Overview of Gastrointestinal Cancers

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CHAPTER 1

Introduction

Cancers of the gastrointestinal (GI) tract include those of the esophagus, stomach, colon, rectum, and anus. Risk factors, incidence, prevalence, and prognosis vary for each site. Many of the risk factors are modifiable, and in a significant number of cases, the cancers are preventable with proper screening and curable when detected and treated at an early stage.

Esophageal cancer is a treatable but rarely curable cancer. Incidence has increased over the past 20 years in the United States, particularly adenocarcinomas of the esophagus, which have increased by 450% in Caucasian men and 50% in African American men (Enzinger & Mayer, 2003). Squamous cell carcinomas are more common in the rest of the world. Treatment involves surgical resection, whenever possible, followed by chemotherapy and radiotherapy. Men and African Americans have a higher incidence of esophageal cancer than do other groups (Posner, Forastiere, & Minsky, 2005).

Gastric cancer ranks second in cancer deaths across the world. In the United States, incidence has been decreasing dramatically and currently occupies 14th place among cancer incidences (American Cancer Society [ACS], 2006). Adenocarcinomas represent 90%–95% of all gastric malignancies. Over the past 25 years, the number of gastric cancers occurring at the gastroesophageal (GE) junction and in the cardia has dramatically increased. Treatment for gastric cancer is surgery, followed by chemotherapy and radiotherapy (Pisters, Kelsen, Powell, & Tepper, 2005).

Colorectal cancer (CRC), a preventable, highly treatable, and often curable cancer, is common in men and women. Surgery is the primary form of treatment and results in cure in more than 50% of the patients. Local or distant recurrences develop in many patients after surgical resection, and those with the highest risk of recurrence receive chemotherapy. More than half of the patients are diagnosed at stage III or with metastatic disease, and minorities are diagnosed more often at advanced stages of cancer than Caucasians (Xiong & Ajani, 2004). For the four major cancer sites (prostate, female breast, lung, and CRC), minority populations have a greater risk of cancer death than other groups (Clegg, Li, Hankey, Chu, & Edwards, 2002).

Anal cancers are uncommon, representing 4% of all cancers (Ryan, Compton, & Mayer, 2000). In the United States, squamous cell carcinomas are the most common histology. The risk of developing anal cancers is rising, with people engaging in receptive anal intercourse and with those having a higher number of lifetime sexual partners being at higher risk (Cummings, Ajani, & Swallow, 2005). A strong correlation exists between human papillomavirus (HPV) infection and anal cancer. Treatment consists of surgery, radiation, and chemotherapy (Cummings et al.).

Incidence and Demographics

CRC is the third most common type of cancer in men and women, with an estimated 106,680 cases of colon and 41,930 cases of rectal cancer expected to occur in 2006 (ACS, 2006). Incidence and mortality rates are greatest in developed Western nations (Van Cutsem & Costa, 2005; Wilkes, 2005). Peak incidence is in individuals older than 60, with more women than men developing colon cancer and more men than women acquiring rectal cancer (Wilkes). Seventy percent of patients present with localized disease, and surgery can be curative in these individuals. However, relapses after complete resection are frequent (Van Cutsem & Costa). Polyp removal and fecal occult blood testing reduced the incidence of CRC by 75% and 20%, respectively (Mandel et al., 2000; Winawer et al., 1993).

Individuals residing in high poverty areas are at an increased risk for developing or dying from cancer (Clegg et al., 2002). ACS estimated that more than 16,000 diagnoses of CRC occurred in African Americans in 2005 (Jemal et al., 2005). African Americans have the highest incidence of CRC of any group, and survival rates are lower than for Caucasians (Agrawal et al., 2005). In an analysis of 57,000 patients
with rectal cancer entered into the National Cancer Institute (NCI)-funded Surveillance, Epidemiology and End Results (SEER) Cancer Incidence Database. African Americans with rectal cancer were diagnosed at a younger age (mean age 64) than Caucasians (mean age 69) (Morris, Billingsley, Baxter, & Baldwin, 2004). African Americans were diagnosed at a more advanced stage of disease than Caucasians (p < .001); Caucasians were more likely to have sphincter-preserving surgeries than African Americans.

