

# CHAPTER 1

## Cancer Prevention, Screening, and Early Detection

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### Introduction

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In 2008, an estimated 1,437,180 new cases of cancer are expected to be diagnosed in the United States, and 565,650 are not expected to survive (American Cancer Society [ACS], 2008). Two-thirds of these cancer deaths will be related to tobacco use, poor nutrition, physical inactivity, and obesity. All cancer deaths related to tobacco and alcohol abuse are entirely preventable. Additionally, more than one million new cases of skin cancer are expected to be diagnosed this year, and many could be prevented by avoiding overexposure to the sun. Cancers related to viral and/or bacterial infections, such as the hepatitis B virus, human papillomavirus (HPV), HIV, and *Helicobacter*, also can be prevented through changes in lifestyle and use of vaccines or antibiotics (ACS, 2006b).

Deaths related to breast, colorectal, uterine, and cervical cancers could be decreased by greater use of screening tests (ACS, 2006c). Only 55% of women 40 years of age and older reported having had a mammogram within the past year, and 79% of adult women reported having had a Pap smear sometime within the past three years (ACS, 2006c). Fewer than half of all Americans have had recent screening for colorectal cancer, according to ACS (2006c). Half of all new cases of cancer are considered preventable or could be detected at an earlier stage. The five-year survival rate for early-stage cancers is 85%, hence the importance of following established screening and early detection guidelines (ACS, 2006b).

A recent analysis of 2005 data from the National Cancer Institute's (NCI's) Health Information Trends Study, which tracks how Americans obtain and use cancer information, documented that most Americans are aware of the current cancer screening modalities but are unsure of the age at which they need to implement these screening tests. Fifty-seven percent of women were unaware that mammography screening for breast cancer begins at age 40. Sixty-one percent of women were unaware of the correlation

between HPV and cervical cancer. Forty percent of Americans surveyed could not name an appropriate screening test for colorectal cancer (NCI, 2005a, 2006b).

In addition to the general knowledge deficits listed here, cultural disparities also were identified in this study. Almost 80% of Hispanic respondents, 75% of African Americans, and 70% of American Indians/Alaska Natives were unaware of the appropriate age at which to begin screening for colorectal cancer, compared to 38% of Caucasians (NCI, 2006b). In general, African American men have higher incidence rates (19%) and higher mortality rates (37%) than Caucasian men (Jemal et al., 2008). African American women have a 6% lower incidence rate but a 17% higher death rate (Jemal et al.). Although part of this disparity is felt to be secondary to various differences in risk factors, knowledge deficits, difficulty with or lack of access to quality screening tests, and delayed diagnosis and treatment also greatly influence ethnic mortality rates (Jemal et al.).

## Role of the Advanced Practice Nurse

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Given the disparities identified among the general public, oncology advanced practice nurses (APNs) are in a unique position to educate their patients and the public regarding recommended cancer risk reduction and screening guidelines. The scope of practice for nurse practitioners includes an emphasis on health promotion and disease prevention (American Academy of Nurse Practitioners, 2002a, 2002b). The Oncology Nursing Society (ONS) recognizes “screening to prevent illness and promote wellness” as part of the role of the oncology APN (ONS, 2003). Therefore, cancer screening and prevention are clearly responsibilities of the oncology APN required to diagnose cancer at the earliest possible stage, if not prevent some cancers entirely.

It is also the position of ONS, as published in its 2002 position statement *Prevention and Early Detection of Cancer in the United States*, that APNs receive educational preparation in the principles of cancer prevention and early detection. Oncology specialty certification examinations (such as the advanced oncology certified nurse practitioner [AOCNP®] and advanced oncology certified clinical nurse specialist [AOCNS®] examinations) include coverage of this topic. In accordance with their state’s scope of practice, nurse practice act, and requirements for educational preparation, oncology APNs must be able to assess, evaluate, and interpret cancer risk assessments and recommend appropriate strategies related to cancer prevention and screening. All oncology nurses must be able to provide culturally sensitive cancer prevention and early detection services and participate in the development of resources that focus on wellness and primary prevention throughout the life span. Evidence-based research on cancer prevention and early detection requires integration into current practice (ONS, 2002).

## Cancer Risk Assessment

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Cancer risk assessment is a vital part of the oncology APN’s role in cancer prevention and early detection. To provide accurate counseling on cancer risk reduction strategies (e.g., tobacco cessation, lifestyle modifications, dietary changes, chemoprevention agents), cancer screening recommendations, and genetic testing (if appropriate), the oncology APN must first perform a comprehensive risk assessment. Cancer risk assessment is an individualized evaluation of a patient’s risk for cancer based on a variety

of both intrinsic and extrinsic factors and begins with a detailed history. This includes thorough past medical, obstetric/gynecologic, and surgical histories and documentation of recent age-appropriate screening tests, or lack thereof. Family history is a critical part of cancer risk assessment and includes at least a three-generation pedigree, particularly if a hereditary cancer syndrome is suspected (see Chapter 2). Medication history (such as hormone use), dietary history, level of physical activity, environmental exposures, history of tobacco and alcohol use, and other lifestyle choices also are important factors to assess when determining cancer risk. A thorough physical examination concludes the cancer risk assessment and includes a breast, pelvic, and rectal examination.

Some cancer risk assessment tools and models are available to help nurses to convey this risk to patients, such as the Gail model, Claus model, and BRCAPRO for breast cancer risk (Euhus, 2001) and the MMRpro model for hereditary colon cancer risk (Greco, 2007). Each of these tools has its strengths and weaknesses. The Gail model is the most commonly used general breast cancer risk assessment tool and is used to estimate a woman's five-year risk and overall lifetime risk for breast cancer. Scores are calculated based on a variety of risk factors, including age, age at menarche, age at first live birth, race, number of first-degree relatives with breast cancer, and number of previous breast biopsies. The score is based on a comparison to that of a woman of average risk and of the same race and age, with elevated risk considered  $> 1.7\%$ . However, this model fails to take into account the age at breast cancer diagnosis in affected family members, history of bilateral breast cancer, second-degree relatives affected with breast cancer, and history of ovarian cancer or lobular carcinoma in situ (LCIS). Both the BRCAPRO and Claus models lack accurate risk assessment for minority women and factors other than family history (such as number of previous breast biopsies), and BRCAPRO may fail to identify hereditary breast cancer syndromes that do not conform to *BRCA* mutations (Euhus).

Some cancer risk assessment tools are available online, such as a lung cancer risk assessment tool through Memorial Sloan-Kettering Cancer Center ([www.mskcc.org/mskcc/html/12463.cfm](http://www.mskcc.org/mskcc/html/12463.cfm)), the CancerGene software from the University of Texas Southwestern Medical Center for Breast Care ([www.utsouthwestern.edu/utsw/cda/dept47829/files/65844.html](http://www.utsouthwestern.edu/utsw/cda/dept47829/files/65844.html)), and the NCI's breast cancer risk assessment tool ([www.cancer.gov/bcrisktool/default.aspx](http://www.cancer.gov/bcrisktool/default.aspx)). The majority of cancers do not have reliable risk assessment tools, and those that do still have weaknesses. Therefore, these models are best used in conjunction with an individualized, comprehensive cancer risk assessment by the APN to best estimate and counsel patients on their overall cancer risk and on interventions to decrease that risk.

## Primary Prevention and Risk Reduction

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Cancer prevention is achieved through primary, secondary, and tertiary methods. Primary cancer prevention is achieved through two mechanisms: the promotion of health and wellness and reduction of risks known to contribute to cancer development (ONS, 2002). Primary prevention aims to reverse or inhibit the carcinogenic process through modifications in a patient's diet or environment or through pharmacologic mechanisms (Turini & DuBois, 2002). Examples of primary prevention include smoking cessation interventions and chemoprophylaxis in women at high risk for breast cancer. Secondary cancer prevention includes screening and early detection. In general, screening for cancer refers to checking for the presence of disease in populations at risk, and early

detection is defined as testing for cancer when no symptoms are present (ONS, 2002). Secondary prevention seeks to detect cancer at the earliest possible stage, when the disease is most likely to be treated successfully. Tertiary cancer prevention is applied to those individuals who have already been diagnosed with a malignancy but are now candidates for screening and early detection of secondary malignancies (ONS, 2002).

## Tobacco Use

Smoking has long been established as a detriment to overall health. As early as 1928, studies pointed to smoking and its association with cancer (Koh, Kannler, & Geller, 2001; Lombard & Doering, 1928). Research culminated with the 1964 U.S. Surgeon General's report, which concluded that smoking was the major cause of lung cancer and was associated with oral and laryngeal cancers in men. Since then, more than 60,000 studies and subsequent reports of the Surgeon General have confirmed tobacco's detrimental health effects (Koh et al.). More than 4,000 chemicals have been identified in tobacco products and tobacco smoke, 55 of which are identified as carcinogens by the International Agency for Research on Cancer (IARC). These carcinogens may induce genetic mutations and ultimately lead to cancer development (Koh et al.). Tobacco use is considered a contributing or causative agent in a multitude of malignancies, including oral, laryngeal, lung, renal, bladder, cervical, gastric, and esophageal cancers, in addition to leukemia (Centers for Disease Control and Prevention [CDC], 2004). Smoking is thought to cause up to 90% of lung cancers and is the leading cause of preventable cancer-related and non-cancer-related deaths in the United States (Koh et al.). Lung cancer is estimated to be diagnosed in close to 215,020 Americans in 2008, of which approximately 161,840 will die, encompassing approximately 30% of all cancer deaths (ACS, 2008).

Tobacco abuse and addiction is perhaps one of the greatest public health concerns of our time, particularly as far as cancer is concerned. In 2005, approximately 21% of adult Americans smoked—equal to 45 million people (Mariolis et al., 2006).

Most adult smokers today began smoking in their youth. Experimentation with cigarette smoking often begins early in adolescence and peaks at 13–14 years of age. Although the smoking rates in adults have been declining in the past decade, smoking prevalence in youths has risen dramatically since the 1990s (Fiore et al., 2000). It is estimated that about 4,000 adolescents per day are smoking for the first time, and more than one-fourth of them will become regular users of tobacco (Lindblom & McMahon, 2006). The percentage of high school students smoking declined from 1997 to 2003; however, from 2003 to 2005, the rate of decline slowed, if not stalled (ACS, 2006b; CDC, 2006a). More than 23% of high school-aged adolescents are current smokers (Lindblom & McMahon). Healthy People 2010 includes reducing smoking prevalence among high school students to 16% or less as one of its objectives. These statistics suggest the emergence of a new generation of smokers unless interventions are implemented to cease tobacco use among adolescents (CDC, 2006a).

