HEALTHY LIFESTYLE AND CANCER PREVENTION

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LEARNING OBJECTIVES

This article will review the current scientific evidence that supports the role of diet and physical activity in the prevention of cancer.

The reader will be able to:

- Understand the multistep process of cancer development;
- Describe the current evidence that supports the role of diet and physical activity in cancer prevention;
- State the current dietary and physical activity guidelines for cancer prevention;
- Describe current recommendations for lifestyle choices to reduce recurrence and promote wellness after a diagnosis of cancer;
- Understand the proposed biological mechanisms by which diet and physical activity are thought to reduce cancer risk; and
- Provide practical tips for clients to modify behavior with the goal of reducing cancer risk.

Key words:
Diet, Physical Activity, Obesity, Inflammation, Oxidative Stress

In terms of disease incidence, the numbers of individuals affected with cancer each year are staggering. Cancer is the second leading cause of death in the United States, with one in two Americans receiving a cancer diagnosis in their lifetime. In 2007, approximately 1.5 million new cases and 559,650 deaths related to cancer are estimated to occur among Americans (1). Overall cancer incidence is slightly higher in men than in women. After excluding common skin cancers, men most commonly have prostate cancer (29%), followed by colorectal and lung cancer. For women, the most common cancers diagnosed are breast (26%), lung (15%), and colorectal cancers (10%). Lung cancer rates were historically higher in men but are now comparable in men and women.

Cancer accounts for an estimated one in four deaths in the United States, with overall 5-year survival estimates approximating 66%. Advances in early detection and treatment have led to increased survival rates, with the cancer survivor population currently estimated at approximately 10.5 million individuals in the United States. Unfortunately, this progress has produced a large population of individuals who present unique, and largely unaddressed, healthcare needs. Cancer survivors have elevated risk for second cancers and numerous comorbidities acquired during their treatments including weight gain, loss of bone and muscle mass, depression, cognitive loss, and decreased mobility. This population represents one of the largest groups of health-motivated people for whom clinical prevention messages are likely to be adopted.

Elevated risk for many of the common cancers (breast, colon, and prostate) has been linked to a number of diet-related factors that range from poor nutrient status (*i.e.,* low fruit/low vegetable intake) to the adverse effects of

chronic excess energy intake (*i.e.,* overfed/sedentary state). The consistent finding across studies that cancer rates are lower among physically active/healthy-weight persons has raised the issue of energy balance to a forefront in nutrition and cancer prevention. This issue has become particularly important, given the growing incidence of obesity and inactivity among people living in the United States and more industrialized countries.

This article will review the current evidence on the role of energy balance, nutrition, and lifestyle in cancer prevention. Furthermore, proposed biological mechanisms of action thought to mediate the risk modification of exposure to chronic positive energy balance also are presented. Finally, practical information for making positive changes in lifestyle behaviors thought to be important in cancer risk reduction will be provided.

CANCER IS A MULTISTEP PROGRESSIVE DISEASE PROCESS

To fully appreciate the complex role of diet in cancer prevention, an overview of the multistep process leading to cancer development is warranted. As shown in the Figure, cancer does not develop overnight; it is a disease that results from decades of insult to healthy cells that over time, and with repeated insult, suffer undesirable changes and a growth advantage. Cancer cells also develop the capacity to ''escape'' from repair or removal by a healthy immune system. The ''path'' to cancer is thought to involve a stepwise progression originating with an initial event where cells are genetically damaged, perhaps related to diet (high fat, dietary carcinogens, etc) or some environmental factor (UV light, smoking, radiation, etc), and become ''initiated.'' Most initiated cells are routinely removed or repaired by the immune system; however, on occasion, an initiated cell escapes these protective mechanisms provided by the immune system and undergoes further damage, eventually resulting in the development of cancer. This change in cells from healthy to cancer is called *carcinogenesis* and encompasses several specific steps labeled as follows: initiation, promotion, progression, and cancer.

