modeling Network (CISNET)*. Rockville (MD), 2009.
121. Gellad ZF, Stechuchak KM, Fisher DA, et al. Longitudinal adherence to fecal occult blood testing impacts colorectal cancer screening quality. *Am J Gastroenterol*. 2011;106: 1125-1134.
122. Liss DT, Petit-Homme A, Feinglass J, Buchanan DR, Baker DW. Adherence to repeat fecal occult blood testing in an urban community health center network. *J Community Health*. 2013;38: 829-833.
123. Smith RA, Cokkinides V, Brawley OW. Cancer screening in the United States, 2009: a review of current American Cancer Society guidelines and issues in cancer screening. *CA Cancer J Clin*. 2009;59: 27-41.
124. Chubak J, Bogart A, Fuller S, Laing SS, Green BB. Uptake and positive predictive value of fecal occult blood tests: A randomized controlled trial. *Prev Med*. 2013;57: 671-678.
125. Shaikat A, Mongin SJ, Geisser MS, et al. Long-term mortality after screening for colorectal cancer. *N Engl J Med*. 2013;369: 1106-1114.
126. 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: 1603-1607.
127. Nadel MR, Shapiro JA, Klabunde CN, et al. A national survey of primary care physicians' methods for screening for fecal occult blood. *Ann Intern Med*. 2005;142: 86-94.
128. Nadel MR, Berkowitz Z, Klabunde CN, Smith RA, Coughlin SS, White MC. Fecal occult blood testing beliefs and practices of U.S. primary care physicians: serious deviations from evidence-based recommendations. *J Gen Intern Med*. 2010;25: 833-839.
129. Collins JF, Lieberman DA, Durbin TE, Weiss DG. Accuracy of screening for fecal occult blood on a single stool sample obtained by digital rectal examination: a comparison with recommended sampling practice. *Ann Intern Med*. 2005;142: 81-85.
130. Centers for Disease Control and Prevention. Vital Signs: Colorectal Cancer Screening Test Use – United States, 2012. *MMWR Morb Mortal Wkly Rep*. 2013;62: 881-888.
131. Inadomi JM, Vijan S, Janz NK, et al. Adherence to colorectal cancer screening: a randomized clinical trial of competing strategies. *Arch Intern Med*. 2012;172: 575-582.
132. Gupta S, Halm EA, Rockey DC, et al. Comparative Effectiveness of Fecal Immunochemical Test Outreach, Colonoscopy Outreach, and Usual Care for Boosting Colorectal Cancer Screening Among the Underserved: A Randomized Clinical Trial. *JAMA Intern Med*. 2013;173: 1725-32.
133. Schenck AP, Peacock SC, Klabunde CN, Lapin P, Coan JF, Brown ML. Trends in colorectal cancer test use in the medicare population, 1998-2005. *Am J Prev Med*. 2009;37: 1-7.
134. National Center for Health Statistics. *Health, United States, 2012: With Special Feature on Emergency Care*. Hyattsville, MD: 2013.
135. Beydoun HA, Beydoun MA. Predictors of colorectal cancer screening behaviors among average-risk older adults in the United States. *Cancer Causes Control*. 2008;19: 339-359.
136. Holden DJ, Jonas DE, Porterfield DS, Reuland D, Harris R. Systematic review: enhancing the use and quality of colorectal cancer screening. *Ann Intern Med*. 2010;152: 668-676.
137. Doubeni CA, Laiyemo AO, Young AC, et al. Primary care, economic barriers to health care, and use of colorectal cancer screening tests among Medicare enrollees over time. *Ann Fam Med*. 2010;8: 299-307.
138. Farmer MM, Bastani R, Kwan L, Belman M, Ganz PA. Predictors of colorectal cancer screening from patients enrolled in a managed care health plan. *Cancer*. 2008;112: 1230-1238.
139. DeBourcy AC, Lichtenberger S, Felton S, Butterfield KT, Ahnen DJ, Denberg TD. Community-based preferences for stool cards versus colonoscopy in colorectal cancer screening. *J Gen Intern Med*. 2008;23: 169-174.
140. Sequist TD, Zaslavsky AM, Marshall R, Fletcher RH, Ayanian JZ. Patient and physician reminders to promote colorectal cancer screening: a randomized controlled trial. *Arch Intern Med*. 2009;169: 364-371.
141. O'Malley AS, Beaton E, Yabroff KR, Abramson R, Mandelblatt J. Patient and provider barriers to colorectal cancer screening in the primary care safety-net. *Prev Med*. 2004;39: 56-63.
142. Green BB, Wang CY, Anderson ML, et al. An automated intervention with stepped increases in support to increase uptake of colorectal cancer screening: a randomized trial. *Ann Intern Med*. 2013;158: 301-311.
143. Myers RE, Bittner-Fagan H, Daskalakis C, et al. A randomized controlled trial of a tailored navigation and a standard intervention in colorectal cancer screening. *Cancer Epidemiol Biomarkers Prev*. 2013;22: 109-117.
144. Sargent D, Sobrero A, Grothey A, et al. Evidence for cure by adjuvant therapy in colon cancer: observations based on individual patient data from 20,898 patients on 18 randomized trials. *J Clin Oncol*. 2009;27: 872-877.
145. Abraham A, Habermann EB, Rothenberger DA, et al. Adjuvant chemotherapy for stage III colon cancer in the oldest old: results beyond clinical guidelines. *Cancer*. 2013;119: 395-403.
146. Wang G, Kelley RK, Gappnet. KRAS mutational analysis for colorectal cancer: Application: Pharmacogenomic. *PLoS Curr*. 2010.
147. Siegel R, DeSantis C, Virgo K, et al. Cancer Treatment and Survivorship Statistics, 2012. *CA Cancer J Clin*. 2012;62: 220-241.
148. Zhu L, Pickle LW, Ghosh K, et al. Predicting US- and state-level cancer counts for the current calendar year: Part II: evaluation of spatio-temporal projection methods for incidence. *Cancer*. 2012;118: 1100-1109.
149. Chen HS, Portier K, Ghosh K, et al. Predicting US- and state-level cancer counts for the current calendar year: Part I: evaluation of temporal projection methods for mortality. *Cancer*. 2012;118: 1091-1099.