Given these startling statistics, primary prevention measures for tobacco-related cancers and tobacco deterrent programs must be aimed at children and adolescents. Recent research shows that adolescents are three times more sensitive to tobacco advertising than adults and are more likely to be influenced to smoke by advertisements for cigarettes than by peer pressure (Lindblom & McMahon, 2006). Tobacco prevention efforts include increased tobacco prices and taxes, public smoking restrictions, and anti-tobacco advertisements. Many studies have identified the aforementioned tobacco

control efforts as being successful in reducing adolescent smoking rates. In 2000, one study estimated that through large-scale media campaigns and a mere \$1 increase in the price per pack of cigarettes, the prevalence of smoking among 18 year olds could be reduced by 26% in the United States and 108,466 lives could be saved (Rivara et al., 2004). This study concluded that efforts to reduce adolescent smoking can affect adult health and mortality. Moreover, continued efforts are needed to focus on implementing statewide tobacco bans; 15 states had done this as of 2006 (ACS, 2006b). Monies to promote tobacco control are also needed—the tobacco industry spent more than \$15 billion on marketing in 2003, which is 23 times the amount spent on tobacco control efforts (ACS, 2006b).

## Smoking Cessation

Despite the known consequences of tobacco abuse on health and society and the proven benefits of smoking cessation (see Table 1-1), most clinicians fail to identify and counsel patients on this topic (Fiore et al., 2000). Reasons for this include inability to quickly identify current tobacco users and a knowledge deficit about what treatments are effective, how they are delivered, and the associated side effects of treatment. Time constraints and lack of institutional support for tobacco cessation counseling also may contribute to the fact that only 21% of clinic visits with current smokers included smoking cessation counseling (Fiore et al.).

Identification of current tobacco users can be achieved by asking all patients at every visit about their smoking status and whether they are interested in quitting. It also may be beneficial to document tobacco use as the fifth vital sign on the chart. It is estimated that up to 70% of current smokers want to quit, but more than a third of those are never asked about their smoking status or desire to quit (Fiore et al., 2000). Even if patients have attempted smoking cessation in the past and have failed, several attempts at smoking cessation are common before long-term abstinence is achieved (Fiore et al.; Rigotti, 2002).

The U.S. Department of Health and Human Services' (DHHS's) *Treating Tobacco Use and Dependence: Clinical Practice Guideline* (Fiore et al., 2000) is a brief set of instructions

**Table 1-1. Health Benefits of Smoking Cessation**

Elapsed Time After Smoking Cessation	Health Benefits
2 weeks–3 months	Circulation, skin tone, oral hygiene, and pulmonary function improve.
1–9 months	Ciliary function in the lungs is restored.
12 months	Risk for coronary heart disease is reduced by 50% compared to persistent smokers.
5–15 years	Risk of stroke is decreased to that of nonsmokers.
10 years	Risk of death from lung cancer is reduced by 50% compared to persistent smokers.
15 years	Risk of coronary heart disease is reduced to that of nonsmokers.

*Note.* Based on information from Fiore et al., 2000.

for clinicians to use in identifying and treating nicotine dependence. ONS (2005) recommends the use of these instructions in all clinical settings in its position *Global and Domestic Tobacco Abuse*. As discussed previously, the first step in treating tobacco dependence is identifying tobacco users. Once identified, smokers can be categorized into one of two groups—those who are interested in making a quit attempt and those who are not. For those patients willing to attempt cessation, the guideline suggests using the “five A’s” strategy as listed in Table 1-2 (Fiore et al.).

**Table 1-2. The Five A’s of Smoking Cessation Counseling**

“A”	Intervention
Ask	Ask about tobacco status of every patient at every visit.
Advise	Advise tobacco users to quit in a strong, clear, and personalized manner.
Assess	Assess the patient’s readiness to quit.
Assist	Assist in the patient’s quit attempt (individualized counseling and pharmacotherapy).
Arrange	Arrange follow-up within one week of the stated quit date.

*Note.* Based on information from Fiore et al., 2000.

Oftentimes, inadequate knowledge of tobacco cessation therapy inhibits clinicians’ ability to assist patients in their quit attempt. Successful smoking cessation interventions contain two components: behavioral counseling and pharmacotherapy. The combined use of these approaches has been shown to improve smoking cessation rates (Fiore et al., 2000; Ranney, Melvin, Lux, McClain, & Lohr, 2006; Rigotti, 2002). Most studies estimate successful smoking cessation rates to be 40%–60% using this combination. This drops to 25%–30% after one year, but it is still higher than the less than 10% of smokers who attain long-term abstinence on their own (Fiore et al.; Rigotti).

Behavioral counseling begins by identifying triggers and stressors unique to each individual smoker. These can be moods, feelings, places, or activities. Some of the most common stressors and triggers include feeling stressed or depressed, talking on the phone or watching television, drinking alcohol or coffee, driving, finishing a meal, managing work and family issues, taking a work break, being with other smokers or seeing someone else smoke, cooling off after a fight, feeling lonely, and having sex (Rigotti, 2002). Making the patient aware of his or her triggers is a valuable method of assisting the patient to stay in control. Once the triggers and stressors have been identified, teaching the patient to cope with these patient-specific triggers involves avoiding these trigger situations and replacing old habits with new ones (Rigotti).

All tobacco users are advised to quit smoking at every encounter (Fiore et al., 2000; Ranney et al., 2006). Even if the individual is uninterested in making a quit attempt at that particular time, discussing the process, benefits, and perceived barriers to smoking cessation can propel the patient closer to seriously contemplating quitting. Therefore, for those who are not willing to attempt cessation, motivational support can be offered using the “five R’s,” listed in Table 1-3 (Fiore et al.).

In addition to behavioral counseling, pharmacotherapy may benefit all smokers ready to make a quit attempt (with the exception of certain populations, such as ado-



**Table 1-3. The Five R's of Smoking Cessation Counseling**

“R”	Intervention
Relevance	Encourage personal relevance of smoking cessation (i.e., health status, impact on family members, economic impact).
Risks	Ask the patient to identify risks of continued tobacco use that are pertinent to the patient, including acute (shortness of breath), chronic (cancer, chronic obstructive pulmonary disease), and environmental risks (cancers and lung diseases in the smoker's spouse and children).
Rewards	Ask the patient to identify potential rewards of smoking cessation, including immediate and long term (e.g., improved overall health for individual and family, money saved).
Roadblocks	Ask the patient to identify potential or actual barriers to smoking cessation (e.g., withdrawal symptoms, weight gain).
Repetition	Repeat the five R's at every encounter with the patient. Also, repeated attempts at smoking cessation are common among smokers.

*Note.* Based on information from Fiore et al., 2000.

lescents, those with specific medical contraindications, and pregnant women) (Fiore et al., 2000; Ranney et al., 2006). Currently, seven U.S. Food and Drug Administration (FDA)-approved first-line therapies are available—five types of nicotine replacement therapy (NRT) (transdermal, oral lozenges, gum, nasal spray, and oral inhaler) and two non-nicotine medications, bupropion sustained release (SR) and varenicline (FDA, 2006a; Fiore et al.; Ranney et al.). Studies show that NRT with or without the use of other FDA-approved medications increases smoking cessation success (Pray & Pray, 2003; Ranney et al.); most studies show a twofold increase in cessation rates with NRT alone over placebo (Fiore et al.; Rigotti, 2002). Most forms of NRT are easy to use and available over the counter, with prices equivalent to or less than cigarettes, depending on the patient's habit. NRT dosing, cost, contraindications, and common side effects are listed in Table 1-4.

Whether through routine office visits or in clinics designed specifically for smoking cessation therapy, several studies have identified the successful role of the APN in smoking cessation interventions (Andrews, Tingen, & Harper, 1999; Christman & Bingham, 1989; Nett & Obrigewitch, 1993; Reeve, Calabro, & Adams-McNeill, 2000). In addition to using the DHHS's guidelines, APNs can take other steps to improve smoking cessation awareness and therapy. For instance, studies have shown that longer, more intensive and individualized interventions for smoking cessation result in higher abstinence rates, up to 40% in some cases (Andrews et al.; Fiore et al., 2000). APNs can have patients set a quit date and sign a smoking cessation contract to facilitate ongoing commitment to their quit attempt. The Fagerstrom Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) is a tool used to identify levels of nicotine dependence and thus gear more or less intense interventions accordingly. Oncology APNs are in a unique position to guide patients along the continuum of nicotine dependence—from identifying tobacco users to providing behavioral counseling and prescriptive and nonprescriptive NRT and aiding in relapse prevention or treatment.

**Table 1-4. Nicotine Replacement Therapy**

Drug	Dose	Common Side Effects	Precautions/Contraindications	Cost
Bupropion SR	150 mg every day for 3 days, then bid	Insomnia, dry mouth	History of seizures or eating disorder	~ \$3.33/day; prescription
Nicotine gum	2 mg (< 25 cig/day); 4 mg (> 25 cig/day); not to exceed 24 pieces in 24 hours	Dyspepsia, mouth soreness	Dentures may prohibit proper use.	< \$7 for 10 of the 2 mg or 4 mg pieces; over the counter
Nicotine inhaler	6–16 cartridges/day	Mouth and/or throat irritation	–	~ \$11 for 10 cartridges; prescription
Nicotine lozenges	2 mg and 4 mg; 9–20 lozenges/day	Mouth irritation, nausea, dyspepsia	–	~ \$43 for 72-count box
Nicotine nasal spray	8–40 doses/day	Nasal irritation	–	~ \$5 for 12 doses; prescription
Nicotine patch	For use > 10 cig/day: 21 mg every day for 6 weeks, 14 mg every day for 2 weeks, 7 mg every day for 2 weeks	Local skin irritation	–	≤ \$4/day; prescription and over the counter
Varenicline (Chantix™, Pfizer Inc.)	0.5 mg once a day for 3 days, then bid for 4 days, then 1 mg bid for total of 12 weeks	Insomnia, nausea	Contraindicated in ages < 18 and during pregnancy or lactation	~ \$124/month; prescription; insurance coverage varies.

*Note.* Based on information from Fiore et al., 2000.

## Sun Exposure

Overexposure to the sun is the greatest risk factor for all types of skin cancer, including melanoma and basal and squamous cell cancers (ACS, 2006c; Hegde & Gause, 2005; Wagner & Casciato, 2004). More than one million people are diagnosed with skin cancer each year; 90% of nonmelanoma skin cancers and 65% of melanomas are felt to be directly related to ultraviolet (UV) rays from the sun (U.S. Environmental Protection Agency [EPA], 2006).