CURRENTLY RECOMMENDED CANCER NUTRITION AND PHYSICAL ACTIVITY PREVENTIVE MEASURES

The American Cancer Society recently released updated dietary and physical activity guidelines for cancer prevention. The current guidelines support the following:

- maintaining a healthy body weight throughout life
- adopting a physically active lifestyle
- consuming a healthy diet with an emphasis on plant food sources
- if you drink alcoholic beverages, limiting consumption

Of interest, these guidelines now emphasize the important role of body weight in reducing cancer risk and suggest that maintaining a healthy body weight throughout adult life may be the single most important behavioral approach to reducing risk for disease. For example, a woman reporting a body weight of 130 lbs at age of 18 years should maintain a weight between 130 and 143 lbs throughout adulthood (thus controlling body weight to within 10% of early adult weight). Yet, despite this recommendation and the ''weight'' of evidence that supports these recommendations, weight gain throughout adulthood is well documented among Americans.

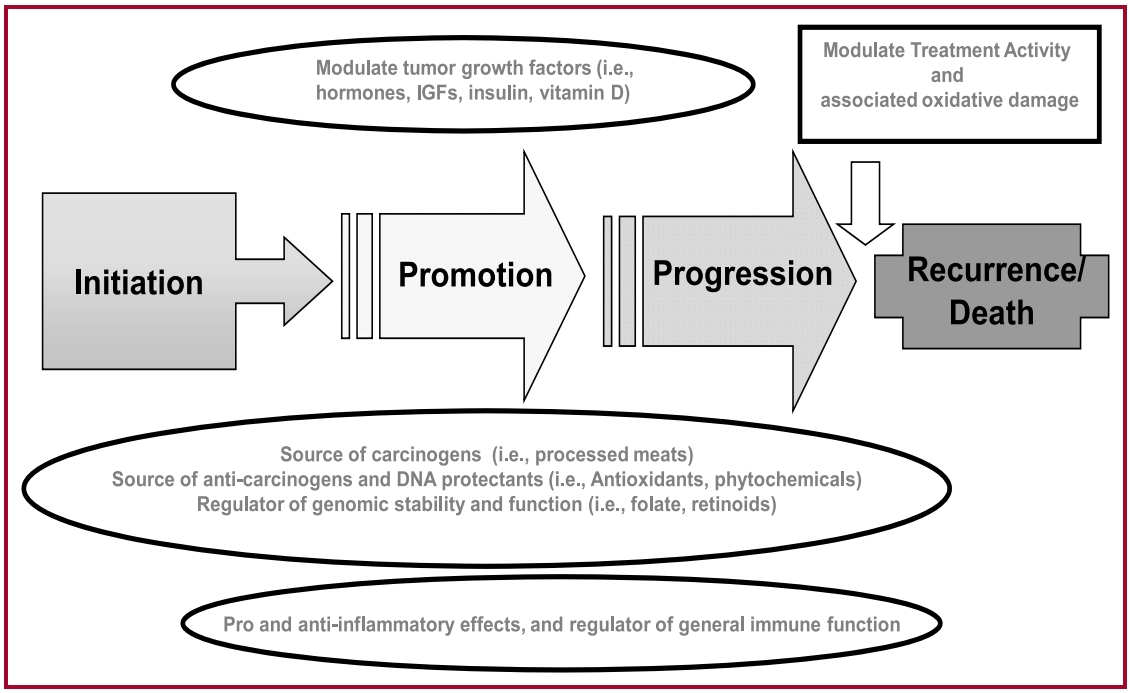

Figure. Dynamic and complex influences of diet on the multistep process of carcinogenesis.

IMPORTANCE OF HEALTHY BODY WEIGHT THROUGHOUT LIFE

According to the National Center for Health Statistics, 65% of U.S. adults over the age of 20 are overweight, with 30% of those considered to be obese (1). Obesity, particularly increased abdominal obesity, is associated with increased risk for the development of reduced insulin sensitivity (2), predisposing to the development of metabolic syndrome, cardiovascular disease, diabetes, and some cancers. Most epidemiological studies support a role for obesity as a risk factor for endometrial (39%), esophageal (12%), kidney, and colon cancers (25%) (3). For colorectal cancers, the effect of obesity as measured by body mass index (BMI) seems limited to the colon with no effect in the rectum, stronger in the proximal colon, and stronger in men than in women. In the case of hormonal cancer, a BMI above 30 kg/m^2 at age of 30 years has been associated with a significant increase in risk for developing hormone-related cancers including breast, ovarian, and uterine cancers (4). For breast cancer, obesity, but more specifically, adult weight gain of 20 kg after age of 18 years, has been most consistently shown to increase risk of postmenopausal breast cancer and is now considered an established risk factor. Unlike postmenopausal breast cancer, premenopausal breast cancer has not been associated with obesity or higher BMI. Premenopausal disease, which occurs earlier in life, also tends to be more aggressive, suggesting that the factors contributing to risk are more likely to be related to genetic background and/or significant exposures to cancer-promoting factors at ''vulnerable'' time points in life when the breast/mammary gland is exposed to higher levels of growth factors such as insulin and estrogen (*in utero*, early life, before puberty).