The estimated incidences of other GI cancers in 2006 are 4,660 cases of anal cancer, with 660 deaths; 14,550 new cases of esophageal cancer, with 13,770 deaths; and 22,280 cases of stomach cancer, with 11,430 deaths (Jemal et al., 2006). Esophageal cancer is uncommon in the United States, with a lifetime risk of less than 1%. However, the risk is increasing, along with a shift in histology type and tumor location. Adenocarcinoma is now more prevalent than squamous cell, with a shift to the GE junction and cardia. African American men are more commonly diagnosed with squamous cell carcinomas, and incidence rates in this demographic have substantially decreased from a peak in the 1980s, along with a steady decrease in mortality. Despite this progress, death rates from esophageal cancer in African American men continue to exceed those of all other populations combined. Conversely, incidence in Caucasian men has increased. Esophageal cancer is five times more common in Caucasians than African Americans, and the ratio of male to female incidence is 7:1. Particularly alarming is an increase in the number of adenocarcinomas by more than 400% during the past 20 years; and in Caucasian women, incidence has increased by more than 300%. These changes in incidence are thought to be related to increased rates of both GE reflux and obesity. Survival rates at five years from esophageal cancer are poor, ranging from 5%–30% (Posner et al., 2005).

Gastric cancer has a worldwide incidence of 875,000 new cases annually but is gradually decreasing in many parts of the world because of dietary and food preparation changes. The United States has seen a dramatic decline in gastric cancer to seventh in cancer-related deaths. It continues to be endemic in Japan, Eastern Europe, and South America. It is twice as common in men than in women and 1.5 times more common in African American men than Caucasian men. Mortality has declined in Japan because of mass screening. Death rates are highest for African American men, followed by Caucasian men, African American women, and Caucasian women, in that order. Gastric cancers are being diagnosed more commonly at the proximal stomach and GE junction (Pisters et al., 2005).

Anal cancers are one-tenth as common as rectal cancers, and in North America, 80% are squamous cell carcinomas. Median age at diagnosis is 60–65 years, and it is more common in women. Incidence of the disease is increasing in Caucasian men. Anal cancer is associated with infection by HPV (Ryan et al., 2000). Risk factors include a history of multiple sexual partners in homosexual or heterosexual relationships and receptive anal intercourse. Individuals who are immunosuppressed also are at an increased risk (Cummings et al., 2005).

Race, Ethnicity, and Poverty

NCI (2005) defined health disparities as differences in the incidence, prevalence, mortality, and burden of cancer related adverse health conditions that exist among specific population groups in the United States. A benchmarking document in cancer health disparities from NCI noted that underserved populations are more likely to be diagnosed and die from preventable cancers, be diagnosed with late-stage disease with cancers for which screening is available for early detection, and receive treatment that does not meet acceptable standards of care (NCI, 2005).

African Americans have overall higher cancer incidence and mortality rates compared to other groups. Socioeconomic status may play a large part in these and other disparities, with poverty affecting the likelihood of developing cancer and securing appropriate and timely treatment (NCI, 2005).

CRC is the second most common cancer in African American women and the third most common in African American men (ACS, 2005b). Incidence rates of CRC in African American men and women, while stabilized since 1975, remain higher than in Caucasians (ACS, 2005a). A disproportionate number of cancer deaths occur among racial and ethnic minorities, especially African Americans, who have a 33% higher risk of dying than Caucasians and are twice as likely to die of cancer as Asians and Hispanics (Shavers & Brown, 2002). When looking at cancer among the five major racial and ethnic groups in the United States, African Americans have the highest overall risk of developing cancer, the highest overall risk of dying from cancer, and the poorest indices of cancer survival (Underwood, Powe, Canales, Meade, & Im, 2004). African Americans are diagnosed with CRC at a younger age than Caucasians, which has implications for screening guidelines (Agrawal et al., 2005). More than 7,000 deaths from CRC are expected to occur among African Americans in 2005, and it is the third leading cause of cancer deaths (ACS, 2005a). SEER data from 1998–2002 showed that incidences of CRC in Asian/Pacific Islanders, Native Americans, and Hispanics are lower than that of Caucasians or African Americans (Edwards et al., 2005).