UV radiation is a known carcinogen, and two types can affect the skin—UVA and UVB. UVA rays penetrate deeper layers of the skin and are responsible for premature aging effects on the skin, whereas UVB rays mainly affect the epidermis and are the primary cause of sunburn (Brannon, 2007; EPA, 2006). UVB rays vary depending on the season and time of day. Engaging in regular activities to decrease exposure to or protect the skin from UV rays can significantly reduce the risk of skin cancer (ACS, 2006c). Primary prevention of skin cancer includes avoiding UV light as much as possible. ACS (2006c) recommends avoiding exposure to direct sunlight from 10 am to 4 pm, when UV rays are



known to be most intense. Avoiding artificial sources of UV light exposure, such as with tanning beds, also is crucial in reducing the risk of skin cancer (ACS, 2006c). According to a recent study, exposure to artificial sunlight increased the risk of basal cell skin cancer by 1.5 and the risk of squamous cell skin cancer by 2.5 (Karagas et al., 2002).

In addition to minimizing exposure to the sun, other sun protective behaviors include protecting the skin with proper clothing and sunscreen. ACS (2007) recommends the following four sun protective measures.

- Slip on a shirt.
- Slop on sunscreen.
- Slap on a hat.
- Wrap on sunglasses.

Wearing hats with wide brims, shirts and pants that adequately cover the extremities, and sunglasses to protect the eyes and covering the rest of exposed skin with sunscreen with a sun protective factor (SPF) of 15 or higher are all pertinent sun protective behaviors (ACS, 2006c, 2006e). Broad-spectrum sunscreens contain ingredients that block or absorb both UVA and UVB rays with chemicals such as avobenzone, titanium dioxide, and zinc oxide (ACS, 2006e; Brannon, 2007; EPA, 2006). The FDA requires that all sunscreens contain an SPF, which correlates to the level of protection from UVB rays. For example, a sunscreen with an SPF of 15 protects against 93% of the sun's UVB rays, and every 15 minutes of wearing sunscreen with an SPF of 15 is equivalent to one minute of UVB exposure without sunscreen (ACS, 2006e; EPA).

In general, use approximately one ounce of sunscreen to cover all exposed areas of skin, enough to form a thin film when first applied (ACS, 2006e; Brannon, 2007). Sunscreen should be applied 30 minutes before exposure to the sun and reapplied every two hours and again after swimming, sweating, or toweling off (ACS, 2006e; Brannon; EPA, 2006). Sunscreens labeled "water resistant" maintain their SPF for 40 minutes of water immersion, and those that are "very water resistant" maintain their SPF for 80 minutes (ACS, 2006e; EPA), but both need routine reapplication. Sunscreen is applied before makeup. When used in combination with insect repellent, sunscreen with a higher SPF should be applied because repellents can reduce sunscreen's effectiveness by up to one-third (ACS, 2006e; Brannon). Most sunscreens expire after one year and should be discarded (ACS, 2006e).

## Diet and Exercise

More than 60 million Americans are obese, equal to roughly 30% of the adult population in this country (CDC, 2005). The National Institutes of Health defines obesity in adults older than 20 as having a body mass index higher than 30 (NCI, 2004a). Maintaining a healthy diet and lifestyle is an important way to reduce the risk of a variety of cancers (ACS, 2006c). In 2002, the IARC reported a positive relationship between obesity and the incidence of several cancers, including postmenopausal breast cancer and cancers of the colon, endometrium, esophagus, and kidney (NCI, 2004a, 2007c). Some correlation also has been identified in cancers of the gallbladder, ovaries, and pancreas (NCI, 2004a). In 2002, an estimated 41,000 cases of cancer were related to obesity (Polednak, 2003). Obesity is linked to cancer mortality as well. Obesity is estimated to account for up to one in seven cancer-related deaths in men and one in five cancer-related deaths in women (Calle, Rodriguez, Walker-Thurmond, & Thun, 2003). Obesity increases the risk of dying from cancers of the esophagus, colon, liver, gallbladder, pancreas, and kidney, in addition to non-Hodgkin lymphoma and multiple

myeloma (Calle et al.). Obese women have an increased risk of dying from breast, endometrial, cervical, and ovarian cancers, and obese men are at increased risk of dying from prostate and gastric cancers (Calle et al.).

Several reasons account for the drastic increase in the prevalence of obesity in the United States over the past several decades. In general, Americans are eating foods that are higher in calories, fats, and sugars. These foods often are cheaper and marketed more intensely than healthier food choices and are served in large portions by fast-food chains and restaurants (ACS, 2006c). In 2003, the IARC published research showing the cancer-reducing effects of eating a diet high in fruits and vegetables (ACS, 2006c). Studies have shown that regular intake of vegetables decreases the risk of oral, pharyngeal, and laryngeal cancers, in addition to esophageal, gastric, lung, renal, and ovarian cancers (ACS, 2006c). Furthermore, the risk of developing colorectal and bladder cancers is decreased with regular consumption of fruits (ACS, 2006c). Yet, recent reports show that only 23.5% of Americans consume the recommended five servings of fruits and vegetables daily (ACS, 2006c).

While obesity rates are climbing in the United States, levels of physical activity continue to decline, further contributing to the obesity epidemic (ACS, 2008). However, as obesity is linked to increased cancer incidence and mortality, physical activity is associated with a decreased risk of breast, colon, endometrial, lung, and prostate cancers (NCI, 2004b). Unfortunately, more than half of all Americans do not engage in regular physical activity (CDC, 2005). The CDC recommends engaging in moderate physical activity for at least 30 minutes five or more days a week or vigorous activity for at least 20 minutes three or more days per week (CDC, 2006b). ACS also has outlined recommendations for diet and exercise (see Table 1-5) to promote physical activity and combat obesity (ACS, 2006a).

## Chemoprevention

Chemoprevention is defined as the use of natural, synthetic, or biologic agents to reverse, suppress, or prevent carcinogenic progression (Tsao, Kim, & Hong, 2004). Chemoprevention primarily is aimed at inhibition or differentiation of cell growth or induction of cell apoptosis (Turini & DuBois, 2002). A variety of agents are being studied, and some have been approved for the prevention of prostate, colon, and breast cancer. To date, no effective chemopreventive agents have been approved for common malignancies such as lung cancer or skin cancer (Tsao et al.). Moreover, certain risks are associated with chemopreventive drugs, such as unwanted side effects and the potential to cause more harm or even higher rates of malignancy, which occurred with the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Trial (ATBC Trial) and the Beta-Carotene and Retinol Efficacy Trial (CARET) (NCI, 1997). In these two studies, no benefit was seen from taking supplements in men at high risk for lung cancer. In fact, in participants taking beta-carotene in the ATBC Trial, 18% more lung cancers were diagnosed, and 8% more deaths occurred. In CARET, 28% more lung cancers were diagnosed, and 17% more deaths occurred in participants taking beta-carotene and vitamin A than in those taking placebos (Clark et al., 1996; Heinonen et al., 1998; NCI, 2006c).

The oncology APN plays an important role in chemoprevention, either through risk assessments that lead to identification of potential candidates for chemopreventive agents, referral to appropriate clinical trials, or referral to qualified colleagues for further evaluation and management. Certain oncology APNs have subspecialized in the area of

**Table 1-5. Guidelines on Nutrition and Physical Activity for Cancer Prevention**

Topic	Recommendations
Diet	Consume $\geq 5$ servings of fruits and vegetables daily. Limit consumption of foods high in fat, such as red meat. Choose whole grains over processed or refined grains and sugars. Limit alcoholic beverages to less than one per day.
Exercise	Adults: Engage in moderate physical activity for at least 30 minutes at least 5 days per week. Children and adolescents: Engage in at least 60 minutes of moderate to vigorous physical activity at least 5 days per week.
Weight control	Balance caloric intake with caloric expenditure. Maintain a healthy weight through the aforementioned diet and exercise guidelines.

*Note.* Based on information from American Cancer Society, 2006a.

risk assessment, genetics counseling, and chemoprevention and are excellent sources of referral or collaboration (Vogel, 2003).

## Tamoxifen and Raloxifene

In 1998, the FDA approved tamoxifen, a selective estrogen receptor modulator (SERM), for the prevention of invasive breast cancer after results from the Breast Cancer Prevention Trial showed a 49% reduction in invasive breast cancer in more than 13,000 high-risk pre- and postmenopausal women (Fisher et al., 2005). Tamoxifen was approved for the prevention of invasive breast cancer for women with a history of noninvasive breast cancer (ductal carcinoma in situ [DCIS] and lobular carcinoma in situ [LCIS]) (Fisher et al.). Tamoxifen is most effective in preventing estrogen receptor–positive breast cancers. Women who benefited most were those with a known genetic predisposition for *BRCA1* or *BRCA2* mutation, history of LCIS, or atypical ductal hyperplasia (Fisher et al.). Additional benefits yielded from the study included a 29% decrease in the risk of osteoporotic bone fractures in women ages 50 and older and a 53% decrease in women younger than 50 (Fisher et al.). Risks associated with tamoxifen use include a higher incidence of thromboembolic events and endometrial cancer (Fisher et al.). Because of these and other risks, the use of tamoxifen is individualized.

Results from the Study of Tamoxifen and Raloxifene recently revealed that raloxifene, a second-generation SERM, has similar effects in reducing invasive breast cancer in high-risk, postmenopausal women as tamoxifen (Vogel et al., 2006). Additionally, fewer cases of uterine cancer, thromboembolic events, and cataracts were seen with raloxifene. Raloxifene was associated with an insignificantly higher number of patients with noninvasive breast cancer compared to tamoxifen (Vogel et al.). Raloxifene is approved for breast cancer risk reduction in postmenopausal women at high risk for invasive breast cancer.

## Celecoxib

Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) have shown activity in the treatment and prevention of colon cancer; however, their gastrointestinal side effects

have limited their applicability (Price, 2002; Turini & DuBois, 2002). Newer NSAIDs, such as celecoxib, selectively inhibit cyclooxygenase-2 (COX-2), a catalytic enzyme in prostaglandin synthesis that is induced in inflammatory conditions, including those involved with tumor proliferation. COX-2 is not normally found in the epithelium of the colon but is overexpressed in a majority of adenocarcinomas and less so in adenomatous polyps of the colon (Price; Turini & DuBois). Celecoxib, at 400 mg bid, has received approval from the FDA for the prevention of adenomatous polyps in patients with familial adenomatous polyposis (FAP) (Price), a hereditary colon cancer syndrome associated with hundreds of thousands of colon polyps and a 100% risk of colorectal cancer if untreated. Celecoxib is not approved for chemoprevention in the general public.