Evidence also has suggested that those adolescents who carry the BRCA1 and 2 familial breast cancer gene mutations, while highly likely to develop breast cancer with age, may delay onset of disease by as much as 12 years if they practice a physically active lifestyle and control their body weight (5). Data on the relationship between obesity and prostate cancer are more inconsistent and complex than those for breast and colon, with obesity most consistently associated with elevated risk for more advanced disease at diagnosis.

PHYSICAL ACTIVITY AND CANCER PREVENTION

One of the most relevant findings in studies of cancer risk factors is the protective role of physical activity for many of the common cancers (6), a protective effect which is related to a wide range of biological mechanisms (7). With the exception of skin cancer, where sun exposure and physical activity are strongly linked, regular physical activity has been associated with a 20% to 50% reduction of risk for cancers of the uterus, colon, and breast. For breast cancer, the evidence for a protective effect of physical activity is strongest for those cancers occurring after menopause, similar to obesity. Unlike colon and breast cancer, limited evidence suggests that physical activity, particularly higher vigorous activity, is associated with lower risk of more aggressive and fatal forms of prostate cancer.

In general, the protective effects of increasing levels of physical activity have been shown regardless of the type of physical activity undertaken—recreational or occupational. To put the effective dose into perspective, in a large study of physical activity conducted in 413,044 participants of the European Prospective Investigation into Nutrition and Cancer, the level of physical activity required to achieve a 20% to 25% risk reduction for colon cancer translated to approximately 1 hour per day of vigorous physical activity (metabolic equivalent [MET] = 6) or 2 hours per day of moderate-intensity physical activity (MET = 3) (8). More recent analyses from the large Nurses Health Study showed protection against recurrent disease when breast cancer survivors participated in regular, moderate-level, physical activity for 30 minutes five times per week or 9 MET-hours per week. For colorectal cancer survivors, the exercise dose required to reduce recurrence risk was approximately double (18 MET-hours per week) (9).

Although studies are limited, the benefits of regular exercise do seem to be strongest for those individuals who maintain a

healthy weight and for those individuals with lower energy consumption. These data suggest important joint effects of physical activity and healthy body weight as primary means of cancer prevention.

Of interest are data that support a protective role for physical activity early in a young woman's life (adolescence). Physical activity seems to have a favorable effect on the breast during development (10). And, activity protects against adult weight gain and metabolic and hormonal factors associated with breast cancer risk. Regardless, these data support the need for early counseling on lifelong behaviors to reduce risk of breast and other cancers—an important message that could be integrated into general pediatric practice and school-based physical education programs.

Current American Cancer Society Guidelines specifically recommend that adults engage in physical activity of moderate-to-vigorous intensity for at least 30 minutes and preferably 45 to 60 minutes 5 or more days of the week. This should be above usual activities such as walking to and from parking lots, meal preparation, dressing, and the like. Maintaining an energy expenditure that controls body weight within a healthy range is paramount to reducing cancer risk.

Several intervention trials have been conducted to evaluate the efficacy of select approaches to enhance daily physical activity and thus reduce cancer risk, mostly targeting the cancer survivor population. In general, cancer survivors report significant fatigue in relation to cancer treatments, particularly radiation therapy—a concern that is commonly expressed even several years after treatment. Thus, early promotion of physical activity is essential for people diagnosed with cancer. Clinicians should discuss the importance of a physically active lifestyle early in the treatment plan and reinforce the need to remain active during and after therapy at ongoing clinic visits. There is no clear evidence that one type of activity is advantageous over another in this setting, although walking is commonly used in clinical trials, and some research suggests that pedometers can be efficacious in goal setting and longer-term maintenance of a physically active lifestyle. Cancer patients frequently experience sarcopenia or loss of muscle mass, related to treatment, and this adverse outcome can be reduced with regular physical activity during treatment. Specifically, weight-bearing exercises and strength training can promote retention of lean body mass and bone mass that also has been shown to be adversely impacted by cancer treatment. Tips for promoting physical activity in the client's daily life are provided in Table 1.