The Institute of Medicine (IOM) documented the disproportionate cancer incidence and death rates in African Americans, regardless of economic and health insurance status, and their lesser chances of receiving the most curative treatments (Smedley, Stith, & Nelson, 2002). The report described a complex interplay among economic, social, and cultural factors. A study of 4,706 patients with CRC showed that Caucasians received standard adjuvant therapy significantly more often than African Americans (Potosky, Harlan, Kaplan, Johnson, &
Lynch, 2002; Shavers & Brown, 2002). Similar findings have been demonstrated in lung cancer (Bach, Cramer, Warren, & Begg, 1999) and in receipt of intensive care in general (Fiscella & Franks, 2000; Fiscella, Franks, Gold, & Clancy, 2000). A comprehensive literature review found limited evidence of differences in treatment effectiveness among racial or ethnic groups but only in receipt of definitive therapies (Shavers & Brown). Freeman (2004) asserted that racial and ethnic bias of healthcare professionals influences the quality of healthcare delivery. Survival rates are lower for African Americans than Caucasians who present with the same disease stage (Potosky et al.). In an equal-access system such as the U.S. Veterans Affairs Health Care System, no survival difference exists between African Americans and Caucasians (Dominitz, Samsa, Landsman, & Provenzale, 1998).

Many African Americans, including older adults and those living in rural and medically underserved communities, do not have ready access to quality cancer care facilities (Underwood et al., 2004). Rural communities, where 14% of African Americans reside, have limited access to quality cancer care; urban communities, with 55% of the African American population, have a shortage of healthcare providers and services. An analysis of doctor visits by Medicare beneficiaries older than 65 years of age found that African Americans were treated by physicians who were less likely to be board certified, and those physicians reported greater difficulties in obtaining specialized care for their patients (Bach, Pham, Schrag, Tate, & Hargraves, 2004). Some authors suggested that African Americans and Hispanics are adequately represented in clinical trials (Hutchins, Unger, Crowley, Coltman, & Albain, 1999), whereas others have noted that racial and ethnic minorities and older adults are less likely to enroll in those trials (Murthy, Krumholz, & Gross, 2004).

In 2002, the U.S. census reported 12% of Americans were poor, 15% were uninsured, and a disproportionate percentage of minorities were poor (Proctor & Dalaker, 2003). Freeman (2004) reported that 20% of African Americans and 32% of Hispanics are uninsured. Poverty is associated with poorer cancer outcomes, regardless of race or ethnic group (Muss, 2001; Wrigley et al., 2003). Five-year survival rates from cancer are 10% lower for those living in poverty (Ward et al., 2004). In a study of 4,675 women with breast cancer, a 49% higher risk of death was found for uninsured people than for those privately insured, and a 40% higher risk of death was reported for Medicaid patients even after adjusting for age, race, income, comorbidities, and stage of disease (Ayanian, Kohler, Abe, & Epstein, 1993).

A group of more than 1,000 patients with CRC was surveyed about their cancer care after nine months of treatment (Ayanian et al., 2005). Those reporting more problems with obtaining health or treatment information, psychosocial care, or coordination of care were more likely to be Asian/Pacific Islander, Hispanic, and African American. Patients who were non-Caucasian and non-English-speaking were less likely to rate their quality of cancer care as very good or excellent. Problems with coordination of care were most strongly correlated with poor quality-of-care ratings. Because differences exist in cancer treatment and outcomes by race, ethnicity, and language (Smedley et al., 2002), perhaps these interpersonal and communication-related problems may be contributory.