## Selenium and Vitamin E

The Selenium and Vitamin E Trial (SELECT) aims to determine the efficacy of these two supplements in preventing prostate cancer. It also aims to identify their effects on lung and colon cancer prevention, although these are not primary end points (NCI, 2005b). Two previous studies alluded to the benefits of selenium and vitamin E on the incidence of prostate cancer, but this was not the studies' primary objective. The Nutritional Prevention of Cancer Trial aimed to determine the relationship between selenium and nonmelanomatous skin cancers. The trial did not do this, but it did identify 60% fewer cases of prostate cancer in men who took selenium for 6.5 years (Clark et al., 1996; NCI, 2005b). In addition to the results of the ATBC Trial discussed earlier, the men taking vitamin E for the prevention of lung cancer were noted to have 32% fewer cases of prostate cancer (Heinonen et al., 1998; NCI, 2005b). SELECT data on more than 35,000 men are expected in 2011 (NCI, 2005b).

## Gardasil® Vaccine

In June 2006, the FDA approved Gardasil® (Merck & Co.) (quadrivalent human papillomavirus recombinant vaccine) for the prevention of cervical cancer, precancerous or dysplastic cervical and vaginal lesions, and genital warts associated with HPV types 6, 11, 16, and 18 (FDA, 2006b). This is the first vaccine approved for cervical cancer prevention, and in clinical trials, it showed nearly 100% effectiveness in preventing precancerous cervical, vaginal, and vulvar lesions and genital warts caused by HPV in women who had not yet been infected (FDA, 2006b). Hence, it is important to administer the vaccine in women who have not been exposed to HPV. Gardasil is approved for use in women aged 9–26 years old and is given as a set of three injections over six months (FDA, 2006b). The most common side effects associated with this vaccine included injection site reactions in the form of erythema, pain, edema, fever, nausea, and headache (Saslow et al., 2007). Gardasil is not approved to treat cervical cancer, nor is it intended to replace routine cervical cancer screening (Saslow et al., 2007). Its impact on cervical cancer incidence and mortality will take decades of vaccinations before becoming evident; however, researchers have suggested a possible 70% reduction in cervical cancer incidence worldwide (Saslow et al., 2007).

ACS has recommended routine HPV vaccination in girls aged 11–12 years and also for those aged 13–18 who need to complete the vaccination series and to provide vaccination to those who may have missed the opportunity to be vaccinated previously. ACS does not support routine vaccination of women aged 19–26 years old because of insufficient evidence for this age group (Smith, Cokkinides, & Eyre, 2007).

## Secondary Prevention and Screening

According to NCI (2007d), screening for cancer in the general population refers to detecting cancer when no apparent symptoms are present, with an overall goal of decreasing cancer-related morbidity and mortality. For almost all types of cancer, improved outcomes are seen when treatment is initiated at the earliest stage possible (NCI, 2007d), hence the importance of early detection. For example, breast, colorectal, cervical, testicular, oral cavity, and skin cancers—which account for half of all cancer cases diagnosed in the United States each year—collectively have a five-year survival rate of approximately 80% but could be improved to 95% if all Americans adhered to routine screening recommendations (ACS, 2006b).

For cancer screening to be effective, screening tests must meet two criteria. First, the screening test must be able to detect cancer at an earlier stage than if it were detected as a result of the development of symptoms. Second, evidence must support that treatment given at an earlier stage results in improved outcomes (NCI, 2007d). The sensitivity and specificity of all screening tests must be considered. *Sensitivity* refers to the proportion of people with cancer that are found to have a positive test—a higher sensitivity means fewer false negative results. Conversely, *specificity* refers to the proportion of people without cancer that have negative results; in other words, the higher the specificity, the fewer false positive results (NCI, 2006a). Potential harms from screening tests also must be weighed against potential benefits. Some screening tests are invasive, such as colonoscopy for colon cancer, and carry risks associated with any invasive procedure, including some serious if not life-threatening complications (such as bowel perforation with colonoscopy) (NCI, 2007d). Other potential harms include the emotional anxiety associated with false-positive results and the dangers of missing an early malignancy with false-negative test results (NCI, 2007d). The financial cost of different screening tests varies widely. Insurance coverage or lack thereof may prohibit individuals from following recommended screening guidelines. For instance, in 2003, only 29% of women without health insurance had received a mammogram within the past year, compared to almost 60% of women with coverage (ACS, 2005).

Multiple organizations recognized in the oncology community have published screening guidelines for a variety of malignancies, both for average-risk and high-risk populations. The oncology APN must have an understanding of each organization's guidelines and realize the differences among them. However, one may opt to follow and recommend one set of guidelines on a routine basis simply because of familiarity with that set of guidelines and ease of use. No one set of guidelines is superior to the other. In general, consensus exists among screening recommendations for the most common malignancies, including breast, cervical, colorectal, and prostate cancer. Variances in screening intervals and ages of screening initiation and cessation are minute. The recommended routine guidelines from ACS, the American Society of Clinical Oncology (ASCO), ONS, and the National Comprehensive Cancer Network (NCCN) are outlined in Table 1-6.

### Breast Cancer

Breast cancer is the most common female malignancy and the second most common cause of cancer death in women (ACS, 2008). In 2008, an estimated 182,460 new cases of breast cancer are expected to be diagnosed (ACS, 2008). The risk of developing breast cancer increases with age and is most common in women; however, approximately 1,990

**Table 1-6. Cancer Screening Recommendations for Average-Risk Population**

Organization	Breast Cancer	Cervical Cancer	Colorectal Cancer	Prostate Cancer	Skin Cancer
National Comprehensive Cancer Network	<p>For women of average risk between 20–40 years of age:</p> <ul style="list-style-type: none"> <li>Clinical breast exam (CBE) every 1–3 years and periodic breast self-exam (BSE) are recommended.</li> </ul> <p>For women &gt; 40:</p> <ul style="list-style-type: none"> <li>Annual mammogram (MMG) and CBE and periodic BSE are recommended.</li> </ul>	<p>Screen with Pap test within 3 years of vaginal intercourse, but starting no later than 21 years of age; continue annually with conventional cervical cytology or every 2 years using liquid-based cytology (vaginal cytology is not indicated in women who have had a total hysterectomy).</p> <p>At age 30, if there have been 3 consecutive normal Pap tests, screening intervals can increase to every 2–3 years.</p> <p>Screening can cease at age 70 with 3 or more consecutive normal Pap tests within the previous 10 years.</p> <p>Continue screening despite age if history of cervical cancer, diethylstilbestrol (DES) exposure, or immunocompromised states.</p> <p>Human papillomavirus (HPV)-positive women continue screening at the discretion of their healthcare providers.</p>	<p>Average risk (age <math>\geq</math> 50, no history of adenoma, inflammatory bowel disease [IBD], or family history of colon cancer):</p> <ul style="list-style-type: none"> <li>Colonoscopy every 10 years is preferred or fecal occult blood test (FOBT) annually and flexible sigmoidoscopy every 5 years or double contrast barium enema (DCBE) every 5 years.</li> </ul>	<p>Risk/benefit discussion and offer baseline prostate-specific antigen (PSA) testing and digital rectal exam (DRE) at 40:</p> <ul style="list-style-type: none"> <li>PSA <math>\geq</math> 0.6 ng/ml, or African American, or positive family history: Screen with PSA and DRE annually.</li> <li>Otherwise, repeat PSA and DRE at age 45.</li> <li>PSA <math>\leq</math> 0.6 ng/ml: Begin annual screening at age 50.</li> <li>PSA &gt; 0.6 ng/ml: Perform annual follow-up.</li> </ul>	Not applicable

(Continued on next page)



**Table 1-6. Cancer Screening Recommendations for Average-Risk Population (Continued)**

Organization	Breast Cancer	Cervical Cancer	Colorectal Cancer	Prostate Cancer	Skin Cancer
American Cancer Society	<p>Yearly MMG beginning at age 40 and continuing as long as the woman is in good health</p> <p>CBE every 3 years for women in 20s and 30s and annually if <math>\geq 40</math></p> <p>BSE is optional for women beginning in their 20s.</p>	<p>Pap test annually (or every 2 years with liquid-based Pap test) beginning within 3 years of vaginal intercourse or starting by age 21</p> <p>At <math>\geq 30</math>, women with 3 consecutive normal Pap tests continue with Pap test screening every 2–3 years.</p> <p>Women <math>\geq 70</math> years old with <math>\geq 3</math> consecutive normal Pap tests within the previous 10 years can cease screening.</p> <p>Discuss screening despite age if history of DES exposure or immunocompromised states.</p> <p>If woman has had total hysterectomy, may stop screening unless surgery was for cervical cancer or premalignant findings.</p>	<p>At age 50, one of the following (with all positive tests followed with colonoscopy):</p> <ul style="list-style-type: none"> <li>• Yearly FOBT or fecal immunochemical test (FIT)</li> <li>• Flexible sigmoidoscopy every 5 years</li> <li>• Yearly FOBT or FIT, plus flexible sigmoidoscopy (preferred over either alone)</li> <li>• DCBE every 5 years</li> <li>• Colonoscopy every 10 years</li> </ul> <p>Discuss early screening if patient has one of the following:</p> <ul style="list-style-type: none"> <li>• Personal history of colorectal cancer or adenomatous polyps</li> <li>• Family history of colorectal cancer or polyps in first-degree relative <math>&lt; 60</math> years of age or in two first-degree relatives of any age</li> <li>• Personal history of IBD</li> <li>• Family history of a hereditary colorectal cancer syndrome (familial adenomatous polyposis or hereditary nonpolyposis colorectal cancer)</li> </ul>	<p>Annual PSA and DRE beginning at age 50 (if patient has <math>&gt; 10</math>-year life expectancy)</p> <p>High-risk men:</p> <ul style="list-style-type: none"> <li>• African American men and men with one or more first-degree relatives diagnosed with prostate cancer before age 65: Annual screening at age 45</li> <li>• Multiple first-degree relatives with prostate cancer at an early age: Consider testing at earlier than age 40; depending on results, may opt to resume screening at age 45.</li> </ul>	<p>Not applicable</p>

(Continued on next page)



**Table 1-6. Cancer Screening Recommendations for Average-Risk Population (Continued)**

Organization	Breast Cancer	Cervical Cancer	Colorectal Cancer	Prostate Cancer	Skin Cancer
Oncology Nursing Society	Offer BSE teaching to women older than 20 and the option to perform these monthly. CBE annually beginning at age 20 Annual MMG beginning at age 40	Not applicable	Not applicable	Not applicable	Not applicable
American Society of Clinical Oncology	Monthly BSE and regular physical exam by health-care provider beginning at age 20 Annual MMG beginning at age 40	Initial Pap test 3 years after beginning sexual intercourse, but starting no later than age 21, and then once every 2–3 years	Beginning at age 50: Annual FOBT, flexible sigmoidoscopy every 5 years, colonoscopy every 10 years, or DCBE every 5 years	Annual PSA and DRE beginning at age 50	Periodic skin self-examination and annual whole body skin examination by healthcare provider, especially after age 40

Note. Based on information from American Cancer Society, 2006a; American Society of Clinical Oncology, 2005; National Comprehensive Cancer Network, 2007a, 2007b, 2007c, 2007d; Oncology Nursing Society, 2002, 2006.

cases of breast cancer are expected to be diagnosed in men in 2008 (ACS, 2008). Other risk factors for breast cancer are linked to reproductive and lifestyle factors (see Figure 1-1). A variety of benign breast lesions also increase the relative risk of breast cancer, depending on the histology (see Table 1-7). Factors that were previously thought to affect risk, but actually do not, include multiparity, lactation, and breast-feeding (Box & Russell, 2004). Factors known to decrease risk include Asian ancestry, early menopause, term pregnancy before age 18, and surgical menopause before age 37 (Box & Russell).