TABLE 1: Tips for Enhancing Physical Activity and Energy Expenditure Daily

Physical activity
take the stairs
wear a pedometer
park car at a distance when shopping or going to work
walk daily with friend or pet
walk during the lunch hour
form work walking club
sign up for local walking event to raise funds for important causes
join a hiking or outing club
cycle to work
treadmill to your favorite music
lift hand weights while watching television
swim with a friend
Increase energy expenditure
stand up during meetings
eat small frequent meals
carry small weights in your pockets
drink caffeinated green tea, black tea, or coffee
consume hot and spicy foods

HEALTHY LIFESTYLE AND CANCER PREVENTION

Numerous nutrients and bioactive food components (BAFCs) have been shown to increase or decrease cancer risk (11). Table 2 below provides an overview of current associations between dietary practices and cancer risk. Much of the evidence for protective associations has resulted from epidemiological evidence where eating practices within a given population are evaluated against cancer rates yielding an estimation of risk. Despite intuitive and biological evidence that diet plays a role in cancer risk, much of the evidence remains inconsistent, and when diet has been shown to be protective, risk estimates indicate relatively small protective effects. There are several major limitations to assessing associations between diet and cancer risk including capturing the correct exposure period, lack of accurate reporting of dietary intake, and quite simply, the complexity of the human diet. These factors are particularly problematic in case-control studies where ''cases'' have already been diagnosed with disease and are asked to recall their eating patterns before diagnosis.

VEGETABLES AND FRUIT: BAFCs

Existing evidence is inconsistent in regard to the relationship between vegetable and fruit intake and cancer risk. Although a large number of case-control studies (where people are asked about their dietary habits after they have been diagnosed with cancer) have supported a protective association, most cohort studies (where self-report of diet is collected before a cancer diagnosis) suggest that commonly reported intake levels in study populations show no significant association. When protective effects are shown, they tend to be on the order of a 10% to 20% reduction in risk, suggesting that an increase in vegetable and fruit consumption alone is unlikely to have a

TABLE 2: **Dietary Factors and Associated Risk for Select Cancers**

	Cancer Type					
Dietary Factor	Breast	Prostate	Colorectal	Lung	Skin	Oral
Obesity	+ (post)	++	+	O	+	O
Protective						
Low fat (<24%)	+	+	±	O	+	O
High omega-3	+	+	+	±	+	±
High fiber	+	+	+	O	O	O
High vegetable	±	±	+	+	±	+
Carotenoid-rich	+	+	+	++	±	++
Cruciferous	±	±	+	++	±	UNK
High fruit	O	O	±	±	±	+
Citrus	+	+	±	+	±	+
Green tea	±	±	+	++	++	+
Lycopene	±	++	+	+	+	+
Plant-based diet	+	+	+	++	UNK	+
Selenium	±	++	+	+	O	UNK
Calcium	O	O	++	O	O	O
Vitamin D	+	+	++	UNK	UNK	UNK
Folic acid	±	UNK	+*	+	UNK	+
Omega-3 fatty acids	+	+	+	UNK	UNK	UNK
Detrimental						
Red meat	UNK	+	++	+	UNK	UNK
Animal fat	+	+	++	+	O	O
Charred meat	+	+	++	+	+	O
Trans fatty acids	+	+	++	±	UNK	±
Alcohol	+	+	+	+	±	++

** Folic acid supplementation has been associated with increased risk for adenomatous polyp recurrence and thus may be detrimental after initiation.*
UNK indicates unknown association or lack thereof; +, an association has been demonstrated; ±, unequivocal; O, no association shown.

significant impact on disease incidence. One explanation for the lack of a significant protective effect is that intake of vegetables and fruit may be insufficient, particularly in terms of upper levels of intake needed to modify risk.