**Age**

The incidence of all GI cancers, as with many other cancers, increases with age. The U.S. Census Bureau estimated that 12.6% of the population is 65 and older, and this percentage is projected to increase to 20.3%, or 70 million people, by 2030 (Kinsella & Velkoff, 2001). Cancer occurs more frequently and causes more deaths in older adults. Sixty percent of malignancies occur in people older than 65, but they receive adjuvant chemotherapy less often than younger people (Sargent et al., 2001). In a medical review of 4,706 people with CRC, those younger than age 55 received standard therapy 78% of the time, whereas those older than 80 received standard therapy only 24% of the time (Potosky et al., 2002). This sharp decline in the use of standard chemotherapy, beginning at the age of 75, is suggestive of age bias even after adjusting for tumor grade and comorbidities. A growing body of knowledge suggests that chemotherapy can be safe and effective in older adults. For many types of cancer chemotherapy treatment, no difference in toxicities is seen in people older than 70 years (Muss, 2001). A meta-analysis of phase III randomized trials in CRC involving 3,351 patients demonstrated that overall survival from CRC was higher in older adults who received adjuvant therapy than those who were untreated, with no significantly higher incidence of GI symptoms, leukopenia, or stomatitis compared to younger people (Sargent et al.). Other studies supported that older adults tolerate and respond to chemotherapy (Benson et al., 1991; Giovanazzi-Bannon, Rademaker, Lai, & Benson, 1994).

Conversely, some studies have suggested that older adults with cancer may be more susceptible to toxicities such as myelosuppression, GI distress, renal impairment, and cardio toxicities (Crivellari et al., 2000; Morrison et al., 2001). When compared to younger patients, older adults with cancer develop myelosuppression more frequently and with greater severity (Morrison et al.), are more susceptible to cardiotoxicity (Kimmick, Fleming, Muss, & Balducci, 1997), and are at increased risk for GI distress such as mucositis, nausea, vomiting, and diarrhea. However, it is important to note that some of those studies included few older adults, were reviews of the literature, or were based on anecdotal information.

Older adults are underrepresented in cancer clinical trials (Hutchins et al., 1999). Eligibility criteria of standard clinical trials often exclude many comorbidities, and little is known about the ability of older adults with normal age-related organ impairments to tolerate treatment. Similarly, physicians may not recommend participation in clinical trials because
of assumptions about the ability of this population to withstand cancer treatments. Benson et al. (1991) reported that in a survey of American oncologists, 50% decided whether a patient was unsuitable for a clinical trial based on age alone. Family members also believed the trials might be too toxic for older patients (Benson et al.). A retrospective analysis of 55 cancer clinical trials reported a statistically significant underrepresentation of older adults (Talarico, Chen, & Pazdur, 2004). This underrepresentation was found in all cancers except for hormonal treatments for breast cancer and was most predominant for those older than 70 years of age.

### Risk Factors

Many risk factors for GI cancers are modifiable, although the most research has been conducted on CRC. Approximately 90% of all instances of CRC and deaths are thought to be preventable through lifestyle changes (Herdman & Lichtenfeld, 2004). Etiologies of CRC have been studied extensively, and diet, physical activity, weight, and smoking have well-documented links to its development. The highly complex interactions between inherited susceptibility and external factors are under active investigation to determine the most effective prevention strategies.

#### Personal and Familial Risk

Average-risk individuals, with no familial history of cancer or polyps, comprise the majority of those who develop CRC. Intermediate-risk individuals have a personal or familial history of colorectal polyps or CRC, placing them at higher risk for the disease. People with a history of a colon cancer are at an increased risk for a second (metachronous) cancer, in addition to risk for recurrence (Winawer et al., 1996). Patients with a familial risk, those who have two or more first- or second-degree relatives with CRC or adenomatous polyps, represent approximately 20% of all cases of the disease. They have approximately twice the risk of developing CRC as someone without a family history. The risk is higher if more than one first-degree relative is affected (Winawer et al., 1996). High-risk individuals are those with a genetic syndrome, which is discussed later.

#### Diet

Drawing conclusions from dietary studies is difficult because recall is imperfect, and amounts of food or methods of cooking rarely are included in the database. Additionally, childhood dietary data rarely are included in studies examining dietary effect on CRC incidence, which is a significant limitation to these data (Willett, 2005). However, migrant and other studies have shown that risk for CRC is modifiable and related to lifestyle factors (Miller et al., 1996). Colon cancer risk changes as people move from low- to high-incidence areas, demonstrating the importance of adult environmental exposure.