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#### Modifiable Risk Factors

- Recent oral contraceptive use or hormone replacement therapy
- Nulliparity or first birth after age 30
- Postmenopausal obesity
- Sedentary lifestyle
- Consumption of one or more alcoholic beverages per day
- Consumption of diet high in fat and low in fruit and vegetable intake

#### Irreversible Risk Factors

- Female gender
  - Caucasian
  - Advanced age
  - Inherited genetic mutation (*BRCA1/2*)
  - Personal or family history of breast cancer
  - Atypical hyperplasia and other benign breast lesions (see Table 1-7)
  - History of high-dose radiation to the chest (as with mantle field radiation in Hodgkin lymphoma)
  - Early menarche and late menopause
- 

### Figure 1-1. Risk Factors for Breast Cancer

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Note. Based on information from American Cancer Society, 2006b; Morrow & Jordan, 2003.

Screening guidelines for breast cancer are divided into two populations: those at average risk and those at high risk. The lifetime risk of developing breast cancer in an average-risk woman is one in eight (in North American women up to age 85) (ACS, 2005; Mirshahidi & Abraham, 2005). Breast cancer screening has been shown to decrease mortality from breast cancer (ACS, 2008). As listed in Table 1-6, the general consensus for breast cancer screening in average-risk women includes counseling regarding the technique, benefits, and limitations of monthly breast self-examination (BSE); clinical breast examination (CBE) beginning at various ages and continued at various intervals; and annual mammography beginning at age 40. This table outlines the recommendations and slight variances among the professional organizations' guidelines.

Racial disparities have been documented among Caucasian and African American women with regard to incidence, death rates, and percentage of women who regularly receive mammograms (Jemal et al., 2008). Incidence rates for breast cancer are higher in Caucasian women, in part because of higher rates of mammography, the incidence of hormone replacement therapy use, and older age at first childbirth compared to African American women (Ghafoor et al., 2003; Jemal et al.). Mortality rates from breast cancer are higher in African American women, partly because of the lack of routine mammographic and CBE screening. Thus, this population is more likely to have a more advanced cancer at diagnosis and a corresponding poorer prognosis (Ghafoor et al.). Because screening for breast cancer is known to improve outcomes and decrease mortality, all women need equal access to appropriate screening exams and diagnostics.

**Table 1-7. Benign Breast Lesions and Breast Cancer Risk**

Type of Lesion	Associated Breast Cancer Risk
Papillomas: Epithelial origin; commonly associated with nipple discharge	Exact risk is debatable but generally is thought to increase risk by 2.3–3.9 times. In younger women, associated with concurrent breast cancer and breast cancer in first-degree relatives
Florid intraductal epithelial hyperplasia: Epithelial origin; most common form of proliferative breast disease	1.5–2-fold elevated risk When associated with papillomas, increases risk by 2.3–3.3-fold
Atypical ductal hyperplasia (ADH): Risk factor for but not a direct precursor of breast cancer; greatest risk is within 10 years of diagnosis	Associated with a 4–5-fold increased risk of breast cancer Relative risk increases to 8.9% when associated with a positive family history of breast cancer.
Atypical lobular hyperplasia: Characterized by changes similar to lobular carcinoma in situ (LCIS), but lack complete criteria	Increases relative risk by 4.3% 8–11-fold increased risk when associated with ADH
Ductal carcinoma in situ: Direct precursor of breast cancer	Relative risk for development of contralateral breast cancer is 2–3-fold.
LCIS: Direct precursor of breast cancer	Relative risk for breast cancer is 5.7 and increases to 8.5 if family history of LCIS exists
Sclerosing adenosis	2.1–2.2-fold increased risk for breast cancer If associated with ADH, 5.3-fold increased risk
Fibroadenomas: Benign tumor most common in women aged 20–30; complex fibroadenomas may have a cystic component, epithelial calcifications, or sclerosing adenosis.	2.17-fold increased relative risk for breast cancer Complex fibroadenomas associated with a higher incidence of breast cancer than simple fibroadenomas

*Note.* Based on information from Gierach & Vogel, 2004; Morrow & Jordan, 2003; Schnitt et al., 1996.

The CDC initiated the National Breast and Cervical Cancer Early Detection Program in 1990 to improve access for low-income women in need of breast and cervical screening and diagnostic tests (Ghafoor et al.). In 1987, an average of 17% of low-income women age 40 and older had received recent mammography screening, compared to 54.6% in 2003 (ACS, 2005).

BSE has been recommended as part of the triad of screening mechanisms for breast cancer since 1933 (ONS, 2006). However, numerous studies have failed to verify consistent results regarding its efficacy, sensitivity, and specificity (Austoker, 2003). Ku (2001) provided an excellent overview of the history of BSE and cited numerous studies that provide a positive relationship between BSE and breast cancer stage at diagnosis. Conversely, several studies also have identified no relationship between BSE and survival or mortality rates (Ku; NCI, 2007b). Therefore, it is the consensus of all professional organizations to discuss the risks and benefits of BSE and use it in combination with other screening modalities. BSE may be ideal for women who are not yet of age to undergo screening mammography and as a means to continue surveillance between

CBEs (Ku). If a woman decides to perform BSE, proper technique is necessary, an ideal educational task for the oncology APN. Women may be taught proper BSE technique using steps similar to the CBE, as described by ACS (2006d) at [www.cancer.org/docroot/CRI/content/CRI\\_2\\_6x\\_How\\_to\\_perform\\_a\\_breast\\_self\\_exam\\_5.asp](http://www.cancer.org/docroot/CRI/content/CRI_2_6x_How_to_perform_a_breast_self_exam_5.asp).

CBE has a sensitivity range of 40%–69% and specificity of 88%–99%. When CBE is combined with mammography, mortality from breast cancer is reduced by 14%–29% (Humphrey, Helfand, Chan, & Woolf, 2002). A study published in 2005 showed even lower rates of sensitivity (28%–36%) for the community clinician, citing the fact that it often is performed along with other time-consuming tasks during the office visit, such as Pap smear (Fenton et al., 2005). In general, the performance of routine CBE has declined, most likely secondary to the prevalence of screening mammography. One study showed that 95% of women in 1987 had undergone a screening mammogram and CBE within the previous six-month period, plummeting to only 50% in 2004 (Goodson, Grissom, Moore, & Dirbas, 2005). However, studies have shown that CBE is an important part of the screening process for breast cancer, detecting between 4.6%–5.7% of breast cancers alone (McDonald, Saslow, & Alciati, 2004). Figure 1-2 outlines the components of a comprehensive CBE, a requisite skill of the oncology APN.

Visual Inspection	Palpation
<p>Position</p> <ul style="list-style-type: none"> <li>View breast from all sides while patient tightly presses hands on hips (to flex pectoralis major muscle) in upright position.</li> </ul> <p>Look for and document (using clock face to record location):</p> <ul style="list-style-type: none"> <li>Skin retraction, dimpling, or ulceration</li> <li>Erythema</li> <li>Skin thickening or peau d'orange</li> <li>Nipple retraction, inversion, deviation, scaling, or discharge</li> <li>Scarring</li> <li>Symmetry.</li> </ul>	<p>Position</p> <ul style="list-style-type: none"> <li>Infra- and supraclavicular and axillary lymph nodes while upright; breast and nipple in supine position with ipsilateral hand placed behind head (to reduce breast tissue thickness)</li> </ul> <p>Palpate</p> <ul style="list-style-type: none"> <li>Use pads of first three fingers to make dime-sized, overlapping circular motions.</li> <li>Apply light, medium, and deep pressure.</li> <li>Use vertical strip method to cover all tissue in between the midaxillary line and the sternum, and from the inframammary ridge to the clavicle.</li> </ul> <p>Document any masses (using clock face to record location) and characteristics:</p> <ul style="list-style-type: none"> <li>Size</li> <li>Shape (round, irregular, linear)</li> <li>Mobility (mobile or fixed)</li> <li>Consistency (cystic, hard)</li> <li>Texture (smooth, irregular)</li> <li>Nipple discharge (spontaneous or forced; color).</li> </ul>

**Figure 1-2. Elements of a Comprehensive Clinical Breast Examination**

*Note.* Based on information from Saslow et al., 2004.

Mammography alone results in a 20%–30% reduction in mortality in women who receive regular screening mammograms after age 50 and a 17% reduction in women between 40 and 49 years of age (Mirshahidi & Abraham, 2005). Mammogram specificity is high at 94%–97%, and the sensitivity for annual mammography reaches up to 96% but is lower in younger women (40–49 years old) with denser breasts (Humphrey et al., 2002; NCI, 2007b). According to the Digital Mammographic Imaging Screening Trial, digital mammography

proved to be more accurate than film mammography in women with dense breasts who were younger than 50 years of age and who are pre- or perimenopausal (Pisano et al., 2005).

Women at high risk for breast cancer include those with prior radiation therapy to the chest, strong family history of or genetic predisposition to breast cancer (*BRCA1/2* mutations), personal history of LCIS, atypical hyperplasia, or invasive breast cancer (NCCN, 2006). Approximately 5%–10% of all breast cancers can be attributed to germ-line mutations of the *BRCA1* or *BRCA2* genes, which are associated with a 40%–85% risk of breast cancer (Mirshahidi & Abraham, 2005). In general, screening in high-risk women begins at an earlier age and occurs at shorter intervals. NCCN has published screening guidelines for women at high risk for breast cancer, which are summarized in Table 1-8. More comprehensive information regarding screening of women with germ-line mutations and breast cancer is covered in Chapter 2.