Another explanation for inconsistent findings from the research may be that the type of vegetables and fruits selected may influence disease risk. Current evidence suggests that diets that include vegetables and fruits providing a wide variety of color —red, green, dark yellow, orange, and purple—are likely to protect against cancer. This is because the presence of color indicates that the plant food also is rich in anticancer phytochemicals (also called BAFC). In fact, colorful vegetables and fruits are known to increase blood levels of one group of phytochemicals called *carotenoids* that have been shown to reduce cancer risk. Of interest, it is the plant source of these compounds that is protective, and studies using supplements to increase carotenoid levels are generally not effective and may even increase cancer risk.

It is the BAFCs, such as carotenoids, as well as others such as polyphenols, resveratrol, monoterpenes, and the like, found in plant foods that have been proposed to be of greatest importance in reducing cancer risk. Table 3 lists several BAFCs that have been examined for their role in cancer prevention. Research has included primarily cell culture models in which cancer cells are exposed to select compounds at variable doses and epidemiological research in which dietary intake of such compounds (or foods rich in BAFC) is evaluated in the context of cancer events.

ROLE OF ENERGY INTAKE

Recent animal studies suggest that an energy- or caloric-restricted diet is protective against the development of tumors (12). In controlled animal feeding studies, a reduction in energy intake of 30% to 50% results in a marked and significant reduction in tumor production. The biology behind this protective effect of energy restriction is not clearly understood

TABLE 3: Food Sources of Cancer-Protective Nutrients and BAFCs

Anthocyanins	red wine, cherries, and grapes
Alpha- and beta-carotene	color-rich vegetables (yams and carrots)
d-limonene	citrus peel, citrus fruit
Indole-3-carbinol	brussel sprouts, kale, water cress, mustard and turnip greens, broccoli, and horseradish
Isothiocyanates	cruciferous vegetables: broccoli, cauliflower, brown mustard, wasabi, kale, cabbage, and horseradish
Lutein	spinach and kale
Lycopene	processed tomato products, tomato, ketchup, watermelon, and guava
Monounsaturated fats	olive and canola oils
Omega-3 fatty acids	nuts and fatty fish
Polyphenols	green tea and cranberry
Resveratrol	red wine, grape juice, red grapes, and peanuts

but may have to do with the reduced oxidative burden on the host or lower exposure to the tumor-promoting effects of insulin and insulin-like growth factor (IGF) 1. It is well known that the metabolism of food (and the carbohydrates, fat, and protein therein) increases oxidative stress, particularly if the diet is higher in fat content. This may explain, to some extent, the phenomena of reduced risk in relation to reduced intake. The magnitude of caloric restriction needed to achieve the same benefit in humans is unknown, and adverse effects of nutrient deficiencies that may arise as a result of severe caloric restriction and impact on muscle and bone health also are unknown. Thus, it is more prudent to promote energy balance and healthy balanced diets to achieve lower growth factor and oxidative stress exposures.

BIOLOGICAL MECHANISMS OF CANCER PREVENTIVE ACTIVITY

Several dietary constituents demonstrate multiple bioactive properties resulting in activities that reduce cancer risk. These include direct effects of dietary compounds to enhance the ability of the cell to self-destruct (apoptosis), stop growth of damaged cells (cell cycle arrest), provide antioxidant protection to the cells, alter hormone levels, activate special enzymes that detoxify the body of select compounds that have been associated with cancer development, and favorably change the levels of insulin/insulin-like growth factor to reduce cancer risk and to reduce inflammation.

Inflammation

Inflammation is a long-standing suspect component in tumor formation (13). For the common cancers, it is increasingly acknowledged that low-level chronic inflammatory states may contribute to the development (initiation) and growth (promotion) of tumors, and this may explain the protective role of nonsteroidal anti-inflammatory agents (such as aspirin) at multiple organ sites. When evaluating the role of inflammation in cancer, researchers commonly measure chemicals in the blood such as C-reactive protein and/or interleukin 6. If either of these markers is present at high levels in the blood, it suggests that some inflammation is present in the body, and on a long-term basis, high levels of these chemicals could place a person at a higher risk for developing cancer. The observed association between obesity/inactivity and cancer risk may be related to chronic exposures associated with proinflammatory chemicals called *cytokines*. Visceral (central body) adiposity, in particular, is marked by elevated levels of several inflammatory compounds that are known to be released from fat cells. Table 4 lists several foods and/or spices that have been shown to reduce inflammatory biomarkers in human studies. Although most have not been investigated in controlled clinical trials enrolling cancer patients, studies in ''healthy'' individuals support the notion that integration of these foods/spices into the daily diet holds potential for controlling inflammation. Furthermore, the addition of these foods/spices to standard anti-inflammatory medications may offer promise of enhanced anti-inflammatory (therapeutic) response, potentially reduced medication requirements, and avoidance of associated toxicities of anti-inflammatory medications.