#### Meat and Fat

In several large, well-designed studies, including a meta-analysis and the Nurses’ Health Study, high meat consumption was related to the development of CRC (Chao et al., 2005; Norat, Lukanova, Ferrari, & Riboli, 2002; Willett, Stampfer, Colditz, Rosner, & Speizer, 1990). The fatty acid content of red meat may be particularly harmful, and promoters of carcinogenesis may be formed when it is cooked (Giovannucci et al., 1995). No increase in risk with meat consumption was seen in two other large studies (Bostick et al., 1993; Thun et al., 1992). High fat intake increased the risk of adenoma recurrence in one study (Neugut et al., 1993), but another found that a low-fat, high-fiber diet did not affect recurrence rate (Schatzkin et al., 2000). A high dietary glycemic load is associated with an increased risk of CRC (Higginbotham et al., 2004). This effect may be related to a relationship between hyperinsulinemia and insulin resistance and tumor growth. Dietary and lifestyle risk factors for developing insulin resistance, such as physical inactivity, obesity, and positive energy balance, also increase the risk of developing CRC and other cancers (Colditz, Canncusio, & Frazier, 1997; Giovannucci et al., 1995; Giovannucci, Colditz, Stampfer, & Willett, 1996).

#### Fruits and Vegetables

Despite common preconceptions, diets high in fiber, fruits, and vegetables have not decreased the rate of adenoma recurrence or overall cancer incidence (Fuchs et al., 1999). The Nurses’ Health Study, with a sample size of 71,910 women, and the Health Professionals Follow-Up Study, with a sample size of 37,725, found no association between fruit and vegetable intake and colon or rectal cancer incidence (Hung et al., 2004). Trials incorporating large doses of fruits and vegetables and beta-carotene also failed to decrease cancer incidence and actually suggested a harmful effect (Willett, 2005). Studies looking at dietary fiber, specifically, have yielded mixed results. No association between fiber intake and CRC was found in a number of large studies (Fuchs et al., 1999; Michels et al., 2000). Other studies have found an inverse relationship between dietary fiber and adenomas (Bingham et al., 2003; Peters et al., 2003). In other studies, the protective effect of fruits and vegetables, particularly those eaten raw, has been shown in esophageal cancer (Posner et al., 2005).

#### Calcium

In some studies, calcium supplementation decreased the risk of all types of polyps, both malignant and benign. Total calcium intake above 1,200 mg is necessary. Subjects with a high intake of total dietary fiber experienced more pronounced effects of calcium supplementation than those with lower fiber intake (Wallace et al., 2004). A higher consump-
tion of calcium products, including dairy, is associated with a lower risk of CRC and polyp recurrences (Baron et al., 1999; Bonithon-Kopp, Kronborg, Giacosa, Rath, & Faivre, 2000; Cho et al., 2004). This inverse association has been consistent across studies and gender. Combinations of calcium and vitamin D may be the preferred preventive regimen. However, a recent report from a randomized trial of more than 36,000 postmenopausal women from the Women’s Health Initiative (WHI) demonstrated that daily supplementation of calcium carbonate with vitamin D for seven years had no effect on the incidence of CRC (Wactawski-Wende et al., 2006). Fifty-four percent of the women were taking calcium supplements at the start of the study, which raises questions about the impact of the intervention. Another limitation was that the average length of intervention was 7 years, which is less than the 10–20 years estimated for the development of CRC. Other possible dietary factors that positively influence risk for CRC include multivitamin and folic acid (Fuchs et al., 2002; Giovannucci et al., 1998).

**Nonsteroidal Anti-Inflammatory Drugs**

Use of anti-inflammatory drugs, particularly aspirin, sulindac, and nonsteroidal anti-inflammatory drugs (NSAIDs), is associated with a decreased incidence of CRC (Thun, Namboodiri, Calle, Flanders, & Heath, 1993; Thun, Namboodiri, & Heath, 1991). Studies in chemoprevention in esophageal and gastric cancers suggest a similar preventive effect (Farrow et al., 1998). These drugs have been found to prevent adenoma formation and to cause adenomatous polyps to regress in patients with histories of CRC, polyp formation, and familial adenomatous polyposis (FAP) (NCI, 2005).