Magnetic resonance imaging (MRI) may be superior to mammography in high-risk women (Mirshahidi & Abraham, 2005). Several studies utilizing MRI screening in high-risk populations are ongoing, but as of yet, MRI screening has not been found to reduce mortality in any group of women (NCI, 2007b; Saslow et al., 2007). ACS recently has adopted annual breast screening with MRI as an adjunct to mammography in women with  $\geq 20\%$ –25% lifetime risk of developing breast cancer (Saslow et al., 2007). This includes women with a genetic predisposition, significant family history of breast or ovarian cancer, or history of mantle radiation therapy associated with treatment for Hodgkin lymphoma. At present, ACS does not support screening women with other risk factors for breast cancer, including a personal history of breast cancer, DCIS, LCIS, atypical hyperplasia, or dense breast tissue, because of insufficient data (Saslow et al., 2007).

## Cervical Cancer

Cervical cancer is the third most common female gynecologic cancer (Posadas & Kotz, 2005). Since the introduction of the Pap smear more than 50 years ago, cervical

**Table 1-8. Breast Cancer Screening Guidelines for High-Risk Women**

Risk Factor	Screening Recommendation
Prior chest radiation therapy	Age < 25: Annual clinical breast exam (CBE) and periodic breast self-exam (BSE) Age > 25: Annual mammogram (MMG) and CBE every 6–12 months starting 8–10 years after radiation therapy or at age 40
5-year risk of breast cancer $\geq 1.7\%$ per Gail model	Annual MMG and CBE every 6–12 months; periodic BSE
Lobular carcinoma in situ/atypical hyperplasia	Annual MMG and CBE every 6–12 months; periodic BSE
Genetic predisposition or strong family history (See Chapter 2 for more detailed information.)	Age < 25 years: Annual CBE and periodic BSE Age > 25 years: Annual MMG (+/- annual magnetic resonance imaging); CBE every 6–12 months (beginning at age 25 for hereditary breast and ovarian cancer syndrome or 5–10 years earlier than the youngest family member with breast cancer if strong family history); periodic BSE

Note. Based on information from National Comprehensive Cancer Network, 2007a.

cancer incidence and mortality rates have declined steadily (ACS, 2006b; Posadas & Kotz). In 2008, 11,070 new cases of cervical cancer are expected to be diagnosed, and 3,870 deaths from the disease are estimated to occur (ACS, 2008). When detected early, localized cervical cancer is one of the most successfully treated cancers, boasting a five-year survival rate of 92% (ACS, 2008). The lifetime risks for developing and dying from cervical cancer in the United States are 0.88% and 0.29%, respectively (Posadas & Kotz). Cervical cancer is more prevalent in lower socioeconomic classes of women, women of minority populations (Latin American, Native American, and African American), and women with lower education levels (Posadas & Kotz). Caucasian women are more likely to have cervical cancer diagnosed at an earlier stage (53%) compared to African American women (44%) (ACS, 2008).

In addition to these risk factors, other risk factors for cervical cancer include those related to sexual history and gynecologic history, smoking, and immunosuppression (see Table 1-9). However, the most significant risk factor for cervical cancer is HPV infection. HPV, a sexually transmitted disease, is the most common cause of and greatest risk factor for premalignant and malignant cervical lesions. More than 200 types of HPV have been identified, 20 of which have been associated with cancer (Einstein & Goldberg, 2002). Benign cervical lesions (genital warts and cervical intraepithelial neoplasia) are most commonly associated with HPV types 6, 11, 42, 43, and 44. HPV strains 16 and 18 are most commonly associated with cervical cancer and are targeted by the HPV vaccine Gardasil, along with HPV strains 6 and 11 (Dunne et al., 2007). An estimated 26.8% of women ages 14–59 are infected with HPV, according to recent data from the National Health and Nutrition Examination Study (Dunne et al.). HPV infection was highest in women aged 20–24, and 15.2% of women overall were infected with high-risk strains 16 and 18 (Dunne et al.).

Routine screening recommendations for cervical cancer are outlined in Table 1-6, and recommendations generally include initiation of Pap smear by age 21 or within three years of vaginal intercourse. Thus far, routine screening for HPV is not recommended, as clinical outcomes and management of cervical cancer with HPV infection have not changed compared to those cases without (Posadas & Kotz, 2005). Most agree it is acceptable to cease screening women at 70 years and older if certain criteria are met (see Table 1-6) and if individualized risk assessments are discussed between the patient and provider.

**Table 1-9. Risk Factors for Cervical Cancer and Associated Relative Risk**

Risk Factor	Relative Risk for Cervical Cancer
History of > 6 sexual partners	2.2
Use of oral contraceptive pills > 10 years	2.2
Sexual intercourse before age 18	1.6
Smoking	1.7
Immunosuppression	
• HIV	2.5
• History of renal transplantation	5.7
Multiparity	1.5

Note. Based on information from Posadas & Kotz, 2005.

## Colorectal Cancer

Colorectal cancer (CRC) is the second leading cause of cancer death in the United States and the third most commonly diagnosed cancer in both men and women (ACS, 2008; Kim, Takimoto, & Allegra, 2005). In 2008, an estimated 148,810 new cases of colorectal cancer are expected to be diagnosed, with 49,960 deaths expected to occur (ACS, 2008). The average lifetime risk of developing CRC is 1 in 18 (Kim et al.). Colon cancer is more common in women, whereas rectal cancer is more common in men (Kim et al.). African Americans have a higher incidence of CRC and a 32% increased mortality rate over Caucasians (Kim et al.).

Several known risk factors exist for CRC, although up to 70% of cases have no identifiable risk factors (Kim et al., 2005). The most common risk factor for the development of CRC is age, with more than 90% of cases found in those older than 50 years of age (ACS, 2008; Kim et al.). Other risk factors include lifestyle factors such as physical inactivity, obesity, heavy alcohol intake, and diets high in red meat and low in fiber, fruits, and vegetables (ACS, 2006b). Smoking is associated with a 2.5-fold increased risk of CRC (Kim et al.) and is estimated to account for 5,000–7,000 CRC deaths annually (Alberts & Goldberg, 2004). Other conditions of the colon (e.g., inflammatory bowel disease [IBD], colon adenomas) and a genetic predisposition or hereditary polyposis syndrome increase the risk of CRC (see Table 1-10) (ACS, 2006b; Goldberg, 2000; Kim et al.). Conversely, several factors are known to reduce the risk of colon cancer, such as regular intake of calcium, aspirin, NSAIDs, or 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (i.e., statins) and postmenopausal use of hormone replacement therapy (ACS, 2006b; Alberts & Goldberg; Kim et al.). Only celecoxib has been approved for prevention of adenomatous polyps in FAP (Price, 2002).

The five-year survival rate for CRC detected at a localized stage is 90%; however, because of a lack of appropriate screening, only 39% of cases are diagnosed at this stage (ACS, 2008). As with most malignancies, a more advanced stage at diagnosis of colon cancer is associated with decreased survival (ACS, 2008). The five-year survival rate for locally advanced CRC (involvement of regional lymph nodes) is 68%, and those with distant metastases have even poorer outcomes, with only a 10% five-year survival (ACS, 2008). Studies of large (> 1 cm) untreated colonic polyps have a 2.5% risk of progressing to malignant tumors within 5 years, an 8% risk at 10 years, and a 24% risk at 20 years. On average, a severely dysplastic polyp will take only 3.5 years to evolve into a malignant tumor, whereas a polyp with only mild atypia may take an average of 11.5 years (Alberts & Goldberg, 2004).

The purpose of screening for CRC is to identify adenomatous or precancerous polyps and remove them before they progress to malignancy, thereby resulting in decreased mortality and better outcomes. Polypectomy and subsequent surveillance with colonoscopy can reduce CRC incidence by 90% (Alberts & Goldberg, 2004). Guidelines for screening average-risk populations (age  $\geq$  50, no history of adenoma or IBD, and negative family history) are outlined in Table 1-6. In general, recommendations include screening beginning at age 50 and either annual fecal occult blood test (FOBT) or fecal immunochemical testing (FIT), sigmoidoscopy or double contrast barium enema every five years, annual FOBT or FIT in combination with sigmoidoscopy every five years, or a full colonoscopy every 10 years. Guidelines for increased-risk or high-risk populations, as recommended by NCCN, are outlined in Table 1-11.



**Table 1-10. Diseases of the Colon and Colorectal Cancer Risk**

Risk Factor	Risk of Colorectal Cancer (CRC)
Ulcerative colitis	<ul style="list-style-type: none"> <li>• Causes 1% of all cases of CRC</li> <li>• Increases with age at onset, extent of disease, and duration of active disease</li> <li>• Cumulative risk is 3% at 15 years, 5% at 20 years, and 9% at 25 years.</li> </ul>
Crohn disease	<ul style="list-style-type: none"> <li>• Twofold increased risk of CRC</li> </ul>
Polyps	<p>70% of polyps are adenomatous or neoplastic:</p> <ul style="list-style-type: none"> <li>• 75%–85% tubular adenomas (lowest risk), 10%–25% tubulovillous adenomas (intermediate risk), ≤ 5% villous adenomas (highest risk)</li> <li>• &gt; 1 cm in size: 2–4-fold increased risk of CRC</li> <li>• Multiple polyps: 5–7-fold increased risk</li> <li>• Time to malignant progression: 3.5 years for severely dysplastic polyps and 11.5 years for mild atypia</li> </ul>
Family history of CRC	<ul style="list-style-type: none"> <li>• One first-degree relative: Relative risk increased to 1.72</li> <li>• Two first-degree relatives: Relative risk increased to 2.75</li> </ul>
Hereditary nonpolyposis colorectal cancer	<p>The Lynch syndromes</p> <ul style="list-style-type: none"> <li>• Lynch I (colonic syndrome) <ul style="list-style-type: none"> <li>– Autosomal dominant trait associated with proximal mucinous or poorly differentiated synchronous or metachronous colonic tumors</li> <li>– Usual development of CRC by age 50; 75% overall lifetime risk</li> </ul> </li> <li>• Lynch II <ul style="list-style-type: none"> <li>– Associated with extracolonic tumors in the ovaries, endometrium, stomach, small intestine, and genitourinary and hepatobiliary tracts</li> </ul> </li> </ul>
Familial adenomatous polyposis	<ul style="list-style-type: none"> <li>• Autosomal dominant inherited syndrome (germ-line mutation in adenomatous polyposis coli gene on chromosome 5q21) consisting of hundreds of colonic polyps developed by late adolescence</li> <li>• 100% lifetime risk of developing CRC</li> </ul>

*Note.* Based on information from Alberts & Goldberg, 2004; Kim et al., 2005.