Oxidative Stress

The role of oxidative stress in cancer etiology has been hypothesized for decades (14), yet no longitudinal human studies exist that demonstrate that cumulative high oxidative stress increases risk for cancer. Furthermore, the capacity for select dietary constituents to modify oxidative stress levels has been demonstrated, and patients diagnosed with cancer have demonstrated elevated levels of oxidative stress biomarkers. Extensive animal and cell culture studies support a major role for oxidative stress as a primary mediator of DNA damage in cells and change in gene expression; thus, it is reasonable to accept a major role for oxidative damage in cancer causation.

TABLE 4: Food/Spices with Anti-inflammatory Properties

Anthocyanins—berries, cherries, and pomegranate
Capsaicin–chili peppers
Cinnamon
Garlic
Ginger
Green tea
Omega-3 fatty acids (salmon, sardines, herring, tuna, flax, and canola)
Turmeric/curcumin

Numerous nutrients and bioactive food constituents demonstrate antioxidant activity *in vivo*. Foods rich in antioxidants are listed in Table 5.

Insulin-IGF Hypothesis

Epidemiological and animal studies provide consistent evidence that chronic elevations in blood levels of insulin (known as hyperinsulinemia) may be a contributory factor for cancer in humans. In the ''Insulin Hypothesis'' put forward by Giovannucci et al. (15), chronic hyperinsulinemia is proposed to increase the bioactivity of insulin-like growth factors and insulin binding proteins that can be measured in the blood and have been shown to promote growth of cancer cells. The major determinant of insulin-associated chemicals is genetic background and more modest influences of diet and physical activity. Severe caloric restriction is one of the main behavior modifications that reduces IGF-1 levels and may explain why animals placed on low-calorie diets have lower tumor rates.

The interplay of diet, physical activity, obesity, metabolic syndrome (insulin resistance), and cancer risk is an area of active cancer prevention research. Numerous similarities between risk factors for diabetes, heart disease, and a number of common cancers including age, inflammation, central adiposity, physical inactivity, high intake of saturated fats and refined sugars, and disturbances in insulin regulation has led to the suggestion that the metabolic changes associated with adiposity, principally insulin resistance, are risk factors for some of the common cancers. Table 6 summarizes the current clinical manifestations of metabolic syndrome. Many of these metabolic disturbances are reversible with lifestyle and diet modifications offering a safe and well-supported strategy to reduce cancer and other chronic disease burdens in the population.

TABLE 5: **Food Sources of Antioxidant Nutrients and BAFCs**

Citrus
Berries
Cranberries
Cherries
Melons
Green leafy vegetables
Yellow-orange vegetables
Tomato-based foods
Cruciferous vegetables
Red wine
Green tea
Brazil nuts
Oils such as olive oil
Seeds
Dark chocolate

TABLE 6: **General Criterion Used in the Definition of Metabolic Syndrome**

Waist circumference of >40 inches for men and >35 inches for women
Triglyceride concentration of >150 mg/dL
HDL cholesterol of <40 mg/dL for men and <50 mg/dL for women
≥130/≥85 mmHg or on hypertensive medication
Fasting glucose level of ≥100 mg/dL

Adult Treatment Panel (ATPIII) of the National Cholesterol Education
HDL indicates high-density lipoprotein.

SUMMARY

Cancer will be diagnosed in one in two men and in one in three women in their lifetimes and remains the second leading cause of death in U.S. adults (1), yet effective lifestyle strategies for reducing risk exist (16). Obesity is a major contributing factor to several types of cancer and is among the most preventable of risk factors. Survival after a diagnosis of cancer has improved considerably in the past decade as our understanding of the biology of this disease is advanced. The improved diagnostic and clinical care for this disease make it a chronic illness and one for which treatment may further compromise health status in relation to bone loss, cardiovascular abnormalities, and/or insulin resistance (17). Physical activity and healthy dietary practices are central to optimizing the health of our growing cancer survivor population.