Taking aspirin daily for as little as three years was shown to reduce the development of polyps by 19%–35% in people at high risk for CRC in two randomized clinical trials, confirming numerous earlier observational studies suggesting that regular use of aspirin lowered rates of colorectal adenomas (Baron, Cole, & Sandler, 2003; Sandler et al., 2003). In a large study of healthcare professionals, aspirin was associated with a 30% reduction in CRC and a 50% reduction in advanced cases (Giovannucci, Rimm, Stampfer, Colditz, et al., 1994). Among a group of more than 600,000 adults enrolled in an ACS study, mortality in regular users of aspirin was approximately 40% lower for cancers of the colon and rectum (Smalley, Ray, Daugherty, & Griffin, 1999; Thun et al., 1991). Another large study found no decrease in CRC at 5 years and later at 12 years (Gann, Manson, Glynn, Buring, & Hennekens, 1993; Smalley et al.; Sturmer et al., 1998). At present, the minimal effective dose and duration of use of aspirin is not well defined. In the Nurses’ Health Study, beneficial effects were not obvious until after two decades of regular aspirin consumption. Risks of treatment include upper GI bleeding, gastric distress, and hemorrhagic stroke (Baron et al., 2003; Sandler et al.).

Cyclooxygenase (COX) has an inflammatory effect biologically. COX-2 is consistently overexpressed in a large percentage and variety of human tumors, including many CRCs. COX inhibitors include indomethacin, sulindac, piroxicam, ibuprofen, and celecoxib. COX-2 inhibitors are being studied as potential agents in the prevention and treatment of cancer after a century of widespread use for inflammation, fever, and pain. COX-2 inhibitors have been shown to reduce the growth of polyps and are being studied for their anticancer effects. The U.S. Food and Drug Administration has approved the use of celecoxib in those with FAP. In a randomized controlled trial of individuals with FAP, the experimental groups who took 800 mg a day of celecoxib had a 30.7% reduction in polyps and had a 14.6% reduction on 200 mg a day (Steinbach et al., 2000).

**Inflammatory Bowel Disease**

Inflammatory bowel disease is associated with an increased risk of CRC, and risk increases with the duration of the disease (Wilkes, 2005). Individuals with ulcerative colitis have an absolute risk of CRC of 30% 35 years after diagnosis; this risk increases to 40% for those diagnosed with colitis before 15 years of age (Ekbom, Helmick, Zack, & Adami, 1990). Smoking and alcohol

**Smoking and Alcohol**

Smokers have a significantly elevated risk of adenomas and CRC (Giovannucci & Martinez, 1996; Giovannucci, Rimm, Stampfer, Hunter, et al., 1994; Neugut, Jacobson, & DeVivo, 1993), but the effect is seen after years of exposure. Men who had smoked for more than 30 years had nearly double the risk of colon cancer compared to nonsmokers, as did women who smoked for more than 40 years. A majority of studies support an association between alcohol use and increased risk for CRC and adenoma formation (Herdman & Lichtenfeld, 2004).

Tobacco and alcohol are major contributing factors to the development of esophageal cancer throughout the world. Ninety percent of the risk for squamous cell cancer can be attributed to tobacco and alcohol. Those who quit smoking experience approximately a 50% reduction in the risk of developing squamous cell cancer of the esophagus. Smoking also is a risk factor for the development of adenocarcinoma, but quitting smoking does not decrease the risk for decades. In the United States, 80% of squamous cell carcinomas in men can be attributed to drinking more than one alcoholic beverage per day (Posner et al., 2005).

**Hormone Supplementation**

In some studies, postmenopausal female hormone supplementation decreased the risk of colon cancer (Calle, Miracle-McMahill, Thun, & Heath, 1995; Terry et al., 2002), but in the WHI data (Chlebowski et al., 2004), women randomized to hormone replacement had fewer invasive CRCs.
than the placebo group. However, the experimental group had higher percentages of more advanced lesions than the control group.