## Prostate Cancer

Prostate cancer is the most commonly diagnosed cancer in American men and the second leading cause of cancer-related death in men (ACS, 2008). In 2008, 186,320 new cases of prostate cancer will be diagnosed, and 28,660 deaths are expected to occur (ACS, 2008). The incidence of prostate cancer has risen steadily with a peak incidence of 191 cases per 100,000 men in 1992, largely as a result of improved detection capabilities with routine prostate-specific antigen (PSA) testing approved by the FDA in 1986 (Gulley & Dahut, 2005; Zisman, Belldregun, & Figlin, 2000). The most common risk factor for prostate cancer is age; the median age at diagnosis is 72 in Caucasian males (Gulley & Dahut; Zisman et al.). African American males have a lower median age at diagnosis (62 years) and higher incidence and mortality rates (Gulley & Dahut; Zisman et al.).

Screening for early-stage prostate cancer with PSA testing or digital rectal examination (DRE) detects prostate cancer in its earliest stage; however, it is largely debatable as to whether screening asymptomatic men with PSA or DRE reduces mortality (Gulley

**Table 1-11. Screening Recommendations for Colorectal Cancer (CRC) for High-Risk Populations**

Risk Factor	Screening Recommendations
History of adenoma	<p>Low-risk adenoma</p> <ul style="list-style-type: none"> <li>• &lt; 3 polyps, &lt; 1 cm, tubular histology</li> <li>• Follow-up colonoscopy every 3–6 years, then every 5 years once normal</li> </ul> <p>High-risk adenoma</p> <ul style="list-style-type: none"> <li>• High-grade dysplasia or carcinoma in situ, &gt; 1 cm, villous histology, or multiple adenomas (&gt; 3 &lt; 10)</li> <li>• Follow-up colonoscopy every 3 years, then every 3–5 years once normal</li> <li>• More than 10 adenomas or &gt; 15 cumulative adenomas in 10 years</li> <li>• Consider polyposis syndrome.</li> </ul>
Personal history of CRC	<p>Colonoscopy one year following diagnosis Repeat in 1–3 years if adenoma is identified. If normal, repeat in 2–3 years.</p>
Personal history of ovarian or endometrial cancer < 60 years of age	<p>Colonoscopies beginning at age 40 and repeated every 5 years if no abnormal findings present</p>
Inflammatory bowel disease (ulcerative colitis or Crohn disease)	<p>Colonoscopy beginning within 8–10 years after onset of disease and continue every 1–2 years</p>
Family history	<p>One first-degree relative with CRC &lt; 50 years old at diagnosis, or two first-degree relatives with CRC at any age, or clustering of hereditary nonpolyposis colon cancers, or polyposis in close relatives and does not meet criteria for hereditary syndrome:</p> <ul style="list-style-type: none"> <li>• Colonoscopy at age 40 or 10 years prior to the age of earliest CRC diagnosis in the family and repeat every 1–5 years</li> </ul> <p>One first-degree family member with CRC or two second-degree relatives with CRC at any age:</p> <ul style="list-style-type: none"> <li>• Colonoscopy at age 40 or 10 years prior to the age of the earliest CRC diagnosis in the family and repeat every 5 years</li> </ul> <p>One second-degree relative or any third-degree relative:</p> <ul style="list-style-type: none"> <li>• Screen according to recommendations of average-risk population with individualized evaluation and risk assessment.</li> </ul>

*Note.* Based on information from National Comprehensive Cancer Network, 2007c.

& Dahut, 2005; NCI, 2007k). Studies have documented that screening for prostate cancer detects disease in some men that would never have caused clinically significant problems, thus resulting in overtreatment in some cases with modalities (e.g., radical prostatectomy and radiation) that carry risks of permanent side effects (Gulley & Dahut; NCI, 2007k). Several studies have demonstrated that more men die with prostate cancer than from it; autopsy data have identified an occult rate of prostate cancer of 75% in men in their 80s (Gulley & Dahut). Despite the controversy, PSA and DRE are widely used in the United States to screen men for prostate cancer.

Most agree that individualized risk assessments and evaluation for screening take place between the patient and provider (ACS, 2006c). Screening guidelines for average-risk and high-risk males (African American heritage, positive family history) are outlined

in Table 1-6 and generally consist of initiating PSA screening at age 50 in average-risk individuals and at age 40 in high-risk individuals.

## Endometrial Cancer

Endometrial cancer is the most common gynecologic cancer and the fourth most common type of malignancy in females overall, representing 6% of or approximately 40,100 new cases and 7,470 deaths in 2008 (ACS, 2008). Almost all cases are diagnosed in postmenopausal women, with incidence peaking in the sixth and seventh decades of life (Memarzadeh, Farias-Eisner, & Berek, 2004). Less than 5% of all cases of endometrial cancer are diagnosed in women younger than the age of 40, and premenopausal diagnosis usually is associated with Stein-Leventhal syndrome or polycystic ovarian syndrome (Memarzadeh et al.).

Multiple risk factors for endometrial cancer exist, mostly pertaining to unopposed estrogen exposure (both exogenous and endogenous) and reproductive history. These include polycystic ovarian disease, anovulatory ovary disease, granulosa cell tumor of the ovary (or other estrogen-secreting tumors), early menarche and late menopause, irregular menses, infertility, and nulliparity (Annunziata & Birrer, 2005; Memarzadeh et al., 2004; NCI, 2007e). Nulliparity carries a twofold increased risk for endometrial cancer compared to the risk in women who have had at least one child (Annunziata & Birrer). Intake of tamoxifen, a weak estrogen administered for treatment and prevention of breast cancer, is thought to carry a twofold increased risk for endometrial cancer (Memarzadeh et al.). Other risk factors include advanced liver disease, hypertension, obesity, and diabetes mellitus (Annunziata & Birrer; Memarzadeh et al.). Lastly, a family history of endometrial cancer increases the risk for endometrial cancer. A woman's risk is tripled if she has one first-degree relative with endometrial cancer and doubled if she has one first-degree relative with CRC (Annunziata & Birrer).

As with other cancers, racial disparities exist in endometrial cancer incidence and mortality rates. African American women have a lower incidence of endometrial cancer than Caucasian women yet have a higher mortality rate. Some studies propose that both biologic factors and lower socioeconomic status play a role in this disparity. For instance, lower income has been associated with a lower probability of undergoing potentially curative surgery with hysterectomy. This leads to advanced disease at diagnosis and, thus, lower survival rates (Madison, Schottenfeld, James, Schwartz, & Gruber, 2004).

Routine screening for asymptomatic women is not recommended (Annunziata & Birrer, 2005; NCI, 2007e). Studies do not support routine transvaginal ultrasound (TVU) or endometrial biopsies, as they have not been proved to reduce mortality. TVU has a low sensitivity and would require additional, more sensitive (and more costly) imaging studies to investigate for the presence of malignancy (NCI, 2007e). Also, endometrial biopsies carry the risk of bleeding and infection, and many endometrial cancers are missed with endometrial sampling (NCI, 2007e).

Screening for endometrial cancer is recommended for certain populations, however. Women with hereditary nonpolyposis colon cancer (HNPCC) carry a 60% lifetime risk of developing endometrial cancer (NCI, 2007e). For these women, routine screening is recommended and consists of TVU and endometrial sampling annually beginning at age 30–35 or 5–10 years earlier than the age at which the first diagnosed case in the family occurred (NCCN, 2007c). Prophylactic surgery with hysterectomy and bilateral salpingo-oophorectomy is an option for women who have completed childbearing (NCCN, 2007c).

## Ovarian Cancer

Ovarian cancer is the fifth leading cause of cancer-related death in American women, expected to take 15,520 women's lives of 21,650 diagnosed in 2008 (ACS, 2008). No major improvement in overall survival has been made in more than 30 years (Memarzadeh et al., 2004). Incidence of ovarian cancer increases with age; the average age at diagnosis is 55 years (Memarzadeh et al.). Nulliparity and continuous ovulation also are risk factors. Therefore, conditions or measures that decrease the number of ovulations, such as oral contraceptives, multiparity, and breast-feeding, have a protective effect (Reed & Altaha, 2005).

As with endometrial cancer, routine screening for ovarian cancer in women without known risk factors, such as a known genetic predisposition or strong family history, is not recommended and has not been shown to decrease mortality (NCI, 2007j; Reed & Altaha, 2005). But, studies do support screening in women who are considered high-risk—those women with strong family histories suggestive of a hereditary cancer syndrome such as *BRCA1* or *BRCA2* mutations or HNPCC. Although only 5%–10% of all ovarian cancers are secondary to germ-line mutations, the associated risk of ovarian cancer is 16%–27% with the *BRCA2* gene and as high as 40%–60% with the *BRCA1* mutation (Memarzadeh et al., 2004; NCI, 2002). Ovarian cancer risk associated with HNPCC is two to four times the risk of the general population (Memarzadeh et al.). In these high-risk women, screening with TVU and the tumor assay CA 125 is recommended and is discussed in greater detail in Chapter 2.

## Skin Cancer

In 2008, 62,480 cases of malignant melanoma are expected to be diagnosed, in addition to more than one million cases of basal and squamous cell skin cancers (ACS, 2008). Most cases of skin cancer are caused by unprotected and/or excessive exposure of the skin to UV light (ACS, 2008); 90% of all skin cancers occur on sun-exposed areas of the skin (Wagner & Casciato, 2004). UV exposure can come from natural sources or artificial sources, such as tanning beds, and both result in skin damage ranging from wrinkling and premature aging to skin cancer (ACS, 2008). Risk factors for melanoma and basal and squamous cell skin cancers include light skin tone, blue eyes, blond or red hair, personal or family history of skin cancer, overexposure to the sun, history of severe sunburn, and freckles or nevi (ACS, 2008; Hegde & Gause, 2005). The incidence of melanoma in Caucasians is 10 times greater than in African Americans and is slightly higher in females than males (Hegde & Gause).

Melanoma has several risk factors (see Figure 1-3). A family history of melanoma may be indicative of familial melanoma. Familial melanoma consists of multiple melanomas in one family, usually developing at a younger age (< 50), and often is associated with dysplastic nevi. Chromosomal abnormalities also have been recognized in familial melanoma, including loss of chromosome 9p21 (Hegde & Gause, 2005).

ASCO (2005) recommends skin cancer screening in the general population with periodic self skin examinations and annual whole body skin examination by a healthcare provider beginning at age 40. Secondary prevention measures include treatments directed at individual lesions (Wagner & Casciato, 2004). Primary prevention of most skin cancers can be achieved by avoiding excessive sun exposure and using sunscreen.