Current recommendations for healthy eating suggest that there is consistency across disease-related health organizations. In fact, control of body weight, increase in vegetable and fruit intake, lowering of fat intake, and promoting higher fiber intake are dietary habits suggested by the American Heart Association, the American Diabetes Association, and The American Cancer

TABLE 7: **Consistency of Dietary and Physical Activity Recommendations Among Chronic Disease Organizations**

Lifestyle Factor	American Diabetes Association	American Heart Association	American Cancer Society/American Institute for Cancer Research
Weight control	+++	+++	+++
Increase fiber	+	++	++
Increase plant foods	+	++	+++
Decrease total fat	NA	+++	+++
Decrease saturated fat	++	++	+
Avoid trans-fatty acids	++	+++	+
Increase omega-3 fatty acids	++	++	++
Alcohol	moderation	moderation	moderation
Daily physical activity	++	+++	++

NA, not addressed.

Society (Table 7). Increasing physical activity also is a consistent recommendation. Although evidence supporting these behavioral strategies remains limited in the setting of cancer prevention (when the limited results from prospective randomized trials are assessed), most researchers and clinicians remain convinced that these behaviors will reduce disease risk. Cancer is a multistep disease that develops over decades, and current short-term lifestyle intervention trials initiated in adulthood are unlikely to have a large impact on cancer events. Efforts to target healthy behaviors earlier in life and sustain healthy behaviors throughout life are essential.

CLINICAL APPLICATION

The frequency of cancer and the potential for unfavorable outcomes warrant continued attention by clinical practitioners to educate the American public on the risk factors, particularly those that are modifiable such as diet and physical activity, for this disease. Preventing or controlling obesity remains a primary treatment strategy. Specifically, practitioners should evaluate the weight, BMI, and waist circumference of their clients on a regular basis and provide feedback to clients regarding changes in these parameters (both favorable and unfavorable). Small increases in any of these clinical measures should be addressed promptly and not ignored. Clients should be educated on the role of energy balance in sustaining health and be given individualized, specific, and measurable goals to achieve optimal health (Table 1) and to increase intake of bioactive food compounds (Table 3).

In addition, as possible, practitioners should partner with the client's physician to evaluate biochemical values that may be indicative of compromised health such as elevations in lipids (cholesterol, low-density lipoprotein, triglycerides, etc). Physical activity in particular will be critical to favorable increases in high-density lipoprotein levels. In terms of bone health, all cancer survivors should be advised to participate in regular weight-bearing physical activity on a daily basis and to meet daily calcium requirements (with or without calcium supplementation). In regard to glucose control, daily physical activity of at least 30 minutes' duration also is advised along with achievement and maintenance of healthy body weight and avoidance of simple sugars and increased intake of fiber-rich foods. Web sites that provide valuable guidance to clients and health-care providers to promote healthy eating and physical activity are listed in Table 8.

TABLE 8: **Healthy Lifestyle Web-Based Resources for Clients and Clinicians**

American Dietetic Association: *www.Eatright.org*
American Cancer Society: *www.Cancer.org*
American Institute for Cancer Research: *www.aicr.org*
Breast Cancer.org: *www.breastcancer.org*
International Food Information Council: *www.ific.org*
USDA MY Pyramid: *www.mypyramid.gov*

So, although the recommendations for optimal health are not significantly different for those trying to reduce their risk of cancer (or cancer recurrence) as compared with overall health recommendations, this population is at an even greater need for guidance in this area. Furthermore, this population is generally sufficiently motivated to make positive changes and to role model healthy behavior for others.

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Patricia C. Thompson, Ph.D., holds a doctoral degree in Microbiology/Immunology from the University of Texas Health Science Center at San Antonio and has led translational research efforts in cancer prevention for the past 8 years. She is well published in the area of lifestyle, genetics, and individual risk of cancer including leading research in the areas of colorectal, breast, and prostate cancer. Her experience includes leading local studies designed to test hypotheses related to the

modulation of metabolic syndrome through lifestyle interventions conducted with cancer survivors and at-risk persons.

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CONDENSED VERSION AND BOTTOM LINE

Healthy food selections, energy balance to promote lifelong weight control, and regular physical activity should be routinely promoted to reduce cancer risk.