**Obesity and Physical Activity**

Obesity is responsible for at least 10% of CRCs and 25%–40% of esophageal, endometrial, and kidney cancers (Gotay, 2005). Obesity is associated with a twofold increase in risk of CRC in women (Terry, Miller, & Rohan, 2002) and a higher incidence and increased mortality from CRC in both genders (Rosen & Schneider, 2004). Exercise, in particular, conferred high levels of protection when vigorous and long-standing (Giovannucci et al., 1996). Physical activity also lowers one’s risk of developing large adenomatous polyps, suggesting it may act at an early point in the carcinogenic process. This effect is seen across all weight levels, suggesting a benefit of physical activity for cancer prevention as well as weight loss (Giovannucci et al., 1995, 1996; Herdman & Lichtenfeld, 2004).

Increased body mass index (BMI) is a risk factor for adenocarcinoma of the esophagus, with the highest risk (sevenfold) for those with the highest BMI. Obesity also is thought to contribute to increased risk of gastric cancer (Calle, Rodriguez, Walker-Thurmond, & Thun, 2003).

When a Mediterranean diet is adopted, with an emphasis on fish, nuts, poultry, and legumes, along with other health lifestyle factors, such as regular exercise, approximately 70% of colon cancers can be avoided (Hu & Willett, 2002; Platz et al., 2000).

**Health Literacy**

A study evaluating patient time and resource use associated with the delivery of chemotherapy and management of neutropenia demonstrated that even relatively simple visits resulted in large disruptions of patient time and lifestyle before, during, and after the visit (Fortner et al., 2004). Cancer treatment is a stressful experience for patients. During the course of diagnosis and treatment, patients experience a myriad of symptoms, psychological reactions, disruptions of normal life, and financial burdens. Research consistently has shown that patients with cancer want information about their disease and treatment, but they continue to report that they are not receiving sufficient information to successfully cope with treatment (Skalla, Bakitas, Furstenberg, Ahles, & Henderson, 2004). Patients with cancer want the maximum amount of information about their illness (Chelf et al., 2001). Benefits of providing patients with information include an increased participation in treatment decision making, enhanced ability to cope with treatment, and increased patient satisfaction. Informational needs change as patients move from diagnosis through treatment, requiring dynamic assessment (Adams, 1991; Mills & Sullivan, 1999).

Although informational needs exist across the continuum of cancer care, multiple factors influence patients’ ability to access care and to learn. These factors include health literacy, coping style, emotional state, motivation to learn, anxiety, fatigue, cultural background, and environmental factors (Chelf et al., 2001). To navigate the healthcare system effectively, an individual should have skills to understand appointment slips, oral and written communication, and health insurance forms while actively participating in healthcare decisions and learning self-care techniques. Anyone can feel overwhelmed by the amount of material to assimilate; however, those with limitations in language, health literacy, or recall are particularly at risk. Health literacy, as defined by IOM, is the degree to which individuals have the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions (Nielesen-Bohlman, Panzer, & Kindig, 2004).

An IOM report on health literacy stated that one-quarter of the U.S. population is functionally illiterate, another 50 million have marginal literacy skills, and the problem is especially prevalent among older adults (Nielesen-Bohlman et al., 2004). Literacy issues raise concerns about individuals’ ability to gain access to and effectively function within the healthcare system. One study reported that one-third to a majority of English-speaking patients, particularly older adults and the chronically ill, at public hospitals could not understand basic health-related materials or instructions about making an appointment and could not comprehend a consent form (Williams et al., 1995). Literacy influences health status and use of health services (Baker, Parker, Williams, & Nurss, 1997; Williams, Baker, Parker, & Nurss, 1998). Literacy was a stronger correlate of health status than education level or sociodemographic variables (Baker et al.).

**Summary**

Patterns of incidence and prevalence of GI cancers demonstrate the need for improved screening techniques and investigation into root causes of cancer disparities. Modifiable risk factors must be addressed on a societal, individual, and community level. Providing the appropriate amount and type of information also should be a priority. The difficulty with linking diet to cancer incidence or prevention limits prescriptive nutritional recommendations.

**References**

Gastrointestinal Cancers