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**Precursor Lesions**

- Dysplastic nevi
- Congenital nevi
- Acquired melanocytic nevi

**Other Risk Factors**

- Xeroderma pigmentosum
  - Familial atypical mole melanoma syndrome
  - Numerous acquired melanocytic nevi
  - Immunosuppression
  - Chemical exposures
  - Trauma
  - Ionizing radiation
- 

**Figure 1-3. Risk Factors for Melanoma**

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*Note.* Based on information from Hegde & Gause, 2005; Wagner & Casciato, 2004.

## Lung Cancer

Lung cancer is the most fatal malignancy and the second most commonly occurring cancer in both men and women (ACS, 2008). Despite this, no routine screening recommendations for lung cancer exist for asymptomatic or even high-risk individuals. In 2001, ACS recognized the need for high-risk individuals (smokers and those with occupational exposures) to explore the option of early detection for lung cancer with their healthcare provider (Smith et al., 2007). The U.S. Preventive Services Task Force (USPSTF) cites insufficient evidence to screen asymptomatic people with low-dose computed tomography (LDCT), chest x-ray (CXR), sputum cytology, or a combination of the three (USPSTF, 2004). Several studies have documented earlier detection of lung cancer through one or all of these means but have not shown a decrease in mortality (Bach et al., 2007; NCI, 2007h; USPSTF). Moreover, both NCI (2007h) and USPSTF concluded that CXR, sputum cytology testing, and LDCT have high false-positive rates and would lead to unnecessary and potentially dangerous invasive tests such as lung biopsy (USPSTF). The National Lung Cancer Screening Trial is comparing spiral computed tomography and CXR in 50,000 current or former smokers in an attempt to determine whether a reduction in mortality from lung cancer is achieved by either modality (NCI, 2006c). Results are expected in 2009 (NCI, 2006c).

## Other Cancers

Although established screening tests and guidelines exist for several cancers, including three of the most common malignancies—breast, colorectal, and prostate—the majority of malignancies do not have established screening recommendations, as insufficient evidence exists to suggest that screening would affect mortality rates. NCI (2007a, 2007h, 2007i) found inadequate evidence to determine whether screening for bladder or urothelial cancer, lung cancer, and oral cancer would reduce mortality in the United States. Moreover, evidence also suggests that screening for esophageal, gastric, hepatocellular, and testicular cancers would not reduce mortality, and potential side effects of testing outweigh benefits—such as the dangers associated with endoscopy for

gastric or esophageal cancers or fine needle aspiration with hepatocellular cancer (NCI, 2006d, 2007f, 2007g). However, clinical trials are ongoing in the search for beneficial screening modalities and recommendations for a majority of malignancies. NCI alone has more than 70 clinical trials dedicated to cancer screening. The National Institutes of Health lists more than 300 clinical trials across the United States that are actively recruiting for cancer screening trial participants.

## Implications for Oncology Advanced Practice Nurses

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The role of the oncology APN encompasses cancer risk reduction, screening, and early detection. Oncology APNs are able to assess, evaluate, and interpret cancer risk assessments and recommend appropriate interventions. Familiarity with known risk factors for various cancers alerts the APN to patients who would benefit from evidence-based interventions to reduce cancer risk. Guidelines are available to guide APN interventions. Strategies are modified based on individual characteristics, population risk variances, and cultural diversities. It is imperative that the oncology APN masters these topics to provide comprehensive, thorough oncology care that begins with risk assessment to reach the goal of cancer prevention.

## Conclusion

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Cancer prevention and early detection are integral parts of the cancer care continuum. Ideally, primary cancer prevention in the form of risk reduction is the best way to decrease morbidity and mortality related to cancer. Certain populations are considered to be at high risk for some malignancies, and the screening and management of these populations differ from that of the general population. Risk models are available to assist the APN in assessment for certain cancers. Evidence-based pharmacologic, nonpharmacologic, and behavioral interventions are available. Education of both the individual and populations is crucial. Education encompasses information about exercise, dietary habits, sun exposure, smoking cessation, and recommended screening practices. Early detection achieved by adhering to routine screening guidelines facilitates cancer being diagnosed in the earliest stage of the disease, when it is most likely to be treated successfully and is associated with the best patient outcomes. The oncology APN has an opportunity and an obligation to offer risk reduction care and appropriate screening to both the individual patient and populations.

### Case Study

A.J. is a 55-year-old African American female seen in the oncology clinic by the oncology APN for follow-up care for anemia. A.J. states she receives health care at a walk-in clinic only when she is ill and that she had been out of work for some time and had not had insurance to cover routine medical care until recently. Review of her family history confirms colon cancer in her 70-year-old mother, diagnosed at age 65. Her 73-year-old father has heart disease and hypertension and was diagnosed with prostate cancer at age 72. She has two brothers, who also have hypertension. She is

married and has no children. She smokes one pack of cigarettes per day and is interested in quitting, but admits she needs help. She denies alcohol use. A.J. works 40–50 hours a week, does not engage in regular exercise, and eats fast food frequently.

Her review of systems is negative except for fatigue and intermittent arthralgia with a previous history of osteoarthritis, for which she takes occasional acetaminophen. She is postmenopausal; her last menstrual cycle was more than two years ago. Other past medical and surgical history is negative. The physical examination (including CBE, pelvic examination, and Pap smear) is also negative, vital signs are stable, and she has no gross abnormalities on examination except for moderate obesity and pale oral mucosal membranes.

1. What risk factors for malignancy can be identified based on this history?
  - Her risks for cancer include tobacco use, sedentary lifestyle, obesity, and poor nutrition. She is nulliparous and has one first-degree relative with a history of colon cancer. Her father's recent diagnosis of prostate cancer at age 72 is noted but does not necessarily affect A.J.'s risk factors at this point.
2. What screening tests does A.J. need, and what cancer risk reduction strategies can the oncology APN discuss with A.J.?
  - As A.J. has not received routine medical care in several years, she has neglected the recommended cancer screening tests. Recommendations include smoking cessation, and because she is willing to make a quit attempt, NRT may be offered in the form of transdermal nicotine. Counseling regarding smoking cessation will increase effectiveness of the intervention. Physical activity of moderate intensity for at least 30 minutes several times per week is a behavioral goal. Dietary counseling is necessary, focusing on eating fewer high-fat foods and consuming at least five servings of fruits and vegetables per day. Counseling on the techniques, benefits, and limitations of BSE will increase her confidence in performing this examination.
  - A screening mammogram is appropriate, as is a referral to a gastroenterologist for a screening colonoscopy. Informational needs include screening recommendations for mammogram and CBE annually, pelvic examination and Pap smear every two to three years, and colonoscopy every five years, given her positive family history (assuming initial colonoscopy results are benign).
3. Before she leaves, A.J. inquires about a television commercial for a vaccine for cervical cancer and wants to know if that is an option for her. How does the oncology APN respond?
  - The oncology APN tells A.J. that Gardasil is a vaccine for prevention of cervical cancer associated with HPV infection and is approved only for administration in females 9–26 years of age. Therefore, A.J. is not a candidate for this vaccination, and the APN recommends she continue with Pap smears for early detection of cervical cancer as discussed previously.

## Key Points

- Primary prevention of cancer is achieved through promotion of wellness and reduction of known risks for cancer.
- Cancer risk assessment involves an individualized, comprehensive patient history and examination to provide accurate cancer risk reduction counseling and screening recommendations.



- Major components of cancer risk reduction for the general population include
  - Avoid or cease cigarette smoking.
  - Minimize UV light exposure, and use sunscreen with an SPF  $\geq 15$  on sun-exposed skin.
  - Maintain an active lifestyle with regular, moderate physical activity.
  - Maintain a healthy weight (avoid obesity).
  - Eat a diet high in fiber, fruits, and vegetables and low in red or processed meats, fats, and sugars.
- Chemoprevention is an option for certain high-risk patients.
- Secondary prevention includes screening and early detection of cancer.
- Screening tests require specificity (few false positives) and sensitivity (few false negatives) for the disease being screened.
- Screening guidelines exist for the general population and for populations at high risk for various cancers, including those with a genetic predisposition for certain cancers.
- Screening for high-risk populations usually begins at an earlier age than screening for the general population and occurs at more frequent intervals.
- Cultural and ethnic disparities exist in almost all malignancies and affect incidence and mortality rates.

## Recommended Resources for Oncology Advanced Practice Nurses

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- Breast Cancer Risk Assessment Tool ([www.cancer.gov/bcrisktool/default.aspx](http://www.cancer.gov/bcrisktool/default.aspx)): Computerized tool for the PDA and desktop computer
- CancerGene software ([www.utsouthwestern.edu/utsw/cda/dept47829/files/65844.html](http://www.utsouthwestern.edu/utsw/cda/dept47829/files/65844.html)): A free computer program that estimates risk for carrying mutation in one of the cancer predisposition genes. Uses BRCAPRO and MMRpro risk models, draws a pedigree, and archives family history and mutation probability.
- CancerPRA™ (Skyscape) ([www.collectivemed.com/jump/capra.shtml](http://www.collectivemed.com/jump/capra.shtml)): PDA tool based on the *Handbook of Cancer Risk Assessment and Prevention* with search capabilities
- Cancer Risk: Understanding the Puzzle (<http://understandingrisk.cancer.gov>): A Web site from NCI for patients that explains cancer risk and provides tools for patients
- *Handbook of Cancer Risk Assessment and Prevention* (Colditz & Stein, 2004): Handbook of risk factors for the most common cancers, a risk assessment tool, and hints to promote risk-reducing lifestyle changes
- Lung Cancer Risk Assessment Tool ([www.mskcc.org/mskcc/html/12463.cfm](http://www.mskcc.org/mskcc/html/12463.cfm)): A prediction tool from Memorial Sloan-Kettering Cancer Center to assess a long-term smoker's risk of developing lung cancer in the next 10 years
- Melanoma Risk Assessment Tool ([www.cancer.gov/melanomarisktool](http://www.cancer.gov/melanomarisktool)): An interactive tool to estimate a person's absolute risk of developing invasive melanoma
- NCCN Clinical Practice Guidelines in Oncology ([www.nccn.org](http://www.nccn.org))
  - Breast cancer risk reduction
  - Cervical cancer screening
  - Colorectal screening
  - Genetic/familial high-risk assessment for breast and ovarian cancers

- RiskyDisky: PDA prediction tool that uses the modified Gail, Claus, and Frank models to predict the five-year risk for breast cancer and consequences of five years of tamoxifen therapy

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