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(510) 893-7900

East Central Division (OH, PA)

Route 422 and Sipe Avenue
PO Box 897
Hershey, PA 17033-0897
(717) 533-6144

Eastern Division (NJ, NY)

6725 Lyons Street
East Syracuse, NY 13057-9332
(315) 433-5603

Florida Division (including Puerto Rico operations)

3709 West Jetton Avenue
Tampa, FL 33629-5146
(813) 253-0541

Puerto Rico

Calle Cabo Alverio #577
Esquina Sargento Medina
Hato Rey, PR 00918
(787) 764-2295

Great West Division

**(AK, AZ, CO, ID, MT, ND, NM,
NV, OR, UT, WA, WY)**
2120 First Avenue North
Seattle, WA 98109-1140
(206) 283-1153

High Plains Division (including Hawaii operations, KS, MO, NE, OK, TX)

2433 Ridgpoint Drive
Austin, TX 78754-5231
(512) 919-1900

Hawaii Pacific Division

2370 Nuuana Avenue
Honolulu, HI 96817-1778
(808) 595-7500

Lakeshore Division (IL, IN, MI)

1755 Abbey Road
East Lansing, MI 48823-1907
(517) 332-2222

Mid-South Division (AL, AR, KY, LA, MS, TN)

1100 Ireland Way
Suite 300
Birmingham, AL 35205-7014
(205) 930-8860

Midwest Division

(IA, MN, SD, WI)
2520 Pilot Knob Road
Suite 150
Mendota Heights, MN 55120-
1158
(615) 255-8100

New England Division (CT, ME, MA, NH, RI, VT)

30 Speen Street
Framingham, MA 01701-9376
(508) 270-4698

South Atlantic Division (DE, GA, MD, NC, SC, VA, Washington, DC, WV)

250 Williams Street
Atlanta, GA 30303-1002
(404) 816-7800



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